Contact Coral Dermatitis Affecting the Ankle

To the Editor:

Contact coral dermatitis has been described in the literature on several occasions. In 1941, Levin and Behrman[1] reported on a dermatitis caused by a stinging coral animal. They described the case of a 43-year-old man who was stung while collecting coral during deep-sea diving. His early symptoms consisted of a stinging sensation followed by the development of a pruritic erythematous, edematous, blistered area. An ulcer then developed; it healed after several months, leaving a thick scar. Later symptoms consisted of a lesion described as a 1-inch serpiginous band composed of flat and conical, variously shaped, reddish-purple papules that were extremely pruritic. The lesion was treated with bland lotions and roentgen radiation. After a total dose of 400 R, the pruritus subsided and the lesion disappeared, leaving a faint brownish pigmentation.

Camarasa et al[2] reported a case of a 26-year-old patient stung on the left arm while diving. The patient immediately experienced a severe stinging sensation followed by the development of a pruritic macular erythematous eruption, which cleared in 3 days with the use of a steroid cream. Blisters were not present. Forty-five days later, the sting sites showed streaks of erythematous, closely set papules 1 cm in diameter, with vesiculation. A perilesional and generalized pruritic papular reaction was also seen. Over the next 7 months, the patient had 3 separate episodes of a generalized pruritic papular eruption, which faded spontaneously in 7 to 10 days in each case.

Addy[3] reported on nine adults who developed coral contact dermatitis while pursuing aquatic recreational activities. In each case, the disease proceeded through four stages. Stage 1 consisted of acute urticaria; stage 2, acute vesiculobullous dermatitis; stage 3, subacute fleshy granulomatous dermatitis; and stage 4, chronic lichenoid dermatitis. Brief contact with the offending coral was followed by intense itching and sometimes a burning sensation. The itching continued after the patient emerged from the water and the area became erythematous and edematous, exhibiting an orange-peel texture. After a few hours, blisters with superficial exudation were noted. The acute dermatitis stage lasted several days and was followed by crusting and the subsequent eruption of pruritic, fleshy, red granulomatous lesions. The lesions gradually hardened, becoming smaller and shiny and assuming a lichenoid texture. The condition remained static and pruritic for several weeks, despite the use of topical corticosteroids. Healing was usually slow, taking up to 15 weeks and accompanied by fine desquamation and flattening of the papules. There was residual postinflammation hyperpigmentation.

The early symptoms reported after coral injury always include a stinging or burning sensation that may or may not be transient in nature.[1-6] In most cases, vesiculobullous eruptions of the injured area follow. The early symptoms usually resolve within a few days, only to reappear at varying intervals in intensified form. This period of recurrence of symptoms may last for several weeks to several months. A residual, prolonged postinflammatory hyperpigmentation is common. Although the dermatitis can affect any exposed part of the body, it is frequently noted on the arms and wrists, with approximately 47% of cases reported involving the upper extremity.[1-3] Twenty-seven percent of the cases involved the thigh and lower leg.[3, 5, 6] The remaining cases were spread evenly throughout the body.

Case Report

A 37-year-old woman presented with a pruritic rash over the medial aspect of her right ankle. She reported that the rash had been present for 12 days. Her medical history was unremarkable. Her social history was significant for a recent vacation in Puerto Rico, where she remembered scraping her ankle while snorkeling. She recalled experiencing an intense burning sensation in the area, but said that she thought she had sustained a superficial abrasion and that the burning was caused by the salt water. Later that evening, she experienced intense pruritus that was unrelieved by hydrocortisone 1% cream. Over the next few days, the area developed a brown eschar that gradually fell off, with the underlying skin appearing normal. The pruritus gradually returned over the next 12 days; it was most severe in the evening and when heat was applied, as during showering. The pruritus continued to be refractory to treatment with hydrocortisone 1% cream. The patient said that approximately 10 days after the initial injury, she developed a red plaque containing linear striations and noted simultaneous lesional pruritus. She pre-

sented with severe itching that was unrelieved by topical medications. She denied experiencing any constitutional symptoms.

Examination of the patient's right lower extremity revealed a 5 × 8-cm plaque on the medial aspect of the right ankle (Fig. 1). The plaque was composed of multiple small, red, well-demarcated, dome-shaped papules in a lichenoid pattern (Fig. 2). The lesion was shiny and erythematous but not warm to the touch. The integument was intact. No cellulitis, lymphangitis, or palpable lymph nodes were noted. The suspected cause of the patient's symptoms was allergic dermatitis due to coral envenomation. Treatment was with betamethasone dipropionate 0.05% cream applied twice daily under occlusive dressing for 2 weeks. At the follow-up visit, the patient reported that this regimen had resulted in some improvement of the lesion and the associated pruritus. Several days after discontinuing the medication, however, the patient returned to the office because the lesion and pruritus had returned. She was instructed to apply flurandrenolide tape over the area daily. Although her symptoms improved greatly with this regimen, the lesion did not disappear for 4 months, and a postinflammatory pigmentation remained.



Figure 1. Clinical photograph of patient's right ankle showing streaked plaque measuring 5 × 8 cm.



Figure 2. Close-up view of lesion depicted in Figure 1 reveals multiple small, red, well-demarcated, dome-shaped papules in a lichenoid pattern.

Comment

The appearance of multiple small, red, well-demarcated, dome-shaped papules in a lichenoid pattern 24 hours after contact with coral is consistent with coral dermatitis as described by Levin et al,[1] Camarasa et al,[2] and Addy.[3] The case presented here, like the others described in the literature, began with intense pruritus and redness but little or no heat. The dermatitis waxed and waned for several weeks, again as described by Levin et al.[1] In the other cases reported in the literature, the patient did not seek medical attention until many weeks to months after the injury; however, in the current case the patient presented soon after the injury. Most of the cases of contact coral dermatitis reported in the literature involve male patients, presumably because of a preponderance of males in the sport of deep-sea diving.

Acknowledgment. Bradley Lamm, a second-year student at the Temple University School of Podiatric Medicine, for research assistance.

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Cutaneous Angioleiomyoma

To the Editor:

Leiomyomas are benign neoplasms arising from smooth (nonstriated) muscle.[1] They are rather uncommon outside the uterus and gastrointestinal tract, but when they occur outside these areas, they are most frequently found in the subcutaneous tissue and skin.[2, 3] Within soft tissue, leiomyomas are usually confined to the superficial layers and occur only rarely in the deeper structures.[4] Most of these lesions arise from structures in these areas that normally contain smooth muscle, such as the arrectores pilorum, the tunica dartos, the erector muscle of the nipple, and the media of large and small vessels.[4]

These lesions may be classified as either superficial or vascular. Superficial leiomyomas are usually derived from arrectores pilorum, dartoid, vulvular, and mamillary smooth muscle. These neoplasms are sometimes also referred to as multiple cutaneous leiomyomas.[2, 3, 5] They can be found in any location but are most common in the extremities, the trunk, and the head and neck regions.[4]

Leiomyomas of the vascular or solitary type are usually larger than the nodules of the multiple type and tend to show no familial tendency, unlike the superficial type. These angioleiomyomas occur in the extremities in 90% of cases, with the majority in the lower extremity.[4]

Clinically, leiomyomas are smooth, freely movable, encapsulated lesions that vary in size from a few millimeters to approximately 3 cm.[3, 5-7] The nodules usually develop slowly into firm masses and vary in color from pink to brown.[3, 5] These neoplasms may have a translucent or waxy appearance.[5]

Leiomyomas are typically associated with pain that occurs spontaneously and may be paroxysmal in nature, with sharp, stabbing, or pinching qualities.[3, 5] The pain is usually attributed to the tumor's directly compressing a cutaneous nerve or to an external force that creates pressure on the lesion.[5, 8] However, it has also been suggested that the pain is related not to neural elements but rather to periodic transient contraction of the vessels, with resultant tissue anoxia.[9]

The etiology of vascular leiomyomas is uncertain. However, they are commonly attributed to the following causes: trauma, infection, hormones, familial and hereditary factors, arteriovenous malformations, and possibly induction of mesenchymal cells of loose connective tissue to form smooth muscle.[3, 10] The incidence of leiomyomas appears to be unrelated to race or ethnic group; they seem to occur most frequently in women between the ages of 30 and 50 years.[3, 11]

Case Report

A 38-year-old man presented with a chief complaint of a painful soft-tissue mass on the back of his right heel that had been present for 2 months. This lesion caused the patient a great deal of pain, which he described as a deep, local, aching type of discomfort. It was aggravated by pressure and certain types of footwear. The patient believed that the lesion was secondary to his driving 100 miles per day. He stated that the lesion had increased in size. He also reported that a similar lesion in the same region had been excised by his family physician a few months previously.

On physical examination, a protuberant soft-tissue mass was noted at the retrocalcaneal aspect of the right heel. The mass measured 1 cm in diameter and was approximately 5 mm high. The mass had an erythematous base with a rim of hyperkeratotic tissue. Palpation elicited mild-to-moderate discomfort. There was no evidence of drainage, odor, or sinus tract formation. The patient's general physical condition and health were good. The radiographic examination revealed no osseous involvement.

The patient was scheduled for surgical excision of the mass. The preoperative examination and laboratory findings were within normal limits. The lesion was removed under local anesthesia. The lesion was found by the pathology department to be consistent with angioleiomyoma (Figs. 1 and 2).

Discussion

Complete excision is the treatment of choice, with the rate of recurrence after this procedure low. However, in his study of ten patients with leiomyomas, Bulmer[12] noted four cases of recurrence despite apparently complete excision. In the case presented here, a similar lesion had been excised previously.

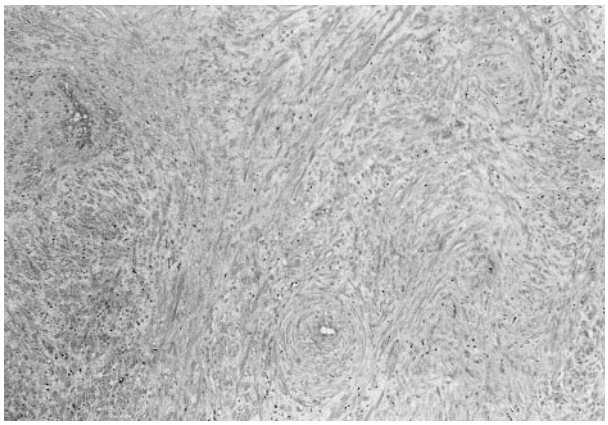


Figure 1. Low-power view of the leiomyoma showing nodular proliferation in the reticular dermis and the presence of vascular channels (H&E, ×100).

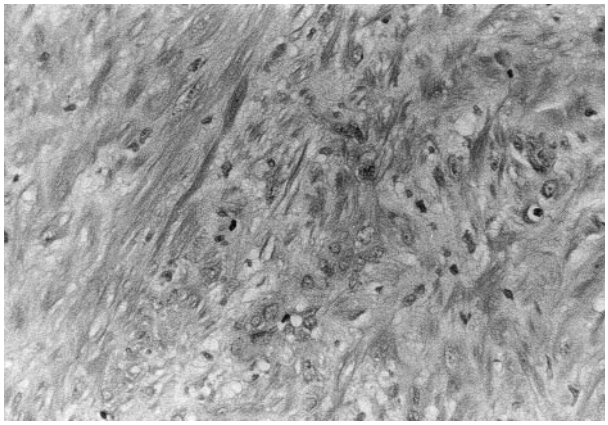


Figure 2. High-power view of the leiomyoma showing spindle cells with regular-to-oval nuclei and cytoplasmic vacuolation (H&E, ×400).

However, the procedure was performed by another physician, and it is unknown whether the excision was complete.

It is generally accepted that leiomyomas rarely become malignant.[3, 13, 14] Leiomyosarcomas account for only 2% to 12% of all soft-tissue sarcomas.[4] However, it has been reported that the lesion may undergo malignant change with metastasis to lymph nodes, bone, and viscera.[13] Thus proper follow-up and pathologic identification are essential.

Also noteworthy is that in the present case the initial diagnosis was a ganglionic cyst. Because leiomyomas often simulate ganglionic cysts, lipomas, fibromas, or glomus tumors, they are frequently misdiagnosed.[3] MacDonald and Sanderson[6] have stated that the incidence of leiomyoma is almost certainly greater than that of glomus tumors, which are more readily diagnosed in the differentiation of small, painful subcutaneous nodules. In fact, glomera are uncommon in the lower extremity except subungually, or on the dorsal aspect of a digit. Painless swelling in the region of a joint is likely to indicate ganglia.[12] Transillumination can aid in the differentiation of cysts and solid lesions. A thorough description of the lesions that may be confused with leiomyomas has been provided by Visalli and McCarthy.[15]

Summary

Leiomyomas are smooth-muscle tumors that may be encountered in the practice of podiatric medicine when they affect the arrector pili muscles or the smooth muscle of a vessel wall. In the present case, the lesion was located on the heel, an area with no hair growth; this further supports the diagnosis, as the most likely origin of the lesion was the smooth muscle of a blood vessel.[16] Vascular leiomyomas show a greater predilection for the lower extremities than their superficial counterparts. Surgical excision is the treatment of choice for these benign lesions, whose prognosis is excellent. When a patient presents with a painful mass in the lower extremity, the diagnosis of leiomyoma should be considered.

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Verifying Successful Off-loading of Neuropathic Pressure Ulcers

To the Editor:

Until recently, the verification of adequate off-loading in the treatment of neuropathic ulcers has been elusive in everyday clinical practice. However, a pressure sensor is now available to help podiatric physicians evaluate off-loading and give them more confidence in their methods. If there is adequate vascular supply, absence of active infection, and good metabolic control, healing of the neuropathic ulcer can be anticipated once the area has been successfully off-loaded (Fig. 1).[1]

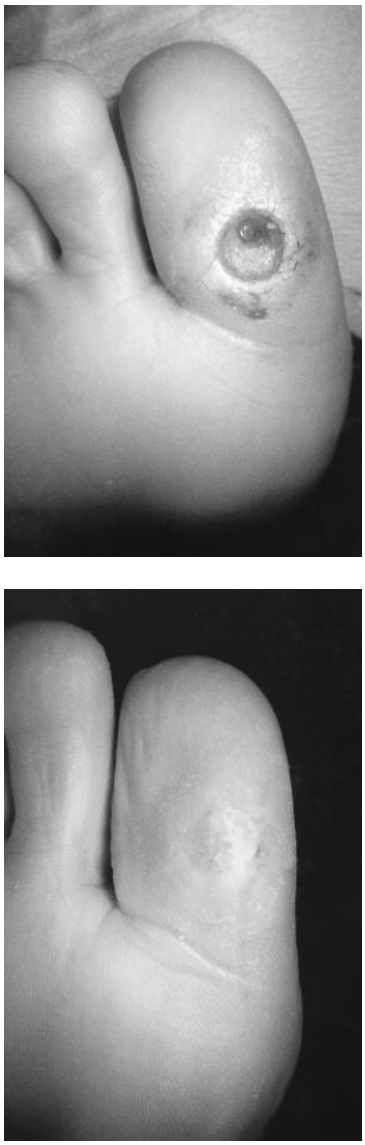


Figure 1. A, Plantar ulcer before off-loading; B, healed ulcer after successful off-loading.

Off-loading of the ulcer and redistribution of normal plantar pressures is the goal of treatment. Treatment modalities include felt or felted foam dressings, total contact casts, molded insoles, healing sandals, rocker-bottom soles, ankle-foot orthoses, and removable walking casts. An extra-depth shoe with a molded insole, a properly placed rocker-bottom sole, and a heel flare, if needed, is the most functional and cosmetically acceptable modality (Fig. 2).[2]

A molded insole of medium-density Plastazote (BXL Plastics Ltd, ERP Division, Croyden, England) is prepared in the following manner. Prior to application of a slipper cast to the foot, a piece of ½-inch-thick felt is cut and skived so that its thickest area covers the ulcer area, with a layer of adhesive tape holding it in place. This creates a positive mold with an enhanced prominence over the area. A 1-inch-thick piece of medium-density Plastazote is heated and form-pressed over this cast. After the mold has cooled, its proximal area is insulated by wrapping layers of towels 3 inches thick around it. It is again heated, and the distal aspect, which includes the ulcer area, is remolded over the cast with the heel cup positioned one-third to two-thirds of an inch proximal on the heel, allowing for elongation of the foot during weightbearing. The insole is then cut and shaped to fit the shoe.

The technique of diagnostic wiring of radiographs is used to determine the proper positioning of the insole beneath the ulceration-prone area (Fig. 3).[3] A



Figure 2. Molded insole, positive cast of foot, and extra-depth shoe with rocker-bottom sole.



Figure 3. Diagnostic wiring showing the desired position of the pressure point in the medial proximal portion of the concavity in the Plastazote insole.

direct weightbearing radiograph is taken of this area with the shoe and insole in place.

Until recently, there has not been any economical means of clinically verifying adequate pressure relief. The FWT Pressure Sensor (FWT Pressure Sensors, Hot Springs, Arkansas) is a small, hand-held unit that is easily used in the clinical setting. It is simply an ohm meter that has been converted to indicate pounds per square inch. The unit measures pressures on an area of approximately 1.2 square inches using a small, thin, round sensor that can be placed over any area on the foot where pressures are to be evaluated

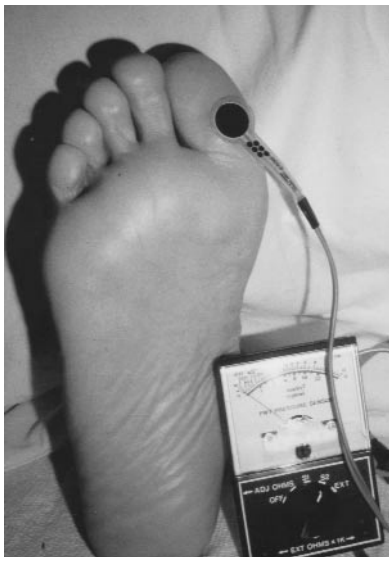


Figure 4. The sensor is placed at the point where pressure is to be evaluated.

(Fig. 4). The sensor is affixed to the treatment area with rubber cement and is covered with one layer of cohesive gauze. The lead from the sensor is brought up the side of the foot beneath the sock with no tension. Just above the sock the sensor lead is plugged into a cable that extends to the pressure gauge. The patient is asked to stand and walk while the readings are evaluated.



Figure 5. Observing peak plantar pressure during gait verifies effective off-loading.

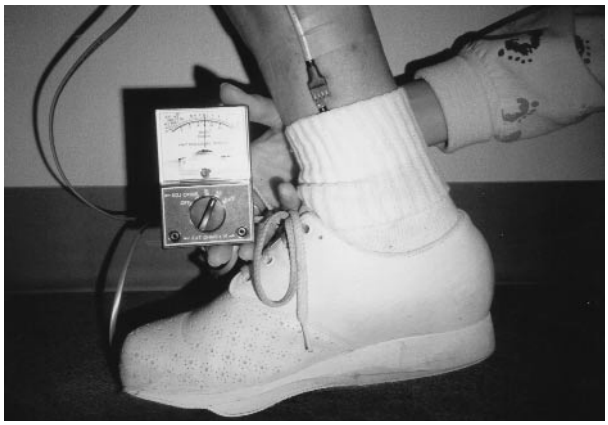


Figure 6. Measurement of peak plantar pressure during toe-off. A reading of 8 psi on the FWT Pressure Sensor usually permits healing of a neuropathic ulceration.

Use of the FWT Pressure Sensor allows the podiatric physician to immediately evaluate the effectiveness of the treatment modality and to make adjustments to ensure successful off-loading. The pressure sensor also aids in the proper placement of metatarsal bars and the apexes of rocker-bottom soles during gait (Fig. 5). The author has found that off-loading of peak plantar pressures to a level less than 8 psi as indicated by the FWT Pressure Sensor will usually allow for the healing of a neuropathic ulceration (Fig. 6). It must be remembered, however, that this device is not a sophisticated research tool but rather a clinical means of estimating relative pressures, so no precise claim can be made.

Use of this sensor enables one to anticipate good results with a much higher level of confidence while also allowing for documentation of effective off-loading. After gaining experience with this device, the podiatric physician can provide much more effective care than before. The FWT Pressure Sensor is bound to become an indispensable aid for any practitioner who treats patients with neuropathic plantar ulcers.

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The Long-term Effects of an Untreated Tibialis Anterior Tendon Rupture

To the Editor:

Despite the fact that the tibialis anterior tendon is the third most frequently ruptured tendon in the lower limb,[1] tibialis anterior tendon rupture has been infrequently reported in the literature since 1934. Most articles written on this subject have suggested two treatment options: surgical repair of the tendon when possible, or nonsurgical conservative treatment in patients who are poor candidates for surgery. These articles also report that there are very few long-term problems associated with an unrepaired tibialis anterior tendon. None of these articles discusses the long-term sequelae in a young patient who is not treated with surgical repair of the tendon.

This article reviews the literature to date and presents the case of a patient with a tibialis anterior tendon rupture that was undiagnosed until 25 years after the initial injury. The long-term consequences of this injury are then discussed.

Literature Review

In 1934, Burman[2] reported two acute cases of tibialis anterior tendon rupture. One case was treated surgically, and the other, involving an elderly patient, was treated conservatively. The conservatively treated patient was followed for 1½ years. He eventually developed a moderate rearfoot valgus, mild flatfoot, and strain of the medial side of the ankle joint of the affected side.

Mann[3] reviewed several surgical techniques in the fifth edition of *Surgery of the Foot* in 1986. He recommended conservative nonsurgical management of the tendon in a patient older than 60 years of age because the disability was not significant enough to risk the possible surgical complications. He also described mild progressive flatfoot in an elderly patient after spontaneous loss of function of the tibialis anterior tendon.

In 1988, Crosby and Fitzgibbons[4] discussed a case of tibialis anterior tendon laceration in a woman who was 65 years old at the time of injury. She was not diagnosed or treated for 8 years. They found the long-term effects to be mild loss of dorsiflexion strength with some pain at the anterior ankle that might have been due to compensation by the remaining anterior leg muscles. No treatment was suggested.

Benzakein et al[5] in 1988 discussed a tibialis anterior tendon rupture in a 57-year-old man that was neglected for 4 months. They found no abnormalities in the

patient's gait or in generalized muscle testing. The painful proximal end of the tendon was removed and was not reattached. Long-term follow-up care was not provided.

Mensor and Ordway[6] in 1953 and Lipscomb and Kelly[7] in 1955 reported on several cases, all of which were treated promptly with reattachment of the tendon. Floyd et al[8] briefly discussed tibialis anterior tendon rupture in 1983, citing surgical repair as the treatment of choice. Dooley et al[9] reported on four cases in 1980, three of which were treated surgically. In the case that was not treated, no long-term follow-up care was provided.

The most recently reported case was that of Rimoldi et al.[10] In 1991, they presented an acute rupture that was repaired surgically.

All of these articles reported success with surgical repair of tibialis anterior tendon ruptures that were discovered early in young, healthy individuals. The authors who discussed long-term effects did not believe that there were significant deformities or gait abnormalities associated with unrepaired ruptures of the tibialis anterior tendon.

Case Report

A 45-year-old man presented to the California College of Podiatric Medicine (CCPM) biomechanics clinic with a painful neuroma in the second intermetatarsal space of his right foot. He had been diagnosed with and treated for this problem at another clinic prior to coming to CCPM. He stated that over a 1-year period he had received three injections of steroid and anesthetic into the right third metatarsophalangeal joint. Each injection gave him complete relief of his symptoms for 3 months.

The patient complained of a burning pain in the second intermetatarsal space of his right foot. The pain worsened during activity and improved with rest. The pain was worst after a few sets of tennis, when he would also feel cramping of the toes of the right foot. The toes would curl up and would relax only after he massaged them. Weeks after the initial history was obtained and subsequent to much of the testing, the patient recalled that he had been in a motorcycle accident 25 years earlier and had sustained a right ankle injury. Although he had been in a cast for several months, he was not sure exactly which body part he had injured. Since the occurrence of that injury, he had not had pain in the anterior ankle or noticed any muscle weakness or walking problems. He did not recall any other injuries to his right ankle.

Physical examination showed pain with medial-to-lateral compression of the foot in the second intermetatarsal space near the metatarsal heads. An audible click was elicited when the intermetatarsal space was palpated. There was no pain with compression or distraction of the second or third metatarsophalangeal joints. The right foot was slightly flatter than the left and exhibited claw toe deformities of all of the lesser digits (Fig. 1). Upon manipulation of the third metatarsophalangeal joint, the proximal phalanx dislocated dorsally onto the metatarsal head and readily reduced. Despite normal articulation during nonweightbearing, the toe immediately dislocated dorsally during weightbearing.

Throughout gait, the digits of the patient's right foot were contracted and raised off of the ground. His subtalar joint was maximally pronated, and there was subluxation of the midtarsal joint. Bowstringing of the extensor digitorum longus and extensor hallucis longus tendons was observed on the right.

Muscle-strength testing was 5/5 in dorsiflexion, plantarflexion, inversion, and eversion bilaterally. Upon inversion testing, it was noted that, although the strengths were equal, the right foot had no visible or palpable tibialis anterior tendon (Fig. 2). More detailed muscle testing showed an overdeveloped extensor hallucis longus and extensor digitorum longus on the right. An H-wave electrical stimulation unit was applied to the origin and insertion of the tibialis anterior muscle, but no response was evoked. Instead, a very large extensor hallucis longus muscle fired, inverting the foot and dorsiflexing the hallux.

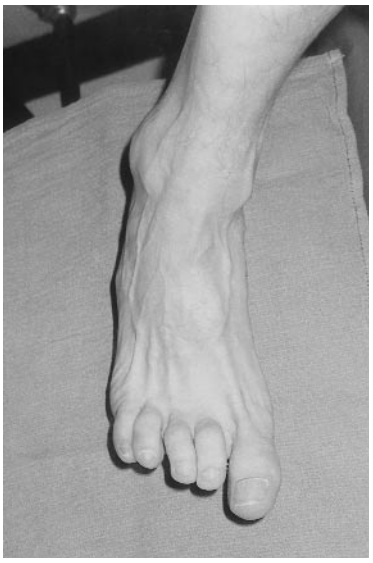


Figure 1. Right foot with contraction of digits 2 through 5 and prominent extensor hallucis longus tendon.

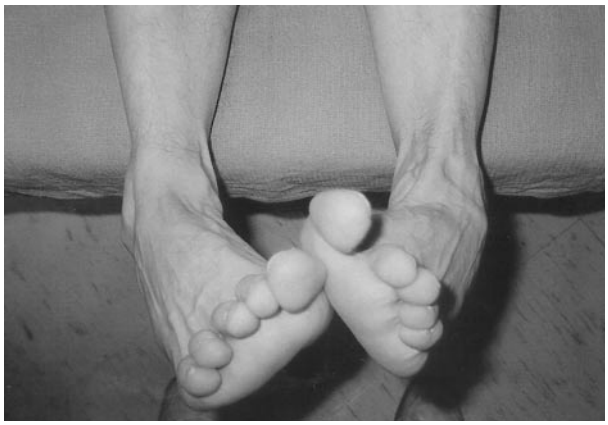


Figure 2. Absence of the tibialis anterior tendon in the right foot.

The following diagnoses were made regarding the right foot: a neuroma in the second intermetatarsal space, an absent tibialis anterior tendon, an easily dislocated third metatarsophalangeal joint, and claw toe deformity.

The patient was treated with custom orthoses with a metatarsal pad to dorsiflex metatarsals 2 through 5. He was also given a buttress pad for his third toe to keep the proximal phalanx from dislocating.

Approximately 1 month later, the third metatarsophalangeal joint was no longer easily dislocated and the neuroma pain had decreased appreciably.

Discussion

The patient described suffered an undiagnosed tibialis anterior tendon rupture, probably from his motorcycle injury 25 years earlier. It is very likely that this injury eventually caused, or at least contributed to, the development of a neuroma. While direct causation cannot be proved, the injury had significant long-term consequences.

The muscles that can dorsiflex the foot are the tibialis anterior, extensor hallucis longus, and extensor digitorum longus muscles. The tibialis anterior is the strongest of these three muscles for dorsiflexion of the ankle.[11] When it is lost, the extensor hallucis longus and the extensor digitorum longus must compensate for the lost strength. Over time, they will become stronger and adapt to better perform this task.

As these muscles overdevelop, the delicate balance between the intrinsic muscles and extrinsic muscles acting on the toes is disrupted. The extensor hallucis longus and extensor digitorum longus muscles become stronger than the flexors and intrinsic muscles, resulting in extensor substitution.[12] Extensor substitution is a common cause of hammer toes and claw toes. It can also be associated with bowstringing of the extensor digitorum longus tendons and the plantar prominence of the metatarsal heads.[12]

Connecting the metatarsal heads is the deep transverse intermetatarsal ligament. It is widely considered to be a major cause of neuroma development because it lies directly over the common digital branches of the medial and lateral plantar nerves, causing mechanical entrapment.[13, 14]

With extensor substitution, the metatarsals can be plantarflexed. The deep transverse intermetatarsal ligament attached to the metatarsal heads is also plantarflexed. When the ligament is forced downward, this causes increased forces on the common digital nerves immediately below the ligament. While these forces may not directly cause a neuroma, they would result in increased mechanical stresses on the nerve. The patient described here had relief of his neuroma symptoms when his metatarsals were dorsiflexed. This supports the idea that the deep transverse intermetatarsal ligament was causing excessive mechanical pressure on the nerve.

Another of the patient's symptoms was mild flatfoot on the affected side. The action of the tibialis anterior muscle is dorsiflexion and inversion. Its primary antagonist is the peroneus longus muscle, which acts at the medial column to evert and plantarflex the foot.[11] When the forces produced by the tibialis anterior muscle are lost, the peroneus longus muscle acts without resistance, causing medial column pronation and midfoot collapse. During gait, this problem manifests by causing even more work for the extensor hallucis longus and extensor digitorum longus muscles. Because their primary actions do not directly antagonize the peroneus longus muscle, they must fire with more force to compensate, further aggravating the extensor substitution.

The rigid functional orthosis that was made for the patient acted to prevent the pronatory actions of the peroneus longus muscle. Thus the orthosis reduced the effort exerted by the extensor hallucis longus and extensor digitorum longus to antagonize the peroneus longus muscle. In gait, this effectively reduced the extensor substitution and aided in relieving the neuroma symptoms.

Conclusion

The previous literature on rupture of the tibialis anterior tendon suggested that the problem could be treated surgically or, in an elderly patient, conservatively without significant consequences. The present

case demonstrates that, in a young person with this injury, adaptations can occur over time that may cause pain and other debilitating complications, including claw toes and neuromas. When this injury is discovered too late, there are very few treatment options for the initial tendon rupture owing to atrophy of the muscle. The authors' observations and treatment of these late complications suggest that early intervention with repair of the injured tendon would have resulted in a more satisfactory outcome.

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Bullae Formation Secondary to Carbon Monoxide Poisoning

To the Editor:

There is overwhelming agreement that bullous lesions associated with carbon monoxide poisoning typically appear within 24 hours and are found primarily on the palmar surface of the hands and the plantar surface of the feet.[1-3] Any areas of pressure or friction are vulnerable to the development of such lesions. These areas include the posterior scalp, wrists, knees, legs, ankles, and dorsa of the feet.[3, 4] No recent reports have directly associated bullous lesions with carbon monoxide poisoning. The case described herein is an unusual presentation of a secondary manifestation of carbon monoxide poisoning.

Case Report

A 37-year-old man presented to the emergency department after attempted suicide by carbon monoxide inhalation. After medical stabilization, the patient was transferred to the mental-health unit. Twenty-four hours after admission, the authors were consulted for evaluation and treatment of two painful bullae located on the left heel.

The patient stated that he had a history of depression and multiple suicide attempts. He had had a cerebral vascular accident secondary to a suicide attempt 4 years previously with no residual effects. He was taking the following medications: fluoxetine hydrochloride, trazodone hydrochloride, thiamine hydrochloride, and acetaminophen. The patient denied the use of alcohol, tobacco, or illicit drugs. He had no known drug allergies. He recalled no history of trauma or any occurrence of a similar lesion.

Upon physical examination, the patient was found to be alert, oriented to person, place, and time, and in no apparent distress. His vital signs were stable, and he was afebrile. The neurovascular status of both lower extremities was intact. The dermatologic examination revealed an 8×9-cm bulla on the plantar aspect of the heel as well as a 2×3-cm bulla beneath the cuboid, both on the left foot. A localized rim of erythema surrounded each bulla. Otherwise, skin temperature, texture, and turgor were normal.

Attention was directed to the bullae; the overlying skin was noted to be tense. Upon drainage, both bullae were found to be multilocular. The fluid was serous in nature. Cultures and Gram's stain performed at this time were negative.

Local wound care consisted of a dry, sterile dressing. The patient remained nonweightbearing with

crutch-assisted ambulation. The bullae were deroofed, revealing contents of an "apple jelly" color and consistency. No granular base was noted. There were no signs of infection. During the next 24 hours, there was copious serous drainage requiring large bulky dressings to be changed as many as ten times (Fig. 1). An estimated 4 L of serous fluid drained from the wounds. Profound epidermal and dermal lysis occurred beneath the initial bullae. Over the following week, the lesions continued to drain profusely and required well-padded dressings. Intradermal and subdermal bullae continued to form at the two sites during this period (Fig. 2).

Laboratory tests consisting of a complete blood count, chemistry profile, urinalysis, and wound cultures were performed, and the results were found to be within normal limits. The patient remained afebrile and stable. The wounds remained benign in appearance over time. As the wounds stabilized and the drainage decreased, wound care was modified (Fig. 3). Aluminum chlorohydrate soaks, followed by application of gentamicin 0.1% ointment, were instituted three times per day. The patient was discharged after 2 weeks with instructions for local wound care.

The patient was seen weekly in the outpatient clinic and a gradual, uneventful course of wound healing was noted. The patient remained nonweightbearing with crutch-assisted ambulation during the healing process. The wounds developed granular bases, which gradually epithelialized with complete resolution after approximately 8 months (Fig. 4).

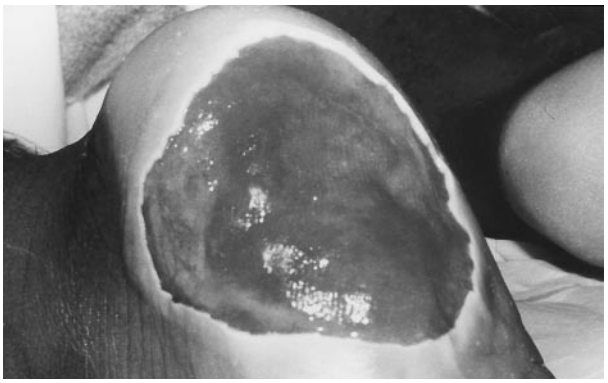


Figure 1. Day 3. Larger bulla (8 × 9 cm) fills the plantar aspect of the left heel and is actively draining a serous fluid.



Figure 2. Day 5. Bullae are observed on the lateral aspect of the left foot. Continued intradermal and subdermal blistering is apparent.

Discussion

The authors found no recent case reports directly associating bullous lesions and carbon monoxide poisoning. The literature on dermatologic lesions associated with carbon monoxide poisoning is scant. Three types of skin lesions have been described: 1) tense vesicular lesions with an erythematous base; 2) dusky, erythematous plaques; and 3) large, tense bullae bordered by a rim of localized erythema.[1] There have been very few reported cases of this latter type of bullae formation from toxic poisoning. These types of cutaneous manifestations have been found primarily in patients who lost consciousness after overdoses from barbiturates, methadone hydrochloride, hydrocodone bitartrate, meprobamate, imipramine hydro-



Figure 3. Day 14. New blister formation has ceased and drainage is greatly decreased.



Figure 4. Eight months. The lesions have healed and the patient is able to bear full weight without pain.

chloride, or glutethimide.[1, 2, 5, 6] It has been reported that only approximately 5% of patients with toxic poisoning develop associated blisters or bullae.[5]

Multiple hypotheses on the etiology of bullous formation in conjunction with carbon monoxide poisoning have been proposed. Four general theories predominate: 1) local hypoxia and trauma[1]; 2) pure toxic effect[7]; 3) pure local friction[2]; and 4) hypoxia, local pressure, and hyperthermia.[8]

No scientific research exists proving or disproving any of these theories. Biopsies of these lesions show significant and dramatic alteration and necrosis of the eccrine sweat glands within the epithelium.[1] Subepidermal bullae demonstrate intraepidermal spongiosis and vesiculations located peripherally about intraepidermal sweat ducts.[1] Interestingly, in the reported cases, reestablishment of apparently normal sweat glands occurred within 10 to 14 days.[1, 3, 5]

Conclusion

An unusual and rarely encountered clinical entity has been presented. The initial lesions, although locular, appeared benign in nature. An infectious process was considered unlikely. The lesions were initially thought to have been caused by localized trauma. It was not until the bullae began to form deeper layers of blistering, with increased drainage, that an extensive differential diagnosis including carbon monoxide–related bullae formation was considered. This case report aims to heighten the physician's awareness of the possibility of bullae formation due to toxic poisoning.

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