Paronychia in Patients Receiving Antiretroviral Therapy for Human Immunodeficiency Virus Infection

Editor's Note: Drs. Sibel, Macher, and Goosby are among the first investigators in the world to report on the occurrence of paronychia in patients receiving antiretroviral therapy for human immunodeficiency virus infection. We are pleased to have been asked by the authors to publish their findings in the Journal. *Because this condition is one that affects the feet, the authors are requesting that podiatric physicians who encounter paronychia in patients receiving antiretroviral therapy contact them so that they can develop a registry of these cases. Such a registry could prove invaluable in future research on the incidence, prevention, and treatment of this condition.*

To the Editor:

In 1998 and 1999, clinicians from Europe reported the first cases of paronychia occurring as a possible complication of antiretroviral therapy in patients infected with the human immunodeficiency virus (HIV).[1-4] In April 1998, Zerboni et al[1] from Italy reported on 12 HIV-infected patients, 11 men and 1 woman (mean age, 38 years; range, 31 to 54 years), who presented with paronychia. During the 3 months preceding the onset of paronychia, lamivudine was the only drug taken by all of their patients. Five patients had involvement of one great toe, five patients had involvement of both great toes, and two patients had fingernail as well as toenail involvement. The mean CD4+ T lymphocyte count was 350 cells per milliliter (range, 44 to 450 cells per milliliter), and the quantitative HIV RNA viral load ranged from 160 to 16,000 copies per milliliter. None of the patients had diabetes or any other risk factors for paronychia.

In June 1998, Bouscarat et al[2] from France reported on 42 HIV-infected patients who presented with paronychia of the great toes during the period September 1996 to September 1997. The 38 men and 4 women had a median age of 36 years (range, 22 to 64 years). Of the 42 patients, 36 had become infected with HIV through homosexual intercourse, 3 through intravenous drug use, and 3 through heterosexual intercourse. Thirty-six of the patients had acquired immunodeficiency syndrome. The mean CD4+ T lymphocyte count was 255 cells per milliliter (range, 16 to 892 cells per milliliter). The quantitative plasma HIV RNA was undetectable in 13 patients and ranged from 600 to 1,000,000 copies per milliliter in the other patients. At presentation, all 42 patients were receiving triple antiretroviral therapy, including indinavir. The median time between the start of indinavir therapy and the onset of ingrown toenails was 120 days (range, 30 to 360 days). Severe skin dryness was present in 28 of the patients. None of the patients had had prior episodes of paronychia, psoriasis, local trauma, or Kaposi's sarcoma.

In February 1999, at the Sixth Conference on Retroviruses and Opportunistic Infections, Bourezane et al[3] from France reported the development of ingrown toenails in 18 patients receiving indinavir; 6 of these patients also developed paronychia. In June 1999, Tosti et al[4] from Italy reported on six HIV-infected patients who developed paronychia during treatment with indinavir and lamivudine. Their lesions appeared 2 to 12 months after starting antiretroviral treatment.

The current authors report on 50 HIV-infected men who were referred to a podiatric medical practice in Washington, DC, from 1995 to 1999 when they developed paronychia while receiving antiretroviral treatment. The antiretroviral medications that were being taken by these 50 patients (aged 25 to 62 years) when they presented with paronychia are listed in Table 1.

The pathogenesis of paronychia in HIV-infected patients who are receiving antiretroviral therapy is unknown. Zerboni et al[1] suggested that lamivudine induced the paronychia in their patients. They also noted that drugs such as cyclosporin, 5-fluorouracil, and methotrexate have been reported to cause periungual inflammation even though their mode of action has not been clarified. Of the 50 patients with paronychia seen by the current authors, 14 were not receiving lamivudine.

Bouscarat et al[2] noted that pyogenic granuloma of nail folds and skin dryness may develop in patients receiving retinoid agents and that homologies between the amino acid sequences of retinoic acid–binding protein 1 and the catalytic site of HIV-1 protease have been reported. Bouscarat et al[2] suggested

Table 1. Antiretroviral Medications of 50 HIV-Infected Male Patients

Patient	Medications	Toe Involvement	Patient	Medications	Toe Involvement
1	Indinavir, stavudine, didanosine	Unilateral	26	Indinavir, zidovudine, nevirapine	Bilateral
2	Indinavir, stavudine, lamivudine	Unilateral	27	Indinavir, zidovudine, lamivudine	Unilateral
3	Zidovudine, lamivudine	Bilateral	28	Indinavir, stavudine, lamivudine	Unilateral
4	Zidovudine, nevirapine	Bilateral	29	Indinavir, zidovudine	Bilateral
5	Indinavir, zidovudine, lamivudine	Bilateral	30	Indinavir, stavudine, lamivudine	Unilateral
6	Indinavir, stavudine, nevirapine	Bilateral	31	Indinavir, zidovudine, lamivudine	Bilateral
7	Indinavir	Bilateral	32	Nelfinavir	Bilateral
8	Stavudine, lamivudine	Unilateral	33	Indinavir, stavudine, lamivudine	Bilateral
9	Zidovudine, lamivudine	Unilateral	34	Indinavir, zidovudine, lamivudine	Bilateral
10	Indinavir, stavudine	Unilateral	35	Indinavir, stavudine, lamivudine	Unilateral
11	Indinavir, zidovudine	Bilateral	36	Indinavir, zidovudine, lamivudine	Bilateral
12	Stavudine, lamivudine, nevirapine, nelfinavir	Unilateral	37	Indinavir, stavudine, lamivudine	Unilateral
13	Indinavir, stavudine, lamivudine	Bilateral	38	Indinavir, stavudine, lamivudine	Bilateral
14	Indinavir, zidovudine, lamivudine, stavudine	Bilateral	39	Indinavir, stavudine, lamivudine	Bilateral
15	Indinavir	Bilateral	40	Indinavir, stavudine, lamivudine	Unilateral
16	Indinavir, delavirdine, nelfinavir	Bilateral	41	Indinavir, stavudine, lamivudine	Bilateral
17	Indinavir, stavudine, lamivudine, ritonavir	Unilateral	42	Indinavir, stavudine, didanosine, nelfinavir	Unilateral
18	Indinavir, stavudine, lamivudine	Unilateral	43	Indinavir, stavudine, lamivudine	Unilateral
19	Indinavir, stavudine	Bilateral	44	Indinavir, zidovudine, lamivudine, delavirdine	Unilateral
20	Indinavir, zidovudine, lamivudine	Unilateral	45	Indinavir, stavudine, lamivudine	Bilateral
21	Indinavir, stavudine, lamivudine	Unilateral	46	Indinavir, zidovudine, lamivudine, delavirdine	Unilateral
22	Indinavir, stavudine, lamivudine, nelfinavir	Bilateral	47	Indinavir, stavudine, lamivudine	Unilateral
23	Indinavir, stavudine	Unilateral	48	Indinavir, zidovudine, lamivudine	Bilateral
24	Indinavir, stavudine, lamivudine	Bilateral	49	Indinavir, zidovudine, lamivudine, nevirapine	Bilateral
25	Indinavir, zidovudine, lamivudine, delavirdine	Bilateral	50	Indinavir, zidovudine, lamivudine	Bilateral

that inhibition of endogenous proteases may explain the initial hypertrophy of the nail fold and the subsequent development of pyogenic granuloma–like lesions. However, unlike the 42 patients in the study by Bouscarat et al,[2] all of whom were receiving indinavir when they developed paronychia, 6 of the 50 patients of the current authors were not receiving indinavir, and 4 of those 6 were not receiving a protease inhibitor.

The authors suspect that HIV-infected patients who have paronychia that is associated with antiretroviral treatment may be visiting the offices of podiatric physicians throughout the United States. The authors would like to create a collaborative registry and request that readers forward their reports of clinical cases to the second author listed below (via mail, fax, or e-mail) to help in this project.

STUART SIBEL, DPM
Podiatric Medicine and Surgery
Washington, DC

ABE MACHER, MD
HIV/AIDS Bureau
Health Resources and Services Administration
US Public Health Service
5600 Fishers Ln, Room 7A-55
Rockville, MD 20857
Fax: 301-443-8143
E-mail: amacher@hrsa.gov

ERIC GOOSBY, MD
Office of HIV/AIDS Policy
Office of Public Health and Science
Department of Health and Human Services
Washington, DC

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A Simple Cure for Morton's Neuralgia

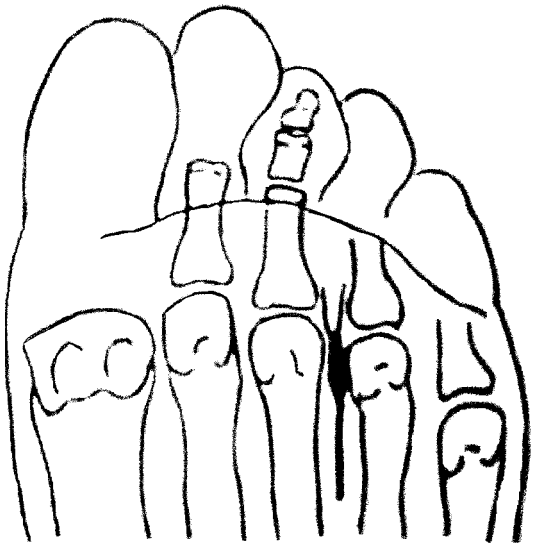


Figure 1. Morton's "neuroma," an inflammatory swelling of the interdigital nerve between the third and fourth metatarsals.

To the Editor:

Morton's neuralgia is a common cause of foot pain. The etiology is an entrapment of an interdigital nerve between two metatarsal heads, most commonly the third and fourth.

The metatarsal bones articulate proximally with bones of the tarsus, and the metatarsal heads articulate distally with the proximal phalanges. The metatarsophalangeal articulations are enlarged relative to the shaft of the metatarsals, and the interdigital nerves, which run between each two metatarsals, traverse this narrow space. The metatarsal bones are usually of different lengths so that two metatarsal heads are not in the same frontal plane. However, when two metatarsal bones are of the same length, which most frequently occurs for the third and fourth metatarsals, the space for the interdigital nerve is markedly narrowed. If, in addition, the patient wears narrow shoes, the interdigital nerve becomes entrapped between the two metatarsal heads. Continued irritation leads to an inflammatory swelling of the nerve, incorrectly called a "neuroma" (Fig. 1). This further increases the force of the entrapment. The pain occurs during walking and may continue when the patient is at rest. It is relieved only when the patient takes off his or her shoes.

Patients with Morton's neuralgia seen in the author's office frequently had received prior treatments, such as use of a metatarsal pad, injections, or surgical resection of the "neuroma." While there may be immediate relief of pain with such methods, recurrences are common. A cortisone injection obviously has only a temporary effect. Surgical resection has a longer effect, but eventually the nerve regrows and pain recurs. The commonly used orthotic correction is a metatarsal pad, but this has only the small effect of spreading the metatarsal heads.

The unconventional treatment method used by the author consists of elevating the fourth and fifth metatarsal heads by approximately 1/8 inch through the application of a 1/8-inch riser to an insole. This is done by marking the patient's fourth and fifth metatarsal heads with lipstick or ink and having the patient step on the insole, thus marking the exact location for the riser. Then a 1/8-inch pad is glued under the insole (Fig. 2). If the patient wears a foot orthosis, the lift can be incorporated. When the patient stands, the weight of the body forces the third metatarsal head down, thus separating the two metatarsal heads vertically and leaving sufficient space to free the "neuroma" (Fig. 3). Although this position of the metatarsal heads is not anatomically correct, it is well tolerated by all patients.

In most cases, the pain with weightbearing is completely and immediately relieved. In the remaining cases, there is immediate improvement, with pain disappearing completely within 2 weeks. There will be no recurrence if the patient wears this orthosis indefinitely.

Gerald G. Hirschberg, MD
1175 Keeler Ave
Berkeley, CA 94708

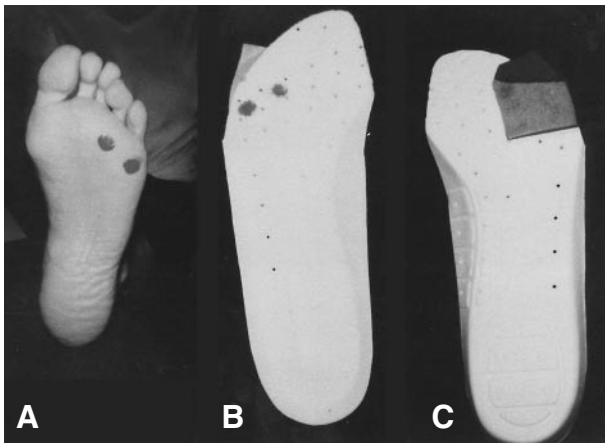


Figure 2. Procedure of placing the 1/8-inch lift under the fourth and fifth metatarsal heads. A, Looking at the sole of the involved foot, mark the fourth and fifth metatarsal heads with lipstick or ink. B, Align the patient's foot over an insole, and apply pressure to transfer the markings to the insole. C, Glue a 1/8-inch patch of cork, leather, or rubber on the underside of the insole directly under the markings.

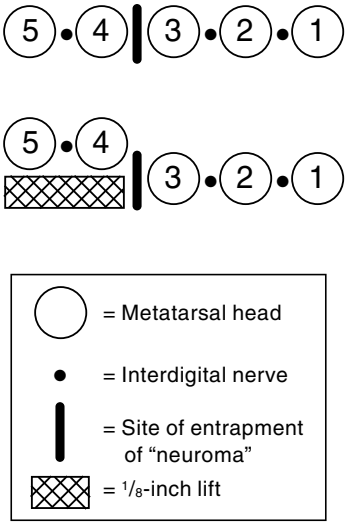


Figure 3. Diagram showing the effect of a lift under the fourth and fifth metatarsal heads. This lift markedly increases the distance between the third and fourth metatarsal heads, thus freeing the entrapped nerve.

Avulsion Fracture of the Plantar Lateral Base of the First Metatarsal

To the Editor:

Avulsion fractures of the plantar lateral base of the first metatarsal are known to occur with tarsometatarsal joint injuries that involve medial displacement of the first metatarsal. Hardcastle et al[1] documented the appearance of this avulsion fracture in combination with Lisfranc B1 and C injuries. Lisfranc B1 injuries are identified by partial incongruity caused by medial displacement of the first metatarsal alone or in association with medial displacement of one or more of the lesser metatarsals. Medial displacement of the first metatarsal with concomitant lateral displacement of one or more of the lesser metatarsals creates a divergent deformity classified as a Lisfranc C injury.

Hodor et al[2] were the first to describe an avulsion fracture of the plantar lateral base of the first metatarsal not related to a Lisfranc B1 or C injury. As the mechanism of action, the authors proposed a strong, sudden pronatory (abductory) force of the peroneus longus tendon on a planted first ray in a closed kinetic chain. The patients described in their article had difficulty recalling their foot position at the time of injury.

This case presentation will document a similar isolated avulsion fracture of the plantar lateral base of the first metatarsal and propose the mechanism of injury.

The peroneus longus muscle originates from the head and upper two-thirds of the body of the fibula and is the most superficial muscle of the lateral compartment of the lower leg. The tendon passes posterior to the lateral malleolus and runs deep to the superior peroneal retinaculum. As the tendon courses distal to the peroneal trochlea on the lateral aspect of the calcaneus, it travels deep to the inferior peroneal retinaculum. At the cuboid, the peroneus longus tendon continues plantarly and anteriorly along the peroneal groove of the cuboid, deep to the long plantar ligament. It advances medially and anteriorly to insert into the lateral aspect of the first metatarsal base and medial cuneiform.

The base of the first ray is not located in the same transverse plane as the cuboid but is positioned above the plantar surface of the cuboid. Only when this relationship exists can the cuboid function as a pulley for the tendon of the peroneus longus.

The primary functions of the peroneus longus muscle are stabilization of the first ray and stabilization of the head of the first metatarsal against the ground.[3] Contraction of the peroneus longus gener-

ates an abductory and pronatory force on the base of the first ray, stabilizing it against the lesser tarsal bones and thereby enabling the first ray to withstand normal ground-reactive forces. The peroneus longus muscle may also exert a strong plantarflexory force upon the first ray when the subtalar joint is in a neutral or supinated position.

Case Report

A 30-year-old man presented to the emergency department at Broadlawns Medical Center in Des Moines, Iowa, complaining of a painful left foot and ankle. The patient described an inversion ankle injury occurring the previous evening as he jumped over a fence. Upon landing, he felt a sharp pain in the arch and lateral ankle. Pedal pulses were palpable and light touch sensation was grossly intact. Moderate swelling was noted along the lateral aspect of the left ankle, extending across the plantar aspect of the foot. Maximum pain was elicited on palpation of the first metatarsal base–medial cuneiform articulation. Radiographs of the left foot revealed an oblique intra-articular fracture of the plantar lateral base of the first metatarsal. Fragment displacement measured approximately 2 to 3 mm, with alignment of the remaining first metatarsal considered anatomical (Fig. 1). Radiographs of the left ankle did not reveal osseous pathology. Later that day, the patient was taken to the operating room for open reduction of the fracture. The fracture fragment was reduced with the use of two 2.7-mm cortical screws in lag fashion (Fig. 2). After surgery, the patient was placed in a Jones compression dressing and discharged on crutches with instructions to remain nonweightbearing. The patient returned 5 days later for a dressing change and the application of a synthetic below-the-knee cast. He failed to keep subsequent appointments and was lost to follow-up.



Figure 1. Preoperative medial oblique radiograph of the left foot shows the avulsion fracture of the plantar lateral base of the first metatarsal.



Figure 2. Medial oblique (A) and anteroposterior (B) radiographs taken immediately after surgery. Note the fixation of the fragment with two 2.7-mm cortical screws. The anteroposterior view demonstrates the normal alignment of the first metatarsal base–medial cuneiform articulation.

Discussion

The first metatarsal base fracture described in this case is similar to the fracture reviewed by Hodor et al.[2] The history, clinical findings, and orientation of the fracture line through the first metatarsal base (perpendicular to the pull of the peroneus longus tendon with lateral displacement) suggest an avulsion fracture associated with an inversion ankle injury.

It is the inversion ankle sprain that provides the basis for the proposed mechanism of injury. Inversion of the ankle at the moment of impact increases tension on the peroneus longus tendon, translating into a traction force applied to the peroneus longus muscle belly. The sudden stretch of the muscle belly elicits a reflex contraction of the muscle known as the myotatic reflex.[4] If the injury is a high-velocity one, these combined actions increase tension at the

insertion of the peroneus longus tendon, ultimately resulting in the avulsion fracture of the plantar lateral base of the first metatarsal.

Other avulsion fractures associated with an inversion ankle injury are well documented in the podiatric and orthopedic literature. These include fractures of the tuberosity of the fifth metatarsal, malleoli, and anterior process of the calcaneus. This article has described an additional avulsion fracture occurring in combination with an inversion ankle sprain. Because of the intra-articular nature of the fracture and the likelihood of the development of post-traumatic degenerative joint disease, open reduction with internal fixation was performed.

Conclusion

Lateral ankle sprains are common, accounting for approximately 75% of all ankle injuries.[5] It is therefore particularly important to understand and thoroughly evaluate an injury. In addition to a comprehensive physical examination, appropriate diagnostic tests need to be done to ensure accurate assessment and treatment.

Even though avulsion fractures of the plantar lateral base of the first metatarsal not associated with tarsometatarsal Lisfranc injuries are rare, clinicians must be aware of this injury as an additional pathology related to lateral ankle sprain.

ROBERT M. YOHO, DPM, MS
HAL HATCHETT, BS
College of Podiatric Medicine and Surgery
Des Moines University–Osteopathic Medical Center
3200 Grand Ave
Des Moines, IA 50312

KARLON OLSON, DPM
Red Oak, IA

FRED GAITHER, DPM
Albuquerque Veterans Affairs Medical Center
Albuquerque, NM

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Retained Digital Foreign Body After a Pellet Gun Injury

To the Editor:

Foreign objects embedded in the foot are commonly seen in podiatric medical practice. Foreign bodies penetrate the foot in a variety of ways, ranging from a person's stepping on an object, usually while walking barefoot, to a projectile's entering the foot. The literature indicates that the most common types of foreign bodies encountered are of metal, glass, wood, hair, and thorns.[1-3] Except in cases of peripheral neuropathy, the individual is usually well aware that something may have penetrated the skin of the foot because of the associated pain. Occasionally, however, the individual is not aware of the injury. In 1991, Assalita[4] reported on a case of a foreign body that had gone undetected by the patient for 20 years. Obviously, when a patient does not recall an injury on initial presentation, a thorough history and physical examination are essential.

Unless a patient presents with an obvious foreign object or knows that something is lodged in the foot, it is easy to exclude a foreign body as a likely diagnosis. A symptomatic foreign body may present superficially as a mass near the skin, thus mimicking other types of lesions. Dowling[5] discussed the importance of considering the possibility of a ganglion or giant cell tumor. Newman and Hunt[6] emphasized the possibility of a granuloma. Hutchinson and Weir[7] found that foreign bodies may even mimic verrucae.

In certain circumstances, foreign objects embedded in the body's tissue cause no symptoms and go unnoticed until some of the chronic effects begin to manifest. Scartozzi and Hoffman[8] attributed these chronic effects to migration of the foreign body, neurovascular compromise, anemia, lead poisoning, and recurrent infection.

Once the foreign object has been identified and its size and location have been determined, the next step is to consider whether it must be removed. In 1993, Stein and Clark[2] proposed five criteria for determining the necessity of removal of foreign objects:

1) injury to vital structures, 2) evidence of infection or significant contamination, 3) an allergic reaction to the object, 4) pain caused by the object, and 5) the ability to superficially palpate the object.

This article presents a unique case involving a foreign object and discusses the circumstances surrounding its implantation as well as preoperative, intraoperative, and postoperative findings, including pathologic findings.

Case Report

A 34-year-old man presented to the Tower Foot and Ankle Institute in Des Moines, Iowa, with the chief complaint of a mass at the distal, medial aspect of his right fourth digit. The patient stated that this mass had been noticeable for several years, but had become progressively larger over the preceding 6 months. He denied experiencing any sharp pain, but stated that he had experienced some numbness, tingling, and burning toward the tip of his toe. Initially, he denied any trauma to the area. This was the first time he had sought treatment for the problem.

Physical examination revealed a firm, circular, immobile mass over the distal, medial, and slightly plantar aspect of the right fourth digit measuring approximately 1.5×1.5 cm (Fig. 1). Mild erythema and edema with a light ecchymosis overlying the taut digit were noted. No pain was elicited with direct or side-to-side compression of the area. With percussion, however, the patient complained of tingling toward the distal aspect of the right fourth digit. The remainder of the physical examination was unremarkable, as were the patient's medical and surgical histories.



Figure 1. Prominent mass over the plantar medial aspect of the right fourth digit.

Dorsoplantar and medial oblique radiographs of the patient's right foot revealed a radiopaque lesion, 4 mm in diameter, located just medial to the distal phalanx of the right fourth digit. Interestingly, the distal phalanx appeared to have grown around the object, with a striking concavity adjacent to the object (Fig. 2).

After the radiographs were reviewed with the patient, the patient recalled being shot in the foot with a pellet gun approximately 25 years before. The patient stated that he sought no treatment at the time of the injury, as there was minimal or no bleeding. He had assumed that the pellet caromed off his foot. At this time, the patient was scheduled for surgery to remove the pellet from his right fourth digit.

Surgical Procedure and Findings

The patient underwent surgical excision of the lesion, which initially revealed a brownish metallic color, similar to that of a penny, staining the subcutaneous tissue (Fig. 3). As dissection continued, the tissue began to appear very darkly stained. Deeper dissection resulted in significant oozing of what appeared to be a tannish purulent liquid with no detectable odor. Owing to the history behind the object, it was suspected that this fluid was the contents of a sterile abscess rather than bacterial in nature. Nonetheless,



Figure 2. Dorsoplantar radiograph demonstrating accommodative growth of the distal phalanx in response to the embedded pellet.



Figure 3. Intraoperative photograph of the encapsulated pellet.

a culture was obtained and sent to the pathology department for analysis. The pellet was then levered into the surgical site, where it appeared to be in one piece, and was removed *in toto*. The postoperative course was uneventful.

Pathologic Findings

Examination of the foreign body revealed a charcoal gray, spherical object approximately 4 × 4 mm consistent with a lead pellet from a pellet gun (Fig. 4). A Gram's stain of fibrotic tissue and purulent fluid obtained from the wound demonstrated no white blood cells or other inflammatory cells such as polymorphonuclear leukocytes or macrophages. However, a few gram-positive cocci were observed. A culture demonstrated light growth of coagulase-negative *Staphylococcus*.

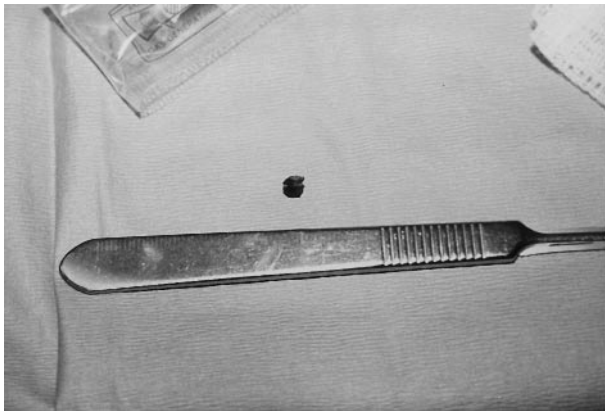


Figure 4. Excised pellet devoid of alloy coating.

Discussion

This case report is very similar to the one published in 1991 by Assalita[4] in that the patient did not recognize the initial traumatic episode and in the significant amount of time that elapsed before the pellet elicited a symptomatic response by the body's tissues. In both of these cases, the pellet's lying dormant for so many years without eliciting a response by the body is inexplicable. Assalita[4] attributes it to the complexities of the human body. The authors concur, as there is no definitive literature to date explaining this phenomenon.

Accidental pellet gun injuries are rather common, especially among school-age children.[9] Fortunately, these types of injuries to the foot are rarely life-threatening. Pellet guns produce low-velocity wounds; the projectile has a velocity of less than 2,000 feet per second and typically induces only an entrance wound, with no exit wound and no extreme damage to surrounding tissues.[10]

In cases of foreign-body penetration, the human body will attempt to either expel, encapsulate, or dissolve the foreign body.[3] In this case, a capsule was identified intraoperatively; it was composed of darkly stained fibrotic tissue, which, according to Fackler,[11] is part of the body's natural defense mechanism against the foreign body. The authors concur that the darkly stained tissue was attributable to a reaction that occurred between the outer coating of the pellet and the cells within the body. Most ammunition, including pellets, is coated with metal alloys that minimize deformation and tissue damage on contact.[10] In this case, the pellet's outer covering was completely removed; this further suggests that some type of reaction occurred. To the authors' knowledge, there is no literature defining the mechanism of the reaction between tissue and the metal alloy that forms the outer covering of pellets. Fackler[11] stated that if the body fails to expel this fibrotic capsule, it will become an abscess. Indeed, in this case, a sterile abscess had formed, although nothing in the patient's history or physical examination was suggestive of infection.

As seen in the pathology review, no white blood cells, polymorphonuclear leukocytes, macrophages, or other inflammatory cells were present. Assalita[4] used the term "foreign body granuloma" to describe the body's response to the foreign body. Granuloma is a generic term to describe the aggregation of mononuclear inflammatory cells or a collection of modified macrophages.[12] Given the pathology results

in this case, the authors cannot consider the term "granuloma" to describe the response to the foreign body. Furthermore, in the pathology review, it was noted that a few gram-positive cocci were observed and that a culture produced a light growth of coagulase-negative *Staphylococcus*, which is probably *Staphylococcus epidermidis*, a component of normal skin flora.[13] These results differ from those obtained by Dowling,[5] who observed many coagulase-positive *Staphylococcus* organisms along with some *Escherichia coli* organisms in a case where a wooden toothpick was the offending foreign body. Perhaps this difference is due to the two different materials in question, the metal pellet and the wooden toothpick, or perhaps it is due to the situation that led to penetration by the foreign object.

As mentioned previously, embedded foreign objects sometimes remain dormant for years until one of the chronic effects begins to manifest and symptoms appear. One of the reasons this patient sought treatment when he did was that he was experiencing occasional numbness, burning, and tingling toward the distal aspect of his right fourth digit. Clinically speaking, he was describing neurapraxia. Neurapraxia refers to the impairment of the conduction of nerve impulses and is usually completely reversible.[14] This patient suffered impairment of the seventh proper plantar digital nerve. At a 1-month follow-up visit, the patient stated that, for the most part, the feeling had returned to the medial aspect of his toe, and he no longer experienced paresthesia.

Summary

A symptomatic foreign body embedded in the human body can be a frustrating problem for physician and patient alike. A unique case of a retained foreign object resulting from a pellet gun injury has been presented. Although the course of treatment in this case was uncomplicated, it is important to understand the complexities of the human body's response to foreign bodies.

ANDREW VINCENT, BS
DAVID W. JENKINS, DPM
College of Podiatric Medicine and Surgery
Des Moines University–Osteopathic Medical Center
3200 Grand Ave
Des Moines, IA 50312

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