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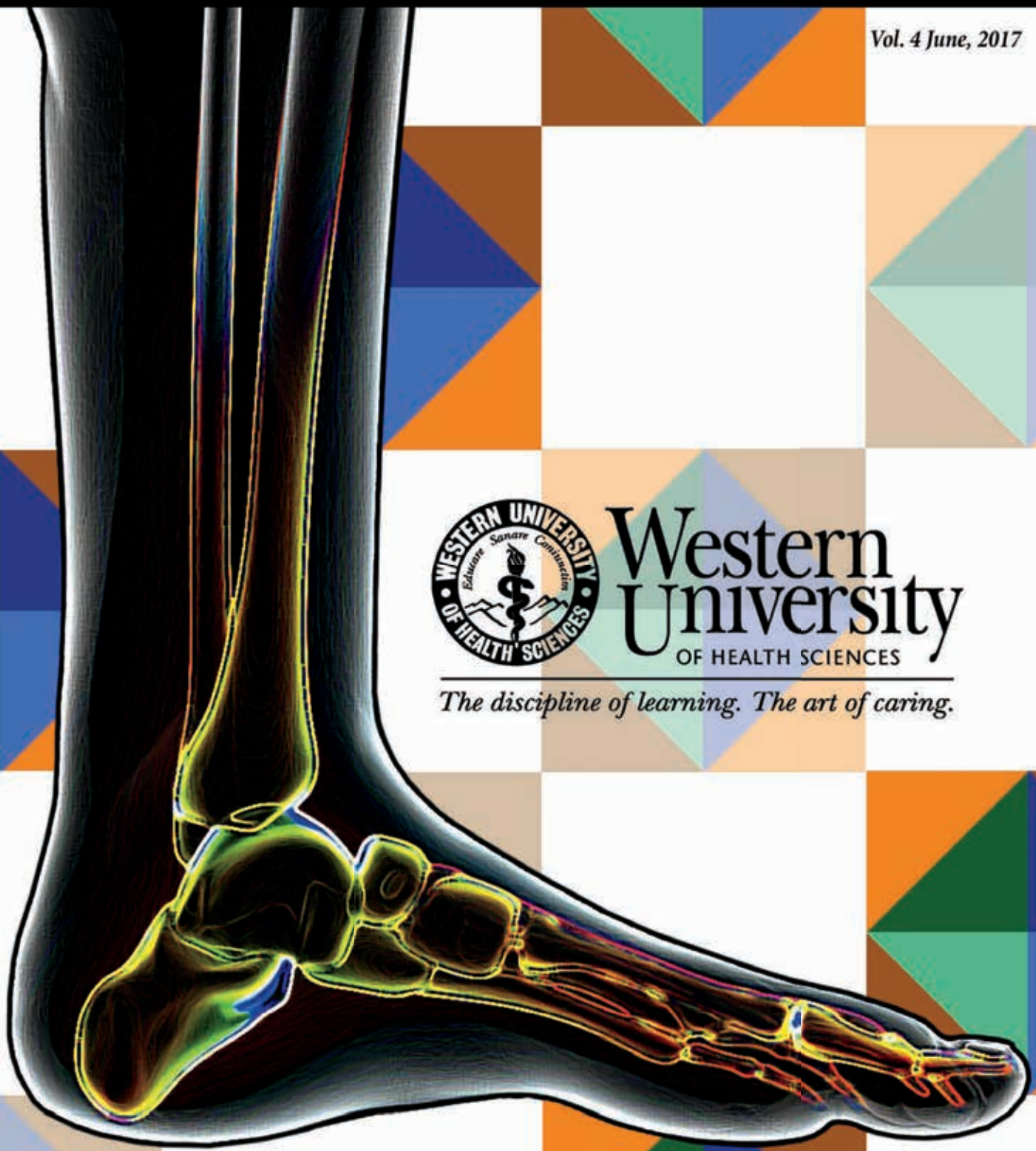

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Dear Readers,

We are excited to present to you the 4th annual issue of Extremitas: Journal of Lower Limb Medicine. This year's edition is a result of a collaboration between WesternU's students and faculty, local podiatric organizations and charities who share a common goal of advancing the care and medical treatment of the lower extremity. Our editorial team would like to invite you to participate in the tradition of peer review research by taking the time to read through some of the articles that our students and faculty have taken the time to produce over the past year.

Topics this year include review articles and case studies on management of diabetic complications, sports medicine, malignancy and original research on medical language interpretation. We hope that our readers find these discussions as intriguing as we have, and we invite all who are curious about these topics to begin their own scientific investigations. Together we can help our communities become more mobile, active, and healthy.

Sincerely,
The Extremitas Team

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Limitations of Ad Hoc Interpreters on Patient Understanding and Compliance in Diabetic Foot Care

Dana Brems[a], MS2 College of Podiatric Medicine*
Marcela Orellana[a], MS2 College of Podiatric Medicine*
Dr. David Shofler[ab], DPM College of Podiatric Medicine
Dr. Janelle Green[ac] , DPM College of Podiatric Medicine

Abstract

When there is a language barrier between healthcare providers and their patients, medical interpreters are used[9]. These might be trained professionals or untrained "ad hoc" individuals[5]. Oftentimes, when ad hoc interpreters such as family members are used, the podiatric physician's instructions to a patient are lost in translation[7]. Studies have found that professional medical interpreters are more accurate than ad hoc interpreters in the general practitioner setting[6]. However, there is limited literature on how ad hoc interpreters convey terminology used specific to diabetic foot care[11]. Language specific to diabetic foot evaluation includes words that describe pain and sensation of the feet, anatomy of the foot, and self-care instructions. These uncommonly used words and phrases might be difficult for an untrained interpreter to accurately relay. In our study, we will look at how often and to what severity common podiatric phrases are misinterpreted by untrained individuals.

Introduction and Purpose

Proper diabetic patient education is essential in reducing the risk of an injury that can lead to ulcer formation[1]. The prevalence of diabetes among the Latino population in the United States is high[3]. Lavery et al found that Mexican-Americans have a greater risk of amputation than non-Hispanic whites[8]. Although the exact reason for this disparity remains unclear, there is a possibility that lack of patient understanding based on language could play a role[4].

Ideally, Limited English Proficient (LEP) patients are seen by a language concordant provider. Studies have shown that this increases compliance and self-care activities[3] like those needed for diabetic foot care. However, many providers cannot communicate with their LEP patients in Spanish, and rely on interpreters who could be either licensed professionals, or ad hoc interpreters[10]. An ad hoc interpreter is any untrained individual, usually a family member in a medical setting, that provides interpretation services[2].

Our study compared the effectiveness of ad hoc interpreters with regards to delivering podiatric medical information to diabetic patients. We surveyed twenty-one untrained bilingual individuals at a local health fair. The subjects underwent an English to Spanish interpretation test with a total of 30 phrases. There were a series of 10 phrases used by podiatrists at screening visits, 10 instructions given to diabetic patients about adequate foot care, and 10 instructions about wound care. We sought to evaluate if there is significant error in the content of a message conveyed the ad hoc interpreter in the setting of diabetic foot care.

Materials and Methods

1.1. ***Location & Subjects***

Our interpretation test was given to twenty-one bilingual volunteer subjects at the Pomona "Relay for Life" health fair in June 2016. Our inclusion criteria included subjects over the age of 18 who consider themselves bilingual. All subjects were informed that they would be recorded and agreed to participate in the survey and verbal test.

1.2. ***Pre-Test Survey***

Before we conducted our verbal interpretation tests, we asked our subjects to fill out a short survey on paper. The questions included the following:

1. Age:
2. Do you consider yourself bilingual in English and Spanish? (Yes/ No)
3. Do you interpret medical information for a

family member?

- o Yes, about once a year
- o Yes, a few times a year
- o Yes, about once a month
- o Yes, a few times a month
- o No

4. What is the highest level of education you have completed?

- o None
- o Elementary school
- o Middle school
- o High school/ GED
- o Some college
- o College (B.A/B.S)
- o Graduate school

5. Where did you go to school?

- o The U.S.
- o Outside the U.S.
- o Both

6. Do you have diabetes?

- o Yes
- o No
- o Unknown

1.3. *Verbal Interpretation Test*

We conducted an English-to-Spanish verbal interpretation test that consisted of common questions and instructions that a podiatric physician would ask a patient. The test was given via the researcher speaking a series of English phrases, and asking the volunteer to interpret them into Spanish. If needed, the phrase could be repeated, but not re-worded. The responses were spoken into a microphone and saved for later evaluation.

1.4. ***Error Evaluation***

We evaluated the voice recordings for accuracy by transcribing participant responses and comparing them to a master translation done by an American Translators Association (ATA) certified language consultant. We quantified how many mistakes were made, and whether the mistake was an omission, addition, substitution, or false fluency. Additionally, we compared the clinical significance of these mistakes based on whether the mistakes affected the meaning of the conveyed message, and could potentially affect the patient's outcome. Finally, we tallied which words and short phrases were most frequently misinterpreted.

1.5. ***Verbal Test English Script***

The following are the scripts for the verbal interpretation test. The script was drafted by investigators Orellana and Brems, then edited by multiple licensed medical interpreters. The edits were made to include words and phrases most often used by podiatrists. Additionally, common false cognates and words which only loosely translate to Spanish were added.

Foot Evaluation Case

1. When did your foot pain start? Was it sudden or gradual?
2. What makes it better? What makes it worse?
3. Can you describe the pain?
4. Is the pain dull or sharp?
5. Is there a burning, tingling, or pins and needles sensation?
6. Do you have that feeling on both feet?
7. Does the pain radiate anywhere else?
8. In a scale of 1 to 10, how severe is your pain?
9. What is your A1C level currently?
10. Record your glucose levels daily.

Diabetic Foot Care instructions

1. Keep your blood sugar levels in check to prevent neuropathy.
2. Neuropathy is nerve damage, and cannot be reversed once it occurs.
3. You are at a higher risk for developing foot ulcers.
4. Inspect feet daily. Be consistent.
5. Check in between the toes looking for redness or discoloration.
6. If you cannot see your feet, use a mirror or ask a family member to inspect them for you.
7. Wear shoes that fit properly.
8. Fit the longest toe, which might be the 2nd toe instead of the 1st.
9. Have your vision checked yearly to ensure that you can see your feet.
10. Don't walk barefoot because you'd be at higher risk of injury.

Wound Care

1. It is normal to see a small amount of blood on you bandages while the wound heals.
2. Remove your bandage daily and replace with clean ones.
3. Moisten the new bandage, without soaking it.
4. Place this wet bandage on the top of your foot, then wrap a dry one all the way around it.
5. Keep weight off the wound while it heals.
6. To prevent infection, don't get your foot wet when you shower.
7. Wash your foot with sterile water and saline. Pat your foot dry.
8. Look for signs of infection such as pain, redness, warmth, or pus.
9. If you notice these signs of infection, go to the nearest emergency room.
10. You will be provided a machine called wound vac to help the healing process.

1.6. *Verbal Test Spanish Script*

The following is the ATA certified Spanish translation of our script. The audio recordings of our subjects were graded against it.

Evaluación de los pies

1. ¿Cuándo inició su dolor en los pies? ¿Fue repentino o gradual?
2. ¿Qué le ayuda a mejorar? ¿Qué lo hace empeorar?
3. ¿Puede usted describir su dolor?
4. ¿El dolor es punzante o sordo?
5. ¿Existe una sensación de quemadura, hormigueo o de agujas y alfileres?
6. ¿Siente esa sensación en ambos pies?
7. ¿El dolor se extiende a cualquier otra parte?
8. En una escala del 1 al 10, ¿cuán severo es su dolor?
9. ¿Cuál es su nivel de A1C actualmente?
10. Registre el nivel de azúcar (glucosa) en su sangre diariamente.

Instrucciones para el cuidado de los pies de un diabético

1. Mantenga sus niveles de azúcar en la sangre controlados para prevenir la neuropatía.
2. La neuropatía es un daño en los nervios, que no puede revertirse si ha ocurrido.
3. Usted estará bajo un mayor riesgo de desarrollar úlceras en los pies.
4. Inspeccione sus pies todos los días. Sea constante.
5. Revise entre los dedos de sus pies que no haya enrojecimiento o descoloración.
6. Si usted no puede mirar sus pies, use un espejo o pida a algún familiar que los inspeccione.
7. Use zapatos que le ajusten apropiadamente.
8. Ajuste de acuerdo al dedo más largo de su pie, que podría ser el 2º en vez del 1º.
9. Sométase a un examen de la vista cada año, para asegurar que usted podrá mirar sus pies.
10. No camine descalzo, pues usted corre un mayor riesgo de lesionarse.

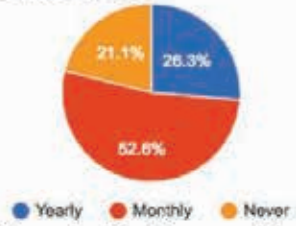
Cuidado de lesiones

1. Es normal ver una pequeña cantidad de sangre en sus vendajes mientras las heridas cierran.
2. Retire su vendaje diariamente, y reemplácelo con uno limpio.
3. Humedezca el nuevo vendaje, sin llegar a empaparlo.
4. Coloque este vendaje húmedo en la parte superior de su pie, y luego envuélvalo completamente con uno seco.
5. No aplique peso sobre la lesión hasta que haya sanado.
6. No moje su pie cuando se bañe, a fin de prevenir infecciones.
7. Lave su pie con agua estéril y salina. Seque su pie con pequeñas palmadas.
8. Busque señales de infección, tales como dolor, enrojecimiento, calor o pus.
9. Si observa estas señales de infección, acuda a la sala de emergencias más cercana.
10. Se le proporcionará una máquina llamada "Wound Vac", que le ayudará con el proceso de curación.

Results

We initially surveyed twenty-one bilingual subjects, two of whom were eliminated from our data for being unable to complete the test. The average age of our remaining participants was 35. The majority completed "some college", and all but one received some formal education in the United States. Only one of our participants had diabetes.

Figure 1 - Ad Hoc Interpreting Frequency Distribution



Over half of our subjects interpret for family members once or more than once a month. (Figure 1)

Figure 2 - Average Number of Clinically Significant Errors

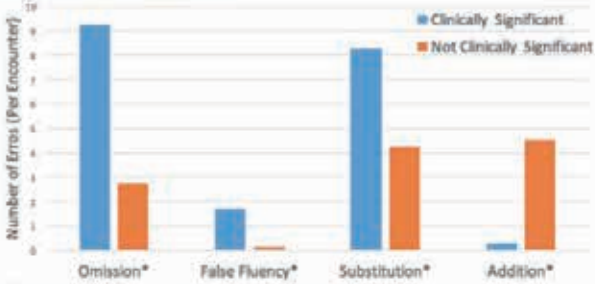


Figure 2 demonstrates the average proportion of errors being clinically significant (CS). For example, the proportion of CS errors of addition is very low. Furthermore, one can see that omission has a high proportion of CS errors, therefore is a serious error of ad hocs. The average number of errors per encounter was 32, and the range was 10-84. 63% of the errors were clinically significant.

Figure 4a – Foot Evaluation Case - Clinically Significant Errors

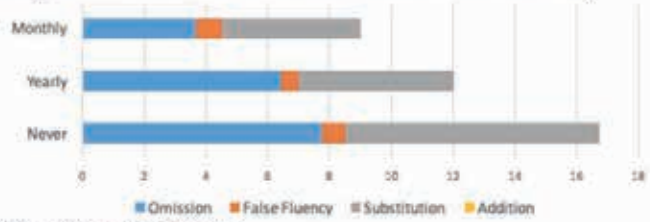
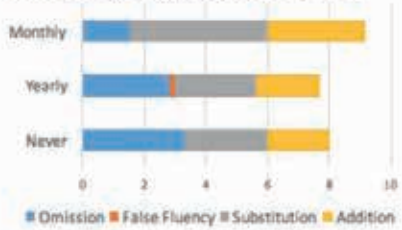


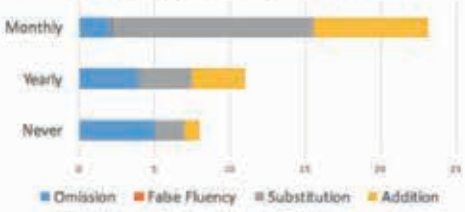
Figure 4b – Foot Evaluation Case – Not Clinically Significant Errors



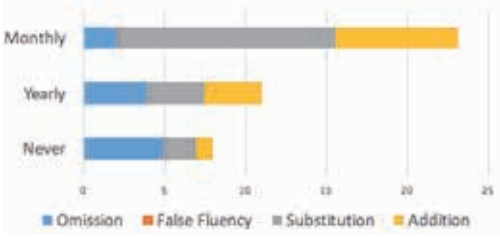
On average, the frequent interpreters made fewer clinically significant errors during a general foot evaluation than those who had never interpreted for family members. (Figure 4a)

Figure 5a – Diabetic Foot Case - Clinically Significant Errors

Figure 5b – Diabetic Foot Case – Not Clinically Significant Errors



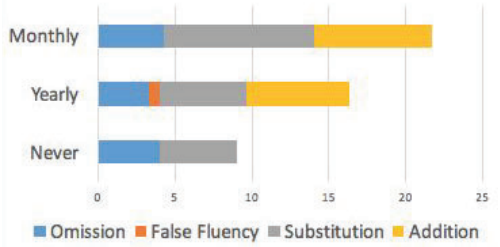
On average, the frequent interpreters made more errors in the diabetic foot encounter, but the errors were more likely to not be clinically significant. (Figure 5b)



On average, the frequent interpreters made more errors in the diabetic foot encounter, but the errors were more likely to not be clinically significant. (Figure 5b)

Figure 6a – Wound Care Case - Clinically Significant Errors

Figure 6b – Wound Care Case – Not Clinically Significant Errors



Overall, the highest numbers of errors were made in the wound care case. (Figure 6a)

Table 1 – Commonly Misinterpreted Words and Phrases

Omission	False Fluency	Substitution
Warmth*	Redness*	"To help the healing process"*
Pain*	Saline*	Bandages**
"While it heals"*	To prevent*	Sharp**
"Pat dry"/"a dry one"*	Neuropathy **	Dull**
Currently*		Gradual*
Tingling*		(to keep) in check*
Pins & needles*		Higher risk*
Be consistent*		
Foot ulcer*		

*Misinterpreted by over 50% of our subjects
**Misinterpreted by over 90% of our subjects

Discussion

Overall we found that ad hoc interpreters, usually family members of the patient, consistently make errors. Virtually all of our interpreters made multiple errors that could be considered clinically significant, with an average of 32 errors per participant, per encounter. This demonstrates the need for professionally licensed interpreters in the podiatric medical setting, even if a bilingual family member is present.

Our data analysis demonstrates that podiatrists cannot assume how well an ad hoc will interpret based on his or her education or experience. We found that there was no correlation between education level and

interpretation accuracy. Furthermore, although ad hocs who interpret for family members made fewer false fluency and omission errors than those who do not, they still made a similar number of CS errors of omission. Conversely, those who interpret monthly for family members had higher rates of CS addition errors than those who do so less frequently. One possible reason is that ad hocs who frequently interpret for family members have experience working with doctors, and may add potentially incorrect comments based on their pre-existing knowledge. Further studies are needed to assess the reason behind this finding.

It is important to mention that most of the clinically significant errors were due to words or phrases that are not necessarily medical jargon. We noticed that words that describe the quality of pain, such as “dull” and “sharp”, were often substituted for words that describe the severity of pain. Knowing the quality of pain is important in any patient encounter, particularly for diabetic patients who may develop neuropathy. Providers can improve the quality of interpretation by using descriptive terms or pictorials when discussing quality of pain.

Furthermore, we noticed that words at the ends of long phrases were often omitted. To prevent these omission errors, we suggest speaking in short phrases when using an interpreter, whether trained or ad hoc.

Future studies are needed to evaluate the use of concise terms to describe pain when using ad hoc interpreters. Specifically, these studies should find replacements for commonly misinterpreted words. This research could also be expanded and applied to other fields of medicine.

In conclusion, understanding physician instructions is critical to diabetic foot care. Our study shows that untrained interpreters are insufficient to accurately interpret for the healthcare provider, therefore we emphasize the need for trained interpreters to best necessitate patient understanding and manage patient health.

Acknowledgements

Thank you to the Dr. Janelle Green and Dr. David Shofler for mentorship on this project. Thank you to the certified medical interpreters that provided feedback on our draft cases. Funding provided by Western University of Health Sciences 2016 Summer Research Fellowship.

References

1. Armstrong, D. G., & Lavery, L. A. (1998). Diabetic foot ulcers: prevention, diagnosis and classification. American family physician, 57(6), 1325-32.
2. Black, S., Maitland, C., Hilbers, J., & Orinuela, K. (2016). Diabetes literacy and informal social support: A qualitative study of patients at a diabetes centre. Journal of clinical nursing.
3. Detz, A., Mangione, C. M., de Jaimes, F. N., Noguera, C., Morales, L. S., Tseng, C. H., & Moreno, G. (2014). Language concordance, interpersonal care, and diabetes self-care in rural latino patients. Journal of general internal medicine, 29(12), 1650-1656.
4. Erfe, B. L., Siddiqui, K., Schwamm, L., & Mejia, N. (2016). The Impact of Professional Medical Interpreter Involvement on the Care of Acute Ischemic Stroke Patients at an Academic Medical Center (S8. 001). Neurology, 86(16 Supplement), S8-001.
5. Flores, G., Laws, M. B., Mayo, S. J., Zuckerman, B., Abreu, M., Medina, L., & Hardt, E. J. (2003). Errors in medical interpretation and their potential clinical consequences in pediatric encounters. Pediatrics, 111(1), 6-14.
6. Hacker, K., Choi, Y. S., Trebino, L., Hicks, L., Friedman, E., Blanchfield, B., & Gazelle, G. S. (2012). Exploring the impact of language services on utilization and clinical outcomes for diabetics. PloS one, 7(6), e38507.
7. Hudelson, P., Dao, M. D., Perron, N. J., & Bischoff, A. (2013). Interpreter-mediated diabetes consultations: a qualitative analysis of physician communication practices. BMC family practice, 14(1), 1.
8. Lavery, L. A., Armstrong, D. G., Wunderlich, R. P., Tredwell, J., & Boulton, A. J. (2003). Diabetic Foot Syndrome Evaluating the prevalence and incidence of foot pathology in Mexican Americans and non-Hispanic whites from a diabetes disease management cohort. Diabetes care, 26(5), 1435-1438.
9. Lor, M., Xiong, P., Schwei, R. J., Bowers, B. J., & Jacobs, E. A. (2016). Limited English proficient Hmong-and Spanish-speaking patients’ perceptions of the quality of interpreter services. International journal of nursing studies,54, 75-83.
10. Nápoles, A. M., Santoyo-Olsson, J., Karliner, L. S., Gregorich, S. E., & Pérez-Stable, E. J. (2015). Inaccurate Language Interpretation and Its Clinical Significance in the Medical Encounters of Spanish-speaking Latinos. Medical care, 53(11), 940-947.

11. Peter-Riesch, B. (2016). The Diabetic Foot: The Never-Ending Challenge. InNovelties in Diabetes (Vol. 31, pp. 108-134). Karger Publisher

Case Study: Eccrine Poroma Conversion to Squamous Cell Carcinoma

Gennady Kolodenker, DPM
Elnaz Hamedani MS1 College of Podiatric Medicine
Brittany Mammano MS2 College of Podiatric Medicine

Abstract

Eccrine poroma is a rare, slow growing, and benign tumor that originates from the eccrine sweat glands. It is reported that 47% of eccrine poroma cases occur on the foot, and it is commonly asymptomatic [1]. We report a patient who presented with a lesion on the lateral aspect of the right foot at the mid-calcaneal body. An initial biopsy of the lesion indicated that it was an eccrine poroma, and the lesion was surgically excised within three months. However, pathological examination of the excised tissue determined that the lesion had converted to a squamous cell carcinoma. Squamous cell carcinoma is a malignant neoplasm and it requires complete excision to prevent the chance of metastasis [4]. Squamous cell carcinoma of the foot is very rare, as only 2.4% of all cases are found on the foot [2]. The conversion of eccrine poroma to squamous cell carcinoma is rare and has previously not been reported in scientific literature. Since eccrine poromas are commonly thought to be benign, the details of this case can be of interest for practicing podiatrists, dermatologists, and general practitioners when evaluating soft tissue masses of the lower extremity. Additionally, it can be a reminder to health care providers of how quickly cells can become cancerous.

Introduction

An eccrine gland consists of a single tubule approximately 6mm in length [3]. The gland is composed of an intra-epidermal portion, or acrosyngium, and a dermal duct, located in the dermis [3]. Eccrine poroma is a slow growing nodular tumor that grows from the acrosyngium [AE1] [BM2] of eccrine sweat glands and can be found on any cutaneous surface. Eccrine poromas are benign and account for less than 1% of primary cutaneous lesions [4]. They are commonly found in middle aged and elderly patients, and studies suggest that long-term radiation exposure and a past history of skin disease increase the likelihood of developing a poroma [4]. Although eccrine poromas have been diagnosed on unique regions of the body such as the face and neck, they are commonly found on the soles of feet [5]. Eccrine poromas are easily treated by excision of the lesion, and have a low probability of recurrence [4].

Unlike eccrine poromas, squamous cell carcinomas are malignant tumors that grow from the squamous epithelium of the dermis [6]. A squamous cell carcinoma is rarely seen in the foot [6]. The preferred method of treatment for squamous cell carcinoma is wide excision to effectively remove all carcinoma cells and prevent metastasis [6]. An estimated 60% of squamous cell carcinoma cases originate from actinic keratosis lesions, which are skin lesions caused by continuous exposure to ultraviolet radiation [7]. Burn wounds can also lead to the development of Marjolin's ulcers, which have a 71% conversion rate to squamous cell carcinoma [8]. Squamous cell carcinoma can also develop from preexisting skin wounds such as ulcers, pressure sores, fistulas, and diabetic wounds [13,14]. Although there have been previous cases of

squamous cell carcinoma development from wounds, there is no literature about the conversion of an eccrine poroma to a squamous cell carcinoma[AE3] [BM4] .

Case Report

A seventy-one-year old female patient presented with a round granular lesion on the lateral aspect of the right foot at the mid calcaneal body. Patient has a past history of hypothyroidism and no known allergies. Lesion was nodular, limited to the skin and subcutaneous tissue, and measured 17x9x7 mm upon excision. A biopsy was performed and the lesion was diagnosed as an eccrine poroma. The lesion was completely excised three months later.

A pathological examination determined that the excised soft tissue mass was a squamous cell carcinoma. The squamous cell carcinoma was a grade one tumor with a maximum dimension of 11 mm. Tumor was well-differentiated and without vascular or perineural invasion. A pathology report showed no evidence of malignancy in the resection margins of the tumor[AE5] [BM6] . Follow up for the patient indicated no complications.

Discussion

Eccrine poroma is a rare benign tumor that originates from the acrosyngium of eccrine sweat glands and is commonly presented in elderly patients. It is a nodular asymptomatic neoplasm that varies in color from pink to red, and has either a smooth or ulcerated surface [9]. Eccrine poromas typically grow on the extremities, and the majority of cases occur on the foot, which has a large amount of eccrine sweat glands. Dermoscopic studies indicate that eccrine poromas contain a variety of glomerular, hairpin, and linear irregular vessels, which can be used to differentiate these benign tumors from other skin lesions [9]. Radiation exposure, past history of skin disease, and the human papilloma virus are thought to cause the development of an eccrine poroma [4,10].

Although eccrine poroma is benign, it does have the ability to mimic other skin cancers. In rare cases eccrine poroma can develop malignant traits, and is known as porocarcinoma, which is characterized by infiltrative growth pattern, nuclear atypia, increased mitotic activity, and necrosis [11]. Clinical case studies have reported that eccrine poromas can develop physical features of malignant skin tumors [10]. Kang et. al present a case of a vascular, bleeding eccrine poroma that was benign despite appearing malignant [10]. Furthermore, a study by Kassuga et al. presents a case of facial pigmented eccrine poroma that simulated a pigmented basal cell carcinoma [12]. Due to the possibility of an eccrine poroma developing malignant traits, the recommended treatment is complete excision with wide margins followed by pathology testing.

The patient presented in this case is a unique instance of eccrine poroma conversion to squamous cell carcinoma. Our patient had a nodular lesion that was indicated by pathology biopsy as eccrine poroma that was completely excised. However, from the date of the diagnosis to the date of the excision procedure, the eccrine poroma had rapidly converted to a squamous cell carcinoma. This result was confirmed by pathological examination of the excised tissue mass.

Squamous cell carcinoma is a malignant tumor that arises from the squamous epithelium. Unlike eccrine poromas, cases of squamous cell carcinoma in the foot are rare. Previous literature indicates that there are instances of squamous cell carcinoma developing from chronic skin wounds. Chronic scars such as burn wounds can convert to malignant tumors, which are called Marjolin's ulcers

[13]. The majority of Marjolin's ulcers become squamous cell carcinoma, although the process of a malignant tumor developing from a wound can take several years to occur [13]. In a 2015 case study by Cho et al., squamous cell carcinoma developed quickly within one month from a chemical burn wound [13]. There have been additional reports of squamous cell carcinoma developing from ulcers, pressure sores, fistulas, and even diabetic foot wounds, although there is no literature about squamous cell carcinoma development from an eccrine poroma [14,15]. Since squamous cell carcinoma can originate from preexisting wounds, it is necessary to thoroughly test skin lesions for this malignancy.

Conclusion:

In this case study we reported a rare eccrine poroma, located at the mid-calcaneal body of the right foot, that converted to a squamous cell carcinoma. This rare case signifies the importance of adequate size biopsy, prompt treatment, and pathology testing of eccrine poroma as a precaution against the development of squamous cell carcinoma. Considering that eccrine poromas are commonly thought to be benign, the details of this case can be of interest for practicing podiatrists, dermatologists, and general practitioners when evaluating soft tissue tumors of the lower extremity. Additionally, it can be a reminder to health care providers of how easy it is for cancer to be undetected.

Future studies of similar cases should include photo documentation of the lesion before biopsy so further cases can be recognized. More research regarding eccrine poromas is needed, including further studies on the prevalence of eccrine poroma conversion to squamous cell carcinomas.

References:

1. Morais P, Guimarães M, Canelhas A, Azevedo F. Unknown: Pedunculated nodule on the foot. Dermatol Online J 18:5, 2012.

2. Rabjohn L, Donohue C, Montilla R, Lavan F. Squamous cell carcinoma of the foot. Podiatry Management Aug 2006:189-198.

3. Wilke, K., A. Martin, L. Terstegen, and S. S. Biel. "Review Article: A Short History of Sweat Gland Biology"." *International Journal of Cosmetic Science* 29 (2007): 169-79. Web. 27 Feb. 2017.

4. Sawaya JL, Khachemoune A. Poroma: a review of eccrine, apocrine, and malignant forms. Int J Dermatol 53:1053-1061, 2014.

5. Kong TH, Ha TH, Eom MS, Park SY. Eccrine poroma of the auricle: a case report. Korean J Audiol 18:151-152, 2014.

6. Wani, I. Metastatic squamous cell carcinoma of foot: case report. Oman Med J 24:49-50, 2009.

7. Goldenberg G, Perl M. Actinic keratosis: update on field therapy. J Clin Aesthet Dermatol 7:28-31, 2014.

8. Tobin C, Sanger JR. Marjolin's ulcers: a case series and literature review. Wounds 26:248-254, 2014.

9. Ferrari A, Buccini P, Vitaliano S, De Simone P, Mariani G, Marenda S, Hagman JH, Amantea A, Panetta C, Catricalà C. Eccrine poroma: a clinical-dermoscopic study of seven cases. Acta Derm Venereol 89:160-164, 2009.

10. Kang MC, Kim SA, Lee KS, Cho JW. A case of an unusual eccrine poroma on the left forearm area. Ann Dermatol 23:250-253, 2011.

11. North, Jeffrey P., MD, Timothy H. McCalmont, MD, and Beth S. Ruben, MD. "Cutaneous Adnexal Tumors." Ed. June K. Robinson. *UpToDate* (2016): n. pag. Web. 14 Jan. 2017.

12. Kassuga LEBP, Jeunon T, Jenoun Sousa MA, Campos-do-Carmo G. Pigmented poroma with unusual location and dermatoscopic features. Dermatol Pract Concept 2:39-42, 2012.

13. Cho HR, Kwon SS, Chung S, Kie JH. Rapidly developed squamous cell carcinoma after laser therapy used to treat chemical burn wound: a case report. World J Surg Oncol 13: 1-4, 2015.

14. Cocchetto V, Magrin P, Andrade de Paula R, Aidé M, Monte Razo L, Pantaleão L. Squamous cell carcinoma in chronic wound: Marjolin ulcer. Dermatol Online J 19(2):7, 2013.

15. Chiao HY, Chang SC, Wang CH, Tzeng YS, Chen SG. Squamous cell carcinoma arising in a diabetic foot ulcer. Diabetes Res Clin Pract 104:54-56, 2014.

Figures

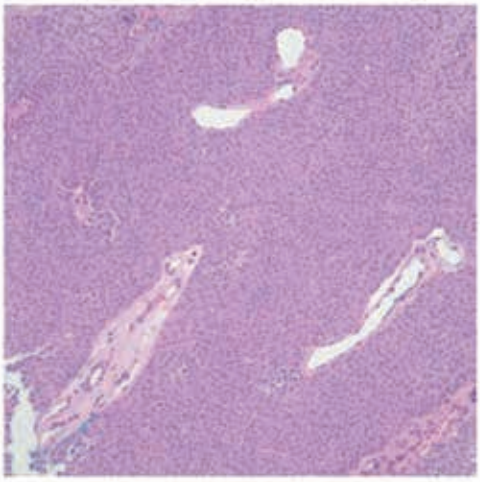


Figure 1 Histomicrograph of eccrine poroma (original magnification 200x, hematoxylin and eosin).

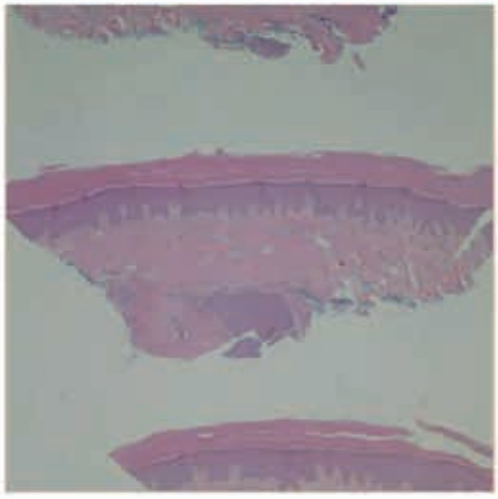


Figure 2 Histomicrograph of eccrine poroma (original magnification 40x, hematoxylin and eosin).

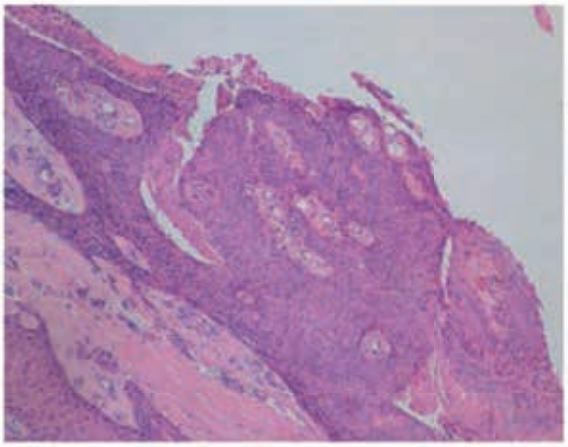


Figure 3 Histomicrograph of the excised squamous cell carcinoma (original magnification 100x, hematoxylin and eosin).

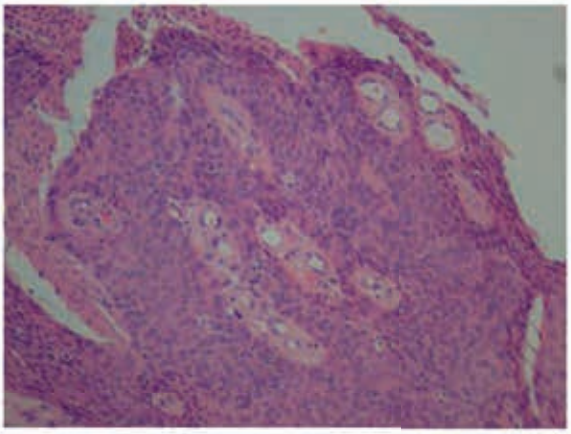


Figure 4 Histomicrograph of the excised squamous cell carcinoma (original magnification 200x, hematoxylin and eosin).

Case Review: Osteonecrosis of the Distal Tibia

Michael Czulinski, MS3 College of Osteopathic Medicine

Introduction

Atraumatic joint pain is common in aging populations. Common etiologies such as osteoarthritis are often at the top of the differential. This case review discusses a presentation of atraumatic talocrural pain due to osteonecrosis (ON) of the distal tibia, a relatively uncommon location of disease occurrence. This study aims to briefly review the common etiology and pathophysiology of ON while also discussing the surgical treatment that patient "T.C." received for management of distal tibial ON.

Patient Background & Presentation

T.C. is a 64 year-old female who presented to the foot and ankle clinic wearing a right ankle brace. Symptoms included insidious onset of right ankle pain in the absence of trauma, redness, swelling, fevers or chills. Radiographic evaluation demonstrates evidence of degenerative joint disease (DJD). Additionally, review of previous radiographic studies of the ankle noted small lucent foci in the fibula which merited a follow up bone scan. NM bone scan showed increased activity in the distal aspect of the right tibia, considered a nonspecific finding. Both sides of the talocrural joint were not involved, thus the increased uptake was determined to be less likely related to DJD at that time. T.C. received triamcinolone injection for possible synovitis and DJD with noted decreased pain at follow up. She was instructed to discontinue the brace and begin physical therapy.

T.C. was managed with moderate success after initial presentation. Approximately 6-8 months later she went to the local urgent care complaining of atraumatic right ankle pain with associated swelling. The ankle was immobilized and repeat radiographs noted soft tissue swelling without fracture (**Figure 1**). A MRI was obtained and revealed a lesion within the distal tibial metaphysis consistent with bone infarct, edema and tibialis posterior tenosynovitis (**Figure 2**). Imaging did not show involvement of the joint nor did it show evidence indicative of early joint collapse.

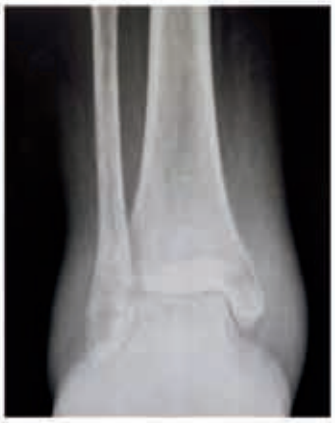


Figure SEQ Figure 1: AP weight-bearing radiograph taken days prior to MRI. Normal with some soft tissue swelling.

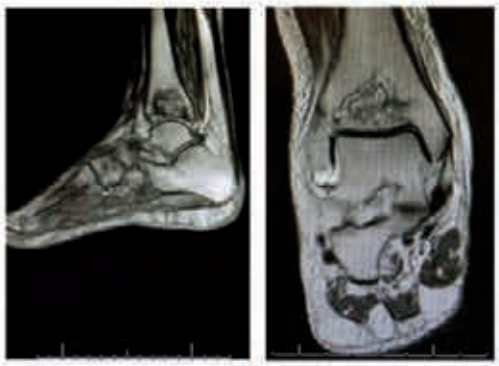


Figure Figure 2: Ankle MRI showing avascular necrosis of the distal tibia without obvious involvement of the talocrural joint.

Physical Exam

On initial presentation, the exam was relatively unremarkable. T.C. was found to have intact 2/4 dorsalis pedis and posterior tibial pulses bilaterally. Brisk and rapid capillary refill was noted in all digits with no observed focal erythema or edema. Digital hair growth was present bilaterally. Neurologic exam confirmed normal proprioception with intact sensation to gross touch. Integument was without blisters, open lesions, maceration, or hyperpigmentation. Musculoskeletal evaluation revealed anteromedial and anterolateral ankle tenderness to palpation. Ligamentous structures were stable and intact. Foot structure appeared to be rectus with very mild eversion. There was no redness, swelling, or heat at the joint. There was no pain with double heel raise exam. When T.C. presented to urgent care 6-8 months later, physical exam remained relatively unchanged. New findings included 10/10 right ankle pain and pitting edema around the right ankle.

Pathophysiology of Osteonecrosis

There are many etiologies associated with ON, including trauma, corticosteroid use, and alcohol. The relative significance of each association is unclear and the pathophysiologic understanding of the various etiologies remains imperfect. Most proposed mechanisms agree that the terminal event leading to ON is the disruption of normal microvasculature, which then leads to nutrient deficient avascular necrosis of the bone [9]. The means by which this disruption occurs vary and are important to distinguish as it may alter possible treatment modalities. Suggested mechanisms of microvascular disruption include trauma, occlusion, or an increase in intraosseous pressure leading to vascular compression [2,9].

Trauma causing interrupted vascular supply is accepted as an etiology leading to ON [2,9]. This is especially true for regions with poor collateral blood supply. Vascular occlusion has also been associated with ON and may be caused by hemaglobinopathies such as sickle cell disease, hypercoagulability, or lipid microthrombi, etc. Extraluminal compression through adipocyte hypertrophy and resulting ischemia has also been suggested as a mechanism of corticosteroid or alcohol related ON [3,9]. Ultimately, the pathophysiology is better understood than it once was and corticosteroid use, alcohol, and trauma are common risk factors for developing ON through these various mechanisms. ON can occur at any point but tends to be diagnosed later in life. In the case of T.C., it remains unclear what the inciting event was as she did not have any major known risk factors.

Surgical Management

Research on conservative management of ON has shown lower success rates in treating the disease in comparison to surgical management [1,5,7]. Some surgical approaches include core decompression (CD), bone grafting, and total joint replacement. CD consists of drilling necrotic lesions. Evidence exists on avascular necrosis of the hip suggesting that CD is useful in the treatment of earlier stages of disease and is associated with better outcomes than non-operative management [5]. T.C.'s presentation was consistent with early stage disease. A survey conducted by McGrory et al. on the treatment of femoral head ON was administered to the American Association of Hip and Knee Surgeon members. This survey was sent to active members and found that CD was the most commonly offered treatment approach for symptomatic pre-collapse ON of the femoral head [4]. A literature review conducted by Tripathy et

al. also noted CD is the most common and cost effective treatment for ON of the hip in early stage disease [10]. This study also discussed the possible advantage of multiple drilling decompression as an improved technique to traditional CD [10]. Since CD is frequently used and has notable success in treating ON elsewhere in the body, it seems reasonable to utilize CD to treat early stage distal tibia ON.

Arthroscopic evaluation of T.C.'s talocrural joint confirmed that the articular surfaces were not involved. T.C. received multiple percutaneous subchondral drillings of the distal tibia with a 0.062 K wire. This was performed for T.C. in hopes of alleviating pain, improving perfusion, and stimulating healing. It is worth noting that CD has been combined with adjunctive therapies like autogenous bone marrow aspirate transplantation or electrical stimulation though neither was performed for T.C.

Conclusions

Given the various possible causes of ON, it seems likely that the process is a multifactorial disease. Etiologies such as corticosteroid use, trauma and alcohol are among the more common risk factors though some cases remain seemingly idiopathic. Each etiology likely causes disruption in bone perfusion through one or more of the mechanisms previously described. Unfortunately, the standard of care for osteonecrosis of the distal tibia has not been clearly defined. CD is commonly used for the treatment of early stage ON. It remains to be seen how T.C. will respond to surgical intervention but at day 12 post-op, she noted decreased pain. T.C. will continue with range of motion exercises, physical therapy, and periodic evaluation to monitor response.

References

1.Hong Y, Zhong H, Lin T, Shi J. Comparison of core decompression and conservative treatment for avascular necrosis of femoral head at early stage: a meta-analysis. International Journal of Clinical and Experimental Medicine. 2015;8 4, p5207-p5216.

2. Kummar, Abbas, Aster. R. Robbins and Cotran Pathologic Basis of Disease Ninth Edition. Philadelphia, PA: Elsevier/Saunders, 2015. pp 1194-1195. Print.

3. Lafforgue P. Pathophysiology and Natural History of Avascular Necrosis of Bone. Joint Bone Spine. 2006; 73(5), pp. 500-7.

4. McGrory B, York S, Teeny S, et al. Current practices of AAHKS members in the treatment of adult osteonecrosis of the femoral head. Journal of Bone & Joint Surgery, American Volume [serial online]. June 2007;89(6):1194-1204.

5. Monte M, Carbone J, Fairbank A. Core decompression versus nonoperative management of osteonecrosis of the hip. Clin Orthop Relat Res. 1996; (324)169-78.

6. Monte M, Ulrich S, Seyler T, Smith J, Marker D, McGrath M, Hungerford D, Jones L. Bone scanning of limited value for diagnosis of symptomatic oligofocal and multifocal osteonecrosis. Journal of Rheumatol. 2008; 35(8): 1629-34.

7. Musso E, Mitchell S, Schink-Ascani M, Bassett C. Results of conservative management of osteonecrosis of the femoral head. Clinical Orthopeadics and Related Research. 1986; (207), pp. 209-215.

8. Powell C, Chang C, Gershwin E. Current Concepts on the Pathogenesis and Natural History of Steroid-Induced Osteonecrosis. Clin Rev Allergy Immunol. 2011;41: 102-113.

9. Shah K, Racine J, Jones L, Aaron R. Pathophysiology and risk factors for osteonecrosis. Curr Rev Musculoskelet Med. 2015; 8(3): 201-209.

10. Tripathy S, Goyal T, Sen R. Management of femoral head osteonecrosis: Current concepts. Indian Journal of Orthopaedics. 2015;49(1):28-45.

The Biomechanics of Cycling, and the Pathomechanics: A Focus on Cycling-Related Achilles Tendinosis

Sara Yancovitz, MS2, College of Podiatric Medicine
Aleena Resendez, MS2 College of Podiatric Medicine

Introduction

Cycling is a popular sport that is especially known as a low-impact activity. It is a sport that can be done over long periods of time with low incidence of overuse injuries [7]. Unfortunately, injuries are possible and do occur despite the low incidence of overuse injuries. Limited research has been done to recognize the pathomechanics behind some of the common injury-related complaints that cyclists face. Clinicians who are unfamiliar with the nature of the sport fail at identifying the source of injury and are unable to adequately elicit injury prevention [7]. This investigation discusses the biomechanics of pedaling and possible pathomechanical issues that arise from cycling, specifically Achilles Tendinosis.

Cycling is a non-weight bearing activity that can boost cardiovascular health and overall fitness with low impact stress on joints. This activity allows for athletes to cycle for longer periods of time, and consequently further develop their lower extremity anatomy. Cycling exercises a primary set of muscles that do the majority of work for the sport-specific movements. A multitude of factors, such as inappropriate bike fit, pedaling technique and continual muscle usage can induce high levels of stress on joints, tendons, and muscles.

Major Anatomical Consideration in Cycling

The anatomy involved in the motion of pedaling a crank on a road bicycle in an "aggressive" bicycle position which we define as a prolonged flexion at the hip, extended back, adducted and retracted shoulders. Cycling involves core musculature, upper body strength, and extensive use of the lower extremity muscles. When riding a bicycle, the upper body is supported by holding on-to handlebars. The majority of the anatomy involved involves the lower extremity at various points in the pedaling cycling (Figure 1).

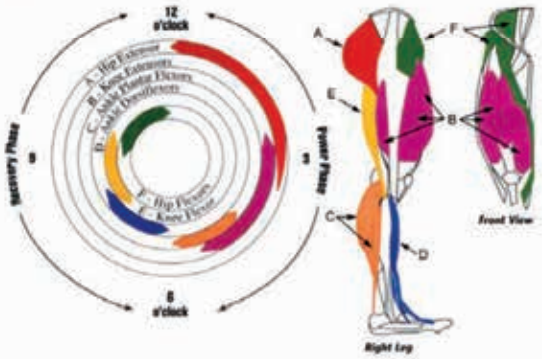


Figure 1 – Muscle Activation during the crank cycle (Adopted from *Success Cycling*, 2015 [3])

Normal Biomechanics of the Crank Cycle

The crank cycle is important in understanding the pedaling biomechanics of cycling. The crank cycle involves various positions of the pedal stroke to propel the bicycle forward without full-body weight bearing. We define the pedal stroke at the 12 o'clock position as the top dead center of force (TDC). The bottom dead center (BDC) is at the 6 o'clock position is 180 degrees apart from the TDC [5].

The crankset installed on road bicycles consist of two cranks -- one for each leg -- that are 180 degrees from each

other throughout the crank cycle. Although the motion occurs concurrently in both leg, each leg motion is examined individually. For simplicity, we will describe the forward pedal stroke in a clockwise motion when looking at the right pedal, also known as *crank-side* (Note: Figure 2).

FIGURE 18.3 Biomechanics use a "clock" diagram to illustrate the forces of ped

Figure 2 – Clock Diagram to illustrate the forces of pedaling (Adopted from Andy Pruitt's "Complete Medical Guide for Cyclists" [5])

Figure 3 – Ankle Position throughout the Crank Cycle (Adopted from *Success Cycling*, 2015 [3])

Electromyographic studies have been done to denote the various muscles activated at the different portions of the crank cycle, and are the basis of future discussion [4]. Muscle coordination using EMG is not quantitative, but qualitative therefore the amount of work each muscle is not precise [6]. Nonetheless, this data tells us at which portion of the crank cycle do certain muscles fire.

There are two different strokes to cycling: the *downstroke* (TDC/0 degrees to 180 degrees) which is considered to be the power phase and the *upstroke* that is known as the recovery stroke (BDC/180 degrees to TDC/360 degrees). The downstroke involves extension of the joints at the hip, knee, and ankle, while the upstroke involves flexion of these joints.

THE DOWNSTROKE

During the first half of the downstroke from TDC to 90 degrees, the hip and knee are at their greatest flexed position, and the ankle is dorsiflexed in most riders. This downstroke uses hip extensors (Rectus Femoris and the Gluteus Maximus), knee extensors (Quadriceps: Vastus Medialis, Vastus Lateralis), Biceps Femoris and the Tibialis Anterior. The hip extensors are active at the TDC and BDC.

In the latter half of the downstroke (90 degrees to BDC), the knee is responsible for the greatest range of motion (75 degrees) [7]. Beginning at it's flexed position at 110 degrees and is extended at approximately 35 degrees [7]. The Quadriceps extends the knee creating a downward force that is applied to the pedal. This generates the major source of torque that continues through the recovery phase of the crank cycle [4]. In the downstroke, the hamstrings extend the knee with help with the Tibialis Anterior muscle. Force transfers through the ankle joint via the gastroc-soleus complex.

At the ankle joint, the Achilles Tendon is responsible for plantarflexing the foot. Providing great leverage inserting at the posterosuperior aspect of the calcaneus. The achilles tendon serves as insertion points for the Gastrocnemius, Soleus, and

Plantaris muscles, which all act synergistically to actively plantarflex the foot. The medial column of the foot is slightly inverted and plantarflexed, and the foot is pronated at the midtarsal and subtalar joints (Note: Figure 3).

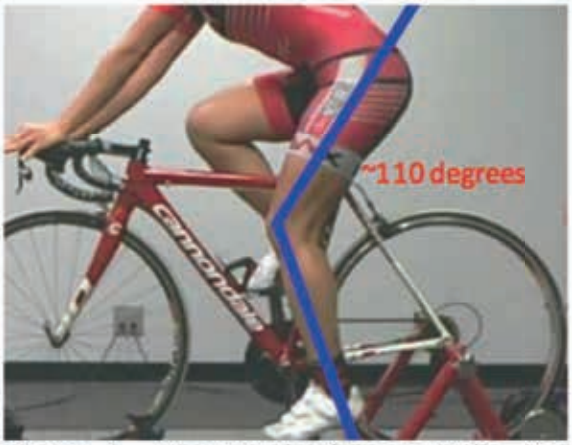


Figure 4 – Downstroke into upstroke, just past BDC

THE UPSTROKE

The upstroke (BDC/180 degrees to TDC/0 degrees) phase, although a less force-producing motion, is considered to be equally important to cyclist motion in propelling the pedal stroke. The rider drives the pedal up and back to the initial TDC position, generating maximum force through the crank cycle.

At the end of the downstroke, the ankle joint is slightly plantarflexed and the hamstrings are in an optimal position to contract (Note: Figures 3 and 4). The hamstrings flex the knee synergistically with the Gastrocnemius muscles. Cyclists flex at the hip through their Iliopsoas and Rectus Femoris muscles, lifting the leg up to follow through with the next rotation of the crank. The Tibialis Anterior muscle assists with the dorsiflexion of the ankle joint back to it's initial position.

For this review, we assume the cyclist has a proper bicycle fit. Some variables include the appropriate saddle height and position (forward/backward), handlebar height and position (closer/farther to the rider), length of the crank, and foot position. Any major deviation from the above can cause alternative muscle recruitment and variation in muscle firing frequency.

Cyclists are prone to pathomechanics when their measurements on the bicycle are less than ideal. As aforementioned, improper bicycle fits can influence alternative muscle recruitment and variation in muscle firing frequency. As a result of constant and repetitive pedaling motion, injury can arise. Although Achilles tendinosis is not the most common cycling injury, it is a chronic and debilitating injury that can hinder the daily activity of a cyclist. The most common ankle problem in athletes is Achilles Tendinopathy [8]. Though the true incidence of Achilles Tendinosis is unknown, it is approximated that over 1 million athletes per year are affected [9]. The following section discusses a case presentation of Achilles Tendinopathy related to this pathomechanical issue.

Case: Cyclist with Achilles Tendinosis

A 22-year-old, 5'10", 142 lb competitive male cyclist complained of right ankle pain. He sought help from a physical therapist who determined his right ankle had limited dorsiflexion. The patient was examined with right-sided gastrocnemius-equinus and was diagnosed with Achilles Tendinosis and prescribed ankle stretching to improve ankle range of motion. Stretching exercises included non-weight bearing active inversion and eversion of the ankle (Note: Figure 5), active plantarflexion and dorsiflexion of the ankle (Note: Figure 6), and gastrocnemius wall stretches (Note: Figure 7). This cyclist rode 15 hours/week for 10 years and had never experienced prior Achilles pain. Every bicycle that he rode underwent a professional precise fit. The

cyclist's position was assumed to be optimal for his body geometry. The cyclist claimed he had an acute increase in training load. He recently had 2 consecutive weeks with more than 20 hours riding/week, as well as, the transition to his new team-issued bike. Though he had undergone a professional bike fit, he had recently disassembled his bike for transport on a trip. He reconstructed the bike built with appropriate measurements except for the saddle height, which he forgot to physically mark on the seat post.

The patient was examined on a stationary trainer by a bike fitter, and the pedal stroke of the cyclist revealed normal pedal stroke throughout the crank cycle. However, the cyclist complained his pedal stroke "felt weird." The bike fitter raised the seat post, increasing the seat height 2mm. At a follow-up evaluation at the physical therapist three weeks later, ankle dorsiflexion increased a few degrees and pain of the ankle during the cycling motion subsided.

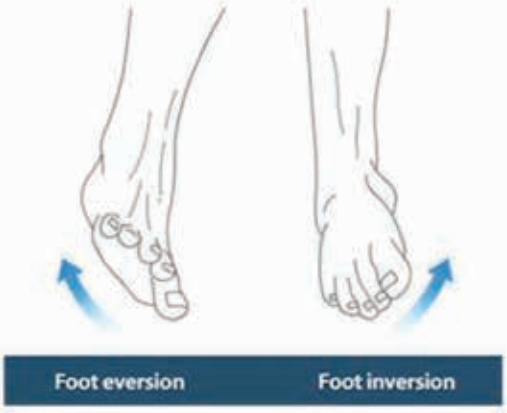


Figure 5- Foot Stretches Demonstrating Ankle Eversion and Foot Inversion [16]

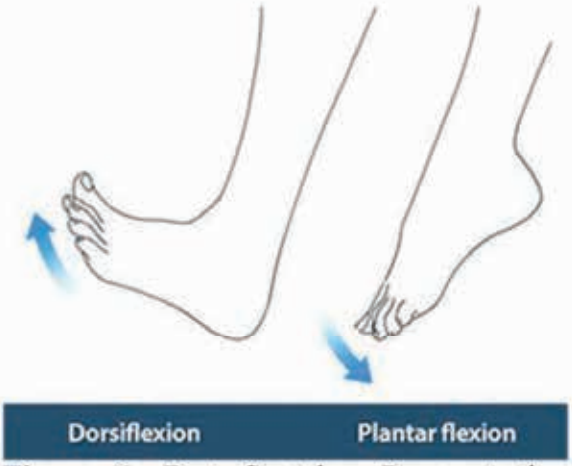


Figure 6- Foot Stretches Demonstrating Dorsiflexion and Plantarflexion [16]

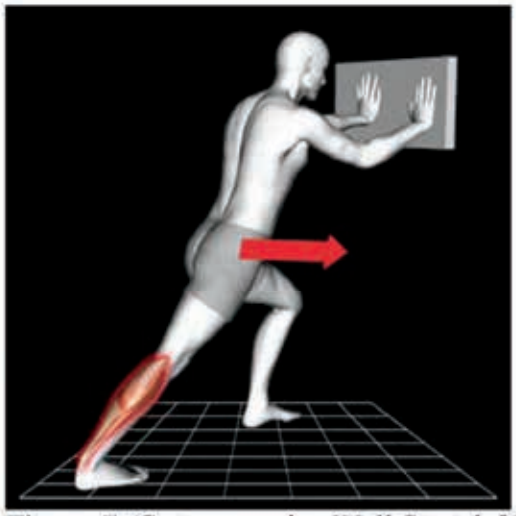


Figure 7- Gastrocnemius Wall Stretch [15]

Discussion:

This case provides an example of how low saddle height was a contributing factor for a pathomechanical position, which resulted in the presentation of Achilles tendinosis. This assumption is made as the seasoned rider does not claim any other changes in bike position. Though it is possible that other factors may contribute to the pathology, there are no other variables to compare this outcome.

Deviations from appropriate saddle height determines the joint range of motion

as well as muscle length and contraction available [17]. In addition, saddle height also plays a role in determining the rider's center of mass and changing the interaction between the seat and handlebars [17]. Though there are multiple interface contact points the rider is subject to, this discussion will only be limited to the lower extremity. Changing the center of mass can contribute to excess strain on joints and requiring more muscle recruitment to propel the rider [17]. There are various methods for determining the appropriate saddle height. One method according to Burke and Pruitt includes measuring from the greater trochanter of the Femur to the point of contact the barefoot makes with the ground [11].

As described in the previous case presentation, a seat that is too low will place the foot in a dorsiflexed position at the BDC, late recovery phase, and early power phase (Note: Figure 8) [12]. This causes the rider's heel to drop, reducing the Gastrocnemius muscle's ability to plantarflex at the ankle [12]. This causes an increase in tension on the Achilles Tendon at the insertion of the Soleus muscle. The increased tension generates the energy needed to plantarflex at the ankle joint [12]. Intensified contraction of the gastroc-soleus complex is a potential source of tendon rupture at the Achilles insertion point [17]. In contrast, a saddle that is adjusted too high will put the rider in a position that overextends the leg at the BDC of the crank cycle (Note: Figure 8). As a result, this causes the Gastrocnemius and Soleus to overcompensate in plantarflexion through transmission at the Achilles Tendon [12].

An additional etiology of Achilles tendonosis is seen with riders who have a low cadence or pedaling rate (under 90 revolutions per minute) Similarly, this is seen when the rider pedals uphill and has to stand on the pedals for a prolonged period of time. Prevention of Achilles tendinopathy could include a higher cadence, pedaling while sitting in the saddle, and also using toe clips that position the foot in a rear facing manner [10].

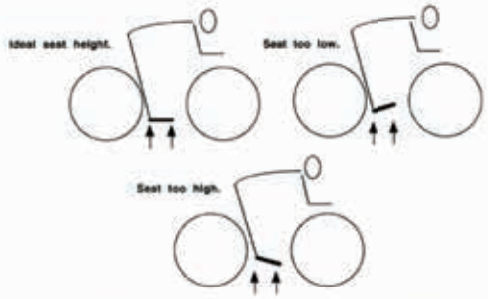


Figure- 8 Foot Position and Saddle Height [14]

Conclusion:

Physicians should be aware of the pathomechanics behind the common lower extremity injuries that cyclists present with, such as Achilles Tendinosis. Solely treating the injury and failure to identify the cause of the injury, such as bike fit, will create a chronic injury for many cyclists which could potentially be more harmful to the patient. More research needs to be done to identify additional sources of cycling-specific injuries, and the many variables that attribute to pathomechanics of the activity. The goal should be to educate healthcare providers accordingly as sport injury prevention is key treatment for cyclists.

If a cyclist is injured, conservative measures should be taken first. Many athletes do not wish to receive inconvenient medical treatment or surgery if they can avoid it. Physicians should be aware of methods of treating avid cyclists, and be willing to educate patients on simple at-home conservative treatments for common cycling injuries like Achilles Tendinopathy. Those treatments include stretching exercises as alluded to in the case presentation.

Physicians should strongly

recommend that cyclists (both healthy and injured) seek appropriate bicycle fit. This is extremely imperative when athletes are attempting high level training. Likewise, it is important to refer avid cyclists to a sport's physical therapist for routine evaluations to prevent chronic and overuse injuries. It is through better understanding and dissemination of information regarding proper cycling biomechanics that healthcare providers can work with athletes to improve their performance and prevent injury.

References:

1. Machin, W. 2013. "Does Riding a Bike Tone Your Arms?," Livestrong. http://www.livestrong.com/article/540278-does-riding-a-bike-tone-your-arms/. Accessed January 12, 2017.
2. MDHealth.com. 2013. "What Muscles Do Push-Ups Work?" MD-Health.com. N.p. Web. 12 Jan. 2017.
3. Success Cycling. 2015. "Pedal Like A Pro." Success Cycling. N.p., Web. 12 Jan. 2017.
4. So RCH, Ng JKF, Ng GYF. Muscle recruitment pattern in cycling: A review. Physical Therapy in Sport. 2005;6(2):89–96. doi:10.1016/j.ptsp.2005.02.004. http://hdl.handle.net/10397/31017. Accessed January 13, 2017.
5. Pruitt AL and Matheny F. 2006. Andy Pruitt's Complete Medical Guide for Cyclists. Velopress.
6. Hug, Francois. 2011. "Review: Can muscle coordination be precisely studied by surface electromyography?" Journal of Electromyography and Kinesiology, Vol 21.1:1-12.
7. WH Sanner and WD O'Halloran (2000) The biomechanics, etiology, and treatment of cycling injuries. Journal of the American Podiatric Medical Association: July 2000, Vol. 90, No. 7, pp. 354-376.
8. Young, J.S. & Maffuli, N. (2007) Etiology and Epidemiology of Achilles Tendon Problems. In The Achilles Tendon, edited by N. Maffuli & L.C. Almakinders. Springer, Verlag, London, pp. 39-49.
9. Hoke, AD. Techniques in Achilles tendon rehabilitation. Tech Foot Ankle Surg. 2003. 2(3):208-19.
10. Deakon RT. Chronic musculoskeletal conditions associated with the cycling segment of the triathlon; prevention and treatment with an emphasis on proper bicycle fitting. Sports Med Arthrosc. 2012;20(4):200-5.
11. Broker and Gregor, 1996 J.P. Broker, R.J. Gregor Cycling biomechanics E.R. Burke (Ed.), High-tech cycling, Human Kinetics, Champaign (1996), pp. 157–163
12. Sanner WH, O'halloran WD. The biomechanics, etiology, and treatment of cycling injuries. J Am Podiatr Med Assoc. 2000;90(7):354-76.
13. Hong-Yun Li and Ying-Hui Hua, "Achilles Tendinopathy: Current Concepts about the Basic Science and Clinical Treatments," BioMed Research International, vol. 2016, Article ID 6492597, 9 pages, 2016. doi:10.1155/2016/6492597
14. Pyle, Steve. "Tri-Guru: Perfect Cycling Position." Tri-Guru: Perfect Cycling Position. N.p., n.d. Web. 18 Jan. 2017.
15. IPC Physical Therapy. 2010. "Soleus Stretch." IPC Physical Therapy. N.p. Web. Available at: http://www.ipcphysicaltherapy.com/SoleusStretch.aspx. Accessed February 26, 2017.
16. Vedi, Viakas. "Foot and Ankle Examination." Mr. Vikas Vedi

Lower Limb Surgeon Consultant Orthopaedic Surgeon Northwood UK. N.p Web. Available at: http://www.vikasvedi.co.uk/foot-ankle-examination-gp-education.html. Accessed February 26, 2017.

17. Jimenez, A. Louis and Kelley, M. Joycelyn. "Insertional Achilles Rupture and Calcaneal Avulsion Fractures." Podiatry Institute. Chapter 29, Pg 151-163. N.p., N.d. Web. 26 Feb. 2017

Case Study: Green Nail Syndrome or Chloronychia in a Young Healthy Female

James Anderson, MS4 College of Podiatric Medicine
Greg Lifferth, MS4 College of Podiatric Medicine

Introduction

Green Nail Syndrome is caused by Pseudomonas Aeruginosa, an aerobic, gram negative rod bacteria. This bacterium flourishes in moist environments or untreated water sources, such as jacuzzis, pools, foam padding of shoes, and bath sponges. This bacterium causes a green pigment when infecting the toenails of the lower extremity.

Green nail syndrome is not a well-researched topic. Treatments that were used on patients included oral ciprofloxacin, topical gentamicin, topical neomycin/polymyxin B galenic unguentum, tobramycin eye drops, and topical nadifloxacin, an acne medication. Our case study will focus on a healthy young female without co-morbidities, displaying clinical features of green nail syndrome. The purpose of this study is to better understand the etiology, review the recommended treatments, and stress that it is important to treat green nail syndrome, also known as chloronychia, to prevent the spread of pseudomonas bacterium to other parts of the body especially in immunocompromised patients.

Background

I.R. is a 21 year-old Hispanic female with no significant past medical history who presents to the emergency department with a chief complaint of green discolored toenails and lesions on her left foot. She states that the nail discoloration has been ongoing for the past six months and has been worsening. She states the discoloration does not improve with scrubbing of her foot with soap and water. Additionally, she relates experiencing bilateral foot pain that is localized to the plantar forefoot. She states the pain is worsened with weight bearing and relieved with rest and elevation. She also describes a blister formation on the plantar left foot.

The patient states she works in a warehouse environment 70-80 hours a week, moving heavy objects and is constantly weight bearing on her feet. She admits to wearing worn out sneakers that are unsupportive and socks that are soaked with sweat by the time she is done with work. She denies any tobacco, alcohol, or illicit drug use. She denies redness, swelling, or any systemic signs of infection such as fever, chills, nausea, and vomiting.

Physical Examination

I.R. presents with moist and malodorous feet, which is worse on the left. The odor is a pungent foot smell, of which she is quite embarrassed. There is noted vesicle formation on the plantar left foot sub first metatarsal head and first interdigital space. Her nails are hypertrophic and yellowish on the right foot, and digits 2-4 on the left foot are hypertrophic and green in color. There are hyperkeratotic lesions noted dorsally on the distal interphalangeal joints of digits two and three and the dorsolateral aspect of digit five of the left foot with a greenish hue and localized periwound erythema. The patient is vascularly intact

with palpable dorsalis pedis and posterior tibial pulses bilaterally and capillary refill time less than 3 seconds on all ten toes. Neurologic exam shows light and sharp sensation is intact in bilateral feet. She has mild tenderness to palpation of bilateral plantar second and third metatarsal heads which is worse on the left. The Hubscher maneuver revealed the windlass mechanism is intact bilaterally. There are noted reducible hammertoe contracture deformities on digits 2-5 on the left foot with no signs of crepitus.

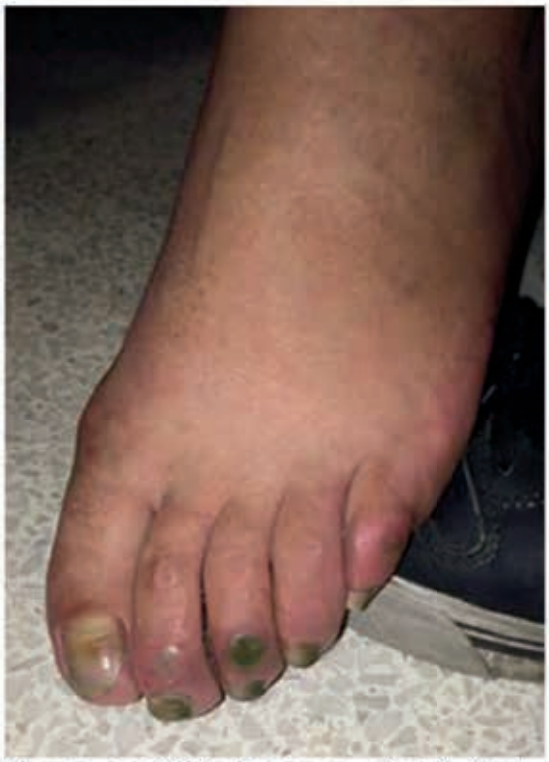


Figure 1: Clinical image of left foot of patient I.R. showing green nail syndrome to nails of digits 2-5 and also a bulla which also appears infected with pseudomonas.

Epidemiology

Although there is limited research or literature on the subject of green nail syndrome or Chloronychia, most of the patients that have been identified with it are immunocompromised, such as the elderly and AIDS patients. It is also more common in patients with occupations such as homemakers, barbers, dishwashers, bakers, and medical personnel.[1]

Pathophysiology

Green Nail Syndrome is caused by Pseudomonas Aeruginosa, an aerobic, gram negative rod bacteria. This bacterium flourishes in moist environments or untreated water sources such as Jacuzzis, pools, foam padding of shoes, and bath sponges. When it grows in isolation, P. aeruginosa is often easily recognized by the hallmark green and blue pigments called pyocyanin and pyoverdin[5]. The condition is commonly associated with onycholysis, the splitting of the nail from the nail bed, which in turn harbors bacterial growth imparting the green color of chloronychia.[6]

Patients who are affected are those who have repeated exposure to water, soaps, and detergents. Also, patients who experience mechanical trauma are prone to green nail syndrome as the pseudomonas bacteria infects the nail beds.[1,3] On clinical examination there is often a triad of greenish or black discoloration of the nail, proximal chronic paronychia, and disto-lateral onycholysis.[7,8]

One such case of repeated micro trauma causing eventual chloronychia is a skier who reported that on a ski trip their hallux was repeatedly forced into the front of the ski boot causing subungual hematoma and onycholysis. Three months later, there was noticeable green discoloration of their hallux toenail.[6]

Differential Diagnosis

Nail Discoloration:

Given the unique clinical presentation of Green Nail Syndrome, the diagnosis is simple. It is worth noting that nail discoloration of varying colors can

result from both chemical exposure, fungal infections, and various systemic diseases.[9,10]

Exposure to chemicals such as resorcinol can lead to discoloration of the nail. Resorcinol, an antiseptic and disinfectant commonly found in soaps, shampoos, and lotions, can lead to brown discoloration of nails.[9] Various fungal infections caused by dermatophyte fungi that target the nail lead to discoloration. Trichophyton rubrum commonly causes white, yellow, or brown discoloration that categorizes distal subungual onychomycosis. Additionally, Trichophyton mentagrophytes may cause dull white spots that characterizes white superficial onychomycosis.[13] Psoriasis is an autoimmune inflammatory process that targets the skin that also may cause a characteristic oil drop discoloration of nails.[11] Additionally, onycholysis, subungual hyperkeratosis, and splinter hemorrhages are common associated findings.[11]

Foot infections associated with P. Aeruginosa:

Pseudomonas aeruginosa infections relating to nail puncture wounds of the foot may lead to osteochondritis, osteomyelitis and septic arthritis. Such infections are shown to be related to tennis shoes of adults and children, where P. aeruginosa is found to grow in the inner sole.[14,15]

Additionally, P. aeruginosa along with other gram-negative mixed bacteria, may lead to interdigital foot infections.[16] Though such infections are relatively common, the process may progress from a mild infection to advanced stages causing sepsis. [17]

Treatment of Pseudomonas Infections:

Fluoroquinolones, such as ciprofloxacin and levofloxacin, are the only oral formulations that effectively treat pseudomonas infections. This would be ideal for green nail syndrome as it can be treated on an outpatient basis. Other antibiotics, such as some penicillins, carbapenems, aminoglycosides, cephalosporins, and aztreonam can treat pseudomonas but require an intravenous infusion.

As with other nail infections, treatment consists of topical and/or oral medications. Topical medications may include 2% sodium hypochlorite solution [7], antiseptic agents (octenidine), and topical antibiotics (nadifloxacin, gentamicin).[11] Oral medications include ciprofloxacin and levofloxacin. One study discussed a patient with dystrophic black-green nails who was treated for months with a conazole with little improvement. A bacterial culture was performed and it grew out pseudomonas. Oral ciprofloxacin 500 mg BID was added to the regimen for 4 weeks along with twice daily 5 minute dilute solution chlorine bleach soaks. Once the pseudomonas infection was resolved the patient was able to see improvement with the antifungal medication.[13] Nails may be infected by both bacteria and fungus so it is important to get cultures taken to see what is growing, especially if there is a green hue to the nails as this is likely pseudomonas.

We were unable to find any research comparing different treatments for green nail syndrome. It was found that the treatment of choice that kept appearing in a handful of the articles read was oral ciprofloxacin. This seemed to work well with most patients.

Green nail syndrome caused by a Pseudomonas infection is not only aesthetically problematic for patients but may also cause more serious infections if the bacteria were to become systemic and travel to other organs of the body, especially in immunocompromised patients. For this reason, patients should be treated promptly.

In this case study we discussed clinical presentation, pathophysiology and different choices of treatment for the nail infection. I.R. was recommended to keep her feet as dry as possible with the use of athletic socks to wick the moisture. She was prescribed Ciprofloxacin 500 mg PO BID for 10 days and was advised to make a white vinegar/water mixture and soak her left foot multiple times per day for the pseudomonas infection of her toenails. She was told to follow up with her primary care physician and was also advised to wear more supportive shoes and take Motrin for the plantar foot pain. She was in agreement with the prescribed plan and was effectively discharged. We were unable to follow up with the patient to see if the treatment prescribed was effective, but the plan that was given to her was similar to what we found in our literature review. There should be more research done about Green Nail Syndrome to find the most effective treatment options.

References

[1] Anca Chiriac, Piotr Brzezinski, Liliana Foia, Iosif Marincu. Chloronychia: green nail syndrome caused by Pseudomonas aeruginosa in elderly persons. Clin Interv Aging. 2015; 10: 265–267.

[2] Giovanni Corsello, Davide Vecchio. Green Nail Syndrome. Pediatrics

International. 2014; 56,801.

[3] Muller S, Ebnother M, Itin P. Green Nail Syndrome (Pseudomonas aeruginosa Nail Infection): Two Cases Successfully Treated with Topical Nadifloxacin, an Acne Medication. Case Rep Dermatol. 2014 Jul 19;6(2):180-4.

[4] Youin Bae, et al. Green Nail Syndrome Treated with the Application of Tobramycin Eye Drop. Ann Dermatology. 2014 Aug;26(4):514-516.

[5] Pier GB, Ramphal R. Pseudomonas aeruginosa. In: Mandell GL, Bennett JE, Dolin R, editors. Principles and Practice of Infectious Diseases. 6th ed. Philadelphia, PA, USA: Churchill Livingstone; 2004.

[6] Nicholas Matheson, Michael Weekes, Sian Coggle. Skier's toe: traumatic onycholysis complicated by Pseudomonas chloronychia. BMJ Case Rep. 2009; 2009: bcr07. 2009.2074.

[7] Maes M, Richert B, de la Brassinne M. Green nail syndrome or chloronychia.Rev Med Liege. 2002;57(4):233–235.

[8] Rallis E, Paparizos V, Flemetakis A, Katsambas A. Pseudomonas fingernail infection successfully treated with topical nadifloxacin in HIV-positive patients: report of two cases. AIDS. 2010 Apr 24;24(7):1087-1088.

[9] Loveman A, Fliegelman M. Discoloration of the nails; concomitant use of nail lacquer with resorcinol or resorcinol monoacetate (euresol) as cause. A.M.A. Archives Of Dermatology [serial online]. August 1955;72(2):153-156. Available from: MEDLINE, Ipswich, MA. Accessed January 7, 2016.

[10] Gupta AK, Jain HC, Lynde CW, et al. Prevalence and epidemiology of onychomycosis in patients visiting physicians' offices: a multicenter canadian survey of 15,000 patients. J Am Acad Dermatol 2000; 43:244.

[11] Jiaravuthisan MM, Sasseville D, Vender RB, et al. Psoriasis of the nail: anatomy, pathology, clinical presentation, and a review of the literature on therapy. J Am Acad Dermatol 2007; 57:

[12] Nenoff P, Paasch U, Handrick W. Infections of finger and toe nails due to fungi and bacteria. Hautarzt. 2014;65(4):337–348.

[13] Elewski BE. Bacterial infection in a patient with onychomycosis. J Am Acad Dermatol. 1997;37(3 Pt 1):493.

[14] Inaba AS, Zukin DD, Perro M. An update on the evaluation and management of

plantar puncture wounds and Pseudomonas osteomyelitis. Pediatr Emerg Care 1992; 8:38.
[15] Jacobs RF, McCarthy RE, Elser JM. Pseudomonas osteochondritis complicating puncture wounds of the foot in children: a 10-year evaluation. J Infect Dis 1989; 160:657.
[16] Aste N., Atzori L., Zucca M., Pau M., Biggio P. Gram-negative bacterial toe web infection: a survey of 123 cases from the district of Cagliari, Italy. *Journal of the American Academy of Dermatology*. 2001;45(4):537–541. doi: 10.1067/mjd.2001.114747.
[17] Westmoreland TA, Ross EV, Yeager JK. Pseudomonas toe web infections. Cutis 1992; 49:185.

Does Predictability in Diabetic Foot Infection Pathogens Rule Out Need for Wound Cultures?

Stephanie Campbell, MS3
Jordan Butterfield, MS3
Dana Day, MS3

Introduction

In the pandemic of diabetes, diabetic foot infections remain to be the most common reason for hospitalization as a consequence of disease, as well as predispose the patient to lower extremity amputation and increased morbidity.[1]

Clinical correlation of the diabetic foot infection (DFI) against microbiology testing and advanced wound care therapies must be ascertained. With the evolution of healthcare in the United States and outcome based reimbursements, new recommendations are being sought in conjunction with clinical care practices to treat infected, non-healing diabetic wounds.

We will address the diabetic wound with respect to signs and symptoms of infection. Are cultures always appropriate to guide therapy of the diabetic wound, especially when suspicion for infection is low? If clinical judgement in treating the DFI is a reliable indicator of prognosis, what does the evidence show in measuring outcomes that ensure the standard of care? Finally, this article will serve as a clinical guide in forming decisions on acute versus chronic, infected versus uninfected ulcers, associated pathogens, options to tailor diagnosis, and indication for treatment.

Studies

The International Working Group for the Diabetic Foot strongly recommends that DFI must be diagnosed clinically, based on presence of local and systemic signs and symptoms of inflammation.[1]

The signs and symptoms of wound infection depend on whether the wound is acute or chronic. Acute wounds show the classic signs of inflammation: erythema, pain, edema, increased warmth, and wound exudate.[2] These signs typically persist beyond the initial inflammatory response. In contrast, chronic wounds, when infected, may not present with the classic inflammatory signs. A compromised immune system diminishes the body's response to infection causing the clinical signs of infection to be more subtle.

In the case of the diabetic patient being immunocompromised, the patient may not exemplify the clinical signs and symptoms of an infected wound as their comorbidities may contribute to chronicity of the wound. Localized manifestations of DFI may include friable skin changes, granular tissue changes, undermining of the wound, and nonpurulent drainage.[1] A diabetic patient with severe neuropathy and diminished sensation may not perceive tenderness to the area, developing a chronic, deeply penetrating wound. Furthermore, a diabetic patient with the comorbidity of peripheral arterial disease, may not allow the local signs of inflammation to manifest due to ischemia of the limb.[3] When initiating an infection evaluation, look for at least two cardinal signs of infection from the ulcer site along with more serious signs of sinus tracking.[4] The obvious clinical signs of infection can tailor your management of infection of the diabetic foot ulcer (DFU). While the clinical presentation for the infection may be low in the diabetic patient, the immunocompromised state of the patient supports use of antibiotics when clinical suspicion of underlying infection is

moderate to high.

A DFU is no different from other wounds in that it is colonized with microorganisms. Performing a microbial culture of the DFU would help determine the type of organisms and microbial load of the wound. Even with the risk of less prominent infectious symptoms and the potential to assess microbes and their loads with cultures, culturing wounds to assess bioburden is unnecessary.[5] The Infectious Disease Society of America reports that the diagnosis of DFI is primarily clinical with most diabetic wounds being colonized, the presence of microbial growth from a wound culture in the absence of supportive clinical findings is not sufficient to make the diagnosis of infection.[6]

Clinical examination of the ulcer with location and extent of involvement of the lesion is pertinent to primary treatment and closure. All classification systems of diabetic wounds establish the extent of induration of infection as it involves skin and superficial soft tissue, subcutaneous tissue, muscle, tendons and/or bone. Gross evaluation of the tissues and bone probing can offer severity of disease process and infection. The majority of DFIs are polymicrobial with more than five specific organisms being identified. The variability of microbial flora is related to the extent of anatomical involvement.[3]

Superficial DFIs, including cellulitic infections and acute ulcers, are most likely infected by aerobic gram-positive cocci. Deep ulcers that are chronically infected or previously treated by antibiotic therapy are most likely polymicrobial with further invasion by enterococci, enterobacteriaceae, Pseudomonas and more severe anaerobic microorganisms. The most severe of infections include wounds that are necrotic, malodorous, and gangrenous, with systemic infection attributable to anaerobic organisms.[3]

The most reliable technique for identifying pathogens present in diabetic foot wounds has been a topic of controversy in the literature. A systematic review of diagnostic techniques for infected diabetic foot ulcers concluded that current available evidence is too weak to suggest an ideal culture method.[7] Swab culturing may be reliable for identification of pathogens in superficial diabetic foot wounds but most researchers consider tissue biopsy to be the best method for pathogen identification. It is believed that deep tissue biopsy is not prone to superficial contamination.[8] Another study suggested that biopsy is not needed, as swab specimens accurately identify the bacterial species present in infected wounds.[9] In the great debate of reliability and necessity of culture specimens, the risk versus benefit of the results must direct clinical judgement in treatment of the wound. Utilizing these tools within their limitations, as it relates to tailored therapy to reduce ulcer size, is all that is required to advocate their use for the goal of wound closure.

With respect to non-healing ulcers, aerobic microorganisms continue to be the most commonly suspected flora. In vitro studies demonstrated that *Staphylococcus aureus* prevailed to be the predominant Gram-positive organism isolated in this study at 18.4% versus *Escherichia coli* at 19.5%. Both *E. coli* and *Pseudomonas aeruginosa* were found to be the most commonly isolated organism of the 70 specimens sampled in non-healing ulcers. This study emphasizes the importance of microbial observation, whilst supporting clinical evaluation and history as strong predictors of medical management. Uncontrolled glycemic index, especially diabetes with neuropathy, hyperlipidemia, and peripheral arterial disease all predispose the patient to develop lesions that resist healing.[10]

University of Texas Diabetic Wound Classification System

Stage	Grade			
	0	I	II	III
A (no infection or ischemia)	Pre- or post-ulcerative lesion completely epithelialized	Superficial wound not involving tendon, capsule, or bone	Wound penetrating to tendon or capsule	Wound penetrating to bone or joint
B	Infection	Infection	Infection	Infection
C	Ischemia	Ischemia	Ischemia	Ischemia
D	Infection and ischemia	Infection and ischemia	Infection and ischemia	Infection and ischemia

Wagner Classification

Grade	Definition
Zero	No ulcer
One	Superficial skin ulcer
Two	Deep ulcer extending through dermis. Tendon, ligaments, joint capsule or bone may be exposed
Three	Deep ulcer with abscess, osteomyelitis or joint sepsis
Four	Localized gangrene - forefoot or heel
Five	Gangrene of the foot

Figure 1. University of Texas and Wagner Classification Systems

Diabetic foot ulcers fail standard wound therapy when improvements in surface area of forty to fifty percent or reduction in wound size are not achieved within 4 weeks.[11,12] Monitoring wound closure at four weeks largely predicts healing at three months and is a reference point in the decision tree of care.[13] Multiple wound classification systems exist: Wagner, University of Texas (UT), PEDIS, and the newly introduced WIfI system for Wound, Ischemia, and foot infection risk stratification. All describe and categorize ulcers based on wound size and depth, as well as the presence of infection, ischemia, and neuropathy. These models preserve the principles in the factors directly responsible for wound healing and direct standard of care treatments.[12]

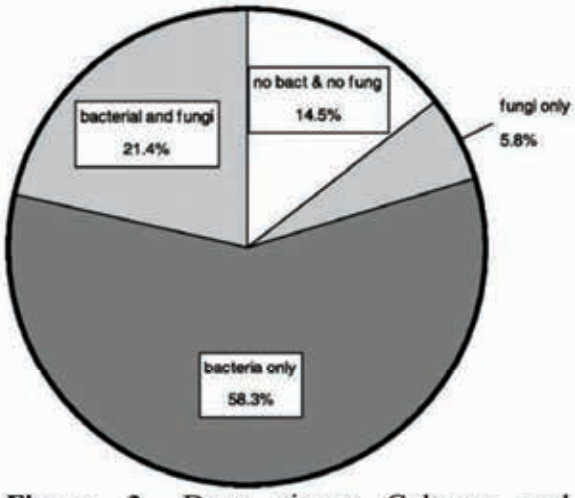


Figure 3. Deep tissue Cultures and Microbrial Flora of Lower Limb Wounds in Patients with Type II Diabetes Mellitus as found by Chellen et al.

Additional therapies provided in limb salvage centers such as hyperbaric oxygen therapy, biologics, and amputation should Be considered at this time when wound closure goals are not being met.

When a diabetic foot wound fails to respond to antibiotic therapy, stratify your treatment regimen to include tough bugs. Methicillin-resistant *Staphylococcus aureus* is a common culprit in DFI. Additionally increasing in prevalence globally are gram-negative bacilli with extended-spectrum beta-lactamase. Both of these pathogens should be considered in patients with previous antibiotic use, hospitalization, or residence in a long term facility.[3]

Macerated ulcers, moisture, and warm climates target *P. aeruginosa* as the most commonly isolated microorganism in as many as 20% of DFI. Yet, in the clinical setting, pseudomonas infections are not the primary pathogen and in that respect. Broader antibiotic coverage should be implemented.[3]

In the DFU that fails treatment and does not clinically correlate with infection, vascularization must be assessed. For patients with DFU and concomitant peripheral arterial disease, vascular surgery

is recommended.[11]

Discussion/Conclusion

It is argued that continuous surveillance of bacterial infection allows for antimicrobial sensitivity panels in generating an algorithm of guided therapy for the patient, while minimizing the risk of complications.[10] It is also argued that diabetic foot ulcers in the absence of clinically correlated infection do not suggest the use of cultures and microbial monitoring -- for no prediction of outcome can be made.[5]

Wounds that fail primary healing require advanced therapies and multidisciplinary teams to coordinate wound care. Multiple factors that delay wound healing must be evaluated and addressed to control glycemic index, edema, nutrition status, vasculature, or biomechanical stressors and efficient offloading. Diabetic foot ulcers that continue to resist healing need to be evaluated for unresolved infection, fungal pathogens -- predominantly Candida, or underlying osteomyelitis with deep tissue or bone biopsy, respectively.[14] Recent literature suggests that diptheroides species, *Cornybacterium striatum,* part of the common skin flora,

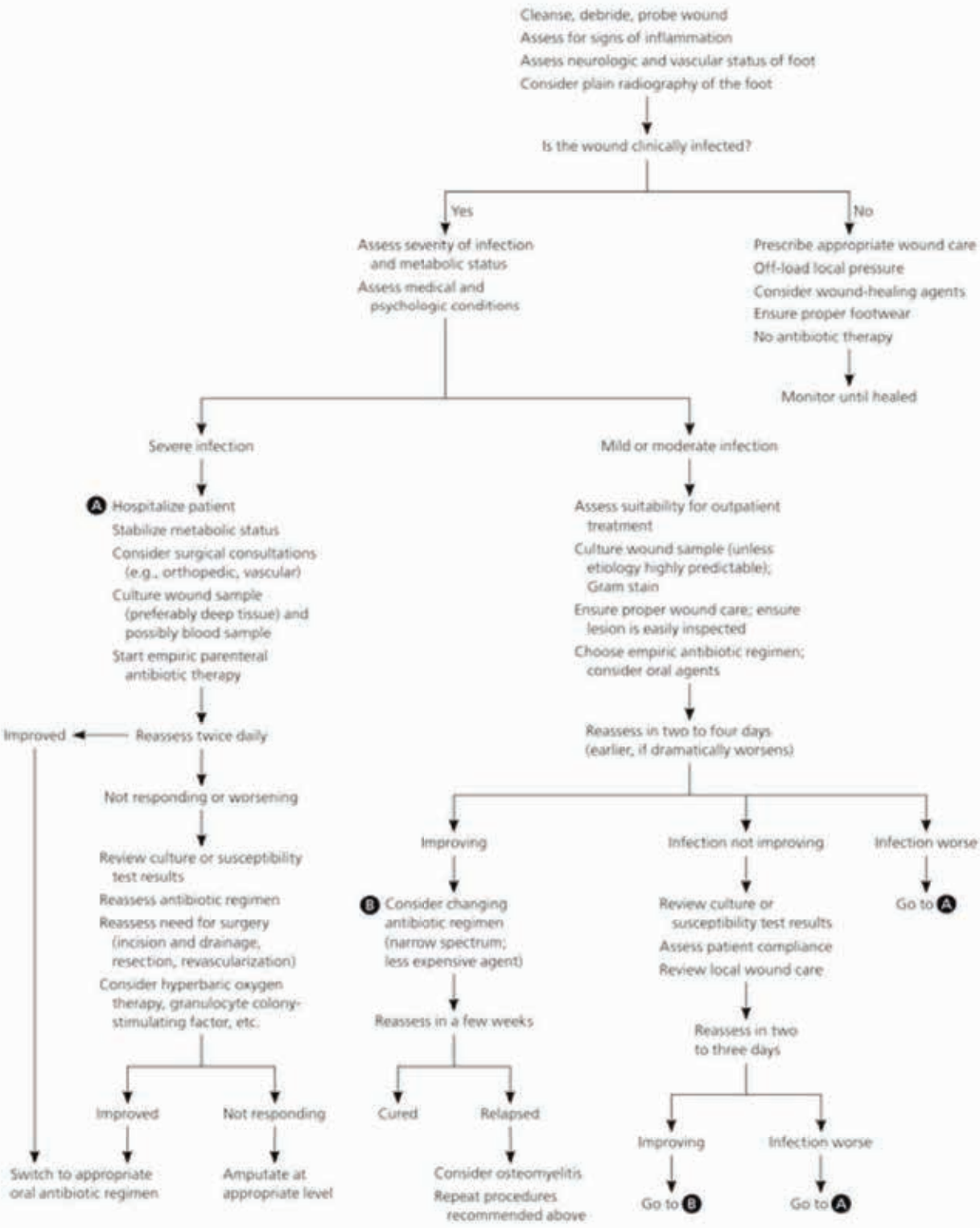


Figure 2. Is the Wound Clinically Infected? Decision to Treat Algorithm by *Bader MS. Am Fam Physician. 2008 Jul 1; 78(1):71-9.*

may actual have a role as the pathogenresponsible for diabetic foot osteomyelitis.[15] Reevaluate the wound for infection or repeat surgical debridement.

Consider culturing before proceeding with soft tissue closure.

Increased understanding of the causative pathogens in diabetic foot infections has improved our ability to diagnose infections and advance empiric antibiotic therapy. Recent studies have clarified the roles of antibiotic and surgical treatment of DFI and osteomyelitis.[16]

Predictability of microbial flora and infection of diabetic foot ulcers can be based largely on clinical suspicion, ulcer location, symptoms, and geographic region such that additional testing and cultures not always being warranted. Wound cultures are recommended for moderate to severe wound infections. The evidence supports clinician based practice in the management of diabetic foot infections. Treating a diabetic foot wound is a complicated task and delayed healing can only complicate the treatment plan further by predisposing the patient to disability, amputation, and increased mortality.

New technologies are under evaluation, but the basic principles of properly diagnosing infection, obtaining a specimen for culture, selecting and refining antibiotic therapy, rapidly undertaking the surgical interventions required, ensuring adequate arterial perfusion, and off-loading pressure remain the keys to good outcomes in limb salvage.

References

1. 1. Lipsky BA, Aragón-Sánchez J, Diggle M, Embil J, Kono S, Lavery L, Senneville E, Urbancic-Rovan V, Van Asten S, Peters EJG. IWGDF Guidance on the diagnosis and management of foot infections in persons with diabetes. 2015.
2. Gardner, S. E., Hillis, S. L., & Frantz, R. A. (2009). Clinical Signs of Infection in Diabetic Foot Ulcers with High Microbial Load. *Biological Research for Nursing, 11*(2), 119–128. http://doi.org/10.1177/1099800408326169
3. Weintrob AC, Sexton DJ. Clinical manifestations, diagnosis, and management of diabetic infections of the lower extremities. In UpToDate, Post Calderwood SB (Ed.), UpToDate, Waltham, MA. (Accessed January 2017)
4. McCulloch DK. Evaluation of the Diabetic Foot. In: UpToDate, Post Nathan DM (Ed.), UpToDate, Waltham, MA. (Accessed January 2017.)
5. Gardner SE, Haleem A, Jao YL, Hillis SL, Femino JE, Phisitkul P, Heilmann KP, Lehman SM, Franciscus CL. Cultures of Diabetic Foot Ulcers Without Clinical Signs of Infection Do Not Predict Outcomes. Diabetes Care. 2014; 37:2693-2701.
6. Lipsky BA, Berendt AR, Cornia PB, Pile JC, Peters EJ, Armstrong DG, Deery HG, Embil JM, Joseph WS, Karchmer AW, Pinzur MS, Senneville E, Infectious Diseases Society of America clinical practice guideline for the diagnosis and treatment of diabetic foot infections. Clin Infect Dis. 2012;54(12):e132.
7. O'Meara, S. et al. "Systematic Review Of Methods To Diagnose Infection In Foot Ulcers In Diabetes". Diabetic Medicine 23.4 (2006): 341-347. Web. https://www.ncbi.nlm.nih.gov/pubmed/16620261
8. Huang, Ying et al. "A Comparison Of Tissue Versus Swab Culturing Of Infected Diabetic Foot Wounds". International Journal of Endocrinology 2016 (2016): 1-6. Web.. https://www.ncbi.nlm.nih.gov/pmc/articles/PMC4829715/
9. Gjødsbøl, Kristine et al. "No Need For Biopsies: Comparison Of Three Sample Techniques For Wound Microbiota Determination". International Wound Journal 9.3 (2011): 295-302. Web. https://www.ncbi.nlm.nih.gov/pubmed/22067000
10. Noor S, Ahmad J, Parwez I, Ozair M. Culture-Based Screening of Aerobic Microbiome in Diabetic Foot Subjects and Developing Non-healing Ulcers. Front Microbiol. 2016; 7:1792.
11. Hingorani A, LaMuraglia G, Henke P, Meissner MH, Loretz L, Zinszer KM, Driver VR, Frykberg R, Carman, TL, Marston W, Mills Sr JL, Murad MH. The management of diabetic foot: A clinical practice guideline by the Society for Vascular Surgery in collaboration with the American Podiatric Medical Association and the Society for Vascular Medicine. Journal of Vascular Surgery. 2016;63:3S-21S.
12. Armstrong DG, Asla RJD, McCulloch DK. Management of diabetic foot ulcers. In UpToDate, Collins KC (Ed.), UpToDate, Waltham, MA (Accessed January 2017).

13. Sheehan P, jones P, Giurini JM, Caselli A, Veves A. Percent change in wound area of diabetic foot ulcers over a 4-week period is a robust predictor of complete healing in a 12-week prospective trial. Plast reconstr Surg. 2006;117(7 Suppl):239S.
14. Chellan G, Shivaprakash S, Ramaiyar SK, Varma A, Varma N, Sukumaran MT, Vasukutty JR, Bal A, Kumar H. Spectrum and Prevalence of Fungi Infecting Deep Tissues of Lower-Limb Wounds in Patients with Type 2 Diabetes. Journal of Clinical Microbiology. 2010;48:2097-2102.
15. Patel S, Lacovella J, Cornell R. Corynebacterium Striatum: A Concerning Pathogen of Osteomyelitis in the Diabetic Patient. Journal of the American Poadiatric Medical Association. September 2016, Vol 106, No sp1, pp.9-9.
16. Uçkay I, Aragón-Sánchez J., Lew D, Lipsky BA. Diabetic foot infections: what have we learned in the last 30 years? International Journal of Infectious Diseases. 2015;40:81-91.

Toe Walking in the Autistic Patient

Ryan Mayberry, MS2 College of Podiatric Medicine

Introduction

Autism is a spectrum of pervasive developmental disorders and is found across all ethnicities and economic groups. Children with autism spectrum disorder (ASD) have difficulty with social interaction, communication, and language skills, with many children demonstrating restrictive and repetitive behaviors[1]. Of note specifically to podiatric physicians are the numerous gait abnormalities that accompany the autistic patient. Previous studies have shown, using instruments such as a GAITRITE system to record plantar pressure, a reduction in cadence, gait velocity, step length, step width, and plantar hindfoot pressure in patients with autism[2]. Another study used camera motion capture systems and force plates to demonstrate significant differences between the autism group and the control group regarding cadence, peak hip, and ankle kinematics and kinetics[3]. One gait abnormality in particular, toe walking, is common among autistic patients. This article aims to discuss the research that has been conducted regarding toe walking in the autistic patient, and to review the various currently available treatment modalities and their effectiveness.

Toe Walking

It has been observed that children with ASD are more prone to idiopathic toe walking (ITW) than in age-matched healthy controls[1]. In a study involving 954 children, 324 of which had an ASD diagnosis, the incidence of persistent toe walking was 12.1% among the control group, with 7.9% experiencing tight heel cords. In the ASD diagnosis group, 20.1% had persistent toe walking, and 12.0% had tight heel cords, which supports the statement that the incidence of persistent toe walking is significantly elevated in patients with ASD[4]. The persistent toe walking associated with non-motor developmental disorders such as ASD differs from those associated with neuromuscular disorders such as Cerebral Palsy by the absence of long-tract signs of upper motor neuroninvolvement (hypertonicity, hyperreflexia, and abnormal Babinski reflexes)[4]. However, despite attempts to discover the cause of persistent toe walking in patient with ASD, the neurophysiological basis for this association largely remains unknown[4].

Persistent toe walking continues longer in autism and can contribute to the development of secondary motor deformity, a heel cord so shortened that the foot can no longer be easily dorsiflexed at, or beyond neutral (90 degrees)[4]. Treatments for this deformity can include stretching exercises, GaitSpot auditory speakers, casting, braces, Botox injections, and, in rare cases, tendo-Achilles lengthening[4].

Treatments

Stretching:

Stretching is often the first attempt to treat toe walking, as it is the least invasive treatment option available. However, it is recognized that stretching and physical therapy offer a limited chance of success for treating ITW, and is more often used in an attempt to maintain range of motion gained by other methods[5].

GaitSpot:

GaitSpot auditory shoe squeakers are simple devices that can be strapped over the foot either inside or over each shoe and

adjusted so that a small squeaker fits squarely over the bottom heel.

Figure 1. The GaitSpot auditory shoe speaker[12]

Once weight is placed on the heel of the shoe as it is placed on the ground, it emits an audible squeak thereby providing the wearer with immediate auditory feedback. In one study, the GaitSpot devices were placed on the subject's shoes for 10 minute sessions. Once the subject elicited 3 consecutive squeaks from the GaitSpot device by successfully heel striking, the subject was reinforced with a reward. The auditory feedback was initially paired with small edibles or tokens, with social praise replacing after an initial period. The GaitSpot speakers were eventually phased out of the trial once the subjects were able to demonstrate zero occurrences of toe walking over several sessions. Results indicated that there was an overall decrease in ITW across all participants though the degree of individual reduction differed[6].

Braces and orthoses:

A study was conducted involving 18 children with idiopathic toe walking, 9 of whom were given an ankle-foot orthosis (AFO), the other 9 of whom received a foot orthosis (FO). After 6 weeks of treatment with the orthoses, the AFO group had better controlled toe walking, however when the orthosis was removed, patients tended to revert to earlier patterns of gait. During a test in which 1495 steps were assessed, 100% of the AFOs were 100% effective at preventing initial contact with the toe. After 6 weeks with the FO group, there was less control of toe walking. In the same assessment of 1495 steps, 13% of the steps with the FO were either flat footed or with toe contact first. However, the FO was considered after the treatment as being less restrictive and more aesthetically pleasing by the parents of the subjects[7].

Botox injections:

A group of 11 children with idiopathic toe walking were treated with a total of 6 units/kg bodyweight of Botox in both the medial and lateral gastrocnemius bellies and the gastrocnemius-soleus complex. Gait analysis was repeated 3, 6, and 12 months after treatment with Botox. In the 12 month follow up, 3 of 11 children had ceased toe walking, 4 of 11 had decreased toe walking, and 4 of 11 continued toe walking[8]. A similar study was conducted in which 5 children were treated with botulinum toxin type A. Prior to treatment, 51% of foot contacts were with the toe. 20 days following treatment, only 8% of foot contacts were toe contact. This improvement was maintained upon a 12-month follow up examination[9].

Surgical treatment options:

Surgical treatment is usually only considered after conservative treatment options are exhausted. Generally, surgical treatment of toe walking has proven to be beneficial. Procedures to lengthen the gastroc-soleus complex have been shown to be an effective treatment of idiopathic toe walking[10]. In similar studies, Tendo-achilles lengthening procedures have also shown to

be effective at treating idiopathic toe walking[11].

Summary

Toe walking is a problem for many patients with ASD. While the etiology of toe walking in the autistic population still remains unknown, treatment options are available to prevent further pathologies from developing secondary to toe walking. There are currently several methods of treatment, however not all are successful, and there is limited evidence based research to support the treatments. Much of the research has involved small sample sizes, and few studies reproducing the findings. In order to better help this population, more research must be conducted not only to support current treatment methods, but also to develop new treatment modalities.

References

1) Kindregan D, Gallagher L, Gormley J. Gait Deviations in Children with Autism Spectrum Disorders: A Review. *Autism Research and Treatment*. 2015;2015:741480. doi:10.1155/2015/741480.
2) Lim B-O, O'Sullivan D, Choi B-G, Kim M-Y. Comparative gait analysis between children with autism and age-matched controls: analysis with temporal-spatial and foot pressure variables. *Journal of Physical Therapy Science*. 2016;28(1):286-292. doi:10.1589/jpts.28.286.
3) Matthew Calhoun, Margaret Longworth, Victoria L. Chester, Gait patterns in children with autism, Clinical Biomechanics, Volume 26, Issue 2, February 2011, Pages 200-206, ISSN 0268-0033,
4) Barrow W, Jaworski M, Accardo P. Persistent Toe Walking in Autism. *Journal of Child Neurology*. 2011; 26(5):619-621
5) Oetgen ME, Peden S. Idiopathic toe walking. *Journal of the American Academy of Othropaedic Surgeons*. 2012; 20(5):292-300
6) Ann Marcus, Brigit Sinnott, Stephen Bradley, Ian Grey, Treatment of idiopathic toe-walking in children with autism using GaitSpot Auditory Speakers and simplified habit reversal, Research in Autism Spectrum Disorders, Volume 4, Issue 2, April–June 2010, Pages 260-267, ISSN 1750-9467,
7) Herrin K, Geil M. A comparison of orthoses in the treatment of idiopathic toe walking: A randomized controlled trial. *Prosthetics and Orthotics International*. 2015; 40(2):262-269
8) Engström P, Gutierrez-Farewik EM, Bartonek Å, Tedroff K, Orefelt C, Haglund-Åkerlind Y. Does botulinum toxin A improve the walking pattern in children with idiopathic toe-walking? Journal of Children's Orthopaedics. 2010;4(4):301-308.
9) Brunt D, Woo R, Kim HD, Ko MS, Senesac C, Li S. Effect of Botulinum toxin type A on gait of children who are idiopathic toe-walkers. *Journal of Surgical Orthopaedic Advances*. 2004; 13(3):149-155
10) McMulkin ML, Baird GO, Caskey PM, Fergusen RL. Comprehensive outcomes of surgically treated idiopathic toe walkers. *Journal of Pediatric Orthopedics*. 2006; 26(5):606-611
11) Hemo Y, Macdessi SJ, Pierce RA, Aiona MD, Sussman MD. Outcome of patients after Achilles tendon lengthening for treatment of idiopathic toe walking. *Journal of Pediatric Orthopedics*. 2006; 26(3):336-340.

12) Image downloaded from http://cdn3.volusion.com/ozdsh.amksr/v/vspfiles/photos/9-031041-118-2.jpg?1472570218

Transmetatarsal Amputation Review: A Biomechanical Analysis

Stephanie Campbell, MS3 College of Podiatric Medicine

Introduction:

Considering the morbidity and mortality rates of major and minor amputations, it is highly favorable to perform distal procedure, clinical presentations permitting. Evans et al. (2011) found that after two years following amputation, 80% of patients with either proximal forefoot or midfoot amputations survived while patient's undergoing below knee amputation face 52% mortality..[5] The transmetatarsal amputation (TMA) has become the procedure of choice when more distal amputations are contraindicated.[6]

The aim of this study is to compare whether guillotine versus parabolic TMA procedures yield better postoperative outcomes by evaluating patients postoperatively for ulcer formation, and whether these ulcers correspond to locations of peak plantar pressures created by surgically-designed parabolas. McGlamry describes TMA procedures as requiring a smooth metatarsal parabola with the second metatarsal as the parabolic apex.[7] However, there are no studies comparing postoperative outcomes of guillotine vs parabolic TMA procedures. Insight into specifications for the TMA surgical technique will guide surgical planning and improve patient postoperative biomechanics and therefore prognosis.[1]

The amputations performed on the patients followed in this study were not performed by the authors or in the institution or clinic in which they were studied.

The authors of this study report no financial disclosures.

Research Purpose

It is the purpose of this study to understand the biomechanical consequence of the surgical parabola and resulting functional benefit for our patients undergoing TMA procedures.

This research will provide insight into specifications for TMA surgical technique and will improve its effects on the patients' resulting postoperative biomechanics. With optimal biomechanics, the patient will have a lower risk of future proximal amputations, pain, callus formation, recurrent ulcerations, and other complications.

Study Methodology:

The metatarsal protrusion angle has been studied for years as the relationship between the first and second metatarsals, exclusively, in order to "establish various values of normality".[2]

Patients were enrolled from the podiatry clinic at Arrowhead Regional Medical Center in Colton, California over a one-year period. A significant number of patients were excluded for previous cardiac injury. A total of 8 patients were included during the time of study.

Inclusion criteria:

1.) Transmetatarsal amputation with or without Achilles lengthening performed at least one year earlier

2.) Patient age greater than 18 and less than 75 years

3.) English speaking

4.) Patients must be ambulatory without the aid of crutch, walker, or wheelchair

Exclusion criteria:
1.) Patient must not have had a previous cardiac event or angioplasty
2.) Patients with active foot infections will be excluded from the study

In this study, the metatarsal parabola will be assessed using weight-bearing AP radiographs and plantar foot pressures will be measured using a pressure plate. Oller's system as described in "Metatarsal Protrusion Angle: Values of Normality" will be used to measure metatarsal lengths and angles.[2]

Patients fulfilling the inclusion criteria subsequently completed informed consent via the supervising podiatrist, and were compensated monetarily for their participation.

The Tekscan HR Mat is calibrated for each patient with the patient weight, and the measurement is recorded in the software with a step calibration. The two-feet step calibration was chosen in place of the single-foot step calibration with the understanding that the pressure points are monitored as a percentage of distributed weight and as a measure of safety for the patients. The technicians became proficient with the Tekscan digital pressure plate scanner in order to calibrate and record computerized scans of the patient's sway stance and gait. After calibration, the patient completed two 30 second stance recordings on the mat. The patients began the recording already standing on the mat, eyes open, looking at a fixed point on the wall ahead with arms relaxed at their sides. Between measurements, the patients were asked to step off the mat, and then step back on the mat, and complete a second recording. It was recorded in the Tekscan Research Software that both feet were on the mat, eyes open, with frames of 0.10 seconds for 30 seconds or 600 frames. Per frame, the software further recorded pressure mapping of both feet, the center of gravity over the recording, as well as distributed pressure percentages in the anterior, posterior, right and left portions of the feet, and peak pressure PSI.

The second phase of study was gait analysis. The parameters input during gait analysis correspond the foot striking the mat during the line of projection with the frame setting at 0.0055 seconds. The recording began with heel strike and ended with last contact on the mat. The patient was asked to walk upright looking at a fixed position on either side of the room. This imprint was recorded as the third step in the patient's gait cycle. The recordings register for approximately seven seconds with three measurements were taken for both the left and right foot.

Results

Table 1. Biomechanical Performance of TMA Patients

Patient	1	2	3	4	5	6	7	8
TMA Side	R	R	L	R	R	R	R	L
TAL	N	Y	N	Y	Y	Y	Y	N
Ulcer Location	2 MH	1 MH	3 DP	1 MH	1 MH	1 MH	1 MH/Heel	1/5 MH
Ulcer Status	Healed	Active	Active	Never	healed	Healed	Never	Healed
Longest Metatarsal	2 MH	1 MH	1 MH	4MH	2 MH	3 MH	2 MH	2 MH
Parabola vs. Guillitine	G	P	P	G	P	G	P	P
Parabola Protrusion Angle	76	62	56	82	50	72	50	53
Average Stance Peak Pressure Left Foot	37.6	40.1	**31.3**	27.7	150.2	59.6	37.3	**16.4**
Stance Peak Pressure Location Left Foot	Heel	2/3 MH	**1 MH**	Heel	Heel	4 MH	Heel	**Heel/ 1 MH**
Average Weight % in Stance Left Foot	54.0	55.5	45.5	66.0	75.5	45.0	56.0	41.5
Average Peak Pressure with Left Foot Stride	148.1	168.0	**167.8**	103.5	282.5	121.4	113.5	**80.8**
Average Peak Location with Left Foot Stride	1 DP	3 MH	**4 MH**	1/2 MH	1 MH	1/5 MH	2 MH/ 1 DP	**1/2 MH**
Average Stance Peak Pressure Right Foot	**59.4**	**40.6**	18.5	**46.2**	**105.4**	**105.9**	**28.5**	78.3
Stance Peak Pressure Location Right Foot	**2 MH**	**5 MH**	1 MH	**Heel**	**1 MH**	**1 MH**	**Heel**	1 MH
Average Weight % in Stance Right Foot	46	44.5	54.5	34	24.5	55	44	58.5
Average Peak Pressure with Right Foot Stride	**290.7**	**224.2**	167.5	**134.3**	**274.5**	**181.2**	**73.2**	162.6
Average Peak Location with Right Foot Stride	**2 MH**	**1 MH**	3 DP	**1 MH**	**1 MH**	**1 MH**	**1 MH/ Heel**	1/5 MH

Table 1. Data collection over 16 trials recording average peak pressures and location of peak pressure for both feet, average (avg) weight distribution in stance, TMA side, tendo-Achilles lengthening (TAL), ulcer location, ulcer status as never/healed/active, longest metatarsal postoperatively, surgery categorized as parabola or guillotine, and parabola protrusion angle (°). MH = metatarsal head, DP = distal phalanx. Data for each patient is averaged over three trials. Highlighted fields indicate the location of the patient's maximum peak pressure points in stance or stride, respectively, that in fact, correspond to locations of postoperative ulcers. Bolded fields emphasize measurements representing the TMA surgical foot.

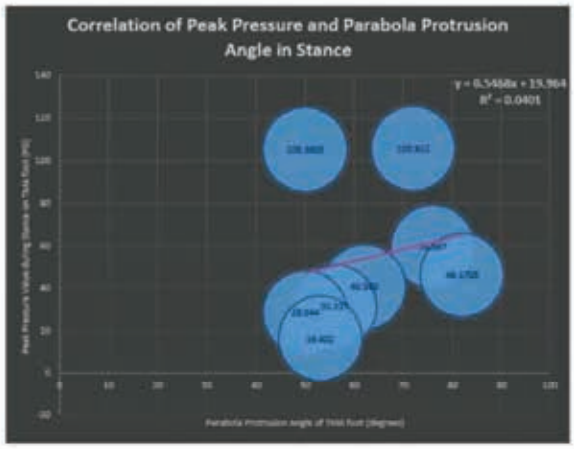


Figure 1. Correlation of Peak Pressure and Parabola Protrusion Angle in Stance and Stride. The graphs above illustrate the relationship during stance or stride between peak plantar pressures in the patient TMA foot versus the respective parabola protrusion angle found in the postoperative TMA foot radiograph using Ollier's classification system. The red line represents a linear regression model of all eight data points with the regression equation and coefficient of determination (r^2).

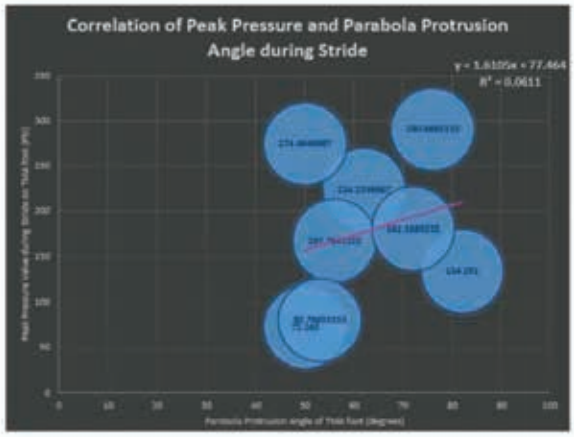
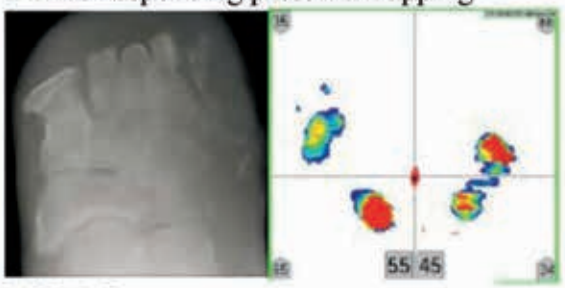


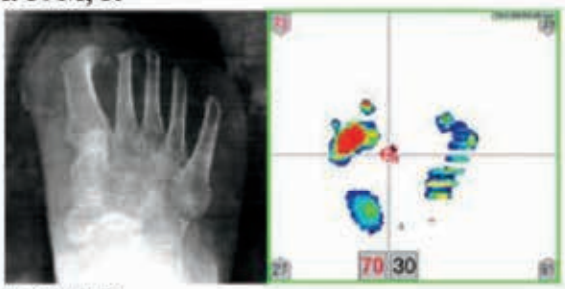
Figure 2. Peak Force versus Time in Stance. 39 year old Male patient with Right TMA, Graph depicting peak force (lbs) over time (s) during a two feet standing, 30 second period of pressure mapping. Initial need to correct balanced forces is significant in the first ten seconds of stance.

Table 2. Postoperative TMA Radiographic Images and correlated bilateral pressure maps of TMA patients standing for 30 seconds. Pressure maps, below, isolated at frame of peak pressures. Pressures are divided into quadrants. Patient center of gravity (0,0) and sway recorded over each trial. Of note, plantar pressures shift medially on the surgical foot.

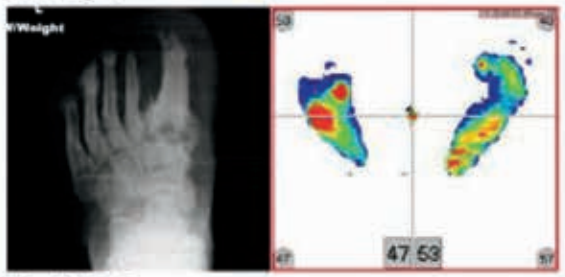
Figure 3. Post-operative TMA Radiographs with corresponding pressure mapping



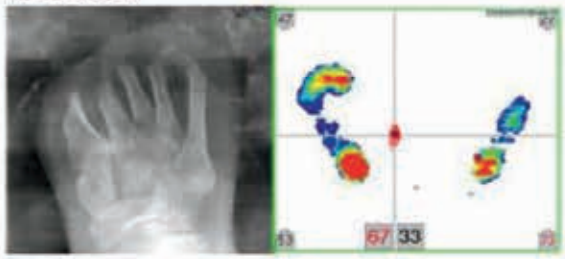
I. 39M, R



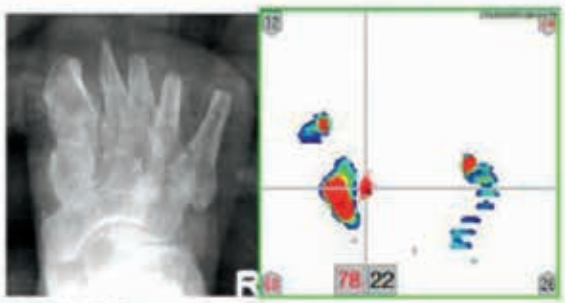
II. 65F, R



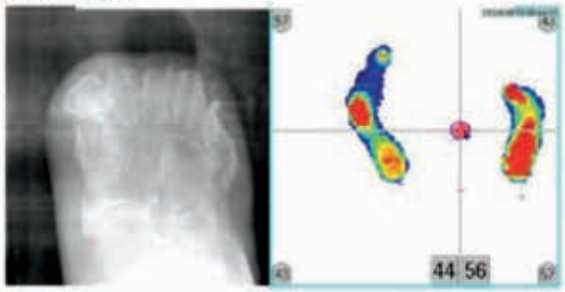
III. 43M, L



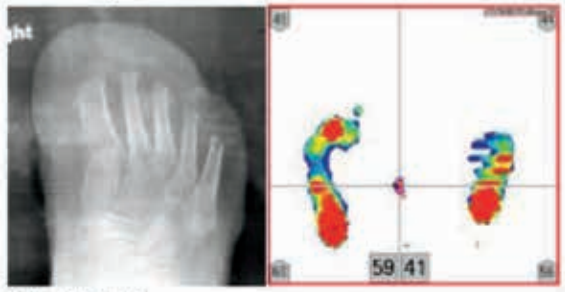
IV. 56M, R



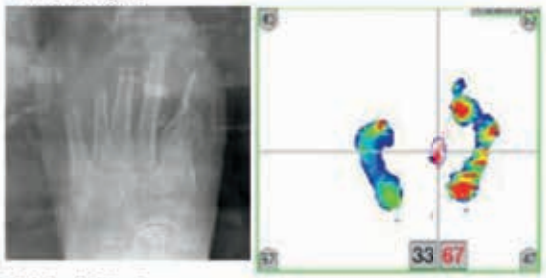
V. 70M, R



VI. 59M, R



VII. 30F, R



VIII. 49F, L

Neither choice in guillotine versus parabola TMA procedure was a predictive indicator of ulcer prevention.

Of the two patients who did not develop ulcers postoperatively, one underwent the guillotine procedure (PPA= 82°) and one underwent the parabolic procedure (PPA= 50°), and both underwent TAL procedures.

Pressure mapping reveals peak pressure points in both the surgical and nonsurgical foot and can be related to the clinical evaluation of ulceration by the patient. Despite the small sample size, 3 out of 6 patients who re-ulcerated had a peak pressure in stance or stride that correlated with the location of their ulcer. The predictive value of measuring peak pressures may be beneficial formulating treatments and precautions for patients.

In the trials of stance pressure mapping, on average, seven of eight patients placed over 50% body weight onto their nonsurgical foot. However, in the trials of patients placing more than 50% body weight onto their TMA foot, pressure maps indicated an active or healed ulcer differing from the location of maximal pressure. It is possible that increased pressure led to guarding compensation in these patients.

In the seven patients placing over 50% body weight onto their nonsurgical foot during stance, five patients had higher peak pressures in their TMA foot relative to the contralateral foot. In the striding trials, four patients had relatively higher peak pressures in their TMA foot. The patient who placed over 50% body weight onto their TMA foot had relatively higher peak pressures in their TMA foot in both standing and striding trials. However, this patient did not develop an ulcer at the location of highest pressure during standing and striding.

Of the six patients with active or healed ulcers, three corresponded to locations of maximal pressure during stride and two corresponded to locations of maximal pressure during stance. Of these, one underwent a guillotine procedure, two underwent parabolic procedures, and two underwent TAL procedures.

In the patients who received parabolic TMA procedures, none of the parabola protrusion angles were found to be within the normal parabola range 95% CI of

54.52-55.65° (metatarsal angle II-V). In the patients with guillotine parabolas, protrusion angles were 4.55 or more standard deviations above the average protrusion angle of 55.09°.[2] There was no correlation between reduced plantar peak pressures and degree of parabola protrusion in stance or during stride (protrusion angle versus peak pressure r^2 values of .04 in stance, .06 in stride).

Conclusions

This study found that pressure mapping following TMA procedures is a useful tool in identifying plantar peak pressure locations in order to proactively prevent postoperative ulcers.

TAL procedures demonstrated a potential protective effect since both patients without any postoperative ulcers underwent TAL procedures, however further research is needed to explore the longevity, internal validity, and external validity of this finding. The indication for concurrent TAL procedures as it relates to this study needs to be further evaluated. Our sample size cannot further support nor negate the effectiveness of TAL procedures in prevention of re-ulceration postoperative amputation.

This study did not find the choice of either the guillotine or parabolic TMA procedures to reliably produce less post-operative ulcerations or decreased peak plantar pressures. Further research is needed to attain statistically significant results for the preference of guillotine versus parabolic TMA, preferred parabola protrusion angle, and utility of adjunctive TAL procedures.

References:

1. Terashi H, Kitano I, Tsuji Y, Hashikawa K, Tahara S. A modified transmetatarsal amputation. **J Foot Ankle Surgery.** 2011 Jul-Aug;50(4):441-444.

2. Metatarsal protrusion angle: values of normality. Dominguez G, Munuera PV, Lomas M. **J Am Podiatry Medical Association.** 2009 Jan-Feb;99(1):49-53.

3. Effects of a tendo-Achilles lengthening procedure no muscle function and gait characteristics in a patient with diabetes mellitus. Hastings MK, Mueller MJ, Sinacore DR, Salsich GB, Engsberg JR, Johnson JE. **J Orthop Sports Physical Therapy.** 2000 Feb;30(2):85-90.

4. Pollard J, Hamilton GA, Rush SM, Ford LA. Mortality and morbidity after transmetatarsal amputation: retrospective review of 101 cases. **J Foot Ankle Surgery.** 2006 Mar-Apr;45(2):91-97.

5. Evans, Karen Kim, Christopher E. Attinger, Ali Al-Attar, Christopher Salgado, Carrie K. Chu, Samir Mardini, and Richard Neville. "The Importance of Limb Preservation in the Diabetic Population." **Journal of Diabetes and Its Complications** 25.4 (2011): 227-31. Web.6. Atnip, Robert G. "Toe and Partial Foot Amputations." *Operative Techniques in General Surgery* 7.2 (2005): 67-73. Web.

7. McGlamry, E. Dalton., and Joe T. Southerland. "Transmetatarsal Amputation."*McGlamry's Comprehensive Textbook of Foot and Ankle Surgery*. 4th ed. Vol. 2. Philadelphia: Wolters Kluwer Health/Lippincott Williams & Wilkins, 2013. 1029-030. Print.

A Student-Friendly Guide for the Selection of a Debridement Method for Foot Ulcerations

Ricardo Navarrete Jr, MS2 College of Podiatric Medicine
Sean Pearson MS2, College of Podiatric Medicine
Lant Abernathy, MS2 College of Podiatric Medicine

Introduction

The transition from classroom to a clinic setting can be daunting; treating ulcers changes from a pre-written case study to a complex wound that contravenes guidelines taught from books. A list of memorized methods are considered when treating patients, however as a beginning clinician, the distinction and selection of debridement method can be challenging as there are several to differentiate from.

There are four main types of debridement: mechanical, surgical, autolytic and enzymatic as well as some lesser known types, such as ultrasonic and bio-surgical. In recognition of the importance of learning appropriate debridement techniques, there seems to be scarce availability of a quick student resource. This article concentrates on the importance of recognizing different debridement technique and the appropriate selection to achieve optimal evidence-based healing for patients.

Debridement is only one aspect of managing an ulcer, however, equally important offloading strategies and an integrated wound care approach to management will not be discussed. Prevention of specific DFUs (i.e., diabetic foot ulcers) is not discussed in this document, but rather a general review of foot and leg ulcerations. The cost benefit and clinical selection process will also be left out of this document, as it furthers complexity in determining a debridement method and strays from theoretical clinical management. - This article aims to offer podiatric medical students a practical, relevant clinical guide to aid in the appropriate decision making and effective methods in people solely based on the presentation of ulceration. The assumption of adequate debridement preparation and general wound care up to debridement will be implied.

Among the many aspects of wound management, tissue debridement is a vital aspect of wound care; "It has been suggested that up to 85% of amputations can be avoided when an effective care plan is adopted [1]" This demarcates a clear need to understand and utilize all aspects of wound management in detail to provide the best treatment and care for patients. Debridement is a process that occurs in all wounds and enables the removal of damaged and necrotic tissue, debris and bacteria from the wound to facilitate the formation of healthy granulation tissue. Lower extremity ulcers present in 4 main patterns: neuropathic, pressure, venous and arterial, all of which require specific care and treatment for the best results. Foot ulcers can be challenging to treat because the 4 main ulcerative lesions may present as infected, ischemic, gangrenous, necrotic and/or all of the above.

Sharp/Surgical

Sharp debridement is considered a form of surgical debridement in which the process of excising devitalized tissue is done by the use of instrumentation (e.g., forceps, scalpels, and scissors). This method requires an experienced practitioner. This technique

in practice is often referred to as the gold standard tissue management for a diabetic foot ulcer [2]. Considered quick and inexpensive, it also limits the use of any analgesics or topical medication to aid in successful management [3]. Sharp debridement is indicated when there is an abundant tissue necrosis and/or when there is an advancing infection [4]. It is important to differentiate viable versus nonviable tissue during debridement and note that there is risk of incising healthy tissue. Additional importance should be given by the practitioner to avoid cutting blood vessels, nerves, and other important anatomic structures. When surgical debridement is contraindicated or has failed to help heal wounds, then other debridement techniques can be used.

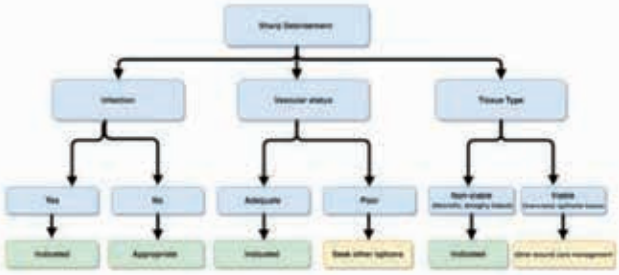


Figure 1. *Sharp Debridement Assessment*

Mechanical

Mechanical debridement offers a similar mechanism except with other forms. One example is Hydrotherapy, in which a saline solution is sprayed at the wound to remove non-viable tissue [5]. Different types of hydrotherapy include whirlpool therapy as well as pulsatile lavage. One study involving the use of a VERSAJET hydrosurgical technique showed that removal of bacteria as well as inflammatory markers from wounds were reduced, although no evidence shows complete removal of biofilm [6]. Disadvantages include high costs and availability.

Another type of mechanical debridement is wet-to-dry dressing. Wet-to-dry dressings are quick and involve administering a wet dressing directly on the wound and waiting for it dry. In theory, once the dressing is dry, it sticks to the unhealthy tissue and thus, is removed when peeled off. While wet to dry methods may be popular, their effectiveness is inconclusive. This method is non-selective, meaning when removing the dressing, granulated tissue may be removed with it. It can also be painful and increases the risk of infection [7].

Ultrasound is another method of mechanical debridement. It involves using an ultrasound machine that emits sound waves into the wound through a saline solution. It is thought that the mechanical forces applied by the ultrasound contribute to angiogenesis, cell division and release of growth factors. Ultrasonic debridement is a quick, can be performed bedside or in clinic and is associated with less pain than other techniques. However, there is no further research to indicate that ultrasound is better than other debridement techniques and this technique requires the use of a trained professional [8].

Figure 2. *Mechanical Debridement Assessment*

Autolytic/Enzymatic

This technique utilizes enzymes made from the body to remove non-viable tissue from wounds [9]. There are many different types of autolysis including honey,

collagenase, urea, and papin. Honey has been shown to be effective in treatment for pressure ulcers as it can provide wound healing and antimicrobial factors [10]. One study found that collagenase ointment acquired from *Clostridium histolyticum* used with other debridement techniques optimizes pressure ulcer wound healing more than by only using collagenase alone [11]. Papain-Urea has also been proposed as another autolytic/enzymatic debridement. One study displayed Papain-urea was effective in wound healing in rats, but needs to be further researched about its debridement properties [12].

As this method relies heavily on the patient's own self healing abilities, it is extremely important to consider the health status of the patient. Systemic imbalances such as diabetes, malnutrition, cancer, vascular disease, smoking, obesity and others will prevent migration and performance of the host's cells. It is recommended that if improvement is not seen in 24-72 hours, another method of treatment should be used [7]. To sum, one may consider the advantages of autolytic debridement for its simplicity, painless action and maintenance of healthy tissue. However autolytic debridement is slow, and can take weeks to complete. It is not useful for individuals who need fast debridement, have an infection or have poor vascularization to the wound [7].

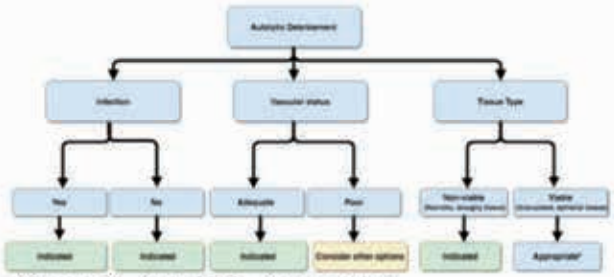


Figure 3. *Autolytic Assessment*
**Commonly used more for moisture balance*

Larval/Maggot

Maggot debridement therapy (i.e., MDT) is an effective therapy for patients who cannot undergo surgical debridement based off medical contraindications[13] . Larvae are sterilized with radiation before administration so that they cannot transform from the larva stage to the pupae stage. The larvae secrete enzymes to degrade necrotic tissue and the biofilm created by bacteria. This nutrient rich liquid is then consumed by the larva [13]. It has been proposed that maggot debridement therapy contains many beneficial effects beyond just dissolving necrotic or infected tissue. Studies have shown that after larval therapy, growth factors that induce neo-granulation as well as angiogenesis are expressed and endothelial cell activity is increased [9, 14].

The sterilization aspect of MDT is likely one of the more beneficial aspects of this therapy. Maggots consume all bacteria, even drug resistant bacteria including and not limited to methicillin-resistant Staphylococcus aureus and vancomycin-resistant Enterococcus [13].
Maggot debridement therapy is a great option for vascular deficient patients who cannot undergo sharp debridement or other therapies. When considering different wounds, MDT is best suited for diabetic ulcers, however, may not be necessarily the best primary treatment for diabetic wounds[13].

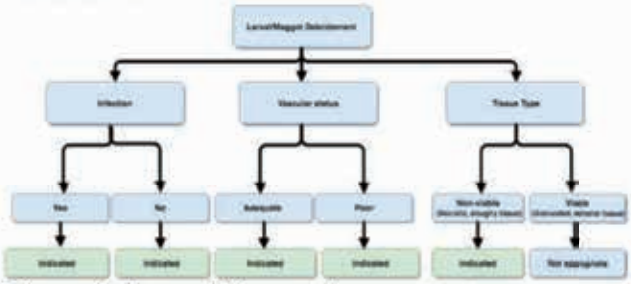


Figure 4. *Larval/Maggot Assessment*

The algorithms seen are constituted on three major factors that are critical in

assessing and discerning a debridement method. Recurrent findings lend the most attention and consideration to infection, vascular status and tissue type of an ulcer when prioritizing a debridement method. Altogether, the algorithm explains a step process in what action will be taken appropriately when assessing an ulcer regarding these factors.

Generally, if the wound is not covered in granulation tissue, such as a sign of healthy or healing tissue, debridement of any type can be performed to progress a wound towards healing. Understanding the adverse impact devitalized tissue has on wound healing highlights the importance of debridement in wound management. It is shown that nonviable tissue becomes a barrier to cell migration and provides an ideal environment for bacterial proliferation. If not addressed, nonviable tissue prolongs the inflammatory response, enhancing the excess production of proinflammatory cytokines [15]. Discerning viable versus nonviable tissue is important in first identifying if the ulcer should be debrided. Therefore it is one of three important factors to consider when considering debridement.

Next, vascular status is very important in assessing the healing potential of an ulcer; it is a vital characteristic and it is well known that reduced vascular perfusion results in poor wound healing. This assessment is especially important before sharp debridement; if a compromised patient is to undergo sharp debridement, further trauma and damage may be done [16].

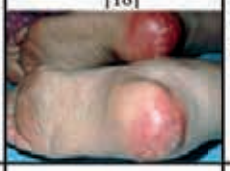
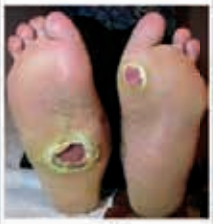
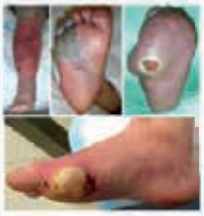
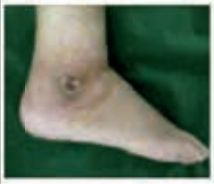
In addition, infection is an important factor to consider when evaluating a wound. If not properly addressed, improper wound healing can take place along with the increased risk of limb amputation. Debridement of necrotic tissue within a wound is critical by helping hinder the progression of infection. Assessment is important in all wound types, however special attention should be made when considering using a wet-to-dry debridement as it is contraindicated in infected ulcers [7]. The table listed at the end provides new clinicians a 'quick glance' of key differences and highlights of each debridement technique in chart form. For example, ease in choosing a method should be made when comparing the debridement advantages versus disadvantages, mechanism of action and any miscellaneous factors.

Conclusion

This article serves as a guide for podiatric students entering clinical rotations to simplify and aid in the process of understanding debridement techniques and their use in different situations. Most wounds will progress through the normal stages of healing and with appropriate care and debridement, healthy tissue will materialize quicker. It is important for the student to understand how to properly assess an ulcer as well as determining which debridement technique is indicated. With all types of debridement, if the other factors of wound healing are not managed, then the process may be arrested. It should be noted that if the wound does not close at a rate around 10-15 percent per week and/or vital structures (nerves, bone, tendon, etc.) are exposed, additional and more advanced therapy may be needed [13]. With the many techniques of debridement, no one surgical debridement method was shown to be more effective in achieving complete ulcer healing when compared to non-surgical management[17]. A six month period showed no significant difference between surgical versus non-surgical management in observing complete ulcer healing[17]. However, knowledge and practice of each of the main techniques will be beneficial to diversify and offer alternative treatments for patients as needed/preferred; such as comfort and efficiency of technique.

As mentioned previously, cost and insurance is not a component of this study; however, they play a large role in deciding which debridement method is preferred. Additionally, it is important to consider the efficacy of a treatment as well. A much more efficacious but expensive treatment may be more cost effective long term [7].

Table 1.

Type	Mechanism of Action	Advantages	Disadvantages	Example of ulcer	Miscellaneous
Autolytic/Enzymatic	Engagement of the bodies own healing resources and/or an application of enzymes for healing.	Use of natural products that are not harmful to the body	Long healing time and cannot be used for infected ulcers	[18] 	Consider health status and systemic imbalances
Larval Therapy (biosurgical)	Secrete enzymes to dissolve and consume dead tissue and bacteria	May express growth factors for angiogenesis and boost endothelial activity. Consumes bacteria and is good for sterilizing wounds	Must be planned in advance. Larval therapy generally takes multiple sessions and is more costly than other treatments.	 [19]	
Mechanical: Ultrasonic (US) Wet to Dry (WD) Hydrosurgical (HS)	**US**: low frequency sound waves projected at the wound **WD**: application of wet dressing and let dry and peel of non-viable tissue. **HS**: high pressure water shot at wound for dead tissue removal	**US:** may provide antibacterial and pro-haling qualities **WD**: simple procedure that does not require specialist **HS:** quick and efficient	**US:** Requires device and practitioner to perform **WD**: Takes time and can remove non-viable tissue **HS**: High Cost with limited availability	[20] 	
Sharp/Surgical	Use of a sharp instrument to discard non-viable tissue	Quick with no use of analgesics or topical medication.	Requires qualified practitioner to perform procedure, human error could result in damaging anatomic structures	[21] 	"Gold standard" of debridement. Contraindicated in arterial, pressure, and any wound with dry eschar covering it.

References:

1. Pecoraro RE, Reiber GE, Burgess EM. Pathways to diabetic limb amputation. The basis for prevention. *Diabetes Care 1990*; 13(5): 513-21
2. *The Johanna Briggs Institute. Surgical and conservative sharp wound debridement for chronic wounds. Wound Prac Research 2011; 19: 29–31.*
3. *Gray D, Acton C, Chadwick P, et al (2011) Consensus guidance for the use of debridement techniques in the UK. Wounds UK 7(1): 77-84.*
4. Woo, K. Y., Keast, D., Parsons, N., Sibbald, R. G. and Mittmann, N. (2015), The cost of wound debridement: a Canadian perspective. Int Wound J, 12: 402–407. doi:10.1111/iwj.12122
5. Haycock S, Chadwick P. Debridement of diabetic foot wounds. Nursing Standard 2012; 26, 24, 51-58.
6. Nick Allan 1 , Merle Olson 1 , Dennis Nagel 2 and Robin Martin 3 1 Innovotech Inc., Edmonton, Canada, 2 Innovotech Animal Resource Centre, Crossfield, Canada, 3 Smith & Nephew Medical Ltd, Hull, UK
7. Wodash, A. J. (2012). Wet-to-Dry Dressings Do Not Provide Moist Wound Healing. *The Journal of the American College of Clinical Wound Specialists*, *4*(3), 63–66. http://doi.org/10.1016/j.jccw.2013.08.001
8. Michailidis, L., Williams, C. M., Bergin, S. M., & Haines, T. P. (2014). Comparison of healing rate in diabetes-related foot ulcers with low frequency ultrasonic debridement versus non-surgical sharps debridement: a randomised trial protocol. *Journal of Foot and Ankle Research,7*(1). doi:10.1186/1757-1146-7-1
9. Zhang, J., Sun, X., Chen, J., Hu, Z. W., Wang, L., Gu, D. M., & Wang, A. (2017). Increasing the miR-126 expression in the peripheral blood of patients with diabetic foot ulcers treated with maggot debridement therapy. *Journal of Diabetes and its Complications,31*(1), 241-244. doi:10.1016/j.jdiacomp.2016.07.026
10. Biglari, B., Linden, P. H., Simon, A., Aytac, S., Gerner, H. J., & Moghaddam, A. (2011). Use of Medihoney as a non-surgical

therapy for chronic pressure ulcers in patients with spinal cord injury. *Spinal Cord,50*(2), 165-169. doi:10.1038/sc.2011.87
11. Carter, M. J., Gilligan, A. M., Waycaster, C. R., & Fife, C. E. (2016). Treating pressure ulcers with clostridial collagenase ointment: Results from the US Wound Registry. *Wound Repair and Regeneration,24*(5), 904-912. doi:10.1111/wrr.12458
12. Majid FA, Suvik A, Effendy MA, Nouri HS (2010), Efficacy of papain-based wound cleanser in promoting wound regeneration. *Pakistan Journal of Biological Sciences*
13. Attinger, C. E., Janis, J. E., Steinberg, J., Schwartz, J., Al-Attar, A., & Couch, K. (2006). Clinical Approach to Wounds: Debridement and Wound Bed Preparation Including the Use of Dressings and Wound-Healing Adjuvants. *Plastic and Reconstructive Surgery,117*(SUPPLEMENT). doi:10.1097/01.prs.0000225470.42514.8
14. 12. Sun, X., Chen, J., Zhang, J., Wang, W., Sun, J., & Wang, A. (2016). Maggot debridement therapy promotes diabetic foot wound healing by up-regulating endothelial cell activity. *Journal of Diabetes and its Complications,30*(2), 318-322. doi:10.1016/j.jdiacomp.2015.11.009
15. Young, T. (2011). Debridement – is it time to revisit clinical practice? *British Journal of Nursing, 20*(Sup6). doi:10.12968/bjon.2011.20.sup6.s24
16. Edmonds ME, Foster AVM. Managing the diabetic foot. Oxford: Blackwell Science, 2005.
17. *National Institute for Health and Care Excellence.* NHS Evidence. Diabetic foot problems: evidence update March 2013. Available at*: http:// www. evidence.nhs.uk.* Accessed April 2013. Nick Allan 1 , Merle Olson 1 , Dennis Nagel 2 and Robin Martin 3 1 Innovotech Inc., Edmonton, Canada, 2 Innovotech Animal Resource Centre, Crossfield, Canada, 3 Smith & Nephew Medical Ltd, Hull, UK
18. Frykberg RG. Diabetic foot ulcers: pathogenesis and management. Am Fam Physician. 2002;66:1655–1662.
19. Spear M. Pressure Ulcer Staging-Revisited. Plastic Surgical Nursing. 2013;33(4):192-194.
20. Pinzur MS. Current concepts review: Charcot arthropathy of the foot and ankle. *Foot Ankle Int.* 2007; 28(8):952-59.
21. Nguyen TV, Margolis DJ. Hydroxyurea and lower leg ulcers. *Cutis*. 1993;52(4):217-219.

Frontal Plane Rotation for Hallux Abductovalgus: A Review of Literature

Samvel Keshishyan, MS4 College of Podiatric Medicine

Introduction

The Lapidus procedure has been a commonly used procedure for patients with severe hallux abductovalgus (HAV) as well as those with HAV combined with a component of hypermobility at the 1st tarsometatarsal joint (TMTJ). The Lapidus involves arthrodesis of the 1st TMTJ, and is widely considered a technically difficult procedure to master. The current standard of treatment for HAV surgically involves metatarsal osteotomies placed depending on the level of deformity. Most commonly, the procedure of choice is the Austin, or a chevron osteotomy at the metatarsal head with lateral translation of the metatarsal head in the transverse plane. This procedure is usually accompanied by a 1st metatarsophalangeal joint (MTPJ) soft tissue release, commonly known as the McBride procedure.

Dayton and his colleagues have attempted to shift the current treatment paradigm for HAV by taking an innovative approach.[1] This new paradigm starts off with a new definition of HAV to resemble the triplanar nature of the deformity, metatarsus primus adductovalgus[2]. The third plane in question, the frontal plane, is the cornerstone to this new paradigm. Dayton et. al. hope to change the landscape of HAV surgical treatment by addressing the deformity at its apex, or CORA (center of rotational angulation) as described by Paley[3], located at the 1st TMTJ. This new paradigm urges the use of the Lapidus procedure while utilizing a derotation of the 1st metatarsal in a varus direction to properly address all three planes of this deformity.[1] According to the theory, frontal plane correction is able to adequately reduce the tibial sesamoid position, proximal articular set angle (PASA), intermetatarsal angle (IMA), hallux abductus angle (HAA), as well as the enlarged medial eminence without the need for any distal osseous or soft tissue procedures. Essentially, this new paradigm seeks to replace the hundreds of procedures described in literature for HAV. This literature review was conducted to take an academic approach to evaluate the validity of each aspect of this new paradigm for correction of hallux abductovalgus.

Frontal Plane Position of the 1st Metatarsal in HAV

The novelty of the new paradigm of frontal plane correction for hallux abductovalgus is the valgus or external rotation of the 1st metatarsal. To effectively present that this valgus rotation is part of the disease progression rather than a normal variant, one must compare the frontal plane position between a group with HAV with a control group. Scranton and Rutkowski did just that in their cadaveric study, concluding that cadaveric feet with HAV (14.5* valgus) contained on average 11.4 * more valgus rotation than those in the control group (3.1 * valgus).[4] Mortier et al found a similar result, with their study resulting in a mean 12.7 * valgus in feet with HAV.[5] These studies showed a trend of frontal plane pathology in patients with HAV. Dayton

et al conducted a radiographic study of cadaveric feet with sequential increase in varus and valgus positioning of the 1st metatarsal.[6] Tibial sesamoid position was found to be directly correlated with frontal plane positioning of the metatarsal (Figure 1). The study also showed that an increase in valgus resulted in a significant increase in the IMA, indicating that increased frontal plane pathology can increase the severity of HAV. Ramdass and Meyr added to the discussion by finding that the distance between the tibial sesamoid and the 2nd metatarsal does not change with an increase in tibial sesamoid position.[7] Dayton et al postulated that this apparent radiographic change in the sesamoid position is due solely to the positional change of the metatarsal, rather than the sesamoids.[6] Based on these findings, it is feasible to conclude that frontal plane rotation is consistently present in the disease process of hallux abductovalgus.

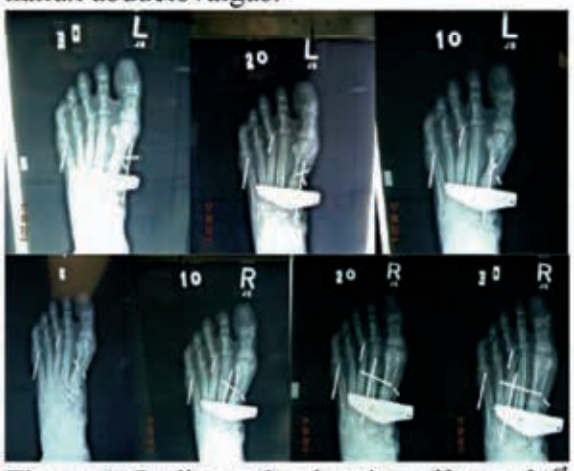


Figure 1: Radiographs showing effects of 1st metatarsal rotation starting at the top left image with 30 * valgus with 10 degrees of increased varus with each successive film, ending with 30 * varus at the bottom right showing correction of tibial sesamoid position with rotation of the metatarsal alone.[6]

Enlarged Medial Eminence

Medial eminence resection, or resection of an enlarged metatarsal head (popularly known as the Silver procedure) has been a hallmark adjunct procedure for surgical correction for HAV. This enlargement of the medial aspect of the first metatarsal head is apparent during clinical examination of patients with HAV. Dayton et al theorized that this so-called medial eminence is a result of valgus rotation of the 1st metatarsal causing the dorsomedial aspect of the metatarsal head to become prominent (Figure 2).[8] The authors state that the oval or square nature of the metatarsal head causes its apparent width to visually seem to be increased when there is valgus rotation at the metatarsal. Grode and McCarthy used advanced imaging with cryomicrotomy and found Dayton's theory to be correct, noting frontal plane rotation is a direct cause of the medial eminence, which is representative of the dorsomedial surface of the metatarsal head.[9] Thorardson and Krewer found that there was no significant difference at the medial eminence between feet with HAV and rectus feet.[10] They attributed the apparent enlargement of the medial eminence to the loss of medial cartilage caused by increased forces from a laterally deviated hallux. Lenz et al measured the sizes of the medial eminence and the metatarsal head of those with and without HAV on AP radiographs.[11] They found a significant difference of 1.12mm larger at the medial eminence and 2.81mm larger at the metatarsal head in patients with HAV compared to those without deformity. The authors attributed these increased widths on 2-dimensional imaging studies to frontal plane rotation of the metatarsal. Based on these studies, the enlargement of the medial eminence is a direct consequence

of the valgus rotation placed on the metatarsal as part of HAV and that routine resection of the medial eminence may be unnecessary. Surgeons should attempt frontal plane correction of the metatarsal prior to evaluation of the apparent enlarged eminence to assess whether the Silver procedure is necessary.

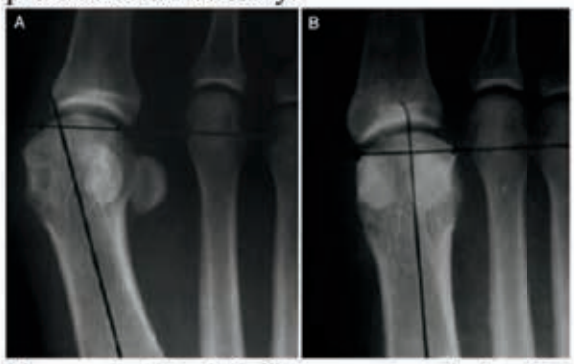


Figure 2: (A) AP film preoperatively. (B) AP film postoperatively after correction of frontal plane rotation and arthrodesis of the 1st tarsometatarsal joint showing apparent reduction of the enlarged medial eminence without bony resection.[1]

Is PASA a Radiographic Artifact?

An increase in the proximal articular set angle has classically been a key indicator in osteotomy placement for the treatment of HAV. An increase in PASA has historically been an indication for a metatarsal head closing wedge procedure, known as a Reverdin, due to the structural nature of the deformity. Although the PASA plays a key role in HAV correction, recently there has been literature suggesting deficiencies in its utility for surgical planning.[1, 8, 12-13, 22] These studies state that the increased PASA is a radiographic artifact caused by frontal plane rotation[8] and that the PASA has poor reproducibility and accuracy.[12] Studies by Vittetoe et al and Robinson et al stated that the PASA changed in correlation to frontal plane rotation of the first metatarsal in a cadaveric model.[13, 22] This demonstrated that the PASA may be more influenced by valgus metatarsal rotation rather than structural deformity of the distal 1st metatarsal. Other researchers found improvement in the PASA utilizing HAV procedures that were proximal to the head, previously thought to have no effect on PASA.[1, 8, 13-15] Dayton et al showed significant reduction of PASA utilizing a derotational Lapidus procedure (Figure 3), and theorized that the apparent increase in PASA could be attributed to medial cartilage loss.[1, 8] Based on these studies, one can infer that the an increased PASA can be caused by frontal plane rotation of the first metatarsal, and that correction of the PASA can be made by utilizing a Lapidus procedure in combination with frontal plane correction. The PASA should be re-evaluated following varus rotation of the metatarsal to appropriately use it as an indication for further surgical correction.

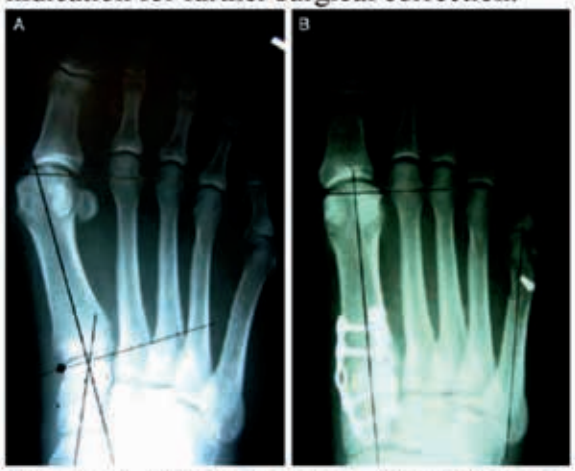


Figure 3: (A) Preoperative AP radiograph. (B) Postoperative AP radiograph following derotation of the 1st metatarsal and 1st tarsometatarsal fusion without resection of the apparent medial eminence with apparent reduction of the PASA.[8]

Are AP Films Appropriate for Evaluation of Sesamoid Position?

Although anteroposterior (AP) radiographs are a widely-used device to assess the sesamoid position pre- and post-HAV correction, there have been many studies that demonstrate the high degree of variability using this assessment technique.[1, 16-19] One such study by Inman found that although the tibial sesamoid position appears to be laterally deviated on the AP films, the sesamoid axial view demonstrates an anatomic position of the sesamoids separated by the median crista and sitting in their respective grooves below the first metatarsal head.[16] This key finding effectively displays that even when the sesamoid complex is in its anatomic location, the AP radiographs may erroneously suggest a lateral displacement. There have been other studies showing that this apparent pseudo-subluxation is caused by a valgus rotation of the metatarsal, putting into question the use of AP films for sesamoid position in bunion assessment.[17-18] Kuwano et al found a statistically significant correlation between the severity of HAV and external rotation of the sesamoid complex using the sesamoid-axial view.[18] This strengthens the argument that frontal plane rotation, HAV, and sesamoid position are interrelated. Based on these studies, one can feasibly conclude that AP films are more significantly affected by frontal plane rotation of the metatarsal than previously thought, which has affected the validity of using these views for assessment of transverse plane translocation of the sesamoid complex. The sesamoid-axial view may prove to be more useful in analyzing the relationship of the sesamoids to their corresponding plantar metatarsal articulation.

Frontal Plane Rotation to Correct Sesamoids

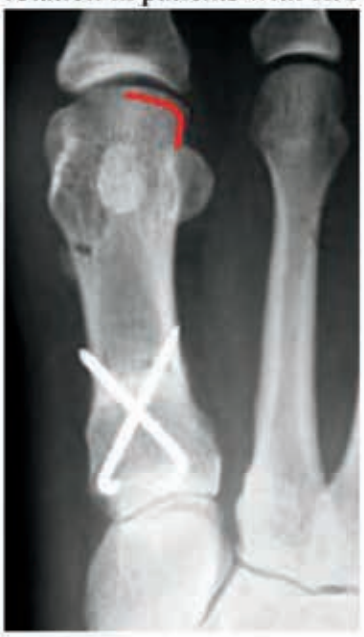
As previously discussed, the tibial sesamoid position is commonly used to evaluate adequate correction of HAV. It was also mentioned that valgus rotation of the metatarsal could potentially be a cause of this increased tibial sesamoid position.[17-18] The next logical question would be: Can correction of the frontal plane deformity in HAV adequately and consistently reduce the tibial sesamoid position? Okuda et al described a technique whereby the surgeon performed a proximal metatarsal crescentic osteotomy with varus rotation of the metatarsal in order to correct the frontal plane deformity for HAV.[15] Using this technique on 31 patients, they were able to reduce the tibial sesamoid position to normal (defined as position 4 or less) in all 31 cases. Dayton et al were able to replicate the findings of Okuda et al with a cadaveric study that showed a correlation between correction of the valgus rotation of the metatarsal in HAV and decrease in tibial sesamoid position (Figure 1).[6] DiDomenico et al described their novel technique for reduction of the tibial sesamoid apparatus without 1st MTPJ soft tissue procedures utilizing frontal plane correction of the metatarsal in a Lapidus procedure.[14] They state that correction of the sesamoids by soft tissue release at the 1st MTPJ is only able to correct the triplanar deformity in the transverse plane, implying inadequate reduction and allowing potential for recurrence. Dayton et al also perform a similar technique albeit with different fixation constructs, and describe that in some cases it may be necessary to thin the medial capsule on cases where the medial eminence still appears enlarged, attributing this to thickening of the medial capsule.[8]

DiDomenico and Dayton performed

a retrospective review of 36 cases whereby they analyzed the degree of varus rotation necessary to realign the 1st MTPJ.[20] They found that when frontal plane rotation was corrected, there was an improvement in the alignment of the 1st MTPJ as well as tibial sesamoid position without the need for capsular balancing, agreeing with previous studies. They concluded that, on average, 22.1 degrees of varus rotation is required for reduction of frontal plane deformity in HAV in order to obtain 1st MTPJ and sesamoidal alignment. Revisiting the initial question, there is overwhelming evidence in literature supporting the theory that frontal plane correction in HAV is able to adequately and consistently reduce the tibial sesamoid position.

Lateral Round Sign

Close examination of the AP radiographic shape of the 1st metatarsal head can potentially give surgeons a pearl for evaluating potential for recurrence of HAV. One such finding evaluates the lateral aspect of the metatarsal head, referred to as the lateral round sign. Okuda et al described the lateral round sign by evaluating the lateral edge of the 1st metatarsal head.[21] If the lateral edge displayed a rounded shape as opposed to having an angled appearance (Figure 4), a positive lateral round sign was determined. In their study, they also examined the presence of this lateral round sign in patients postoperatively following HAV correction. Patients that possessed the lateral round sign post-operatively had a statistically significant increase in both hallux abduction angle as well as intermetatarsal angle at the most recent follow up (mean forty-eight months), while those that did not possess the lateral round sign did not have a significant increase in either HAA or IMA. The authors attributed this presence of the lateral round sign to frontal plane rotation of the 1st metatarsal. Based on the evidence presented, one should expect a patient with a positive lateral round sign to have an eventual recurrence of HAV due to inadequate reduction of frontal plane rotation. Additionally, presence of the lateral round sign should be evaluated intraoperatively and surgical modification must be made to address the frontal plane rotation in patients with HAV.



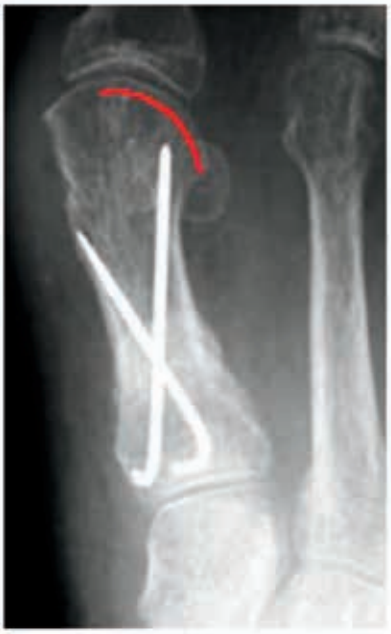


Figure 4: (A) Postoperative AP radiograph displaying a negative lateral round sign, due to the angular shape of the lateral aspect of the 1st metatarsal head. (B) Postoperative AP radiograph showing a positive lateral round sign at the lateral 1st metatarsal head.[21]

Conclusion

Hallux abductovalgus deformity is a triplanar deformity[1-2], with frontal plane valgus rotation of the 1st metatarsal being a historically overlooked aspect until recently in literature. Correction of this frontal plane valgus rotation of the 1st metatarsal has been shown to significantly reduce IMA, HAA, PASA, and tibial sesamoid position without the need for distal soft tissue releases or resection of the medial eminence.[6, 8, 14-15] AP radiographs have been shown to be of less utility for evaluation of the sesamoid position in assessing adequacy of HAV correction.[1, 16-19] Surgeons should order a sesamoid-axial view for a more complete evaluation of the sesamoid position in relation to the 1st metatarsal. When performing a Lapidus procedure, derotation of the metatarsal in a varus direction should be performed to assess reduction of medial eminence and sesamoid position prior to resorting to bony resection of the metatarsal head or soft tissue release. Intraoperative assessment of a lateral round sign should also be evaluated following any HAV procedure as this finding has been noted to have an increased rate of recurrence of HAV when present post-operatively.

Due to the fact that most of the studies involving rotation of the metatarsal have been centered on radiographic evaluation of HAV with complete negligence of clinical findings, further research is required to validate the necessity to perform a Lapidus procedure over distal metatarsal osteotomies. An argument can be made that, although the distal chevron procedure does not adequately address all three planes of deformity in HAV, the clinical and functional outcomes are satisfactorily improved without unnecessary fusion of the 1st tarsometatarsal joint. Further studies will have to disprove this argument in order to successfully shift the current paradigm of HAV correction. These future studies ideally will involve a prospective approach whereby patients with HAV are randomly grouped into either a derotational Lapidus group or a distal metatarsal osteotomy group with evaluation of radiographic correction as well as functional and clinical outcomes in mid and long term.

References

1. Dayton P, Kauwe M, Feilmeier M. Is our current paradigm for evaluation and management of the bunion deformity flawed? A discussion of procedure philosophy relative to anatomy. J Foot Ankle Surg.

2015;54(1):102-11.
2. Dayton P, Kauwe M, Feilmeier M. Clarification of the anatomic definition of the bunion deformity. J Foot Ankle Surg. 2014;53(2):160-3.
3. Paley D, Herzenberg JE, Tetsworth K, Mckie J, Bhave A. Deformity planning for frontal and sagittal plane corrective osteotomies. Orthop Clin North Am. 1994;25(3):425-65.
4. Scranton PE, Rutkowski R. Anatomic variations in the first ray: Part I. Anatomic aspects related to bunion surgery. Clin Orthop Relat Res. 1980;(151):244-55.
5. Mortier JP, Bernard JL, Maestro M. Axial rotation of the first metatarsal head in a normal population and hallux valgus patients. Orthop Traumatol Surg Res. 2012;98(6):677-83.
6. Dayton P, Feilmeier M, Hirschi J, Kauwe M, Kauwe JS. Observed changes in radiographic measurements of the first ray after frontal plane rotation of the first metatarsal in a cadaveric foot model. J Foot Ankle Surg. 2014;53(3):274-8.
7. Ramdass R, Meyr AJ. The multiplanar effect of first metatarsal osteotomy on sesamoid position. J Foot Ankle Surg. 2010;49(1):63-7.
8. Dayton P, Feilmeier M, Kauwe M, Hirschi J. Relationship of frontal plane rotation of first metatarsal to proximal articular set angle and hallux alignment in patients undergoing tarsometatarsal arthrodesis for hallux abducto valgus: a case series and critical review of the literature. J Foot Ankle Surg. 2013;52(3):348-54.
9. Grode SE, Mccarthy DJ. The anatomical implications of hallux abducto valgus: a cryomicrotomy study. J Am Podiatry Assoc. 1980;70(11):539-51.
10. Thordarson DB, Krewer P. Medial eminence thickness with and without hallux valgus. Foot Ankle Int. 2002;23(1):48-50.
11. Lenz RC, Nagesh D, Park HK, Grady J. First Metatarsal Head and Medial Eminence Widths with and Without Hallux Valgus. J Am Podiatr Med Assoc. 2016;106(5):323-327.
12. Chi TD, Davitt J, Younger A, Holt S, Sangeorzan BJ. Intra- and inter-observer reliability of the distal metatarsal articular angle in adult hallux valgus. Foot Ankle Int 23:722–726, 2002.
13. Vittetoe DA, Saltzman CL, Krieg JC, Brown TD. Validity and reliability of the first distal metatarsal articular angle. Foot Ankle Int. 1994;15(10):541-7.
14. Didomenico LA, Fahim R, Rollandini J, Thomas ZM. Correction of frontal plane rotation of sesamoid apparatus during the Lapidus procedure: a novel approach. J Foot Ankle Surg. 2014;53(2):248-51.
15. Okuda R, Yasuda T, Jotoku T, Shima H. Supination stress of the great toe for assessing intraoperative correction of hallux valgus. J Orthop Sci. 2012;17(2):129-35.
16. Inman VT. Hallux valgus: a review of etiologic factors. Orthop Clin North Am. 1974;5(1):59-66.
17. Boberg JS, Judge MS. Follow-up of the isolated medial approach to

hallux abducto valgus correction without interspace release. J Am Podiatr Med Assoc. 2002;92(10):555-62.
18. Kuwano T, Nagamine R, Sakaki K, Urabe K, Iwamoto Y. New radiographic analysis of sesamoid rotation in hallux valgus: comparison with conventional evaluation methods. Foot Ankle Int. 2002;23(9):811-7.
19. Talbot KD, Saltzman CL. Assessing sesamoid subluxation: how good is the AP radiograph? Foot Ankle Int. 1998;19(8):547-54.
20. Dayton P, Kauwe M, Didomenico L, Feilmeier M, Reimer R. Quantitative Analysis of the Degree of Frontal Rotation Required to Anatomically Align the First Metatarsal Phalangeal Joint During Modified Tarsal-Metatarsal Arthrodesis Without Capsular Balancing. J Foot Ankle Surg. 2016;55(2):220-5.
21. Okuda R, Kinoshita M, Yasuda T, Jotoku T, Kitano N, Shima H. The shape of the lateral edge of the first metatarsal head as a risk factor for recurrence of hallux valgus. J Bone Joint Surg Am. 2007;89(10):2163-72.
22. Robinson AH, Cullen NP, Chhaya NC, Sri-ram K, Lynch A. Variation of the distal metatarsal articular angle with axial rotation and inclination of the first metatarsal. Foot Ankle Int. 2006;27(12):1036-40.

Morton's Neuroma: Diagnosis and Management Without Nerve Destruction

Dana Lin, MS3 College of Podiatric Medicine

Introduction

Morton's neuroma is a common forefoot pain condition. When a neuroma is located in the space between the 3rd and 4th metatarsals, it is called a Morton's neuroma. This is the most common area for a neuroma to form.[1] Morton's neuroma is an entrapment condition where the common digital nerve is compressed under the deep transverse metatarsal ligament (DTML). In the 3rd intermetatarsal space, the common digital nerve consists of the terminal branches of both medial and lateral plantar nerves and lies beneath the DTML (Fig. 1). It has been thought that these terminal branches come together to make a thicker nerve that is tethered against the DTML.[2] On clinical presentation, patients often complain of pain to the plantar aspect of the foot between the metatarsal heads. [2] Other symptoms include burning, tingling, or cramping pain that radiates to the toes as well as numbness to digits adjacent to the webspace.[3] Occasionally, patients describe feeling a "lump" in the plantar aspect of their foot and that their symptoms worsen in tight or closed-toe shoe gear.[4]

The exact cause of a Morton's neuroma is unclear but there are some risk factors such as poorly fitting shoe gear with a narrow toe box and high-heeled shoes. Morton's neuromas occur more commonly in women, as their toes are hyperextended at the MTPJ by the prolonged use of high-heels shoes.[7] Therefore, one form of treatment can simply be a change in shoe gear. Additional conservative treatments include orthotics, oral NSAIDs, corticosteroid injections, and other injections involving neurolysis. If these conservative treatments have failed, then surgical intervention is warranted such as nerve decompression or neurectomy. Generally, neurectomy is the primary surgical course[5]; however, patients are often looking for a minimally invasive solution. Patients who undergo endoscopic or minimally invasive procedures also typically have less pain and postoperative complications than those who have had open procedures for the same condition.[6] Systematic reviews have indicated that there is insufficient evidence to validate the efficacy of whether any one particular treatment is better than others.[7] The purpose of this article is to

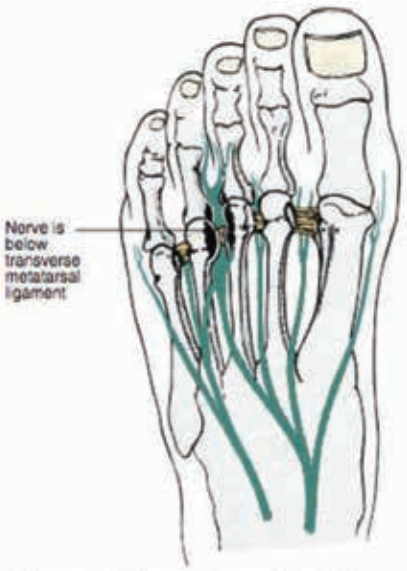


Figure 1. Illustration of the 3rd branch of the medial plantar nerve. Note that the nerve courses plantarward, under the transverse metatarsal ligament.[2]

present alternatives treatments to neurolysis/ neurectomy as well as supportive evidence of conservative and surgical management in not damaging the nerve when treating Morton's neuroma.

Terminology & Histology

The terminology of a condition can affect the types of treatment. In the case of Morton's neuromas, there is a contradiction with its terminology in that it is not identified as a "true neuroma," which would refer to a more proliferative histologic process of a tumor growing from a nerve. A true neuroma would damage to the nerve fibers,[8] while a Morton's neuroma is seen as benign and should be defined as an entrapment neuropathy that causes painful metatarsalgia and occasional loss of sensation. Histologically, Morton's neuroma differs from a neuroma in that there is fibrosis around the perineurim and thus often is defined as a perineurial fibroma.[9] This misnomer may have misconstrued the etiology and introduced a misperception of treating this nerve entrapment, continuing the support for surgical resection as opposed to decompression. Conservative treatments and surgical management of Morton's neuromas such as decompression are practical options that should be considered before proceeding to more invasive procedures of damaging the nerve through methods of neurolysis or neurectomy.

Diagnosis

An accurate diagnosis is important for successful management of a patient suspected of having a Morton's neuroma. Diagnosis primarily relies on the patient's symptoms as well as obtaining a thorough history and physical exam. Physical exam findings may comprise of one or all of the following: Mulder's sign, Sullivan's sign, Gauthier's sign, Bratkowki's sign, and Tinel's sign.[10] Most often clinical findings from the physical exam alone can confirm the diagnosis without imaging. For example, the Mulder's sign, which is a palpable "click," results from the thickened nerve running through the bones upon manipulation of the joint. Mulder's sign is done in the physical exam by squeezing the forefoot while pressing on the neuroma and can elicit pain. The Sullivan's sign demonstrates the splaying of toes like a "peace sign" when standing due to the growing mass pushing on bone and soft tissue and indicates a more chronic neuroma formation. Although these clinical findings may confirm the diagnosis, it is essential to rule out other causes of forefoot pain such as stress fractures, intermetatarsal bursitis, synovitis, capsulitis, arthritis, soft tissue tumors, tarsal tunnel syndrome. Diagnostic testing may likewise be performed to further solidify the diagnosis of a Morton's neuroma and can rule out other differential diagnoses. These diagnostic testing includes radiography, ultrasound, magnetic resonance imaging (MRI), nerve conduction study, and toe tip sensation. Ultrasound and MRI also help to obtain a better view of the neuroma and its location within the forefoot. Radiographs are necessary to visualize any structural issues, which play an important role in surgical planning, as the structural issue should be addressed before any nerve component. Furthermore, when the diagnosis is still unclear despite imaging and a thorough physical exam, a diagnostic injection of local anesthetics may be helpful in

localizing the pain and differentiating adjacent interspace lesions.[8]

Treatment

Conservative management for Morton's neuroma

The initial management approach is a change of shoe gear such as avoiding constrictive shoes and choosing a wider toe box shoes. [10] Shoe modifications allow the metatarsal heads to spread out and relieve the plantar pressure on the nerves.[2] Applying metatarsal pads may also help offload the inflamed nerve. Similarly, custom orthotics with a metatarsal dome alleviates pressure on metatarsal heads.[10] Further shoe modifications include metatarsal bars and rockers.[2] Other conservative treatments comprised of the RICE (rest, ice, compression, elevation.) method, orals NSAIDs, physical therapy, and activity modifications.[2] When these treatments fail to provide pain relief, then corticosteroids injections can occasionally be effective.[11] Corticosteroid injections only provide a temporary relief and can be associated with complications such as plantar fat pad atrophy, plantar plate rupture, and discoloration of the skin; thus, this type of injection should be used with some caution.[12]

Other injection therapies that may be used is through neurolysis methods such as phenol and alcohol sclerosing injections. These methods produce injury to the nerves and surrounding tissue. In 1999, the Journal of Foot and Ankle Surgery published a study by Dockery that established the use of alcohol sclerosis as a viable alternative treatment to Morton's neuroma when conservative therapy fails.[13] This study utilized sclerosing alcohol at 4% strength over course of 3–7 injections and included a sample population of 100 patients. Dockery's study resulted in an 89% success rate of patients being subjectively improved. Despite these results, many of the articles on alcohol sclerosing have been low-level studies with insufficient evidence and most have warranted further research. Recent studies found that alcohol sclerosing therapy was not an effective treatment and advocated to avoid the use of alcohol solution due to being associated with poor results.[14] In the one study, symptomatic relief was reported in only 7 of 32 patients (22%) who were treated with alcohol sclerosing injections.[15] Though other therapeutic neurolysis options such as cryogenic and radiofrequency ablations have also been increasingly available as of late, the results of these procedures are not considered to have long-term efficacy. Furthermore, one study found that there is a potential in worsening pain because of nerve regeneration and that the nerve did not have to be completely destroyed for patients to get pain relief.[14]

There are emerging novel therapeutic modalities that offer pain relief without destroying the nerve. One alternative method to conventional radiofrequency is pulse radiofrequency. Unlike conventional radiofrequency, pulse radiofrequency temperatures do not exceed 38° to 42°C, protecting the nerves from destruction and leads to a less painful procedure.[16] Studies have also demonstrated favorable outcomes in pulse radiofrequency compared with conventional radiofrequency.[17] In treating Morton's neuroma, one original article found a decrease in pain level in 18 of 20 (90%) patients.[18] Although the sample size is small, this evidence shows that ultrasound-guided pulsed radiofrequency is a promising

management modality. Another possible novel modality presented by a pilot study is treating Morton neuromas with *Botulinum toxin A*, which is also non-neurodestructive as it essentially prevents any signals transmitted from the nerves by blocking acetylcholine release at the motor endplate.[19] Nevertheless, there are still very few high-leveled evidence studies proving the efficacy of these various injection modalities. Destructive measures to the nerves of a nerve entrapment would be better avoided. When conservative treatments have failed and symptoms reoccur, then surgery is indicated.

Surgical management for Morton's neuroma

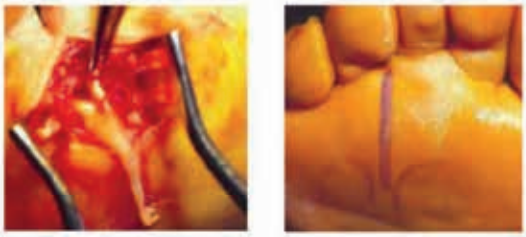


Figure 2. Plantar incisional approach where the nerve is transected proximal to

Even after conservative treatments, the majority of patients continue to have symptoms and 60-70% choose to do a neurectomy, an excision of the neuroma.[2] A neuroma excision is a very common procedure, as is the only location in the body in which nerves are generally resected. The overall success rates following neurectomies procedures also vary widely from 57% to 93% for both the dorsal and plantar (Fig. 2) surgical excision approach.[3] Though this is a common surgical treatment of Morton's neuroma, it should be made clear to the patient that a true neuroma will frequently be formed. A true neuroma forms at the resected end of a common digital nerve and often demonstrates proliferative histologic changes. When this happens it is called a recurrent Morton neuroma or also known as a stump neuroma. Stump neuroma may produce worsening symptoms as a complication of the initial surgical resection method, which would then require a revisional surgery.[14] In one retrospective review of 120 patients treated with neurectomy, only 50% good or excellent patient ratings resulted at an average follow-up of 67 months.[20] Other than developing recurrent Morton neuroma or a painful stump neuroma, one additional complication of neurectomy is postoperative numbness.

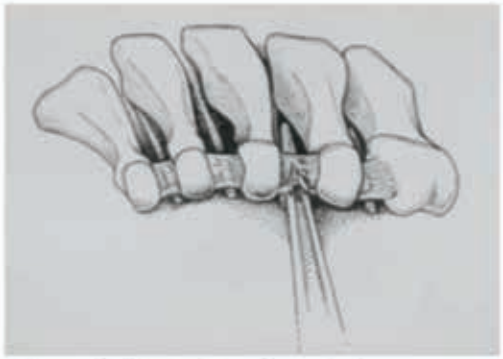


Figure 3. Illustration of surgical technique for decompression of the intermetatarsal nerve. The deep transverse metatarsal ligament is being transected from distal to proximal.[21]

Rather than electing neurectomy as the initial surgical approach for Morton's neuromas, endoscopic nerve decompression has been shown to have fewer risks and complications by releasing the deep transverse metatarsal ligament (Fig. 3), thus salvaging the nerve. The benefit of decompression without excision is that there is no loss of sensation or possible subsequent formation of a stump neuroma, which is more difficult to treat than the primary neuroma before.[21] Other advantages of decompression are the smaller incision

site and faster recovery with minimal risk of hematoma and infections, which are known complications to occur in open techniques. A retrospective review of 69 patients reported an overall success rate of 86.2% while 13.7% had poor results.[22] However, patients in the poor category had a history of prior treatment with alcohol sclerosing injections, which had done previous damaged to the nerve. When those patients with past nerve destructive procedures were removed from the study, there was an overall success rate of 91.3%. This result compares very positively to not only studies of neurectomy but also of neurolysis.

Conclusion

There are alternative therapeutic options to the commonly used neurolysis and neurectomy. Much supportive research as mentioned in this article has advocated not destroying the nerve when treating Morton's neuroma. Moreover, there is no other chronic nerve compression in the human body that is predominantly treated with excising the nerve with expected loss of sensation as the mainstay treatment. With the less than encouraging results and further possible complications, primary neuroma resection is not the best surgical approach for treatment. Subsequently, the initial surgical management for Morton's neuroma should be decompression instead of neurectomy as destroying an entrapped nerve has the potential to release a storm of neural consequences. Endoscopic decompression is also the less invasive surgical technique for treating Morton's neuroma. If decompression of the entrapped nerve has failed, then secondary neurectomy may be deemed appropriate.

References

1. Kay D, Bennett GL. Morton's neuroma. *Foot Ankle Clin N Am*. 2003; 8:49-59.
2. Coughlin MJ, Saltzman CL, Anderson RB. *Mann's Surgery of Foot and Ankle*. 9th edition. 2014; (1): 622-63
3. Southerland JT, Boberg JS, Downey MS, Nakra A, Rabjohn LV. McGlamry's Comprehensive Textbook of Foot and Ankle Surgery, 4th edition. 2012: (1): 119
4. Thomas JL, Blitch EL, Chaney DM, Dinucci KA, et al. Clinical Practice Guideline: Diagnosis and Treatment of Forefoot Disorders. Section 3. Morton's Intermetatarsal Neuroma. J Foot Ankle Surg. 2008; 40(2): 251-256.
5. Singh SK, Ioli JP, Chiodo CP. The surgical treatment of Morton's neuroma. *Curr Orthop*. 2005; 19:379–84.
6. Tomczak RL, Haverstock BD. A retrospective comparison of endoscopic plantar fasciotomy to open plantar fasciotomy with heel spur resection for chronic plantar fasciitis/heel spur syndrome. *J Foot Ankle Surg*. 1995; 34(3):305–11.
7. Thomson CE, Gibson JN, Martin D. Interventions for the treatment of Morton's neuroma. *Cochrane Database Syst Rev*. 2004;(3):CD003118.
8. Giakoumis M, Ryan JD, Jani J. Histologic evaluation of intermetatarsal Morton's neuroma. *J Am Pod Med Ass*. 2013; 103(3):218- 222.
9. Giannini S, Bacchini P, Ceccarelli F, Vannini F: Interdigital neuroma: clinical examination and histopathologic results in 63 cases treated with excision. *Foot Ankle Int*. 2004; 25:79–84.
10. Jain S, Mannan K: The diagnosis and management of Morton's neuroma: a literature review. *Foot & ankle specialist*. 2013; 6(4):307-317.
11. Younger AS, Claridge RJ. The role of diagnostic block in the management of Morton's neuroma. *Can J Surg*. 1998;41(2):127–30.
12. Basadonna PT, Rucco V, Gasparini D, Onorato A: Plantar fat pad atrophy after corticosteroid injection for an interdigital neuroma: a case report. *Am J Phys Med Rehab*. 1999; 78(3):283-

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