CHAPTER 25

METABOLIC AND ARTHRITIC CAUSES OF PAINFUL HEEL

Sanford Hartman, M.D.

While the vast majority of cases of a painful heel are purely mechanical in nature, and therefore potentially responsive to either conservative or aggressive correction of the mechanical abnormality, there are distinct subsets of other causes that may either not be truly mechanical or that may be mechanical, but of such etiology that straightforward correction would represent a minimal approach to a much greater overall problem.

The painful heel itself is a generic description of multiple possible areas of involvement, and it is obviously imperative that the specifically involved tissue be determined before any corrective approach, be it medical or surgical, can be undertaken. The painful area could lie in such diverse tissues as the Achilles tendon (Achilles tendinitis), calcaneus (calcaneal spur), retrocalcaneal bursa (retrocalcaneal bursitis), calcaneal fat pad (traumatic panniculitis), plantar fascia (plantar fasciitis) or skin (blister). Once the area is determined, it is then incumbent upon the physician to determine why it has become painful. Common causes include trauma (either abrupt or repetitive), degenerative changes, inflammatory conditions (septic or non-septic), and abnormal metabolic states, while less common causes would include certain other metabolic, heredofamilial, infectious, or inflammatory conditions. As the endpoint or end-organ (i.e., the heel) is the same in all states under discussion here, it is the specific etiology that must be sought and discovered, so that adequate therapeutic and preventive measures can be instituted in proper sequence.

Of the many causes, trauma is certainly the most common, and if one is arbitrarily categorizing all causes as either requiring the care of the surgeon or the non-surgeon, this cause most frequently falls on the surgical side of the ledger. Straight-forward Achilles tendon rupture, retrocalcaneal bursitis, exostoses, plantar fasciitis, calcaneal fat pad degeneration, shoe trauma to the skin, and bursitis between the tendo Achillis and the skin would only be seen and treated by the non-surgeon insofar as rest and/or local injection may be helpful; otherwise the patient should be referred to a physician more experienced in such matters. Degenerative joint disease (DJD or osteoarthritis) could be seen by either the surgeon or non-surgeon, but when it is symptomatic in the heel, there is very little the medical approach is likely to accomplish.

Other common causes of heel pain include inflammatory and metabolic conditions. Inflammatory diseases, for purposes of this discussion, would be classified as either septic or non-septic, and if sepsis is present, a close medical-surgical approach and cooperation is mandatory, especially if the offending agent is bacterial or tuberculous. This is true whether there is septic bursitis, tendinitis, fasciitis, or osteomyelitis. Some debate exists in regard to the non-septic inflammatory causes, but in general these are medically treated diseases. The surgeon's role is one of supplementing the medical approach, since mechanical correction is not likely to be permanent, totally satisfactory, or complete, although palliation for long periods of time or for specifically involved areas is often helpful and necessary.

Examples of diverse causes of heel pain in a patient with rheumatoid arthritis (RA) might include an Achilles tendinitis, a rheumatoid nodule, or an Achilles tenosynovitis with erosion at the calcaneal insertion. The seronegative spondyloarthropathies may also present with different causes of heel pain, including retrocalcaneal bursitis, plantar fasciitis, bursitis between the tendo Achillis and skin and even with specific types of exostoses. The gamut of these conditions covers the most common ankylosing spondylitis with extremely rare heel involvement, the less common enteropathic or psoriatic arthritides with heel involvement more likely, to the patient with Reiter's syndrome with the rather classical periostitis of the os calcis (Lover's heel).

The common metabolic causes include gout, which may certainly attack the heel area or involve that area with tophaceous deposits, pseudo gout which uncommonly attacks the heel unless hyperparathyroidism is present, hydroxyapatite crystal

deposit disease which seems to have particular affinity in the heel for tendons, and finally, type II hyperlipoproteinemia (hypercholesterolemia) with xanthomatosis (tendinous, periosteal, or of the plantar fascia).

With all of these more common inflammatory causes, surgical intervention is likely to be necessary only in those patients in whom medical therapy has failed, those that have the most severe cases, or those that have been less than ideally compliant with medical approaches.

Some rules of thumb to bear in mind in regard to the more common cause of heel pain include the following:

1. If DJD exists out of proportion to what one expects for the age and/or sex of the patient, consider unusual causes of degenerative disease such as hemochromatosis.
2. Always consider sepsis in the singly-involved acutely inflamed area.
3. As noted previously, there are multiple causes of heel pain in rheumatoid arthritis, and excised rheumatoid nodules frequently regrow.
4. The only seronegative spondyloarthropathy in which heel pain is a common symptom is Reiter's Syndrome; always ask about eye inflammation, urethral discharge, arthritis, and skin lesions in the young male with a painful heel for which no obvious etiology exists. The HLA B27 can be helpful in such individuals, but has marked limitations which must be borne in mind:
   a. While it is found in about 92% of ankylosing spondylitics, it is found only in 60-80% of Reiter's Syndrome patients.
   b. HLA B27 is of particular value only in the white population (to whom the statistics above apply); the percentages are much lower in blacks.
   c. 8% of the normal white population is HLA B27 positive.
   d. The test is not needed if you are sure of the diagnosis (either positively or negatively).
   e. Be prepared to answer or refer-for-answers questions about genetic tendencies in other family members of those patients that are B27 positive.
   f. The test must be run on plasma (not serum) that has not been refrigerated.
   g. At a cost of about $160 or $165, it is important to know, and let the patient know, the limitations of a positive or negative result.
5. An acute attack of gout requires no surgical intervention; chronic gouty arthritis or trophi suggest either severe disease or a lack of compliance, or both.
6. Since pseudogout (calcium pyrophosphate dihydrate or CPPD) is much more common in the knee or wrist, and in an older population, various underlying diseases must be considered when it is found in the heel, especially hyperparathroidism; a long list of other causes can be found in Table 1, some of which one will undoubtedly encounter over the lifetime of a practice.
7. Hydroxyapatite crystal deposit disease is usually of the heel, ankle, or knee, and patients are usually younger than those with gout or CPPD; unfortunately a definitive diagnosis requires electron microscopy.
8. Patients with xanthomatous disease in tendons associated with hypercholesterolemia will usually have the xanthomas recur after excision, and also often have multiple other medical problems; removal should be undertaken if necessary, but cosmesis is probably not an indication.

There is a long list of metabolic or heredofamilial diseases that can be associated with heel pain, but since these are primarily medical diseases, and are likely to have already been diagnosed by the time the patient is referred to the surgeon, they are merely listed in Table 2. Occasionally, however, a patient will have only minimal expression of such conditions, and so it is necessary to be attuned to the patient who does not appear quite normal for his or her sex, age, symptoms, or other findings. Of the long list in Table 2, you will likely at one time or another see at least an occasional patient with amyloidosis, osteogenesis imperfecta of some form, and/or hemophilia.

The rare infectious causes of heel pain are just that, rare, and are listed for interest in Table 3. Only actinomycosis is seen in the heel with any frequency at all, and that is primarily in Central or South America. Involvement is by direct invasion (Madura Foot).

The uncommon non-septic inflammatory causes of heel pain are outlined below because, despite the fact that they are uncommon, it would be unusual to go through the lifetime of a surgical practice and not see at least a few patients with each condition (except possibly for scleroderma).

Juvenile Rheumatoid Arthritis (JRA)

Juvenile rheumatoid arthritis of the polyarticular variety not uncommonly has heel involvement, but it would rarely be the presenting symptom. Those patients are also more likely to have nodules which clinically and pathologically resemble those of acute rheumatic fever rather than rheumatoid arthritis.

Sarcoidosis

In sarcoidosis, heel pad pain is not uncommonly encountered and ankles and knees are also often involved, although this is not likely to be the presenting complaint.

Acute Rheumatic Fever (ARF)

Acute rheumatic fever will frequently have nodules on the Achilles tendon which are painless, last between 7 and 30 days, and are usually felt by the patient before they can be seen.

Gonococcal (GC) Arthritis

Gonococcal arthritis can involve the heel either with the well-described superficial skin lesions, usually pustules, or with the peripheral arthritis itself.

Calcinosis Universalis or Calcinosis Circumscripta

Finally, calcinosis universalis or calcinosis circumscripta are often accompanying signs of various inflammatory connective tissue diseases, most frequently progressive sclerosis (scleroderma), the CREST syndrome (Calcinosis, Raynaud's, Esophageal motility dysfunction, Sclerodactyly, Telangiectasia) or polymyositis/dermatomyositis. Additionally, scleroderma has a propensity for tendon involvement, including the Achilles tendon. Surgical therapy for any of these diverse causes of heel pain is rarely indicated and may even be ill-advised. The JRA and ARF patients will usually remit on their own, including the spontaneous disappearance of nodules. Patients with sarcoidosis will usually require corticosteroid therapy which almost completely alleviates their musculoskeletal symptoms. The GC patient's symptoms and disease virtually totally resolve with appropriate antibiotic therapy. Calcinosis universalis or circumscripta patients often have severe cases of their underlying diseases with the quite common recurrence of the subcutaneous calcifications in the same area as well as in the surgical scar, if surgery is performed for any reason. Additionally, these patients tend to heal poorly.

While patients with this group of diseases are generally not surgical candidates for their heel pain, surgery would certainly be appropriate if: medical therapy totally fails, the patient is insistent because of the magnitude of his or her symptoms and risks are clearly outlined for the patient, or the patient has a cause of heel pain amenable to surgical intervention that is unrelated to his or her systemic disease process.

Table 1

POSSIBLE UNDERLYING CAUSES OF CPPD DISEASE

Most Common

- Hyperparathyroidism
- Hemochromatosis
- Hemosiderosis
- Hypophosphatasia
- Hypomagnesemia
- Gout
- Hypothyroidism
- Neuropathic Joints
- Aging

Less Common

- Hyperthyroidism
- Nephrolithiasis
- Ankylosing Hyperostosis
- Ochronosis
- Wilson's Disease
- Hemophilia Arthritis

Least Common (But Still Possible)

- Diabetes Mellitus
- Hypertension
- Azotemia
- Hyperuricemia
- Gynecomastia
- Inflammatory Bowel Disease
- Rheumatoid Arthritis
- Paget's Disease of Bone
- Acromegaly

(It is also associated with, and may well be a part of osteoarthritis.)

Although the medical causes of heel pain outlined in this review are many and varied, it should be remembered that all together they make up a very small proportion of all patients with heel pain. The plus side of this is that most of the patients that you find in your office will likely stand to gain from seeing you. The minus aspect is that it would be easy to overlook the trickle of systemic processes causing heel symptoms in the flood of the majority of patients with similar or identical symptoms.

Table 2

UNCOMMON METABOLIC AND HEREDOFAMILIAL CONDITIONS ASSOCIATED WITH HEEL PAIN

Ochronosis	rare despite cartilage involvement
Hemochromatosis	causes premature osteoarthritis
Wilson's Disease	(OA) and heel not usually affected unless concomitant chondrocalcinosis exists
* Amyloidosis	- may be primary or secondary - suspect if rapidly growing nodules are present - joints will usually look like RA - carpal tunnel syndrome occurs frequently - suspect in a patient with multiple myeloma associated with articular symptoms
Marfan's Syndrome	- heel involvement quite rare
Hemocystinuria	- heel involvement quite rare Ehlers-Danlos - heel involvement quite rare
Genetic Mycopolysaccharidoses	- heel involvement quite rare
* Osteogenesis Imperfecta	- several forms, mainly autosomal dominant - loose jointedness, thin skin, easily bruised - short extremities, deafness (2nd decade) - brittle bones, discolored teeth (wear away) - blue sclerae - Achilles tendon rupture (patellar tendon too)
* Hemophilia	

* Ones most likely encountered

Table 3

RARE INFECTIOUS CAUSES OF HEEL PAIN

Coccidiomycosis Blastomycosis Histoplasmosis Sporotrichosis Cryptococcosis Candidiasis	Although therapy is usually medical, diagnosis may need to be made surgically and debridement may be necessary.
Actinomycosis	(see text)