

# OFPP

Osteopathic Family Physician

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FAMILY PHYSICIANS

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## EDITOR'S MESSAGE

Summertime When  
Feet & Ankles are Bare

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## REVIEW ARTICLES

Common Orthopaedic Foot & Ankle  
Diagnoses Encountered in the  
Primary Care Setting

Empathy and Its Role in Quality of Care

Etiology, Evaluation and Osteopathic  
Management of Adult Constipation

Dysuria

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## CLINICAL IMAGES

Uvulitis

Inherited Patterned Lentiginosis:  
A Diagnosis of Exclusion

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## PATIENT EDUCATION HANDOUT

Plantar Fasciitis



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Osteopathic Family Physician

# JOURNAL

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- » Anxiety  
*(with OMT treatment component)*
- » Current Management of the Menopausal Woman  
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- » Direct Primary Care: Emerging Practice Alternative
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*(with OMT treatment component)*
- » Movement Disorders - Parkinson's Disease, Essential Tremor, Restless Leg Syndrome *(with OMT treatment component)*
- » Patient Engagement *(Help define the science of engaged research, provide tangible examples of the impact of engaged research, or answer a question or controversy related to patient engagement.)*

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# CONTENTS

- 8 **EDITOR'S MESSAGE**  
**Summertime When Feet & Ankles are Bare**  
*Amy J. Keenum, DO, PharmD*
- 10 **FROM THE PRESIDENT'S DESK**  
**A Future Path for Our Members**  
*Larry W. Anderson, DO, FACOFP dist.*
- 12 **REVIEW ARTICLES**  
**Common Orthopaedic Foot & Ankle Diagnoses Encountered in the Primary Care Setting**  
*Matthew Martell, DO; Adam Bitterman, DO; Brett Auerbach, DO; Simon Lee, MD*
- 20 **Empathy & Its Role in Quality of Care**  
*Sherri J. Howell, DO*
- 24 **Etiology, Evaluation, & Osteopathic Management of Adult Constipation**  
*Jessica Watari, BS, OMS IV; Melinda Danowitz, BA, OMS IV; Samuel Jacob, BS, OMS IV; To Shan Li, DO*
- 32 **Dysuria**  
*Joe Kingery, DO; Brittany Bobrowski, DO*
- 37 **CLINICAL IMAGES**  
**Uvulitis**  
*Kelsey Graven, OMS IV; Lindsay Tjiattas-Saleski, DO, MBA, FACOEP*
- 40 **Inherited Pattern Lentiginosis: A Diagnosis of Exclusion**  
*Nadia Hasan, DO; Mari M. Batta, DO; Tamara B. Fedec, DO*
- 45 **CALENDAR**  
**Calendar of Events**
- 46 **PATIENT EDUCATION HANDOUT**  
**Plantar Fasciitis**

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# EDITOR'S MESSAGE

## Summertime When Feet & Ankles are Bare

Amy J. Keenum, DO, PharmD, Editor, Osteopathic Family Physician

Our feature article this month is a detailed review of foot and ankle injuries. Google® trend shows that most Google® queries of the foot are done in June but that ankle queries peak in October. The OFP editors pay attention to the season when we consider relevance of articles, so on this one we split the difference between June and October and published in July/August. The article is well written and a good review with illustrations, worthy of your time.

Other articles this month are constipation and dysuria, both common in osteopathic family medicine. Drugs are a common cause of constipation and the present attention to opioids should not be missed in this connection as well. Dysuria, while commonly due to infection, has other causes (some serious).

OFP is continuing with our decision to run two Clinical Images. This month the items are on the theme of “name that tune” so the reader is left to read each for a diagnosis. They are short and sweet, a quick reminder of what we see every day.

So as the summer continues with days in flip-flops and sandals, take care of those feet, as ankle season is approaching.



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## FROM THE PRESIDENT'S DESK



### A Future Path for Our Members

Larry W. Anderson, DO, FACOFP *dist.*  
2016 - 2017 ACOFP President

Every 3-4 years one of the most important tasks for the ACOFP Board of Governors is creating a new Strategic Plan that outlines a future path for our members, specialty and ACOFP.

The 2016-2018 ACOFP Strategic Plan<sup>1</sup> approved by our Congress of Delegates in April:

- Family Medicine for America's Health
- Practice Enhancement and Quality Reporting
- Continuing Medical Education
- Single Accreditation System and Osteopathic Distinctiveness

Now how did we come up with these four initiatives? We asked a simple question: "What are the desired outcomes for our members?"

For individual family physicians the answer involves daily career satisfaction, practice transformation and payment based on value. For the specialty in general, a desired future is one where the specialty is recognized and respected for its contribution to primary care.

It also means attracting osteopathic and allopathic medical students to osteopathic-recognized family medicine residency programs. Also, under the Single Accreditation System, it requires highlighting and advancing the contributions of osteopathic medicine.

To address these desired outcomes, the Board had to also ask what external challenges our members face today.

Payment reform is a major factor that is, and will continue to be, an issue for family physicians. We are rapidly shifting away from fee-for-service to a new model of value-based payment. CMS will soon require between 50 and 80 percent of payment be based on quality metrics that will also apply to private payors.

Another major factor is AOA/ACGME Single Accreditation System that is affecting ACOFP-administered osteopathic family medicine residency programs.

What this could mean is that fewer DOs will participate in family medicine residencies that provide osteopathic distinctive training. This would result in fewer AOA/AOBFP certified physicians.

Other issues include that more family physicians are employed and work in restricted integrated networks. Also scope of practice issues, burden of federal legislation, regulation and EHR paperwork requirements remain challenges.

With that in mind, the ACOFP for the next three year will be focusing on four Cornerstone Initiatives.

**Family Medicine for America's Health:** The ACOFP joined seven other medical associations to form FMAHealth, which is a national campaign focusing on six areas that impact family medicine: payment reform, practice transformation, workforce development, technology application, patient engagement and research initiatives.

The ACOFP encourages members to get involved with FMAHealth initiatives. These many initiatives vary on expertise and time commitment, so there's many ways to promote family medicine through FMAHealth.

By promoting the family medicine profession to patients, payors and policymakers, it's hoped that will positively impact physician pay and satisfaction and lead to more medical student opting for the specialty.

For more information, go to [fmahealth.org](http://fmahealth.org).

**Practice Enhancement and Quality Reporting:** The ACOFP will focus on helping members understand and apply the principles of the Patient-Centered Medical Home. We have developed a practice transformation toolkit and are considering a practice management response team to answer members' questions.

The ACOFP Quality Markers Program attempts to address this initiative. It's a tool that collects, analyzes and reports patients on 19 chronic care and wellness suites. We hope to have between 750-1,000 subscribers in three years.

The ACOFP has also hired Debbie Sarason, who is ACOFP, Manager of Practice Enhancement and Quality Reporting. Please feel free to contact her at 847-952-5523 or [debbies@acofp.org](mailto:debbies@acofp.org) with questions.

**Continuing Medical Education:** While the ACOFP provides outstanding CME at ACOFP Annual Convention, ACOFP Intensive Update and Board Review and OMED, we are looking at expanding on demand opportunities, including podcasts, webinars and videos.

**Single Accreditation System & Osteopathic Distinctiveness:** To address the five-year phase in process for the AOA/ACGME Single Accreditation System, the ACOFP is considering many options. The options range from sending an ACOFP representative to ACOFP residency programs to provide program directors with individual mentoring support from dual program directors. Also, the ACOFP may establish a hotline for program directors to ask question about the process.

To promote osteopathic distinctiveness the ACOFP will promote the ACOFP textbook, Somatic Dysfunction in Osteopathic Family Medicine, ACOFP educational curriculum, OMT video and apps.

Please go to the ACOFP website, [www.acofp.org](http://www.acofp.org) to read about our Strategic Plan.

If you have any questions or comments, please contact me at [president@acofp.org](mailto:president@acofp.org).

Sincerely,



Larry W. Anderson, DO, FACOFP *dist.*  
ACOFP President

**REFERENCES:**

1. The American College of Osteopathic Family Physicians (2015). 2016-2018 Strategic Plan of the American College of Osteopathic Family Physicians. Unpublished manuscript

## REVIEW ARTICLE

# Common Orthopaedic Foot & Ankle Diagnoses Encountered in the Primary Care Setting

Matthew Martell, DO,<sup>1</sup> Adam Bitterman, DO,<sup>2</sup> Brett Auerbach, DO,<sup>3</sup> & Simon Lee, MD<sup>4</sup>

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## Keywords:

Foot, Ankle

Achilles Tendon

Ankle Sprains

Ankle Fractures

Plantar Fasciitis

Sports Medicine

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Peroneal Tendon Injuries

Foot and ankle disorders are commonly encountered in the primary care setting. Many of these disorders can be successfully managed by primary care physicians, allowing for early detection and prompt treatment. However, there are circumstances when patients require a referral to a foot and ankle specialist to decrease potential complications of these disorders. This article will review ten common foot and ankle disorders to aid in the improved understanding of when conservative management is appropriate and when referral to a specialist is necessary.

## INTRODUCTION

Foot and ankle disorders are commonly encountered in the primary care setting. Many of these disorders can be successfully managed by primary care physicians, allowing for early detection and prompt treatment. However, there are circumstances when patients require a referral to a foot and ankle specialist to decrease potential complications of these disorders.<sup>1</sup> This article will review ten common foot and ankle disorders to aid in the determination of when conservative management is appropriate and when referral to a specialist is necessary.

## ANKLE SPRAINS

Ankle sprains are one of the most commonly encountered orthopaedic injuries, comprising 15-20% of all sports injuries.<sup>2</sup> Ankle sprains are most commonly due to inversion and adduction of the plantar flexed foot, resulting in injury to the lateral ligamentous complex. Medial sprains are less common, resulting from an eversion and abduction force. The anterior talofibular ligament is the most common ligament injured in a lateral ankle sprain, followed by the posterior talofibular and calcaneofibular ligament. In one-third of cases all three ligaments are injured.<sup>3</sup>

Ankle sprains can be difficult to differentiate from other conditions, including fractures, tendon ruptures and midfoot injuries. Patients may present with bony tenderness to palpation (TTP) if there is an avulsion rather than a mid-substance ligament tear. Ankle stability can be assessed by performing the anterior and posterior drawer tests, the talar tilt test, Kleiger's test and the dorsiflexion torque test.

The anterior and posterior drawer tests are performed by grasping the foot and stabilizing the tibia while applying an anterior or posterior force to the ankle. A positive test is indicated by translation of the ankle joint relative to the uninjured side. The talar tilt test is performed by applying an inversion stress to the ankle in neutral dorsiflexion with the knee flexed to 90°.

Many injuries occur concomitantly with ankle sprains, and for this reason Fallat et al. suggested that these injuries be referred to as "Ankle Sprain Syndrome." Commonly associated injuries include ankle syndesmotomous injuries, Achilles tendonitis, peroneal tendonitis, medial and intermediate dorsal cutaneous neuritis, ankle avulsion fractures, and fifth metatarsal base fractures.<sup>3</sup>

Ankle sprains are classified as grade one through three based on increasing severity of injury and stability of the ankle joint, which serves to guide treatment.<sup>2,4</sup> Low grade sprains with a stable ankle joint are managed using functional bracing: taping, elastic bandage, lace-up or semi-rigid ankle braces, which yield superior outcomes as compared to rigid immobilization. Lace-up braces result in less

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persistent swelling than elastic bandages, but more dermatologic complications. Grade III sprains, those with an unstable ankle joint, may benefit from a short period of immobilization in a short leg cast, removable cast boot, splint, or air-cast, followed by a supervised rehabilitation program. Surgical management, consisting of suture repair or tendon transfers is controversial. There is little evidence supporting routine surgical management of acute ankle sprains, except in cases of chronic recurrent ankle sprains failing non-operative treatment.<sup>3,4</sup>

Like the talofibular and calcaneofibular ligaments, those comprising the ankle syndesmosis can also be sprained or torn as a result of rotational ankle injuries, a so-called “high ankle sprain.” The ankle syndesmosis is comprised of the anterior inferior tibiofibular ligament, the posterior inferior tibiofibular ligament, the inferior transverse ligament, the interosseous membrane and the interosseous ligament. The mechanism of injury is usually external rotation and can be purely ligamentous or associated with a fracture. Clinically, patients will present with pain between the tibia and fibula more proximal to the ankle joint. A positive squeeze test, eliciting pain when the proximal tibia and fibula are squeezed together, can aid in the diagnosis. Radiographic evaluation will be discussed in a later section. Isolated syndesmotic sprains rarely result in ankle instability and can be managed nonoperatively with cast immobilization for 2 to 3 weeks followed by progressive weight bearing in a walking boot.<sup>5</sup>

## ANKLE FRACTURES

Fractures of the malleoli typically occur as a result of rotational forces to a planted foot, most commonly an external rotation force applied about a supinated foot. Two-thirds of ankle fractures are unimalleolar, with bimalleolar and trimalleolar fractures resulting from increasingly higher energy injuries. Radiographs of the ankle

typically include AP, lateral, and mortise views. Additionally, full length x-rays of the tibia and fibula should be obtained due to possible proximal fibula fractures occurring in conjunction with ankle fractures.<sup>6</sup> Particularly in cases of tenderness to palpation over the proximal lateral calf.

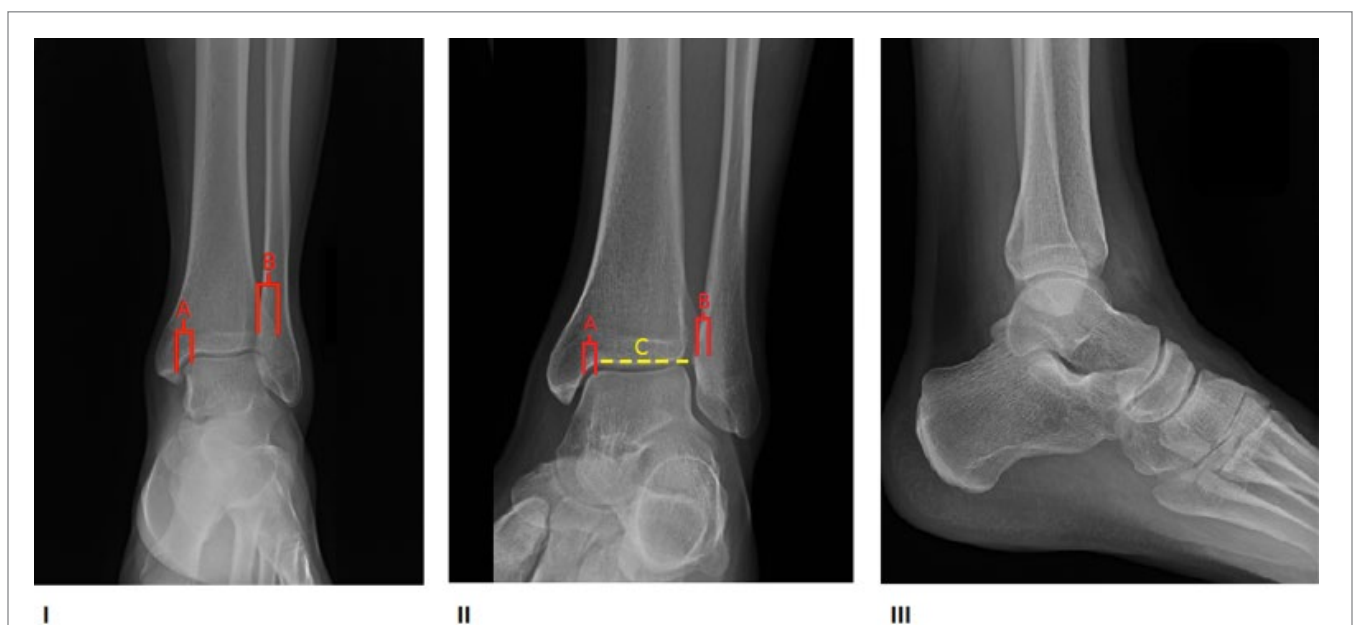
Management of ankle fractures depends on the stability of the injury, which can be determined by a number of radiographic parameters, which are illustrated in Figure 1. The medial clear space (A) between the talus and medial malleolus should equal the tibio-talar joint space (Normal is <6mm) on AP or mortise views.<sup>5</sup> Medial clear space widening is indicative of deltoid ligament injury, seen in bimalleolar and trimalleolar equivalent fractures (injuries with a lateral and posterior malleolar fracture and medial deltoid disruption). Also on AP and mortise x-rays, the tibiofibular overlap (B) should not be less than 10mm, as this is indicative of injury to the tibiofibular syndesmosis.<sup>5</sup>

The location of the lateral malleolus fracture also assists in determining stability. Lateral malleolus fractures at or above the level of the tibial plafond, the distal tibial articular surface (C), are more likely to have associated tibiofibular syndesmotic injury than those below the level of the plafond. Any injury to the medial side of the ankle including medial malleolus fractures, bimalleolar and trimalleolar fractures with deltoid ligament disruption indicates an unstable injury requiring operative management.<sup>5,7</sup>

Dislocation of the ankle will require prompt reduction and immobilization to minimize swelling and soft tissue damage.<sup>7</sup> Isolated lateral malleolus fractures below or at the tibial plafond, including lateral malleolus avulsion fractures, with no medial clear space widening, talar shift or medial tenderness can be managed nonoperatively with an ankle brace and full weight bearing as tolerated. Isolated lateral malleolus fractures at or above the tibial plafond without medial clear space widening and without talar shift can

### FIGURE 1:

AP (I), Mortise (II), and Lateral (III) radiographs of a left ankle. Medial clear space is demonstrated by “A.” Tibiofibular overlap is demonstrated by “B.” The tibial plafond is demonstrated by “C.”



also be managed nonoperatively, with immobilization and non-weight bearing. Isolated medial malleolar fractures, including avulsion fractures, can be treated conservatively with immobilization if there are no radiographic indicators of ankle instability. Additional consideration of stress radiographs may be indicated in suspected unstable injuries. Suspicion of an unstable ankle fracture pattern is an indication to refer to an orthopedic specialist to further evaluate and stress an ankle for instability, as these may require operative intervention. Bimalleolar and trimalleolar fractures, and any fracture-dislocations are unstable injuries requiring operative management, following reduction and splinting. The goal of operative fixation is the restoration of fibular length and the congruity of the tibiotalar joint. Injury to the syndesmosis, which may be found intraoperatively, requires additional fixation; however, the exact technique for syndesmotic fixation, remains controversial.<sup>6,8</sup> Ankle fractures in skeletally immature patients should generally be referred to a pediatric foot and ankle orthopaedic specialist as these injuries frequently involve the physis.

## ANKLE ARTHRITIS

Approximately 1% of the population suffers from ankle osteoarthritis (OA). Compared to the hip and knee, the ankle is rarely subjected to primary OA, with most cases being post-traumatic. Brown et al found that 79.5% of patients with ankle OA had a history of at least one joint injury, versus 1.6% and 9.8% in hip and knee OA, respectively. Ankle fractures have been shown to be the primary cause of ankle post-traumatic osteoarthritis (PTOA), followed by ligamentous ankle injuries.<sup>9,10</sup>

There are two underlying mechanisms involved in PTOA of the ankle, direct damage to the chondral surfaces from the initial injury, and the resultant chronic abnormal overloading of the joint secondary to incongruity of the articular surfaces and instability. Risk factors for PTOA include lateral malleolus fractures above the tibial plafond, medial malleolus fracture, dislocation, increasing BMI, age, time since surgery and severity of chondral damage at the time of injury.<sup>10,11</sup>

Patients will present with progressively worsening deep ankle pain. Early on, pain occurs during the day with activities that load the ankle joint, whereas advanced ankle OA will cause pain at rest and at night. With mid-stage and end-stage disease, significant lower extremity muscle atrophy can be seen. Reduced ankle ROM is the earliest physical exam finding, which can be attributed to many factors including joint incongruity, cartilage loss, soft tissue contractures or muscle spasm, along with osteophytes or loose bodies. Over time, crepitus, joint effusion, joint line tenderness and hindfoot instability can develop.<sup>10,11</sup>

Weight loss, activity modification and restoration of articular surfaces in ankle fractures are key to prevention of PTOA. Conservative treatment options include NSAIDs, orthotics, and physical therapy. Viscosupplementation with hyaluronic acid is controversial and has limited evidence supporting its efficacy in ankle OA. Steroids are marginally more effective and longer lasting than NSAIDs, which are often first-line treatment of arthritis. Orthotics and bracing can be used to help unload the affected side of the ankle and provide stability, but their effectiveness is limited by patient compliance. Finally, physical therapy can play a role in prevention of OA progression, as well as preserving independent ambulation.<sup>10-12</sup>

Surgical treatment varies in scope and effectiveness. Osteophytes, loose bodies, and chondral defects can be treated with arthroscopy. Osteotomies can be performed in early OA to correct bony deformities. Arthrodesis in neutral dorsiflexion with roughly 5 degrees of external rotation is reserved for end-stage ankle OA to allow for near normal gait and pain relief. Total ankle arthroplasty is an emerging option for severe ankle OA, resulting in improved pain relief, gait and patient satisfaction, but potentially has a higher reoperation rate when compared to arthrodesis.<sup>13-15</sup>

## ACHILLES TENDON INJURIES

Despite being the largest and strongest tendon in the body, the Achilles tendon is the most commonly injured tendon in the lower extremity. Achilles tendon disorders exist along a spectrum ranging from tendinosis to acute tendon rupture. Achilles tendinosis is a non-inflammatory degeneration secondary to repetitive micro-trauma and aging. Patients are often asymptomatic, however some patients with partial ruptures of the tendon can experience focal tenderness over the area of rupture. Areas of tendinosis can be visualized as hypoechoic lesions on ultrasound or as areas of altered signal on MRI. Treatment of Achilles tendinosis is largely conservative, consisting of rest, anti-inflammatories, heel lifts and activity modifications. Those with severe pain may respond well to a period of immobilization followed by physical therapy consisting of eccentric heel stretching and calf strengthening exercises.<sup>16,17</sup>

Achilles tendon ruptures occur most commonly in males in their fourth or fifth decade. Sixty-eight percent are sports-related, resulting from an eccentric contraction of the triceps surae, especially in episodic athletes ("weekend warriors"). Approximately 75% of ruptures occur 2-6cm from the calcaneal insertion due to a vascular watershed area here. Other common sites of rupture include the distal insertion (10-20%) and the musculotendinous junction (5-15%).<sup>17,18</sup>

Corticosteroids, fluoroquinolone antibiotics, and chronic tendinopathy such as from a Haglund's deformity, an enlargement of the posterosuperior calcaneal tuberosity, are also associated with Achilles tendon ruptures. Patients will describe feeling a "pop" or a sensation of being hit in the back of the leg following resisted plantar flexion. Patients may also complain of inability to bear weight and plantar flexion weakness, but often will not complain of pain after the acute episode.<sup>17,18</sup>

Diagnosis of acute ruptures requires at least two physical exam findings: a positive Thompson Test, decreased plantar flexion strength, decreased resting plantarflexion of the ankle, a palpable defect over the tendon, or increased passive dorsiflexion. Intact plantar flexion does not rule out an Achilles rupture, as the tibialis posterior, long flexor tendons of the hallux and toes and plantaris muscles can still weakly plantarflex the foot. Ultrasound, radiography, and MRI are not necessary to confirm the diagnosis, but can be beneficial in the management of chronic ruptures, and to differentiate partial and complete tears.<sup>17</sup>

Patients should be immobilized in a posterior splint in plantar flexion, until a definitive treatment course is determined. Recent studies has shown when nonoperative management includes an early functional rehab program, re-rupture rates and plantar flexion strength appear to be equivocal between operative and non-



operative management. Operative management does however carry surgical risks including infection, adhesions, nerve injury, and wound problems, therefore one must carefully consider pursuing operative management in patients at risk for poor wound healing.<sup>18,19</sup>

## PERONEAL TENDON INJURIES

Peroneal tendon injuries are often overlooked as a source of ankle pain, and commonly misdiagnosed as ankle sprains. The peroneus brevis and longus tendons pass posterior to the lateral malleolus within the retromalleolar sulcus, beneath the inferior and superior peroneal retinaculum (SPR) to insert at varying sites on the foot. The peroneal tendons are subject to two types of acute injuries, tendon subluxation and tendon tears. Peroneal tendon subluxation occurs with a tear or defect in the SPR, allowing the tendons to subluxate out of the retromalleolar sulcus.<sup>20,21</sup>

Following a dorsiflexion or inversion injury, patients will complain of a “popping” sensation, lateral ankle pain, and possible instability. Patients will have tenderness and swelling behind the lateral malleolus and along the lateral calcaneus, weakness and pain with resisted eversion, and pain with passive inversion stretching. Also, with resisted eversion and active dorsiflexion, patients may feel apprehension or physical subluxation of the peroneal tendons.<sup>21,22</sup>

Standard foot and ankle radiographs should be obtained to rule out fracture or other osseous abnormalities. Ultrasound can visualize dynamic subluxation of the tendons, as well as tears and fluid collections associated with peroneal tendonitis.<sup>23</sup> MRI can be used as an adjunct to evaluate for variations in normal anatomy of the peroneal muscles and tendons, as well as determination of whether tendon tears are isolated or combined injuries.<sup>20</sup>

Treatment of peroneal tendon subluxation depends on the acuity of the injury. Acute subluxations can be treated nonoperatively with a short leg cast in a plantar flexion and inversion for approximately 6 weeks, after reduction of the tendons. With chronic subluxation (greater than 4 weeks), nonoperative treatment has a poor success rate. Chronic subluxations have multiple surgical options, including direct SPR repair, retromalleolar groove deepening, soft tissue transfers, and fibular osteotomies. Operative management is indicated in athletes wishing a rapid return to sport, with direct SPR repair the most common surgical treatment.<sup>20,22</sup>

Isolated peroneus brevis tears can be treated conservatively with NSAIDs, cast or walking boot, and activity modification, however the success rate is low and surgical management should be strongly considered. Isolated peroneus longus tears are rare, occurring in patients with predisposing conditions including diabetes, hyperparathyroidism, rheumatoid arthritis, and psoriasis.<sup>20</sup> Patients with full function and minimal symptoms can be managed non-operatively, while those with more severe or recalcitrant symptoms can be managed operatively, similar to isolated peroneus brevis tears. With concomitant tears of both tendons, surgical management is indicated, ranging from tenodesis to repair, depending on the condition of each tendon. Most patients return to full activity following operative treatment.<sup>21</sup>

Peroneal tendinopathy is a more chronic degenerative condition affecting the peroneal tendons, resulting from repetitive micro-trauma in patients such as runners and dancers, or patients with

chronic ankle sprains or high arch feet. Patients present similarly to those with tendon tears, with pain and tenderness along the length of the peroneal tendons. Ultrasound and MRI can be effective in visualizing fluid surrounding the tendon sheath as well as tendon thickening. Treatment is generally conservative, with NSAIDs, cast or walking boot and activity modification. If patients fail 3-6 months of conservative therapy, operative tendon debridement and tenosynovectomy can be effective.<sup>22</sup>

## POSTERIOR TIBIALIS TENDON DYSFUNCTION

The tibialis posterior is the most important dynamic stabilizer of the arch of the foot. Contraction causes elevation of the medial longitudinal arch, locking the midfoot and hindfoot, thus increasing rigidity during gait. More commonly, laxity of the hind and midfoot results in gradual degeneration of the subtalar, calcaneocuboid, talonavicular, and tibiotalar joints. Posterior tibialis tendon dysfunction (PTTD) is the most common cause of acquired flat foot deformity in adults, occurring most commonly in middle-aged, obese women. Other risk factors include hypertension, diabetes, local steroid injection and seronegative arthropathies, such as ankylosing spondylitis and psoriatic arthritis.<sup>25</sup> The pathophysiology of PTTD is a tendinosis resulting from a tenuous blood supply to the tendon as it passes posterior to the medial malleolus in addition to chronic stresses. Direct rupture of the tendon is rare.

Swelling and TTP along the tendon may be noted posterior to the medial malleolus. In PTTD, hindfoot valgus and forefoot abduction results in flattening of the medial longitudinal arch leading to the “too many toes sign” as compared to the normal.<sup>25</sup> The ability to perform a single-leg heel rise is the most commonly used functional test in PTTD; patients are unable to perform the normal 5-10 repetitions of a single-leg heel rise. Alternatively patients can be asked to walk a short distance on their toes. Imaging in early stages should be limited to standard ankle and foot radiographs to rule out other causes of ankle pain. In later stages, radiographs and CT scan can reveal arthrosis in the hind and midfoot joints. MRI is useful for evaluating abnormal pathology along the tendon course.<sup>25</sup>

Conservative treatment of early PTTD consists of weight bearing in a cast or fracture boot for 4-weeks, followed by arch support and medial heel wedge orthotics. Physical therapy involves strengthening the tibialis posterior. Initial surgical management may entail tendon transfers and osteotomies that can be used to correct flat foot deformities failing conservative management. At this stage preserving remaining motion is still possible. In late stages, involving the subtalar, calcaneocuboid, and talonavicular joints, a triple arthrodesis can be considered to correct deformity and relieve pain, with subsequent loss of motion. Finally, with advanced disease involving the tibiotalar joint, a pan-talar arthrodesis can be performed as a salvage procedure.<sup>25</sup>

## METATARSAL FRACTURES

Metatarsal fractures comprise approximately five to six percent of all fractures in the primary care setting.<sup>26</sup> In all cases suspicious for fracture, standard AP, lateral and oblique radiographs of the foot should be obtained (*Figure 2, page 16*).

**FIGURE 2:**

AP (I), Lateral (II), and Oblique (III) radiographs of a left foot.



Metatarsal shaft fractures result from twisting injuries or direct blows. These fractures are generally stabilized by adjacent metatarsals, and the majority can be treated nonoperatively with reduction and immobilization using a compressive wrap, posterior splint or stiff-soled shoe with weight-bearing as tolerated. Unstable fractures will require operative fixation. Metatarsal base fractures can be categorized into first through fourth metatarsal base fractures and fifth metatarsal base fractures.<sup>26</sup>

Fractures of the base of the first through fourth metatarsals are usually caused by crush injuries, direct blows or axial loads to a plantarflexed foot. These fractures rarely occur in isolation as adjacent structural ligaments may also be damaged and frequently require surgical intervention.<sup>26</sup> Of note, special attention should be paid to fractures at the base of the second metatarsal as these can be fractured in LisFranc joint injuries, a disruption of the articulation of between the base of the second metatarsal and the medial cuneiform. One must have a high suspicion for LisFranc injuries in any patient presenting with forefoot pain following a hyper-plantarflexion injury, as these injuries are highly unstable and routinely require operative management. Weight bearing foot radiographs will aid the primary care physician in making this diagnosis.

Fifth metatarsal base fractures are more common, and result from inversion injuries to the ankle, often being misdiagnosed as ankle sprains. These fractures are grouped into zones, depending on location, as illustrated in figure 3 (Figure 3). There is typically a high level of confusion in regards to these injuries with the main differentiation between a Zone 1 and 2 injury. Zone 1 (pseudo Jones fractures) or tuberosity fractures can be treated with protected weight bearing in a soft dressing or hard-soled shoe, or a splint initially if the patient has significant pain.<sup>27</sup> Zone 2 or metaphyseal-diaphyseal junction fractures occur at the fourth-fifth metatarsal articulation, and are commonly referred to as “Jones Fractures.” This zone coincides with the avascular watershed area, increasing the risk of nonunion by disrupting the already tenuous vascular supply. Zone 3 or diaphyseal stress fractures occur distal to the fourth-fifth metatarsal articulation. Zone 2 and 3 fractures may be treated with 6-8 weeks of nonweightbearing in a short leg cast.<sup>27</sup>

In higher level athletes or individuals consideration for surgical intervention of Zone 2 and 3 injuries should be discussed. It is important to differentiate Zone 1 fractures in adolescents from Iselin’s disease, traction apophysitis of the base of the fifth metatarsal, which is the result of repetitive traction by the lateral plantar aponeurosis and can be treated with rest, ice and activity modification.

Metatarsal stress fractures also require special consideration. These injuries occur most commonly in athletes with an acute increase in activity level, or by chronic repetitive overloading. Additionally they can occur in the cohort of patients with osteopenia or osteoporosis with a relatively minor history of trauma. Radiographs are often negative, in which case MRI or bone scan can be useful to confirm diagnosis. Often the only presenting complaint is difficulty with weight bearing and dorsal foot swelling. Stress fractures with no sclerosis at the fracture site are treated nonoperatively with protected weightbearing precautions in a cast or cast boot for 6-8 weeks. When sclerosis is present, operative management is required, consisting of curettage, bone grafting and fixation, followed by six weeks of non-weightbearing.<sup>28</sup> The navicular and cuboid tarsal bones are also subject to stress fractures. These injuries also commonly result from overuse injuries in athletes. These injuries can be treated successfully in most cases with non-weightbearing and cast immobilization.

## HALLUX VALGUS

Hallux valgus is the most common cause of forefoot pain in adults. It is a chronic condition characterized by the progressive lateral deviation of the hallux and medial deviation of the first metatarsal, leading to the subluxation of the first metatarsophalangeal (MTP) joint.<sup>30</sup> Predisposing factors to hallux valgus are divided into intrinsic and extrinsic factors. Intrinsic factors include genetic predisposition, female gender, ligamentous laxity, osteoarthritis and rheumatoid arthritis, foot deformities such as pes planus, increased age and neuromuscular disorders such as cerebral palsy. Extrinsic factors include high heeled or narrow toe box shoes, ballet dancing, trauma and excessive weight bearing.<sup>29,31,32</sup>

**FIGURE 3:**

Oblique radiographs of a left foot. Zone I demonstrated by "A", Zone II is demonstrated by "B", Zone III is demonstrated by "C."



Any factor compromising the medial structures of the first MTP joint can initiate the progression of hallux valgus. When the medial collateral ligament becomes attenuated, the metatarsal head deviates medially and the proximal phalanx is pulled into valgus. This deformity can induce a bursitis over the medial eminence (bunion), and the extensor and flexor hallucis longus tendons bowstring laterally, exaggerating the deformity. Patients will present with pain and varying degrees of deformity at the first MTP joint.<sup>31</sup> Special attention should be paid to the wear patterns of patients' footwear as this can be a large contributing factor to the patient's symptoms.<sup>30</sup> Patients will frequently present early on with callus formation of the first MTP.

Conservative management should be initiated in all patients and particularly is the first-line for pediatric hallux valgus, elderly patients, patients with neuropathy, and other poor surgical candidates. As there is no evidence that conservative treatment can correct deformity, the goal of conservative management is to relieve symptoms while avoiding lifestyle modifications. Avoiding narrow toe box shoes, over the counter cushions and pads, anti-inflammatories and possibly steroid injections can all be attempted. Orthotics have not been shown effective in slowing the progression of hallux valgus.<sup>30</sup> Additionally hallux valgus should not be treated with surgical intervention prophylactically or for cosmetic purposes. Indications for surgery are pain and functional limitations not responding to shoe wear or activity modifications.

### INTERDIGITAL (MORTON'S) NEUROMA

Interdigital neuroma of the foot is another common cause of adult forefoot pain, occurring primarily in middle-aged women. The most common location is within the third webspace, between the third and fourth metatarsal heads, affecting the third common digital nerve – commonly referred to as a Morton's neuroma. Possible causes include narrow toe box shoes and the inherent anatomy of the third interdigital space predisposing to neuroma formation. Regardless of the cause, the third common digital nerve becomes compressed, leading to a fusiform swelling of the nerve.<sup>32,33</sup>

Patients complain of burning pain between the metatarsal heads radiating to the third and fourth toes, particularly with narrow toe box shoe wear. There is tenderness on the foot plantar surface with standing and walking along with paresthesias, which are reproducible with palpation. A positive Mulder's Sign, eliciting a palpable click in the affected interspace with reproduction of the patient's symptoms when the metatarsal heads are squeezed together, is the most sensitive diagnostic tool. Radiographs can be obtained to rule out osseous conditions, while MRI and ultrasound can be used as an adjunct for diagnosis.<sup>32,33</sup>

Initial management consists of changing to wider toe box shoes, activity modification, NSAIDs and possible steroid injections. Surgical treatment is indicated in those cases failing conservative management. Open or endoscopic release of the transverse intermetatarsal ligament can be performed to remove the mechanical irritation of the common digital nerve. Alternatively, the neuroma and nerve itself can be excised.<sup>33</sup>

### PLANTAR FASCIITIS

Plantar fasciitis (PF) is the most common cause of plantar heel pain in adults. It occurs primarily in patients in their 40s and 60s, and with a bimodal distribution in younger patients who are runners and older patients who are relatively sedentary.<sup>34</sup> It is usually unilateral, occurring bilaterally in about one third of cases. The plantar fascia serves as one of the static stabilizers of the longitudinal arch of the foot. PF occurs as a result of repetitive microtrauma and excessive strain to the plantar fascia. Risk factors for the development of PF include Achilles tendon or triceps surae tightness, obesity, chronic weight bearing professions, increased age, poor footwear, over training in athletes, and decreased mobility of the subtalar joint.<sup>34,35</sup>

Patients present with pain that is worst with their first steps in the morning, or after a prolonged period of rest. On exam there will be tenderness at the medial plantar calcaneal tuberosity and along the length of the fascia. Maneuvers that stretch the plantar fascia, including passive dorsiflexion can also elicit pain. The Silfverskiöld test can be used to differentiate between Achilles tendon contracture and gastrocnemius muscles tightness, both of which tension the plantar fascia.<sup>34</sup> To perform the Silfverskiöld test, the patient's ankle dorsiflexion is measured first with the knee extended then flexed to 90°. Decreased dorsiflexion in extension which increases with knee flexion indicates a tight gastrocnemius. If knee flexion and extension does not affect dorsiflexion, the Achilles contracture is the likely cause. The diagnosis of PF is usually clinical, however standard weight bearing radiographs of the foot should be obtained to rule out skeletal causes of heel pain.

Activity modification, rest, stretching, structured physical therapy programs, heel cushions, orthotics, NSAIDs and weight loss are all components of successful conservative management. Night splints are used to help prevent the contracture of the plantar fascia and the triceps surae.<sup>34</sup> Studies have shown steroid injections and platelet rich plasma (PRP) injections into the plantar fascia as equally efficacious, however given the potential risk of fascia atrophy and rupture, PRP is technically a safer alternative, albeit substantially more expensive, requires specialized equipment and typically is not reimbursed through insurance.<sup>36</sup>

Surgical management of PF is usually reserved for recalcitrant cases. Plantar fasciectomy, both open and endoscopic, has been shown to provide relief of symptoms in about two thirds of patients. Open plantar fascia release has the added benefit of releasing the first branch of the lateral plantar nerve, further reducing pain.<sup>35,37</sup> Gastrocnemius recession may also be an option for treatment. Recalcitrant patients should be referred to a foot and ankle specialist to evaluate for additional causes and possible surgical intervention.

## CONCLUSION

The ten foot and ankle conditions reviewed in this article are but a small fraction of the myriad of musculoskeletal complaints commonly encountered in the primary care setting. With a better understanding of the disorders and their initial evaluation and management, indications for referral to a musculoskeletal specialists and the need for operative management, primary care physicians can better aid in the diagnosis, care and recovery of their patients afflicted by these common disorders.

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## REVIEW ARTICLE

# Empathy & Its Role in Quality Care

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## Keywords:

Empathy

Quality

Patient Satisfaction

Patient Care

Compliance

Disease Prevention

Wellness

Empathy is widely viewed as essential to every patient encounter. There is evidence that empathy in the patient-physician encounter improves increases diagnostic accuracy, compliance, and patient outcomes. Patient satisfaction is higher when physicians demonstrate empathy. Empathy also reduces malpractice risk exposure. Empathy is a learnable skill at any point in a physician's career that can significantly improve patient care.

## INTRODUCTION

Empathy has become the subject of much discussion in the lay press as well as the medical literature. In April 2015, U.S. News and World Report published an article entitled "Why nice doctors are the best doctors," and stresses empathy as a key component to patient care and satisfaction.<sup>1</sup> An article in Harvard Business Review called for an "epidemic of empathy" in order to improve patient care. The author argues that healthcare has become too complicated, reducing the patient to feeling that they are "being treated like disease and organs rather than human beings."<sup>2</sup>

The need for empathy is also recognized by health organizations worldwide. The World Health Organization issued a report in 2008 calling for an increased empathy in health care delivery.<sup>3</sup> The American College of Obstetricians and Gynecologists has issued an opinion paper in 2011 and reaffirmed in 2014 entitled Empathy in Women's Health Care.<sup>4</sup> Both organizations state that patient centered, empathetic care is necessary to increase communication and empower patients.

The patient-physician relationship is complex. The nuances of the interaction are subtle and go far beyond the words that are spoken. Empathy is an important learnable skill that greatly enhances the patient-physician relationship. But what is empathy? Can we measure it? Does it affect patient outcomes? Does empathy reduce risk exposure in healthcare? Can it be learned?

A sentinel article written by Mercer, which is widely used in empathy research, defined empathy as the ability to understand the patient situation, communicate that understanding, and check its accuracy.<sup>5</sup> This definition appears straight forward, but there are numerous steps that must occur in order to fulfill it. The physician must gather large amounts of information while building an atmosphere of trust. Physicians should allow patient adequate time to tell their story, and when necessary, encouraging them to provide additional information. The physician must also be able to convey the desire to help the patient with their concern.

Empathy involves active listening. In other words, the clinician must listen to the patient's words and monitor their nonverbal expressions. Listening to the patient's words provides cognitive clues. Watching the patient's facial expression, body language, and integrating the tone with which the words are spoken provides the emotions and details the words lack. Active listening also requires the clinician to respond in such a way that the patient feels understood. In other words, the body language, tone, and words of the clinician must reflect that the patients has been not only heard, but also understood. The words draw the outline, but the body language and tone provide the color to complete the picture.

Empathy also involves empowering the patient. For the patient to be empowered, the physician must take time to help the patient understand the diagnosis and treatment plan. This may come in the form of educating the patient on the diagnosis, discussing treatment options, or enabling the patient to cope better with their diagnosis. It may take the form of encouraging the patient to make lifestyle changes, making decisions about or understanding the use of medications.<sup>5</sup> In short, the physician creates an environment that allows the patient to feel comfortable in asking questions and engages them in their own care.

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## CAN EMPATHY BE MEASURED?

In 2001, Blasi et al undertook an extensive literature review to evaluate evidence that there is a therapeutic relationship between physician and patient. Unfortunately, there was little empirical evidence to support the clinical benefit of the physician-patient relationship. Since then instruments have been developed to evaluate empathy and subsequent research into its value in patient care.<sup>6</sup>

The two instruments commonly used to evaluate physician empathy are the Jefferson Scale of Empathy (JSE)<sup>7</sup> and the Consultation and Relational Empathy (CARE) measure.<sup>8</sup> Both use Likert formats that enable the participant to choose the strength of their responses (i.e. always, sometimes, never) consistently on a series of statements. JSE is completed by physicians to rate themselves on 20 items on a seven-point Likert-type scale. CARE is a 10 item five-point Likert-type scale that is completed by patients rating the physician after an encounter. Both instruments include aspects that define empathy such as listening, showing compassion, and understanding the patients' concerns.

## DOES EMPATHY AFFECT PATIENT OUTCOMES?

With the development of validated instruments, empathy has been measured and correlated favorably with a variety of patient outcomes.

The common cold accounts for numerous office visits seasonally. In a study published in 2009 the interaction between the physician and the patients were evaluated for length of illness, severity of symptoms, and provider empathy. They also had nasal swabs to measure the immune cytokine interleukin-8 (IL-8) at baseline and 48 hours. The patients that rated their provider with "perfect" CARE scores (50 points out of 50 possible), had higher rise in IL-8 and trended toward less severity scores. The patients also had shorter duration of illness than those who scored the providers with less than perfect scores.<sup>9</sup>

Diabetes management is difficult for both patient and physician. There are two studies that evaluated the role of empathy in diabetic patients. The first was a study that evaluated long-term glucose control and LDL control. The physicians were surveyed with the Jefferson Scale of Empathy. The study found that patients that reached glucose control (A1c <7%) and LDL goals (<100) were cared for by physicians with higher empathy scores than those that failed to reach their goals. The authors concluded that more empathy enhances mutual understanding and trust, which in turn leads to better alignment between patient needs and treatment plan.<sup>10</sup>

A second study reviewed the charts of 20,961 patients for metabolic complications and compared them to the 242 treating physicians using the JSE measure. There were significantly fewer hospital admissions for diabetic ketoacidosis, coma, and hyperosmolar state for the physicians with empathy scores in the top third. These results were independent of the patient age, gender, and the length of time the patient had been cared for by the physician. The authors concluded that empathy had a significant correlation in clinical outcomes and should be considered an important component of clinical competence.<sup>11</sup>

Trauma patients were engaged in a study to rate their surgeons' empathy. The CARE measure was administered six weeks and one year after their discharge. Those patients that rated their physicians as having high empathy scores (41-50 points) were more likely to perceive their treatment and outcome as successful. The study also found that the empathy scores were independent of age, gender, and severity of initial injury. The authors concluded that "the interpersonal treatment aspects such as emotional care are associated to a more positive valuation of the medical treatment and its effects."<sup>12,13</sup>

Migraine management includes life style modifications, trigger avoidance, and medications. Patients that rated their physicians higher in empathy were more likely to be compliant with diet, exercise, destressing, sleep pattern modifications, and medications. A decrease in migraine symptoms and disability was also noted.<sup>14</sup>

The patient-physician interaction can produce anxiety in many patients. Surgical patients reported less preoperative anxiety when they perceived an empathetic attitude from the anesthesiologist. Patients also felt that the information provided was higher quality and their overall satisfaction was higher for those physicians that responded more empathetically.<sup>15</sup>

The authors of each study concluded that the positive relationship between the patient and the physician was a significant factor in patient outcomes. Some authors hypothesized that physicians with higher empathy scores had a better understanding of the patients' individual situation. Therefore, the authors concluded the patient plan facilitated improved compliance.<sup>10,11</sup> Increased diagnostic accuracy can result from physicians obtaining more clinically relevant information. It is also reasonable to conclude that empathetic engagement enhances mutual understanding leading to increased trust and communication between patient and provider.<sup>11</sup> Other authors concluded that providing the patient with better understanding of the diagnosis framed patient expectations and improved outcomes.<sup>9</sup>

## DOES EMPATHY REDUCE YOUR RISK IN PATIENT CARE?

A retrospective study published in JAMA determined that the majority of patient complaints were made against a minority of providers. Furthermore, those complaints resulted in the highest number of open risk management files, risk management expenditures, and lawsuits. The authors noted that the risk was not related to complexity of illness or physician technical skills. Rather it was noted that risk was related to patient "dissatisfaction with their physicians with ability to establish rapport, provide access, administer care and treatment consistent with expectations, and communicate effectively."<sup>15</sup>

Empathy takes time and time is money. Or is it? In 1997, a study was published that compared communication behaviors of physicians that had had no malpractice claims versus those that had had two or more lifetime claims. Ten routine office visits were recorded and reviewed for empathetic behaviors such as soliciting patients' opinions, checking understanding, and encouraging patients to talk. Out of 59 family physicians and internists, those with

no claims spent a total of 18.3 minutes compared to 15 minutes for those with two or more claims.<sup>16</sup> The small amount of additional time spent engaged in effective communications could be viewed as an investment in reducing the liability risk in patient care.

## CAN EMPATHY BE TAUGHT?

Empathy has been recognized as an essential element in healthcare. It is required within the undergraduate and postgraduate course curriculums of all training institutions. Healthcare organizations also recognize the importance of empathy. The Cleveland Clinic sponsors an annual seminar entitled Patient Experience: Empathy and Innovation Summit which is attended by profession within the healthcare delivery system.<sup>17</sup> There are also online resources such as [www.vitaltalk.org](http://www.vitaltalk.org) and [www.empathetics.com](http://www.empathetics.com) which can be accessed by individuals and organizations for empathy training. Regardless of the methods of delivery, there is compelling evidence that each can be effective in improving communication skills and empathy scores.

For example a study published in 2011 randomized oncologists to either receive a lecture (the control group) or a lecture and one hour interactive CD-ROM (the intervention group). The oncologists were then videotaped during patient visits. The tapes were scored by trained personnel listening for statements of empathy. There was a statistically significant increase of empathetic statements in the intervention group over the control group. The patients were surveyed one week later regarding trust and quality of communication. The patients' also perceived greater empathy and trust in the intervention group of physicians.<sup>19</sup>

Massachusetts General Hospital developed E.M.P.A.T.H.Y. as an acronym used in teaching empathy. It stands for Eye contact, Muscles of facial expression, Posture, Affect, Tone of voice, Hearing the whole patient, and Your response. It was studied by randomizing 99 residents and fellows to receive either the standard residency or fellowships empathy training or three one-hour modules. Patients were surveyed using the CARE measurement before the physicians underwent training and after the training modules. Those physicians that received the intervention showed statistically significant improvement on patient ratings of physician empathy.<sup>20</sup> The results were published in 2012. Since then the modules have been taught nationally and internationally as a simple way for health professionals to perceive and respond to nonverbal emotional cues.<sup>21</sup>

## WHAT ARE SOME PHRASES THAT BUILD EMPATHY?

Empathy involves gaining understanding and communicating that understanding back to the patient. A physician may need to ask for more information or clarify information, then respond so that the patient confirms that the information is correct. Table 1 has some words and phrases that facilitate empathy.<sup>22</sup>

TABLE 1:

<b>Queries</b>
“Would you tell me a little more about that?”
“What has that been like for you?”
“Is there anything else?”
“Are you okay with that?”
<b>Clarifications</b>
“Let me see if I have this right..”
“I want to make sure I really understand what you’re telling me.”
“I don’t want to go further until I’m sure I’ve gotten this right.”
<b>Responses</b>
“That sounds very difficult.”
“Any one in your situation would feel that way.”
“I can see that you are . . .”
“I can imagine that this might feel. . .”

## CONCLUSION

Empathy is the ability to step into another’s shoes and understand their perspective and emotion. In healthcare, the display of empathy in the patient-provider relationship has been shown to improve patient outcomes in diabetes, migraines, trauma, and the common cold. It has also been shown to reduce patient anxiety. Improved perception of empathy reduces patient complaints and risk of litigation. The corollary is that it also increases patient satisfaction. Empathy can be learned and have long-term and persistent effects.

Empathic behaviors and phrases are taught in multiple settings and formats. These are available to physicians in all levels of training and have shown to improve patient care and perceptions. Empathy takes a small amount of time but is cost-effective because it puts the patient at the center of care.<sup>23</sup>



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## FINANCIAL DISCLOSURES: NONE

## REVIEW ARTICLE

# Etiology, Evaluation, & Osteopathic Management of Adult Constipation

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## Keywords:

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Intestinal Microflora

Gastrointestinal Tract

Osteopathic Manipulative Medicine

Gastroenterology

Constipation in adults is a common complaint seen in family practice that can broadly be defined as infrequent stools and/or difficult stool passage. Constipation can be classified as primary (functional) constipation or secondary constipation. Primary constipation can further be differentiated as normal transit, slow transit, or outlet obstruction. Secondary constipation may be due to medications, mechanical issues, metabolic disturbances, neurological causes, or myopathies. The autonomic nervous system segmentally innervates the gastrointestinal tract and plays an important role in colonic muscle tone, contractility, and mucous secretion. Intestinal motility is one of multiple factors that can influence the intestinal microflora, which research has shown to be altered in those with constipation. Evaluation of constipation involves gathering a thorough history regarding the patient's definition of constipation, medication use, and any symptoms indicative of organic disease. Physical examination should include examination of the abdomen, perineum, and rectum. Osteopathic structural examination should focus on assessing regions of sympathetic and parasympathetic influence as well as identifying any sacral or pelvic restrictions. If the history and physical exam reveals any symptoms or signs of organic disease, further work-up is warranted. Initial therapy for patients with primary constipation is lifestyle modifications such as exercise, high fiber diet, and increasing water intake. If this is not effective, pharmacological agents can be used such as osmotic laxatives and bulking agents. Additionally, osteopathic manipulative treatment (OMT) techniques, such as rib raising, suboccipital release, sacral rocking, abdominal lifts, and abdominal and pelvic diaphragm release may improve symptoms and disease severity.

## DEFINITION OF CONSTIPATION

Functional constipation can be defined using the most recent Rome III criteria. The criteria must be fulfilled for the last 3 months with symptom onset at least 6 months prior to diagnosis (*Table 1*). In order to be diagnosed with functional constipation, a patient cannot meet the criteria for irritable bowel syndrome (IBS) (*Table 2*). Although the Rome criteria provide a standardized diagnostic criteria, it is argued that it cannot feasibly be used in practice as most patients with constipation do not meet the criteria. The American College of Gastroenterology Chronic Constipation Task Force recommends using the following broader definition of chronic constipation: "unsatisfactory defecation characterized by infrequent stools, difficult stool passage or both."<sup>2</sup> Difficult stool passage includes straining, a sense of difficulty passing stool, incomplete evacuation, hard/lumpy stools, prolonged time to stool, or need for manual maneuvers to pass stool. In order to be defined as chronic, symptoms must be present for at least three of the previous 12 months.

## ETIOLOGY

There are both primary and secondary causes of constipation. Primary, or functional, constipation can be classified as normal transit, slow transit constipation, or obstructive defecation. Normal transit constipation is the most common form and may be due to perceived difficulty with passing hard stools. It will typically respond to increased dietary fiber or an osmotic laxative. Slow transit constipation results from decreased or uncoordinated motor activity in the colon leading to hard, small feces.<sup>6</sup> Obstructive defecation may be due to insufficient rectal forces or inadequate anal relaxation. Obstructive defecation can also be attributed to paradoxical contraction of the puborectalis and external anal sphincter during defecation.<sup>6,7</sup> Patients with defecatory disorder will typically present with prolonged straining, difficulty passing soft stools, and rectal discomfort.

Secondary causes for constipation are numerous and deserve the necessary work-up if clinically warranted. Secondary constipation may be due to medications, myopathies, mechanical, metabolic, neurological, and psychological causes (*Table 3*).<sup>8,9,10</sup>

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TABLE 1:

Rome III Criteria for Functional Constipation<sup>1</sup>

<p><b>Must include 2 or more of the following:</b></p> <ul style="list-style-type: none"> <li>• Straining during at least 25% of defecations</li> <li>• Lumpy or hard stools in at least 25% of defecations</li> <li>• Sensation of incomplete evacuation for at least 25% of defecations</li> <li>• Sensation of anorectal obstruction/blockage for at least 25% of defecations</li> <li>• Manual maneuvers to facilitate at least 25% of defecations (e.g. digital evacuation, support of the pelvic floor)</li> <li>• Fewer than 3 defecations per week</li> </ul>
<p><b>Loose stools are rarely present without the use of laxatives</b></p>
<p><b>There are insufficient criteria for IBS</b></p>

TABLE 2:

Rome III Criteria for Irritable Bowel Syndrome (IBS)<sup>1</sup>

<p><b>Recurrent abdominal pain or discomfort at least 3 days per month in the last 3 months associated with 2 or more of the following:</b></p> <ul style="list-style-type: none"> <li>• Improvement with defecation</li> <li>• Onset associated with a change in frequency of stool</li> <li>• Onset associated with a change in form (appearance) of stool</li> </ul>
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TABLE 3:

Secondary Causes of Constipation<sup>8,9,10</sup>

Medications	Mechanical	Metabolic	Neurological	Myopathies	Psychological
<ul style="list-style-type: none"> <li>• Anticholinergics</li> <li>• Diuretics</li> <li>• Beta blockers</li> <li>• Opioids</li> <li>• Antacids, especially with calcium</li> <li>• Iron supplements</li> <li>• Calcium channel blockers</li> <li>• Antidepressants</li> <li>• Acetaminophen</li> <li>• Aspirin</li> <li>• NSAIDs</li> </ul>	<ul style="list-style-type: none"> <li>• Anal fissures</li> <li>• Colon cancer</li> <li>• Stricture</li> <li>• Rectocele</li> <li>• Sigmoidocele</li> <li>• Enterocele</li> <li>• Rectal prolapse</li> <li>• Megacolon</li> </ul>	<ul style="list-style-type: none"> <li>• Electrolyte disturbances (<i>hypercalcemia, hypokalemia, hypomagnesemia</i>)</li> <li>• Diabetes mellitus</li> <li>• Hyperparathyroidism</li> <li>• Hypothyroidism</li> <li>• Chronic renal failure</li> </ul>	<ul style="list-style-type: none"> <li>• Dementia</li> <li>• Parkinson Disease</li> <li>• Multiple Sclerosis</li> <li>• Cerebrovascular Disease</li> <li>• Spinal cord injury</li> <li>• Cauda Equina Syndrome</li> </ul>	<ul style="list-style-type: none"> <li>• Scleroderma</li> <li>• Amyloidosis</li> </ul>	<ul style="list-style-type: none"> <li>• Depression</li> <li>• Personality Disorders</li> <li>• History of abuse</li> </ul>

## ANATOMICAL CONSIDERATIONS OF THE GASTROINTESTINAL TRACT

The adult distal gastrointestinal tract has a variable length, typically averaging around 150 cm.<sup>11,12,13</sup> It extends from the ileocecal valve to the anus and consists of the cecum, ascending, transverse, descending, sigmoid colon, rectum, and anus. The blood supply and autonomic innervation corresponds to the embryonic foregut, midgut, and hindgut divisions (*Table 4, page 27*).

Each segment of the distal gastrointestinal tract has a different function regarding stool formation and movement. The primary function of the colon is water absorption; decreased transit time is associated with increased water absorption, leading to harder feces. The ascending and transverse colon are sites of fecal storage, and the descending and sigmoid colon are involved in fecal transport.<sup>11</sup> The anal canal is surrounded by both voluntary and involuntary muscles that exhibit tonic contraction to keep the anal canal closed and therefore prevent defecation. Retraction of both the external and internal anal sphincter allows for defecation.

## MICROFLORA OF THE INTESTINES

The microflora of the intestines can be altered in pathologic states, including constipation. Gut microbial analysis has revealed that over 1,000 bacterial species can inhabit the human digestive system, with a relative predominance of Bacteroidetes and Firmicutes species.<sup>14,15,16</sup> The number and specific species of microbial organisms vary throughout the digestive tract, becoming more abundant and diverse distally. This is largely dependent on several factors including luminal pH, intestinal motility, mucus abundance, and acid secretion.

Intestinal microflora possibly contributes to the pathogenesis of several colonic conditions, including constipation. Bacterial abundance as well as bacterial species differ between healthy and constipated patients. One study comparing patients with refractory constipation to healthy volunteers found a decrease in probiotic bacteria (Bifidobacteria and Lactobacilli) but no significant difference in the predominant bacteria (Bacteroidetes, and Clostridium coccoides and Clostridium Upturn). Another study examining the gut micro-organisms of constipated patients found a decrease in Prevotella and an increase in Firmicutes, which presumably would result in an increase in butyrate production and possibly contribute to constipation. Substantial research has also been done on the microfloral components of both the constipation and diarrheal subsets of irritable bowel syndrome (IBS) patients, and it is thought that alterations in digestive microflora are linked to irritable bowel symptoms. This is likely due to gut inflammation, disruptions in the intestinal mucosa, and nerve involvement. Research has thus increased our understanding of the potential role that intestinal flora plays in colonic function.

## EVALUATION

### History

The initial evaluation of a patient presenting with constipation involves gathering a thorough medical history. The history should include onset of complaint, patient's definition of constipation, bowel movement frequency, stool consistency, medical history, medication and laxative use, fluid intake, dietary fiber intake, and exercise.<sup>9</sup> A surgical and gynecological history should be obtained as adhesions are implicated in chronic lower abdominal pain, constipation, and ileus. Symptoms such as bloating, pain, and malaise may suggest irritable bowel syndrome. Excessive straining as well as the need for perineal or vaginal pressure during defecation or direct digital evacuation of stools is suggestive of defecatory disorders. Asking for the presence of these symptoms is vital in identifying an evacuatory disorder as it does not respond to standard laxative therapy.<sup>7</sup>

### Physical examination

Physical examination of the constipated patient should include examination of the abdomen, perineum, and rectum. The abdominal exam should assess for masses and hepatomegaly. Inguinal areas should be assessed for hernias and enlarged lymph nodes. The perineal exam should evaluate for external hemorrhoids, skin tags, anal warts, and fissures.<sup>8,10</sup> During the rectal exam, sphincter tone and anal reflex should be assessed as well as presence of rectocele or rectal masses.<sup>9</sup> To test for pelvic floor dysfunction, patients

should be instructed to attempt to expel the examiner's finger. The anal sphincter and puborectalis muscle should normally relax and the perineum should descend. Pelvic floor dysfunction can also be assessed by having patients contract or squeeze the pelvic floor muscles which should result in lifting of the pelvic floor.

## Osteopathic Structural Exam

The osteopathic structural exam expands on the physical exam described above by considering additional structural factors that can impact the function of the colon. This is based on the osteopathic tenet that structure and function are reciprocally inter-related. Proper colonic function depends on the balance of sympathetic and parasympathetic activity to the gut. Sympathetic stimulation results in decreased colonic muscle tone and contractility, therefore slowing fecal movement. Parasympathetic stimulation on the other hand leads to an increase in colonic muscle tone and contractility, as well as secretion of colorectal glands, facilitating stool transport. Inhibition of the sympathetics leads to unopposed output of the parasympathetics, or vice versa. In practice, proper recognition of the levels of autonomic innervation to the gastrointestinal tract allows the physician to target the osteopathic structural exam to evaluate for somatic dysfunction in key regions that can influence colonic activity (Table 4).

Somatic dysfunction in the T10-L2 distribution should be specifically assessed as sympathetic facilitation from the colon is reflected in this region. Increased sympathetic output from the thoracic and lumbar region can reduce peristalsis and increase sphincter tone. The abdominal ganglia, consisting of the celiac, superior mesenteric, and inferior mesenteric sympathetic ganglia, can be assessed by palpating for tension and tenderness between the xyphoid process and umbilicus. The subocciput, pelvis, sacrum, and sacroiliac joint should be assessed as they may affect parasympathetic output via the vagus nerve and pelvic splanchnic nerves.<sup>27</sup> Lastly, both abdominal and pelvic diaphragm movement should be evaluated for any restrictions as these can have mechanical effects on the superior and inferior aspects of the abdominal cavity.

## WORK-UP

Lab testing such as thyroid function tests, calcium, glucose, electrolyte levels, complete blood count, and urinalysis are frequently used in the evaluation of a constipated patient.<sup>4</sup> However, according to the American College of Gastroenterology Chronic Constipation Task Force, in patients without alarming signs or symptoms, there is inadequate data on the routine use of thyroid function tests, serum calcium, or other diagnostic tests. Metabolic testing such as glucose, calcium, and thyroid stimulating hormone levels are only recommended if the clinical presentation warrants it. For example, if signs or symptoms are indicative of organic disease such as hypothyroidism, specific diagnostic tests may be performed. The Task Force also states that there is inadequate data on the routine use of flexible sigmoidoscopy, colonoscopy, and barium enema in patients without alarming signs or symptoms.<sup>2</sup> However, in patients with symptoms such as new onset or worsening constipation, blood in stool, weight loss, fever, anorexia, nausea, vomiting, or family history of inflammatory bowel disease or colon cancer, a complete exam of the colon is required.<sup>4</sup> Additionally, routine colon cancer screening is recommended for patients starting at 50 years of age.<sup>2</sup>

TABLE 4:

Innervation and Blood Supply of the Gastrointestinal Tract<sup>13,28,29,30</sup>

Location	Sympathetics		Parasympathetics		Somatic Innervation	Arterial / Venous Supply
	Nerve (Vertebral Level)	Area to Examine	Nerve (Vertebral Level)	Area to Examine		
Mouth to Second Part of Duodenum (foregut)	Greater Splanchnic Nerve (T5 - T9)	T5 - T9	Vagus Nerve (Cranial Nerve X)	OA, C1 - C2	_____	Celiac Trunk / Splenic and Portal Veins
Third Part of Duodenum to Proximal 2/3 of Transverse Colon (midgut)	Lesser Splanchnic Nerve (T10 - T11)	T10 - T11	Vagus Nerve (Cranial Nerve X)	OA, C1 - C2	_____	Superior Mesenteric Artery / Vein
Distal 1/3 of Transverse Colon to Upper Anus, Internal Anal Sphincter (hindgut)	Lumbar Splanchnic Nerve (L1 - L2)	L1 - L2	Pelvic Splanchnic Nerve (S2 - S4)	Sacroiliac, Sacrum, Pelvis	_____	Inferior Mesenteric Artery / Vein
External Anal Sphincter	_____	_____	_____	_____	Pudendal Nerve and S4	Middle and Inferior Rectal (Internal Iliac) Arteries and Veins

Physiologic testing should only be done in patients with refractory constipation not due to a secondary cause or in patients that did not respond to a high fiber diet and laxatives.<sup>4,7</sup> Slow transit constipation is most commonly diagnosed with the Sitzmark transit study while obstructive defecation can be assessed using anorectal physiologic studies such as anorectal manometry and balloon expulsion test.<sup>6,9,10</sup>

**TREATMENT**

Lifestyle modifications including high fiber diet, exercise, and increased fluid intake may lead to symptomatic improvement.<sup>10</sup> Although lifestyle modifications are usually attempted prior to medical therapy, there are limited controlled trials supporting their use.<sup>9,31</sup> Available studies suggest benefit with these lifestyle measures only when there is a true deficiency present.<sup>9</sup> Patients with normal transit or slow transit constipation should gradually increase their dietary fiber intake to 20-25 g per day followed by supplement use if necessary.<sup>4</sup> Additionally, bowel retraining may be of benefit to the constipated patient. Bowel retraining, a type of behavior modification, involves developing a regular daily routine with time set aside for bowel movements, preferably after meals to utilize the gastrocolic reflex. Such a routine helps the patient recognize and respond to defecatory signals.

If lifestyle modifications do not improve symptoms, an osmotic laxative such as magnesium hydroxide or lactulose can be used. If this is not effective, polyethylene glycol, another osmotic agent, should be used.<sup>7,8</sup> The American College of Gastroenterology

Chronic Constipation Task force have found both polyethylene glycol (17 g/d) and lactulose (15-60 mL/d) to be effective in improving stool frequency and consistency.<sup>2</sup>

Other available pharmaceutical agents for constipation include bulking agents. A systematic review found good evidence for the use of the bulking agent psyllium.<sup>31</sup> Due to insufficient data, the American College of Gastroenterology Chronic Constipation Task Force has not made a recommendation on the use of other bulking agents such as calcium polycarbophil, methylcellulose, and bran. Additionally, the Task Force found that stool softeners had minimal effect on symptomatic control of chronic constipation. Tegaserod, a 5-HT4 selective agonist, has been well-studied and found to improve the frequency of bowel movements and stool consistency.<sup>2,31</sup> Other treatments such as stimulant laxatives, herbal supplements, lubricants, and combination laxatives have not been adequately studied for the Task Force to make a recommendation.<sup>2</sup> Of note, stimulant laxatives have been reported to be associated with cathartic colon which is characterized by colonic dilatation and loss of haustration. However, this effect is not associated with currently available stimulant laxatives and it remains debatable if long-term stimulant laxative use is associated with permanent damage to colonic mucosa or the enteric nervous system. Overuse of stimulant laxatives can reduce colonic tone leading to dependency on laxatives for defecation.

Probiotics have also been of interest as a possible treatment option for constipation. A systematic review of available studies found that there was symptomatic improvement following treatment

with *Bifidobacterium lactis* DN-173 010, *Lactobacillus casei* Shirota, and *Escherichia coli* Nissle 1917. However, additional studies with improved study design are needed in order to determine the role of probiotics in the treatment of constipation.<sup>35,36</sup>

For patients with defecatory disorders, patients can be retrained in the evacuation process by using biofeedback.<sup>4,6,9</sup> The goal of biofeedback is to restore a normal defecatory pattern by improving abdominal push effort, relaxing pelvic floor muscles, performing simulated defecation training, and enhancing rectal sensory perception. Biofeedback is recommended over laxative therapy for defecatory disorders.<sup>7</sup>

If symptoms persist, referral to a specialist may be necessary for further management.

## ROLE OF OSTEOPATHIC MANIPULATIVE TREATMENT IN CONSTIPATION

OMT can improve functioning of the colon by normalizing the autonomic nervous system. Treating the abdominal and sympathetic chain ganglia and surrounding tissues allows for optimal functioning of the nerves and improved regulation of colonic tone, motility, and gastrointestinal secretions. In addition, OMT can address myofascial strains in the viscera and the structures that make up the abdominal cavity and thus help with colonic function from a mechanical perspective (*Table 5*). A.T. Still, the founder of osteopathy, understood this when he described that constipation is propagated by dysfunction of the nervous system, fascia, mesentery, and peritoneum of the gut. He also described that constipated patients have a strain on their abdominal viscera and pelvic overcrowding, with the large bowels being forced into the pelvic cavity, blocking the passage of stool and fluid circulation. Visceral OMT on the abdomen improves the circulation of blood and lymph to and from the viscera which is required for the bowels and fecal matter to remain in a soft condition. Additionally, visceral OMT can decongest the intestines, improve smooth muscle tone, and reestablish the normal resilience, mobility, and motility of the involved organ.<sup>26,40</sup>

Myofascial treatments can address the abdominal container in which the intestines reside. The abdominal container is bordered by the abdominal muscles, lumbar muscles (e.g. psoas muscle), and the abdominal and pelvic diaphragms. Dysfunctions in these areas may cause or be the result of constipation. Therapies targeting pelvic floor hypertonicity can improve constipation symptoms. Treating the abdominal diaphragm can also aid in relieving abdominal distention. Potential OMT that can be used include iliopsoas muscle release and abdominal and pelvic diaphragm release. Prior to diaphragmatic release, thoracic outlet release should be performed to ensure maximal lymphatic and venous return.

Chapman's points, which are viscerosomatic reflex points, may also arise in response to constipation (*Table 6*). These points represent visceral dysfunction and are mediated by the sympathetic nervous system. Additionally, they provide both diagnostic and therapeutic utility as treatment of the Chapman's points can break positive feedback cycles through the somatovisceral pathway.<sup>26</sup>

Two pilot studies have shown that OMT may play a role in constipation management. One study included 6 subjects with chronic constipation that were treated with techniques such as paraspinal

inhibition, passive joint mobilization of the thoracolumbar spine, visceral techniques, and muscle energy technique to the sacro-pelvic region, thoracolumbar spine and atlanto-occipital joint. After six treatments over one month, subjects had significant improvement in symptom severity, colonic transit time, and quality of life.<sup>40</sup> A second study included 13 children with cerebral palsy that were diagnosed with chronic constipation. One study group received OMT which included fascial release, iliopsoas release, sphincter release, and bowel mobilization while the other group received OMT and medical therapy. Both groups had significant improvement from baseline evaluation.<sup>44</sup> Pilot studies have demonstrated the benefit of OMT in other gastrointestinal pathologies including irritable bowel syndrome and post-operative ileus<sup>45,46</sup> prompting the need for further research in the field.

## Clinical Case

41-year-old male presents with chief complaint of straining during bowel movements and hard stools for 4 weeks. He has no past medical or surgical history and is currently not taking any medications. He denies melena, hematochezia, fever, and weight loss. Structural examination reveals T11-L2 bilateral paraspinal hypertonicity, tension/tenderness over the inferior mesenteric ganglion, and a restricted abdominal diaphragm.

## OMT

From a neurological perspective, autonomic tone can be addressed by using paraspinal inhibition to T11-L2 and inferior mesenteric ganglion release, thereby normalizing sympathetic tone to the lower GI tract. From a mechanical/structural perspective, diaphragm releases, starting with thoracic outlet release, relieves tension in the myofascia allowing for maximal venous and lymphatic flow.

## ROLE OF COMPLEMENTARY AND ALTERNATIVE (CAM) TREATMENT IN CONSTIPATION

Complementary and alternative medicine (CAM) therapies such as massage therapy, certain forms of acupuncture, and chiropractic care can potentially ameliorate constipation and have shown to be effective.<sup>47,48</sup> Discussion of these therapies is beyond the scope of this article, but exploring these alternative options is important due to the increasing frequency of constipation related emergency department visits, cases of constipation refractory to conservative medical treatment, and the associated costs of care.<sup>49</sup> There is also public awareness and interest in CAM,<sup>50</sup> so a basic understanding of such options can promote dialogue with the patient and enhance patient satisfaction.

**TABLE 5:**

Selected OMT techniques for treating constipation

Treatment Target	Treatment Goal	OMT
Autonomics	↓ Sympathetics	Rib Raising* Paraspinal inhibition* Ganglionic Release (celiac, superior, inferior)
	↑ Parasympathetics	Address restrictions to cervical area* (e.g. suboccipital release) Jugular foramen, and sacrum* (e.g. sacral rock)
Viscera	Remove restrictions to mesentery and flow of blood or lymph	Mesenteric Lift Colonic Stimulation ◊
Myofascia	Remove Restriction	Iliopsoas release*◊ Pelvic diaphragm release and Abdominal diaphragm release* (treat thoracic inlet first)

\*Refer to reference 40 ◊ Refer to reference 44

**TABLE 6:**

Chapman's reflexes for gastrointestinal tract<sup>43</sup>

	Anterior Point	Posterior Point
Duodenum	Between the 8 <sup>th</sup> and 9 <sup>th</sup> ribs near the costochondral junction	Between T8 and T9 midway between the spinous processes and tips of the transverse processes
Jejunum	Between 9 <sup>th</sup> and 10 <sup>th</sup> ribs near the costochondral junction	Between T9 and T10 midway between the spinous processes and the tips of the transverse processes
Ileum	Between the 10 <sup>th</sup> and 11 <sup>th</sup> ribs near the costochondral junction	Between T10 and T11 midway between the spinous processes and tips of the transverse processes
Cecum	Upper one-fifth of the right thigh anteriorly on the tensor fascia lata	Triangular area from the transverse process of L2 to L4 and extending laterally to the iliac crest bilaterally
Ascending Colon	Middle three-fifths of the right thigh, on the anterior aspect of the iliotibial tract	
Right half of the transverse colon	Proximal to the right knee, on the anterior aspect of the iliotibial tract	
Left half of the transverse colon	Proximal to the left knee, on the anterior aspect of the iliotibial tract	
Descending Colon	Middle three-fifths of the left thigh on the anterior aspect of the iliotibial tract	
Sigmoid Colon	Upper one-fifth of the left thigh, anteriorly on the tensor fascia lata	Sacrum, close to the ilium at the lower end of the sacroiliac articulation
Rectum	Proximal inner thighs over the lesser trochanters	

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## REVIEW ARTICLE

# Dysuria

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## Keywords:

Dysuria

Urinary Tract Infection

Cystitis

Prostatitis

Pyelonephritis

Urology

Dysuria is defined as burning, pain, or discomfort with urination. Dysuria is a very common presenting complaint in family medicine clinics accounting for 5% to 15% of visits. It does occur more commonly in females, but may occur in males as well, especially in older males. Dysuria can be caused by infectious etiologies as well as non-infectious etiologies. Some of these are relatively benign, but some are much more serious. Many causes can be identified by history, exam, and simple in-office tests. Further laboratory tests and imaging are sometimes required to diagnose more complex or unusual etiologies. Laboratory and imaging studies include urinalysis, urine culture, vaginal smear, vaginal culture, ultrasound, CT scan, MRI, and cystoscopy. Acute cystitis is one of the most common causes, accounting for roughly 650,000 to seven million office visits per year and can usually be diagnosed accurately on history alone.

## INTRODUCTION

Dysuria is defined as burning, pain, or discomfort with urination. Dysuria is a very common presenting complaint to family medicine clinics. Approximately 5% to 15% of visits to family medicine clinics are for dysuria.<sup>1</sup> One of the most common etiologies of dysuria is acute cystitis accounting for 650,000 to seven million office visits per year.<sup>2</sup> However, dysuria can be a presenting complaint of many other etiologies, some of which are life-threatening. The etiology can be infectious as well as non-infectious. Evaluation of dysuria begins with a thorough history to identify any possible etiology, followed by physical exam and then laboratory testing as guided by the history and physical. Urinalysis is the single most useful, yet technically easy test in evaluation of dysuria. There are several other laboratory studies and imaging that can be utilized if the history and exam dictate further evaluation.

## CAUSES OF DYSURIA

Dysuria may be caused by several etiologies. The patient history will help greatly with diagnosis. One way to classify the causes of dysuria is by dividing the causes into infectious and noninfectious. While cystitis is a very common cause of dysuria, other infections, structural abnormalities, hormonal changes, inflammation, psychogenic, and even neoplastic processes have to be included in the differential. The differential depends, in part, on if the patient is male or female. Tables 1 and 2 are useful in differentiating between causes.

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## Infectious

Urinary tract infections (UTIs) are one of the most common bacterial infections encountered in family medicine with estimates of 650,000 to seven million office visits per year.<sup>2</sup> It is estimated that approximately one-half of women will experience at least one urinary tract infection during their lifetime.<sup>3</sup> A history of diabetes, abnormal bladder function, kidney stones, and enlarged prostate or current pregnancy are risk factors for UTI. UTIs are divided into two main categories: lower urinary tract infections and upper urinary tract infections.<sup>4</sup> Lower UTIs are also referred to as cystitis.<sup>5</sup> Pyelonephritis is an upper urinary tract infection, additional symptoms include low back pain, fever, and nausea and/or vomiting. The majorities of urinary tract infections are acute uncomplicated cystitis and are relatively easy to treat, although increased resistance to some antimicrobials have continued to occur. Pyelonephritis is more difficult to treat and if left untreated, can lead to sepsis.<sup>6</sup>

Other infections to consider include prostatitis, urethritis, cervicitis, epidymo-orchitis, and vulvovaginitis.

## Noninfectious

Aside from infection, many other conditions can cause dysuria. In women, these include estrogen deficiency, endometriosis, and vaginal or vulvar cancer. In men, conditions include benign prostatic hyperplasia, prostate cancer, and penile cancer. A few conditions can exist in both men and women that include urethral strictures or diverticula, renal cell cancer, bladder cancer, trauma (e.g. catheter placement), inflammatory disorders, medication side effects, and psychogenic conditions such as somatization disorder anxiety, depression, and anxiety.

**TABLE 1:**

Differential diagnosis of dysuria in women

If Patient Has ...	Consider ...
Acute, colicky flank pain or history of kidney stones	Kidney stone, complicated cystitis
Costovertebral angle tenderness, fever	Pyelonephritis
Vaginal discharge	Sexually transmitted disease, bacterial vaginosis, candidiasis, Pelvic Inflammatory Disease
Diabetes/immunosuppression	Complicated cystitis, unusual pathogens
Vaginal atrophy	Estrogen deficiency
Joint pains	Spondyloarthropathy (i.e. Reiter or Bechet syndrome)
History of childhood UTI or urologic surgery	Abnormal anatomy, complicated cystitis
Recurrent symptoms after treatment	Abnormal anatomy; abscess; stone; chronic prostatitis; resistant organism; inadequate length of treatment; Munchausen syndrome; somatization disorder

**TABLE 2:**

Differential diagnosis of dysuria in men<sup>7</sup>

If Patient Has ...	Consider ...
Acute, colicky flank pain or history of kidney stones	Kidney stone, complicated cystitis
Costovertebral angle tenderness, fever	Pyelonephritis
Urethral discharge	Sexually transmitted disease
Diabetes/immunosuppression	Complicated cystitis, unusual pathogens
Testicular pain	Torsion, epidymo-orchitis
Prostate Tenderness on exam	Prostatitis
Joint pains	Spondyloarthropathy (i.e. Reiter or Bechet syndrome)
History of childhood UTI or urologic surgery	Abnormal anatomy, complicated cystitis
Recurrent symptoms after treatment	Abnormal anatomy; abscess; stone; chronic prostatitis; resistant organism; inadequate length of treatment; Munchausen syndrome; somatization disorder

## EVALUATION

As with all presenting symptoms, a thorough, detailed history and exam are vital. In many cases a history and exam will lead to an accurate diagnosis without needing further workup or only require a simple urinalysis. This is especially true in the case of acute cystitis.<sup>1</sup> However, as outlined above and in Tables 1 and 2, there are several complicated causes that do warrant further investigation. Possible studies include simple in-office tests such as urinalysis to MRI or cystoscopy.

## HISTORY

A thorough history, including previous urinary tract infections, symptoms of dysuria, urgency, frequency, and suprapubic pain with or without hematuria should direct the clinician to the diagnosis of urinary tract infection.<sup>8</sup> Family history should focus on any history of urogenital cancers and renal stones in the family. Social history should include any tobacco use, which increases cancer risk of the urinary system including renal and bladder cancer. A sexual abuse history increases the likelihood of psychogenic causes and possibly sexually transmitted infections.<sup>1</sup> Medication history should be reviewed (including herbal medications) that may cause dysuria including penicillin G, cyclophosphamide, ticarcillin, and saw palmetto. Vaginal douches and vaginal sprays can cause a change in vaginal flora leading to bacterial vaginosis as well as irritate the urethra causing dysuria from the inflammation. Bubble baths can also cause urethritis and thus complaints of dysuria.

## EXAM

Physical examination, including vital signs and temperature aid the clinician in determining likelihood and the severity of infection as well as the possibility of pyelonephritis This is helpful in determining the need for more aggressive or prolonged treatment.<sup>9</sup> Costovertebral angle tenderness suggests pyelonephritis or urinary stone. A vaginal exam is warranted if the history includes vaginal discharge, vaginal itching, vaginal pain, history of vulvovaginitis, sexually transmitted infection exposure, or dyspareunia. For men, a testicular exam or prostate exam may be warranted if the history suggests testicular or prostate etiologies. A pre- and post-prostatic massage or the Meares-Stamey 4-glass test urine sample can be obtained for analysis.<sup>10</sup> A modified 2-glass test also can be used and is more common. However, neither one is utilized that much in practice.

In cases of urinary tract infections, distinguishing between uncomplicated and complicated infections (those which may require additional investigation and extended treatment) should be initially assessed by the clinician. Evaluation of symptoms such as such as fever (>38°C, 100.4° F), chills, flank pain, costovertebral angle tenderness and nausea and/or vomiting may point the clinician to the possibility of a more complex infection (pyelonephritis). See Table 3 (page 34).

Information obtained from the history and physical examination should provide guidance to the clinician for the appropriate treatment regimen or further workup.

**TABLE 3:**  
Symptoms of Uncomplicated and Complicated Urinary Tract Infections

Symptoms of Uncomplicated UTI	Symptoms of Complicated UTI
Dysuria	Fever > 38° C (100.4° F)
Increased frequency of urination	Flank Pain
Suprapubic pain	Costovertebral angle tenderness
Hematuria	Nausea with or without vomiting

## DIAGNOSTIC TESTS

There are a variety of diagnostic options when trying to determine the cause of dysuria in a patient. Diagnostic options include urine studies, vaginal and urethral studies, and imaging studies. The diagnostic method to be used is determined by the practitioner following information collected during patient's history and physical examination upon presentation.

### Urine Studies

Urine studies are the most common and useful method of diagnosis for patients with a complaint of dysuria. These studies are inexpensive, non-invasive, and return results quickly. Urinalysis is the most commonly used diagnostic exam and can help to quickly confirm a diagnosis of urinary tract infection or aid in diagnosis of possible urinary stone.<sup>11</sup> A simple urine dipstick can show the presence of leukocyte esterase and nitrites, either of which indicate a probable UTI. One thing to keep in mind is that pyuria can be seen in infections other than UTI such as *Trichomonas vaginalis* and *Chlamydia trachomatis* as well as nephrolithiasis and urologic neoplasms. A microscopic exam of a clean-catch, mid-stream urine sample that is spun down is the gold standard. This allows visualization of white blood cells and red blood cells, as well as bacteria.<sup>1</sup> Other organisms such as *Trichomonas* can be seen on microscopic exam as well. Urine cultures allow for verification of UTI as well as determination of the most effective antibiotic treatment regimen and are especially useful in patients where complications can develop such as pregnant patients, patients with diabetes or in male patients with UTI. Other than cystoscopy, urine cytology is a method for detection of bladder cancer as a cause of dysuria. The only time urine studies are not used is when the history and physical findings suggest other diagnostic methods would be more appropriate.

### Vaginal and urethral smears and cultures

Vaginal and urethral smears and cultures, along with ligase and polymerase chain reaction tests, are preferred when patients have dysuria with vaginal or urethral discharge or in instances of rape or child sexual abuse. Vaginal and urethral smears are useful for detection of *Trichomonas vaginalis*, *Candida* species, and with the use of gram staining the detection of *Neisseria gonorrhoeae*. Vaginal cultures are nearly 100% specific for *Neisseria gonorrhoeae* and

*Chlamydia Trachomatis* and are required as part of a patient work-up in cases of rape or child sexual abuse. Ligase chain reaction and polymerase chain reaction (PCR) tests also detect *Neisseria* and *Chlamydia*. They are not as specific, but results are available faster than cultures. These can also be detected by PCR in urine. This makes testing quicker and easier overall. *Chlamydia* particularly can mimic a urinary tract infection very closely and should be high on the differential list.<sup>12</sup>

### Imaging Studies

Imaging studies, both radiologic and non-radiologic, are useful when the diagnosis is in doubt, when complications are suspected, when patients are not responding to antibiotic therapy, and in patients who are severely ill or immunocompromised. Ultrasonography and plain film radiography are relatively inexpensive and allow quick assessment of kidney stones, diverticula in the bladder or pyelonephritis that could all lead to the symptoms of dysuria.<sup>13</sup> The drawbacks to these particular tests are they are not very effective in obese patients or in cases where there are obstructions that could cloud the images such as gas pockets or feces in the bowel or masses in the area of the bladder or kidneys. IV pyelography and voiding cystourethrography are useful in cases of recurrent UTI to help detect obstructions and anatomical causes of decreased urine flow leading to dysuria. CT with or without contrast and helical CT are useful in detection of tumors, cysts, abscesses and areas of infarction that could all be causing decreased urine flow, and in turn symptoms of dysuria. MRI is preferred for visualization of masses and the renal vasculature in patients with renal insufficiency or an allergy to contrast media.<sup>14,15</sup> Cystoscopy is an invasive procedure, but does allow for performance of a biopsy for a histological diagnosis and also allows direct visualization of the bladder and urethra. A smoking history with persistent hematuria, either micro- or macroscopic, should raise suspicion for bladder cancer and warrant referral for cystoscopy.<sup>16,17</sup>

## SUMMARY

Dysuria is a very common presenting complaint to family medicine physicians. It occurs in women more often than men. Etiologies include both infectious and noninfectious causes. The most common etiology for dysuria is acute cystitis, with 650,000 to seven million clinic visits per year. History and exam are very important and in many cases can lead to an accurate diagnosis without need for extensive workup. However, there are several differentials, some of which are serious, that can cause dysuria, and thus; if the history and exam dictate it, further testing is recommended with laboratory and/or imaging studies. Urogenital cancer of both men and women can occur and should be part of the differential. Proper treatment depends on identifying the cause of the dysuria. In cases of urinary tract infection, it is important to understand resistance rates of common bacteria. Resistance rates do vary by geographic and even institutional locations and thus should be understood prior to prescribing antibiotics. Early treatment of cystitis is important to prevent complications including pyelonephritis and sepsis. Several laboratory and imaging tests are available to aid in diagnosis as outlined in this article.

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# Uvulitis

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A 53-year-old African-American female presented to the emergency room with sudden onset of dysphagia. The patient was driving to the grocery store when she developed difficulty swallowing, sore throat and drooling. She denied shortness of breath, fevers, chills, rash, rhinorrhea, cough or any sore throat previously. She denies new medications, but the patient does have a history of hypertension for which she is on nisoldipine, torsemide, carvedilol and aspirin. Prior to arrival at the emergency room, the patient did not attempt any intervention. The patient was fully immunized as a child.

Physical exam shows an uncomfortable, afebrile, hypertensive patient with an injected pharynx bilaterally and symmetrical, erythematous tonsils without exudates (*Figure 1*). The uvula has moderate edema. No stridor or trismus was noted. Tongue swelling and elevation are absent. The patient appears to have difficulty swallowing with some drooling. There is no perioral or facial swelling noted. During her workup she had a negative strep test and a negative soft tissue x-ray of the neck. Her basic metabolic panel showed elevated blood glucose and low potassium. The complete blood count had a hemoglobin of 10 grams/deciliter (13-17.5g/Dl normal) without leukocytosis or left shift.

## QUESTIONS:

### 1. What is the most likely diagnosis?

- Angioedema
- Epiglottitis
- Streptococcal Pharyngitis
- Uvulitis

### 2. Which of the following is a recommended treatment for uvulitis?

- Corticosteroids, antihistamines and epinephrine
- High dose intravenous immunoglobulin and aspirin
- Penicillin V or amoxicillin
- Treatment of uvulitis depends on the specific cause

FIGURE 1:



## ANSWERS

### 1. What is the most likely diagnosis?

The correct Answer is:

D) Uvulitis

The patient has an inflamed and enlarged uvula on exam in association with clinical history consistent with uvulitis. Angioedema is an inherited or acquired edema of the dermis, most commonly involving the periorbital or perioral regions. Some triggers include food, medications or exercise.<sup>1</sup> Large, swollen wheals frequently involving the eyelids and lips, but can erupt in the pharynx and larynx causing airway obstruction.<sup>1</sup> Epiglottitis is rapid progressive inflammation of the epiglottis.<sup>1</sup> Patients usually have a toxic appearance with drooling, dysphagia, "hot potato voice" and foreign body sensation. Lateral neck films may show the classic "thumb print" sign.<sup>1</sup> Streptococcal pharyngitis is generally associated with swollen, enlarged tonsils with or without exudates, cervical lymphadenopathy and fever.<sup>1,2</sup>

### 2. Which of the following is a recommended treatment for uvulitis?

The correct Answer is:

D) Treatment of uvulitis depends on the specific cause

Treatment varies due to the wide range of causes.<sup>3</sup> For patients with uvulitis and pharyngitis caused by Group A streptococcus, the first line treatment is oral penicillin V or amoxicillin.<sup>2,3,4</sup> High dose intravenous immunoglobulin and aspirin is the treatment of choice for Kawasaki Disease associated uvulitis.<sup>5</sup> Uvulitis suspected to be due to allergic reaction can be treated with corticosteroids, antihistamines and epinephrine if the clinical situation indicates.<sup>6</sup>

## DESCRIPTION

Uvulitis is an inflammatory condition of the uvula caused most often from either an infectious process or trauma.<sup>3</sup> It is an acute cellulitis from direct invasion of microbes or direct injury resulting in uvular edema and erythema.<sup>3</sup> It is typically found in children and adults with predominance in spring and winter.<sup>3</sup> Infectious causes include Group A streptococcus (GAS) most commonly, and Haemophilus influenzae type b (Hib) secondly, both of which are normal nasopharyngeal flora.<sup>3</sup> Less common organisms include Fusobacterium nucleatum, Prevotella intermedia, and Streptococcus pneumoniae.<sup>7,8</sup> In immunocompetent children, Candida albicans has also been reported as an etiology of uvulitis.<sup>3</sup> Noninfectious causes of uvulitis range from trauma, inhalation or ingestion of chemical irritants, vasculitis (Kawasaki disease), allergic reactions, or angioedema of the uvula.<sup>3,4,5,9,10</sup>

The presentation of uvulitis is often varied and dependent on the causative agent. Patients may present with fever, sore throat, difficult or painful swallowing, drooling, and/or respiratory distress.<sup>3,9</sup> Group A streptococcus uvulitis is often less severe with low-grade fever and sore throat in association with GAS pharyngitis.<sup>2,3,4</sup>

The clinician should have increased suspicion for uvulitis caused by Hib with presentations in younger children or non-immunized patients.<sup>3</sup> Hib uvulitis may be associated with potentially life-threatening epiglottitis.<sup>3</sup> Severe symptoms of drooling and/or respiratory distress are more commonly seen in patients with noninfectious causes such as trauma.<sup>11</sup>

Oropharynx examination may be challenging depending on the cause of uvulitis. If the patient is able to open their mouth adequately for a thorough exam, the uvula will be erythematous and edematous with or without purulent exudates.<sup>3</sup> Pharyngeal edema, tonsillar enlargement, and exudates may be observed if the patient has associated GAS pharyngitis.<sup>2,3</sup> Oral lesions and mucosal inflammation can be present in patients with uvulitis secondary to noninfectious causes due to allergic reactions, trauma, and inhalation of marijuana.<sup>4,12</sup> If there is a high suspicion of concomitant epiglottitis based on the patient's presentation (anxious, drooling, respiratory distress) a physical exam should be avoided in order to not precipitate complete airway obstruction. An otolaryngologist or an anesthesiologist should be consulted for rapid direct laryngoscopy under anesthesia. If a patient is at high risk for epiglottitis (not vaccinated for Hib) and has less extreme symptoms but is unable to open their mouth adequately for a thorough oropharyngeal exam, a lateral neck radiograph or CT with contrast of the neck should be considered for evaluation to rule out subclinical epiglottitis.<sup>3,4,9,10</sup>

Laboratory workup should be guided by clinical presentation. The causative organism for uvulitis and pharyngitis is most commonly GAS.<sup>3</sup> Surface cultures of the uvula and/or rapid antigen detection tests for GAS are recommended to confirm the etiology.<sup>2,3</sup> In patients with isolated uvulitis without pharyngitis, surface cultures of the uvula and throat should be obtained if an infectious cause is suspected.<sup>7,13,14</sup> It is important to consider Hib in patients younger than 5 years old who have not received the Hib vaccine or are not properly vaccinated against this organism.<sup>2,3</sup>

Treatment varies due to the wide range of causes.<sup>3</sup> For patients with uvulitis and pharyngitis caused by Group A streptococcus, the first line treatment is oral penicillin V or amoxicillin.<sup>2,3,4</sup> A macrolide can be substituted for penicillin allergic patients.<sup>2</sup> An advanced generation cephalosporin such as cefotaxime or ceftriaxone should be the initial antibiotic of choice for uvulitis secondary to Hib.<sup>3</sup> In isolated uvulitis, empiric therapy should cover for both Hib and Streptococcus pneumoniae.<sup>2</sup> Uvulitis with epiglottitis is an emergent situation in which the airway must be secured and appropriate antimicrobial treatment started quickly.<sup>3,4,5,9,10</sup> High dose intravenous immunoglobulin and aspirin is the treatment of choice for Kawasaki Disease associated uvulitis.<sup>5</sup> Uvulitis suspected to be due to allergic reaction can be treated with corticosteroids, antihistamines and epinephrine if the clinical situation indicates.<sup>6</sup>

This patient was treated with diphenhydramine and methylprednisolone initially during her ED stay. Her laboratory workup did not indicate infection and she was afebrile in the ED. The case was discussed with otolaryngology who recommended empiric clindamycin and outpatient follow up. It is suspected that the uvulitis was initially infectious or allergic in etiology. However, the strep swab procedure was traumatic to the uvula and caused



the hemorrhagic uvulitis as depicted. The patient's symptoms improved during her ED stay and she was discharged home. Of note, previous records were reviewed and approximately one year prior to this presentation, the patient was seen in the ED for sore throat and foreign body sensation. She had a negative soft tissue neck x-ray, normal CBC and was sent home on antibiotics. She was diagnosed with uvulitis at that time as well.

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## Inherited Patterned Lentiginosis: A Diagnosis of Exclusion

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A 55-year-old female presented to her family physician with multiple dermatological complaints. She reported a skin lesion on her left thigh. It had been present for many years, but was enlarging and darkening over the past few months. The patient also reported a pruritic rash on her torso that was present for one week. She noticed it randomly and denied any recent outdoor camping or hiking activity, new lotions or detergents, or ingestion of new medication or food. She stated that she had multiple tan “spots” on her face and arms that she was never concerned about as they had been present her “whole life” and was a feature most of her family members had. The spots were not pruritic, and she had not noticed a change in appearance for as long as she could remember.

The patient’s past medical history was significant for hypothyroidism, eczema, and chronic irritable bowel syndrome. She had no allergies. Her medications included levothyroxine 25 mcg daily and over-the-counter psyllium fiber daily. She revealed a family history of a father who died of an esophageal carcinoma at an unknown age, a living brother with myeloma, a living sister with systemic lupus erythematosus and a living mother with coronary artery disease. She reported that her mother, all four sisters, daughter, and son had the same tan macules on their face along their nose and cheeks, while sparing the oral mucosa.

The patient had a minimal smoking history of a few cigarettes per day for 5 years – she quit 32 years ago. She consumed alcohol socially and denied drug use.

Review of systems was positive for skin lesions, dry itchy skin, and irritable bowel with intermittent loose stool and constipation. Pertinent negatives included absence of fever, chills, hot or cold flashes, sudden weight gain or loss, hearing loss, chest pain at rest or on exertion, shortness of breath, and blood in her stool. All other review of systems was negative.

On physical exam, there was a violaceous red papule with one focal area of brown pigmentation on the left anterior proximal thigh. Biopsy revealed a benign hemangioma. There were pink hyperpigmented macules with fine scale located diffusely on the anterior trunk consistent with tinea versicolor, which was treated with a topical antifungal cream. There was a single 3 mm blue macule consistent with a blue nevus located on the right frontal scalp. The patient also had numerous 1-3 mm light tan macules in a photo distribution over her face, including eyelid margin and lips, consistent with lentiginosities [Figure 1]. These lentiginosities were also noted on the arms [Figure 2]. There were no lentiginosities seen on her bilateral palms or soles or her feet. There were grouped speckled brown macules with a solitary lighter tan patch located on the right posterior thigh indicating nevus spilus. Finally, there was a well marginated oval light tan macule under the right breast consistent with a café-au-lait macule.

Pertinent negatives on exam included the absence of lentiginosities on the buccal mucosa, palms of her hand and soles of her feet. She had a normal cardiac and abdominal exam, and there was no thyromegaly detected.

The patient had a normal colonoscopy with no evidence of polyps. A thyroid ultrasound was normal, as was the patient’s electrocardiogram. The patient received an echocardiogram that was negative for structural or wall motion abnormalities and notably absent of cardiac myxomas. Pertinent lab values included hemoglobin 13.3 g/dl, fasting glucose 82 mg/dl, and a thyroid stimulating hormone of 3.86 mIU/L – all within normal limits.

**QUESTIONS:**

1. After a thorough imaging and lab work up that was negative for abnormality, what is the diagnosis?
  - a. Carney Complex
  - b. Inherited Patterned Lentiginosis
  - c. LEOPARD syndrome
  - d. Peutz Jeghers syndrome
  - e. Squamous cell carcinoma
  
2. What differentiates the lentigines of Inherited Patterned Lentiginosis from Peutz Jeghers syndrome (PJS)?
  - a. Lentigines can be present on buccal mucosa in PJS, whereas the buccal mucosa is spared in Inherited Patterned Lentiginosis.
  - b. Lentigines of PJS histologically resemble ephelides (freckles)
  - c. The lentigines seen in Inherited Patterned Lentiginosis are autosomal dominant.
  - d. The lentigines seen in PJS get darker in adulthood
  - e. There are no differences; the two conditions cannot be differentiated based on physical appearance alone.
  
3. The most serious and potentially fatal manifestation of Carney Complex syndrome is:
  - a. Ovarian carcinoma
  - b. Metastatic melanoma
  - c. Thyroid storm
  - d. Cardiac myxoma
  - e. Thrombotic thrombocytopenia purpura (TTP)
  
4. Differences between lentigines and ephelides include all of the following EXCEPT:
  - a. Ephelides do not generally indicate systemic disease
  - b. Lentigines will darken with sun exposure, whereas ephelides are not affected by UV light
  - c. Ephelides histologically show increased melanin and a normal amount of melanocyte, whereas lentigines present histologically with increased melanocytes
  - d. Lentigines are seen predominately in people of African descent, whereas ephelides are most common in northern and western European descent
  - e. Lentigines appear on non-sun exposed skin, ephelides are typically are confined to sun-exposed skin

**FIGURE 1:**

Facial lentigines

**FIGURE 2:**

Lentigines on forearms



## ANSWERS

### 1. After a thorough imaging and lab work up that was negative for abnormality, what is the diagnosis?

The correct Answer is:

B) Inherited Patterned Lentiginosis

### 2. What differentiates the lentigines of Inherited Patterned Lentiginosis from Peutz Jeghers syndrome (PJS)?

The correct Answer is:

A) Lentigines can be present on buccal mucosa in PJS, whereas the buccal mucosa is spared in Inherited Patterned Lentiginosis.

### 3. The most serious and potentially fatal manifestation of Carney Complex syndrome is:

The correct Answer is:

D) Cardiac myxoma

### 4. Differences between lentigines and ephelides include all of the following EXCEPT:

The correct Answer is:

B) Lentigines will darken with sun exposure, whereas ephelides are not affected by UV light

## DISCUSSION

The distribution and characteristics of the macules on the face and arms appeared consistent with lentigines. However, because the patient also presented with nevus spilus, blue nevus, and a café-au-lait macule, as well as gastrointestinal (GI) complaints, a family history of GI malignancy, and endocrine abnormality of hypothyroidism, further studies were obtained to rule out familial lentiginosis syndromes such as Carney complex and Peutz Jeghers syndrome.

Through a diagnosis of exclusion, the patient was determined to have Inherited Patterned Lentiginosis. Inherited patterned lentiginosis is an uncommonly described benign cutaneous condition originally described by John F O'Neill and William D James in 1989 in the Archives of Dermatology.<sup>1</sup> Although rarely studied or noted in research articles, this condition appears to be inherited in an autosomal dominant fashion and is most commonly seen in lighter-pigmented African Americans, particularly those with mixed American Indian heritage.<sup>2</sup> Cutaneous findings include lentigines, which are small hyperpigmented macules that present in early childhood<sup>1</sup> and often increase in amount as a child ages and enters puberty.<sup>2</sup> These lentigines are commonly found on the

central face, lips, as well as hands, elbows, and buttocks. There is sparing of the mucous membranes. In contrast to other lentiginosis syndromes, there are no associated systemic diseases and it is therefore a benign condition.<sup>3</sup>

While lentigines often clinically resemble ephelides (freckles), lentigines typically do not darken with sun exposure and can appear on non-sun-exposed skin. Histologically, a lentigo will show basal cell layer hyperpigmentation with increased number and hyperplasia of melanocytes.<sup>4</sup> In contrast, ephelides generally present with increased melanin within basal keratinocytes and a normal number of melanocytes.

The differential diagnosis for facial lentigines are familial lentiginosis syndromes, including Peutz-Jeghers syndrome (PJS), Carney Complex (CNC), LEOPARD syndrome, and Cronkhite-Canada syndrome, as well as more rare disorders, such as Laugier-Hunziker syndrome. Peutz-Jeghers syndrome (PJS) is an autosomal dominant disorder that usually presents in early teenage years. It is a mutation of the STK11 gene on chromosome 19p13.3.<sup>5</sup> Cells overgrow characteristically in the GI tract and manifest as multiple hamartomatous polyps. Rarely, polyps have been reported in ureters, nasal and respiratory tracts, and the gallbladder.<sup>6</sup>

The hamartomatous polyps of PJS have a high risk of turning into malignant carcinoma, and patients have a greater likelihood of developing other cancers including breast, cervical, GI, pancreatic, and endometrial carcinoma. One meta-analysis has cited a 93% cumulative risk of developing cancer.<sup>6</sup> It is therefore imperative that this condition be diagnosed early with colonoscopy and endoscopy so cancer screening can be implemented immediately.<sup>5,6</sup>

Cutaneous manifestations of PJS that are similar to inherited patterned lentiginosis include multiple 1-5 cm blue-gray to brown macules found around the eyes, nostrils, mouth, and occasionally on hands, feet and anal region.<sup>6</sup> The lentigines are seen in 95% of patients affected with PJS, and tend to be most visible in childhood, fading by adulthood.<sup>7</sup> A key difference between PJS and inherited patterned lentiginosis is the presence of lentigines on buccal mucosa in PJS.

Diagnostic criteria for PJS require at least one of the following: 1) 2 or more polyps histologically confirmed to be PJS, 2) any number of polyps plus a family history of PJS, 3) mucocutaneous pigmentation plus a family history, 4) Peutz Jegher polyps and mucocutaneous pigmentation.<sup>8</sup>

Carney Complex (CNC) is an autosomal dominant disorder associated with a mutation of the PRKAR1A gene on chromosome 17q22-24.<sup>7</sup> Cardiac myxomas are the most serious manifestation of CNC with a 16% sudden cardiac death rate.<sup>9</sup> Endocrine tumors and therefore endocrine hypo- and hyper-activity are also a common finding. Primary pigmented nodular adrenocortical disease, growth hormone-secreting pituitary adenomas, thyroid carcinomas, testicular tumors and ovarian cysts have all been associated with CNC.<sup>9</sup>

Cutaneous manifestations of CNC include lentigines located on the conjunctiva and vermilion border of lips that are most noticeable in adolescence and fade with age. Blue nevi of less than 5 mm have been reported on the face, trunk and limbs. Café-au-lait macules, nevus spilus, and cutaneous myxomas are also present, with the myxomas seen on the face, ears and trunk in 30-55% of patients.<sup>4</sup>

Diagnosis usually begins with findings suggestive of CNC based on the cutaneous manifestations and family history. Testing for endocrine abnormalities initially include thyroid panels, blood glucose, and urinary cortisol. If abnormal, plasma adrenocorticotropic hormone (ACTH), growth hormone (GH), insulin-like growth factor and dexamethasone suppression testing can be performed. Patients should be evaluated with echocardiogram to rule out a cardiac myxoma. Other imaging may be performed based on lab values and clinical suspicion, including adrenal CT scans, thyroid US, testicular US, ovarian US, and pituitary MRI.<sup>7</sup>

LEOPARD syndrome is a rare autosomal dominant disorder caused by a mutation of the PTPN11 gene on chromosome 12q24.1.<sup>10</sup> Its name is an acronym for the various manifestations of this syndrome: Lentigines, Electrocardiogram abnormalities, Ocular hypertelorism, Pulmonary stenosis, Abnormal genitalia, Retardation of growth, and sensorineural Deafness. The lentigines of LEOPARD syndrome are found primarily on the upper trunk and face, but not oral mucosa – a feature that distinguishes it from PJS but is similar to inherited patterned lentiginosis. Unlike PJS and CNC, the lentigines start in infancy and then increase in number with age. Often, lentigines are the first clinical clue to diagnosis,<sup>10</sup> and it is made if lentigines plus two of the other features are present. If lentigines are absent (only 10% of cases), diagnosis is established if a first degree relative is affected and three of the aforementioned features are present.<sup>7</sup>

Cronkhite-Canada syndrome (CCS) is a rare, non-familial syndrome that presents around the 6th decade. It is characterized by hamartomatous polyps throughout the GI tract that are phenotypically similar to those seen in PJS. These polyps are associated with other mucosal changes and protein losing enteropathy that lead to severe malabsorption.<sup>11</sup> A patient will present with sudden onset severe malnutrition, as well as alopecia, onycholysis and lentigines of the palms and dorsal hands. Lentigines have not been reported on the face or buccal mucosa.<sup>12</sup>

Laugier-Hunziker syndrome (LHS) is a benign acquired syndrome where 2-5 mm blue-black and brown macules appear as either solitary or multiple lesions commonly on the tongue and gingiva. They are seen after puberty and histologically look more like ephelides than lentigines, with increased melanin and normal melanocytes. Additionally, buccal mucosa and nails can be involved 60% of the time.<sup>13</sup>

## CONCLUSION

A 55 year old African American female presented with lentigines and other cutaneous and systemic abnormalities that raised suspicion for systemic disease. A full work up was obtained and all results were benign, ruling out familial lentiginosis syndromes. The lentigines on her face were not bothersome, and the patient opted to conservatively manage with monthly self-skin checks, daily sunscreen application and strict photo protection, and routine follow up with her dermatologist. If the patient had opted for skin treatment, her PCP could refer her to dermatology for intense pulsed light source (IPL) therapy, which has been shown to completely clear facial lentigines caused by PJS.<sup>14</sup> There are multiple other types of lasers that have also been shown to lighten or completely remove lentigines.<sup>15</sup> There are skin-lightening agents that can be prescribed that have been shown to lighten lentigines, but a

prescriber should be experienced in such agents and use with caution, especially if they are to be applied to face. Agents used alone, in combination therapy or as an adjuvant to cryotherapy include fluocinolone acetonide 0.01%, hydroquinone 4% and tretinoin 0.05%.<sup>16</sup>

Inherited patterned lentiginosis, a diagnosis of exclusion, is highly prevalent in the African American community, but has gained minimal attention in medical research and literature due to its benign nature. It poses no harm to patients, does not progress, and usually does not get formally diagnosed.<sup>1,3</sup> However, if a patient presents with the cutaneous presentation of inherited lentigines along with any of the signs and symptoms of an underlying systemic disease, it is imperative to be aggressive in ruling out other conditions, as lentigines may be the first indication of a more serious issue.

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## CALENDAR OF EVENTS

### 2016

#### July 27 - 31, 2016

FSACOFPP & FOMA Convention & Family Medicine Update  
Omni Orlando Resort  
Champions Gate, Florida  
[www.fsacofp.org](http://www.fsacofp.org)

#### August 3 - 7, 2016

TOMA & Texas ACOFP Joint Convention  
LaCantera Hill Country Resort  
San Antonio, Texas  
[www.txacofp.org](http://www.txacofp.org)

#### August 4 - 7, 2016

California ACOFP 40<sup>th</sup> Annual Scientific Medical Seminar  
Disneyland Hotel  
Anaheim, California  
[www.acofpca.org](http://www.acofpca.org)

#### August 4 - 7, 2016

MAOFP Summer Family Medicine Update  
Grand Traverse Resort & Spa  
Acme, Michigan  
[www.maofp.org](http://www.maofp.org)

#### August 5 - 7, 2016

POFPS 41<sup>st</sup> Annual CME Symposium  
Hershey Lodge  
Hershey, Pennsylvania  
[www.poma.org](http://www.poma.org)

#### August 11 - 14, 2016

North Carolina Society of the ACOFP Annual Meeting  
Courtyard Carolina Beach Marriot  
Carolina Beach, North Carolina  
[www.nc-acofp.org](http://www.nc-acofp.org)

#### August 12 - 14, 2016

ACOFPP Intensive Update & Board Review  
Loews Chicago O'Hare Hotel  
Rosemont, Illinois  
[www.acofp.org](http://www.acofp.org)

#### September 17 - 20, 2016

OMED 2016: ACOFP / AOA's 122<sup>nd</sup> Annual  
Osteopathic Medical Conference & Exhibition  
Anaheim Convention Center  
Anaheim, California  
[www.acofp.org](http://www.acofp.org)

#### November 3 - 6, 2016

Inaugural Joint IOMS Annual Meeting & Scientific Seminar  
Oak Brook Hills Conference Center  
Oak Brook, Illinois  
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#### December 2 - 4, 2016

IOA Annual Winter Update  
Sheraton Hotel at Keystone Crossing  
Indianapolis, Indiana  
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## PLANTAR FASCIITIS

Peter Zajac, DO, FACOFP, Author

Amy J. Keenum, DO, PharmD, Editor • Ronald Januchowski, DO, FACOFP, Health Literacy Editor



Plantar fasciitis is an inflammation of the tissue that extends from the heel to the ball of the foot. The plantar fascia absorbs the stresses placed on the foot during walking, running, and jumping. When irritated, the plantar fascia causes a sharp pain under the heel and sole of the foot. The pain is often worst when stepping onto the foot, especially when first getting out of bed in the morning or when getting up after being seated for some time. Plantar fasciitis is usually caused by shoes that do not fit, excessive athletic training, stress on the arch of the foot, weakness of the foot, or some combination of tightness of the foot and calf. Other possible factors that may increase the risk of plantar fasciitis include obesity, prolonged standing, and limited ankle flexibility.

### Medical Care & Treatment Options:

If you have any questions about plantar fasciitis, please contact your Osteopathic Family Physician. Your physician can diagnose plantar fasciitis with a thorough history and physical exam along with appropriate tests. Management includes the right treatment plan and regular visits with your doctor. Your family doctor will help you choose which current recommended treatment(s) will work best for you. In case of any emergency, you should call your doctor or 911 right away.

### Home Management Includes:

- Wear shoes with more arch support. Sometimes, arch supports or special shoe inserts may be used to cushion and support your heel.
- Athletes and active people may need to rest and limit activities that cause repetitive impact to the heel such as walking, running, jumping, and marching.
- Stretching and strengthening exercises are an important part of the treatment for plantar fasciitis and must be done daily at least until you have had no symptoms for three months. Some of these include:

**Plantar Fascia Stretch** – sit with your ankle resting on your opposite knee. Grasp your toes and pull them gently backward until a stretch is felt in the arch of your foot. Hold for 30 seconds and repeat six times. Doing this exercise two to three times a day will help prevent future episodes of pain.

**Towel Curl** – sit with your foot flat on the end of a towel placed on a smooth surface. Keeping your heel on the floor, pull the towel toward your body by curling up the towel with your toes.

**Marble Pick-ups** – put a few marbles on the floor near a cup. Keeping your heel on the floor, pick up the marbles with your toes and drop them in the cup.

**Toe Taps** – you will lift all your toes off the floor and, while the heel is on the floor and the four toes are in the air, tap the big toe to the floor. Next you will change the order and tap the outside four toes to the floor a number of times while keeping the big toe in the air. Start with 10 taps and work up to 50 taps per session.

- An ice massage or ice pack can help with inflammation. Rub ice over the painful heel using a circular motion and medium pressure for 5-10 minutes. An ice pack can be made by putting crushed ice in a plastic bag wrapped in a towel and molding it to the foot. Use the ice pack for 20 minutes up to four times daily. Applying ice to your heel after exercising, stretching, strengthening, and working can help with symptoms.
- A night splint, which also may be helpful, is a hard plastic splint that is worn at night to maintain your foot and ankle in a neutral position after stretching. Splints can be found at most drug or medical equipment stores and help speed healing.
- Other treatments, that your Osteopathic Family Physician may prescribe, include Osteopathic Manipulative Therapy, anti-inflammatory medication, cortisone shots into the heel to decrease pain, casting, shock wave therapy, and surgery. Please talk with your doctor about the possible benefits, risks, and side effects of these treatments.

**SOURCE(S):** American Family Physician, Plantar Fasciitis. Gov, and Up-To-Date.

The Osteopathic Family Physician Patient Handout is a public service of the ACOFP. The information and recommendations appearing on this page are appropriate in many instances; however, they are not a substitute for medical diagnosis by a physician. For specific information concerning your personal medical condition, ACOFP suggests that you consult your family physician. This page may be photocopied noncommercially by physicians and other health care professionals to share with their patients.

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The ACOFP congratulates Dr. Jan Zieren on becoming the 2016 Outstanding Female Leader Award, which was given to her during the ACOFP's 53<sup>rd</sup> Annual Convention this past April.

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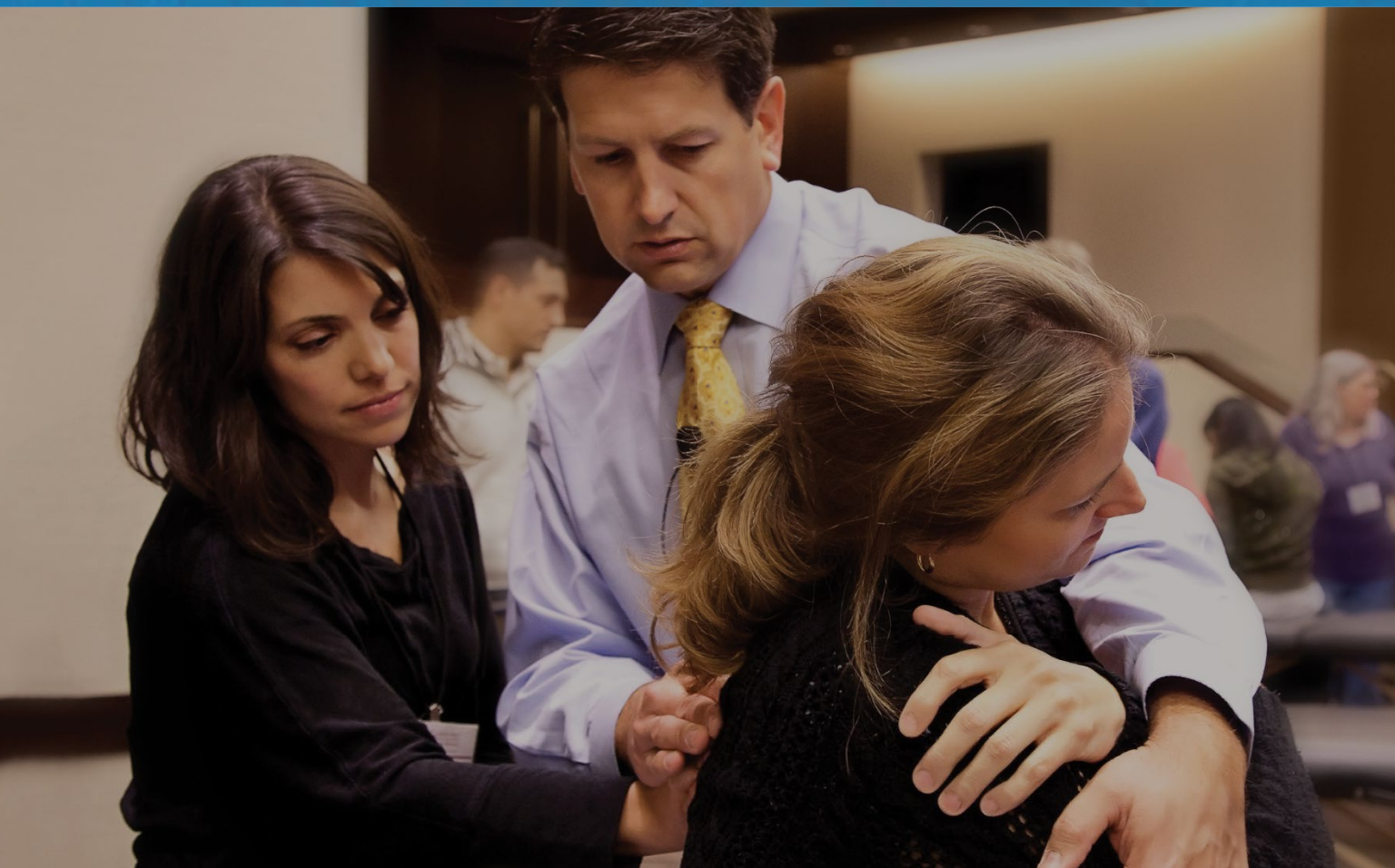


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