Surfer’s Toe: Trauma-Induced Idiopathic Acro-Osteolysis in the Toes of a 46-Year-Old Surfer

A Case Report

Larisa M. Lehmer, MA*
Bruce D. Ragsdale, MD†
Daniel Hoffman, AAS‡
Steven J. Clark, DPM§

Acquired acro-osteolysis (AOL) is defined as the resorption of bone from the tufts or shafts of the terminal phalanges. Acquired acro-osteolysis can manifest as a primary osteolysis syndrome and also appears in a number of disease states including rheumatologic disorders, neuropathic diseases, the result of prolonged exposure to polynvinyl chloride, and in rare cases, as a response to repeated mechanical stress. In this report, a 46-year-old surfer was evaluated for AOL as a complication of sports-related repetitive trauma to the right second and third toes. Radiography showed the bony tips of his right second and third toes had been eroded away. Acquired acro-osteolysis in the surfer’s toes resulted from increased blood flow initiated to repair microdamage caused by repeated trauma to the distal ends of his second and third right toes due to the habitual dragging of the affected toes across a surfboard. The always initial lytic phase of bone repair was magnified by the increased arterial input to warm the extremities after prolonged exposure to cold. At 6-years’ follow-up, the use of a protective bandage while surfing has permitted full regeneration of the affected toes. (J Am Podiatr Med Assoc 102(2): 165-166, 2012)

Acro-osteolysis (AOL) refers to the dissolution of bone at the distal ends of the extremities, specifically the phalanges, as a result of vascular (inflammatory or noninflammatory), neurovascular, metabolic, toxic, or mechanical processes. Clinical features include swollen fingertips and watchglass nails. Radiographically, AOL is characterized by tuftal resorption often accompanied by zones of “bandlike” areas of radiolucency across the waist of one or more terminal phalanges.

Case Report

The patient (and co-author, D.H.), a 46-year-old male, has been surfing since age 13, and for the past 26 years has surfed 6 to 10 hours a week in the chilly 8 to 12° C (47 to 54° F) waters off California’s Central Coast. The patient reports the cold temperature of the water causes a mild numbness of the hands and feet such that he is unaware of the occasional cuts and bruises he has sustained on his feet until after he has come ashore. Over the 40 years and counting that he has surfed, he has used a “goofy foot” stance (right foot forward on the board, the left foot to the back) (Fig. 1). In transition to standing, he drags his right foot forward across the board. At age 46, he started to notice some mild pain, swelling, and deformity of the nail on his right second toe. A radiograph showed the bony tips of his right second and third toes had been eroded away. He developed second-toe weakness occasionally forcing him to limp, and later, greater swelling and severe pain that spread through his entire foot.

Symptoms of mild pain, nail bed and joint deformity, and swelling in the right second toe began at age 46. Two months after symptoms started, radiographs were taken demonstrating lucency consistent with acro-osteolysis of the second and third right toes. Symptoms progressed to severe pain occasionally affecting his entire foot and swelling of the two affected toes. The develop-
ment of second-toe weakness intermittently induced a limp. The toes were a maroon red with evidence of mild bruising after surfing. The surfer noted that cold water aggravated the condition, because he experienced little to no symptoms while surfing for 6 hours a day for 12 days in the balmy 26° C (80° F) waters off the coast of Mexico. He went to a podiatric physician for the swelling and weakness in his toes.

Radiographically discovered lytic change (Fig. 2A) motivated needle biopsies of the second and third terminal phalanges of the right foot under a clinical diagnosis of neoplasm vs osteomyelitis. The second toe distal phalanx biopsy revealed fracture and repair (Fig. 3A), and in the third toe, osteoclasts were clearing out injured trabeculae while osteoblasts were compensating with new bone on trabecular surfaces (Fig. 3B). Neither biopsy showed osteomyelitis or neoplasm. Cryoglobulinemia was ruled out histopathologically because of an absence of the characteristic thrombi.

To protect it from further injury, the patient routinely wrapped a strip of moleskin and self-adhesive nylon bandage around his second toe but did not alter his surfing habits or routine in any other way. Follow-up at 6 years shows radiologic reconstitution of the second and third toe distal phalangeal tufts (Fig. 2B) while chronic mild pain and weakness persist to the present.

Discussion

The mechanism resulting in AOL can be any of the three basic influences that explain all changes in bone density: vascular, metabolic, or mechanical (ie, trauma or stress).

Vascular

Vascular—Too little blood flow occurs in vasculitic and vaso-occlusive infarction in rheumatoid arthritis, scleroderma, severe Reynaud's syndrome, polyvinyl chloride toxicity, and systemic lupus erythematosus, periarteritis nodosa, polymyositis, serum sickness, Takayasu's disease and Wegener's granulomatosis. Reduction of blood flow can also accompany primary vascular lesions, such as atherosclerosis, Buerger's disease, and diabetic osteopathy. Angiography in patients with rheumatic disease, scleroderma, severe Reynaud's syndrome, and other vascular disorders, and who have erosions of terminal tufts, can reveal multiple vascular occlusions and stenoses, most prominent in the proper digital arteries adjacent to points of manual stress. In all of the above, the sequence is bone infarction followed by resorption of dead bone.

Excessive flow, ie, active hyperemia, drives mitochondria-rich osteoclasts in neurovascular dysregulation in diabetes, leprosy, Sudeck's atrophy, and causalgia (reflex osteodystrophy). In psoriatic arthritis, AOL usually occurs with typical distal interphalangeal joint synovitis, because the bone shares the same vascular trunk as the synovium. Although osteoarthritis often involves the distal interphalangeal joints, true AOL is an uncommon finding in wear-and-tear osteoarthritis.

Metabolic

Metabolic—hyperparathyroidism with elevated parathyroid hormone classically causes resorption of phalangeal tufts.

Mechanical

Mechanical—trauma (ie, fracture), pressure erosion (neoplasms and cysts), tight skin (progeria, Rothmund's syndrome, epidermolysis bullosa), or a fibrous band of chronic inflammation in ainhum. In a 110-patient survey of fractures of the distal phalanges of the hands, DaCruz et al 11 observed osteolysis only in comminuted tuft fractures in 20 patients at 3 months, and 26 patients at 6 months with an 8% full-recovery rate at 6 months' follow-up, compared with a 28% recovery rate for non-osteolysis comminuted tuft fractures.

Whatever the trigger, necrosis, unabated active

Figure 1. "Goofy foot" surfing stance demonstrated by patient (co-author, D.H.).
hyperemia, or mechanical damage, AOL is essentially an over-expression of the homeostatic process of bone resorption in physiologic turnover. An increase in arterial blood flow (active hyperemia) shifts local bioelectric charge to the positive and elevates the partial pressure of oxygen to favor the modulation and activity of mitochondria-rich osteoclasts that carry out the osteolysis.

Primary idiopathic osteolysis is very rare and may continue for years, resulting in severe deformity and serious functional disability until eventually ceasing spontaneously. Histologic studies of osseous lesions in patients with primary osteolysis syndromes reveal fibrovascular marrow transformation without cellular inflammation. More relevant to the present case, AOL was reported in the left fingers of a healthy, 18-year-old guitar player as well as in the left phalanges of an 8-year-old guitar player diagnosed with rheumatoid arthritis. But perhaps incorrectly, both instances were deemed to result from repetitive finger placement and pressure on steel guitar strings leading to osteonecrosis.

In the present case of the surfer, AOL was the
result of increased blood flow initiated to repair microdamage caused by repeated trauma to the distal ends of the second and third right toes attributable to the habitual dragging of the affected toes across a surfboard. The always initial lytic phase of bone repair was magnified by the increased arterial input to warm the extremities after prolonged exposure to cold.

The evolution of thought in orthopedic pathology has seen a trend to initially ascribe various peculiar bone changes to “osteonecrosis,” only to have the true mechanism later clarified as something else: witness Freiburg’s infraction (a fracture), Osgood-Schlatter’s disease of the tibial tubercle (avulsion of the patellar ligament), Kienbock’s disease (lunate fractures), etc. Destouet and Murphy propose that vascular occlusion explains the bands of osteolysis across terminal phalanges in their guitar player while stress reaction and microfractures likely explain them, as Young et al. proposed for their guitar player. The associated “swelling” (edema) described in both musicians’ fingertips would not be expected with vascular compromise. Maturation in thought regarding etiology should proceed in regard to acro-osteolysis attributable to repeated mechanical stress (trauma), ie, matrix wear plus gross or microscopic fractures evoke active hyperemia to support accelerated remodeling and repair, the initial phase of which is always osteolysis.

Conclusions

“Surfer’s toe” joins the list of other surfing-related sports maladies such as surfer’s asthma, surfer’s knots/nodules, surfer’s ear, surfer’s rib, surfer’s elbow, surfer’s myelopathy (nontraumatic), and surfer’s neuropathy.

Financial Disclosure: None reported.
Conflict of Interest: None reported.
References