Dupuytren’s Disease: An Overview

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Learning Objectives: After studying this article, the participant should be able to: 1. Describe the epidemiology and etiology of Dupuytren’s disease. 2. Understand the biology and biochemistry of the diseased palmar fascia. 3. Describe the pathologic anatomy of Dupuytren’s disease. 4. Discuss the options for surgical management. 5. Understand the importance of postoperative rehabilitation and management of potential complications.

HISTORY

The earliest reference in surgical history to contracture of the palmar fascia appears in the writings of Felix Plater of Basel, Switzerland. In his book, Observationum in Hominis Affectibus, published in 1614, he incorrectly attributed contractures of the ring and little fingers to flexor tendon shortening. It was not until Henry Cline, Sr., of London first dissected two cadaveric hands with this affliction in 1777 that the causal relationship of the palmar fascia was correctly identified. Cline first proposed palmar fasciotomy as a surgical cure in 1787.

Baron Guillaume Dupuytren was born in 1777, ironically, the same year that Henry Cline identified palmar fascia as the cause of the affliction that now bears Dupuytren’s name. It is likely that this disorder was named after Dupuytren because his lecture series were reported verbatim in many of the weekly medical journals of Paris. In 1831, before a class of medical students, Dupuytren described the differential diagnosis, suspected pathology, clinical course, and surgical treatment of this malady by lecture, cadaveric illustration, and operative demonstration. He performed a fasciotomy through a transverse incision at the level of the distal palmar crease and stressed the importance of postoperative splinting. He associated the disease with an occupational predisposition caused by local trauma and chronic damage of the palmar fascia. Dupuytren’s contributions to the study of this disease ended in 1833, when an illness cut short his career.

Epidemiology and Etiology

Population studies have shown that Dupuytren’s disease is almost unique to Caucasian races, particularly those of northern European descent; it is a relatively rare occurrence in the black population. The prevalence of Dupuytren’s disease in selected populations has been reported to be as low as 2 percent to as high as 42 percent. It is thought to be inherited as an autosomal dominant condition with incomplete penetrance. The incidence increases with advancing age and is exceedingly rare in children. Men are 7 to 15 times more likely to have a clinical presentation requiring surgery than women, who tend to develop a more benign form of the disease that appears later in life. By the ninth decade, the relative prevalence between the sexes is nearly equal.

Dupuytren’s diathesis is a term introduced by Hueston to describe the phenomenon of rapidly progressing contractures in a young person. A diathesis can be recognized when there is a strong family history in the patient who is young at the time of onset and presents with bilateral disease, especially with radial-sided disease or diffuse dermal involvement. Dupuytren’s diathesis does not seem to demonstrate a sex predilection. A propensity for a flare response with early recurrence or extension of the disease is an indicator of a strong diathesis. An association of ectopic disease is also common. Ectopic deposits have been described on the soles of the feet (Lederhose’s disease), over the dorsum of the proximal in-

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terphalangel joints (Garrod’s knuckle pads), and on the penis (Peyronie’s disease).

A simple cause-and-effect relationship has not been identified; however, anecdotal reports have linked Dupuytren’s disease with a multitude of etiologies. There is a higher incidence in the alcoholic population, in which the causal factor appears to be the effect of alcohol on the liver. Because of the association between smoking and microvascular changes in the hand, some believe that tobacco may play a role in this disease. A link to diabetes mellitus is generally accepted and may be involved with the associated microangiopathy. Epileptic patients also seem to have an increased incidence. It was once believed that this link was altered tissue metabolism secondary to drug therapy; however, there are now conflicting reports in the literature. Although not usually related to hand trauma, Dupuytren’s disease occasionally develops after significant hand injuries, including surgery. Rheumatoid arthritis is the only condition reported with a statistically significant lower incidence of Dupuytren’s disease and is likely to be secondary to the effect of anti-inflammatory medications. Although Dupuytren himself originally proposed an occupational predisposition, no studies have consistently revealed a clear occupational association with the disease.

**BIOCHEMISTRY AND CELL BIOLOGY**

The biochemical aspects of Dupuytren’s disease have received the attention of several investigators. A clearer understanding of the collagen components, extracellular matrix, and growth factors that may enhance cell proliferation and collagen synthesis is beginning to emerge. The major collagen type of normal palmar fascia is predominantly type I, although small levels of type III are present. Many studies have confirmed that there is an increase in the ratio of type III to type I collagen in Dupuytren’s disease. Badalamente and Hurst summarized some effects of the extracellular matrix components. Prostaglandin-F2α and lysophosphatidic acid seem to promote myofibroblast contraction, whereas prostaglandin E1 and E2α and calcium-channel blockers have been shown to cause relaxation of myofibroblasts. Gamma-interferon has been shown by Pittet et al. to decrease fibroblast replication and collagen formation. These studies begin to provide a rational basis for further investigation into the use of these agents to treat Dupuytren’s disease.

Current evidence suggests a role of increased levels of growth factors in the diseased palmar fascia of Dupuytren’s disease. Basic fibroblast growth factor, platelet-derived growth factor, and transforming growth factor-beta have been implicated in the pathogenesis of Dupuytren’s disease through myofibroblast proliferation. Unfortunately, at present there is no clear knowledge of what actually is responsible for proliferation, contraction, or involution of myofibroblasts in Dupuytren’s disease.

**HISTOLOGY AND PATHOGENESIS**

Dupuytren’s disease is characterized by the development of nodules and cords in the palmar and digital fascia. The diseased tissue possesses the biologic features of benign neoplastic fibromatosis; however, it behaves similarly to the contracture and maturation of wound healing. In 1959, Luck described the nodule as the fundamental lesion. The nodules occur in specific locations along longitudinal tension lines in the palm and digits. Luck stated that nodules then evolve into cords. His classification of Dupuytren’s disease into three biologic stages has become widely accepted. The first is the proliferative stage, characterized by an intense proliferation of myofibroblasts (the cells believed to generate the contractile forces responsible for tissue contraction) and the formation of nodules. The second, involutional stage, is represented by the alignment of the myofibroblasts along lines of tension. During the third, residual stage, the tissue becomes mostly acellular and devoid of myofibroblasts, and only thick bands of collagen remain.

Several theories have been proposed to explain the pathogenesis of Dupuytren’s disease. McFarlane believed that this entity is the result of pathologic changes in the normal palmar fascia, whereas Hueston proposed that the fibrotic process begins with the de novo appearance of the nodule and then progresses to the cord. Gosset suggested that cords and nodules represent different forms rather than different stages of Dupuytren’s disease, with nodules arising de novo and cords arising from palmar fascia. Murrell et al. stated that the patients’ age, as well as genetic and environmental factors, may contribute to microvessel narrowing, which results in localized ischemia and the release of superoxide free radicals,
which in turn stimulate myofibroblast proliferation.

**Pathologic Anatomy**

The lesions of Dupuytren’s disease do not occur in a random pattern but rather follow certain well-defined anatomic pathways determined by the longitudinal lines of tension. Dupuytren’s disease primarily affects the longitudinal layer of the palmar fascia and usually spares the deeper fascial layers. The normally supple bands thicken to become deforming cords, resulting in contractures at the metacarpal phalangeal joint, the proximal interphalangeal joint, and, occasionally, the distal interphalangeal joint. In the palm, the diseased cords are pretendinous cords or natatory cords. Within the digit, they are the spiral, central, and lateral cords (Fig. 1).

The pretendinous bands are the longitudinal extension of the palmar aponeurosis, which divide distally at the level of the metacarpal head to blend with the spiral band and lateral digital sheet. Dupuytren’s disease causes thickening of the pretendinous band, which results in a pretendinous cord and subsequent metacarpal phalangeal joint flexion contracture. This joint flexion deformity is the result of the cord attachment to the skin in the distal palmar crease. The neurovascular bundles are not usually displaced by the formation of the pretendinous cord.

The natatory ligaments, which pass transversely across the web spaces linking the flexor tendon sheaths, may also become involved in Dupuytren’s disease as the natatory cord. The thickening and shortening of these ligaments converts the normal U-shaped web space fibers into a V-shape, resulting in limitation of abduction and progressing to adduction contractures of the fingers. The neurovascular bundles normally run dorsal to this diseased tissue and are not displaced by the formation of this cord.

The distal extension of the pretendinous band, the spiral band, lateral digital sheet, and Grayson’s ligament thicken to form the spiral cord of Dupuytren’s disease. The typical cord begins proximally as the pretendinous cord and passes dorsal to the neurovascular bundle just distal to the metacarpal phalangeal joint. The cord then runs lateral to the neurovascular bundle as it involves the lateral digital sheet.

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**Fig. 1.** The change in normal fascia bands to diseased cords of Dupuytren’s disease. (Left) Normal fascia bands that may become involved in Dupuytren’s disease. (Right) The deforming cords of Dupuytren’s disease that result in joint contracture. (Reprinted with permission from McFarlane, R. M. The Finger. In R. M. McFarlane, D. A. McGrath, and M. Flint (Eds.), Dupuytren’s Disease Biology and Treatment. Edinburgh: Churchill Livingstone, 1990. Pp. 155–167.)
and finally becomes superficial to the neurovascular bundle as it joins Grayson’s ligament. Progressive thickening, shortening, and straightening of this cord produces a flexion contracture of the proximal interphalangeal joint. Because the neurovascular bundles normally run within the spiral fascial fibers, they are usually displaced proximally, superficial to the spiral cord, and toward the midline of the finger, thus becoming susceptible to injury during surgical release of the proximal interphalangeal joint contractures (Fig. 2). The nerve displacement is progressive and occurs most often in patients with significant proximal interphalangeal joint contractures.

The diseased lateral digital sheet may thicken to form the lateral cord of Dupuytren’s disease. It is often associated with the natatory cord and attaches to the skin or to the tendon sheath through Grayson’s ligament. It can contribute to flexion contracture of the proximal interphalangeal joint and, occasionally, to distal interphalangeal joint contracture. This cord may also displace the neurovascular bundle toward the midline in the little finger.

A central cord, which has no fascial precursor but is usually an extension of the pretendinous cord, may form within the fibrofatty tissue located between the neurovascular bundles. The central cord is the most common cause of proximal interphalangeal joint contracture. However, it does not normally displace the neurovascular bundle.

**CLINICAL PRESENTATION AND DIFFERENTIAL DIAGNOSIS**

The diagnosis of Dupuytren’s disease in its early stages may be difficult. Most cases of Dupuytren’s disease present after the disease has progressed, at which time the diagnosis can be made more easily. In the classic scenario of typical disease, a nodule develops, followed by formation of a cord that gradually contracts, leading to a progressive flexion deformity of the digit.

The diagnosis is based on the palpable nodule, characteristic skin changes, changes in the fascia, and progressive joint contracture. It is generally agreed that palpable nodules are diagnostic of Dupuytren’s disease, but they are not an invariable feature inasmuch as they may disappear in the latter stages of the disease. Skin changes are caused by retraction or involvement of the skin and may present as dimples or pits. Pathologic changes in the diseased fascia result in cord formation, and joint contractures appear as the disease progresses.

The typical patient who presents for surgical evaluation of Dupuytren’s disease is a 50- to 60-year-old Caucasian man of northern European origin who has had progressive contracture of the palmar fascia for approximately 10 years. The disease is usually bilateral, with one hand being more severely involved; however, there is no association with hand dominance. The patient may have one, two, or three rays involved in the more severely affected hand. The most commonly involved digit is the little finger, which is involved in approximately 70 percent of patients. In decreasing order of frequency, the involved digits are the little, ring, middle, thumb, and index.

The differential diagnosis of Dupuytren’s disease should include hand abnormalities that cause nodules or contractures. These pseudo-Dupuytren’s contracture conditions are caused by soft-tissue changes that can mimic early Dupuytren’s disease and pathologic processes that could be mistaken for an established Dupuytren’s contracture. These include intrinsic joint contractures, palmar ganglions, inclusion cysts, stenosing tenosynovitis, occupational hyperkeratosis, callous formation, soft-tissue giant cell tumors, epithelioid sarcomas, and changes secondary to rheumatoid arthritis.

Dupuytren’s disease in children or in teenagers must be differentiated from camptodactyly.

**FIG. 2.** With increasing flexion contracture of the proximal interphalangeal joint, the neurovascular bundle is displaced proximally, superficially, and toward the midline of the finger, thus becoming susceptible to injury during surgical release of contractures of this joint. (Reprinted with permission from McFarlane, R. M. The Finger. In R. M. McFarlane, D. A. McGrouther, and M. Flint (Eds.), *Dupuytren’s Disease Biology and Treatment*. Edinburgh: Churchill Livingstone, 1990. Pp. 155–167.)
**NONSURGICAL MANAGEMENT**

Reports of regression and even complete spontaneous resolution of Dupuytren’s disease without surgical intervention have led many researchers to investigate nonsurgical treatment options such as splinting, radiation, di-methylsulfoxide, vitamin E cream, anti-gout medications, physical therapy, and ultrasonic therapy. Most of these therapies have not proven to be clinically useful or of any long-term value in the treatment of established contractures. Percutaneous needle fasciotomy, continuous slow skeletal traction, calcium channel blockers, steroid injection, and gamma-interferon show some promise.

Enzymatic percutaneous fasciotomy, as described by Hurst and Badalamente, has shown encouraging results. With this procedure, 10,000 U of collagenase diluted in calcium chloride is injected percutaneously into the Dupuytren’s cord. Before the injection, ultrasonographic imaging is performed to determine the depth of the underlying flexor tendons to avoid inadvertent injection. On the day after the injection, the patient undergoes gentle manipulation of the finger, which often provides correction of the contracture. Patients are instructed to wear an extension splint at night for 4 months and to perform at-home finger extension and flexion exercises. With an average of 9 months of follow-up, the authors report “excellent results” in more than 90 percent of metacarpal phalangeal joint contractures and in more than 66 percent of proximal interphalangeal joint contractures.

These findings give hope that a reliable alternative to surgical therapy may be eventually developed, but long-term, double-blinded studies have not yet been done. At the current time, operative intervention remains the mainstay of treatment in Dupuytren’s disease.

**SURGICAL MANAGEMENT**

The diagnosis of Dupuytren’s disease is not necessarily an indication for surgical intervention. It is necessary to consider the patient, the hand, and the disease. The patient’s age, comorbid conditions, expectations, and willingness to undergo postoperative rehabilitation are important factors that must be taken into account. The surgeon must also balance the severity of functional limitation with its importance to the patient. The age, duration, amount, and speed of the contracting process are measures of the aggressiveness of this disease. It is usually better to rely on functional difficulty and the rate of progression when deciding on surgery rather than a set amount of joint contracture. Nevertheless, flexion contractures of 30 degrees at the metacarpal phalangeal joint and 20 degrees at the proximal interphalangeal joint are generally considered to be indications for intervention.

The goal of surgery is to restore hand function, not “cure” the disease. This goal may be achieved by a variety of different surgical options. An integrated treatment plan takes into account case selection, timing of surgery, the patient’s expectations, the actual operation, and the postoperative rehabilitation program. The surgeon who treats Dupuytren’s disease must first understand the nature of the disease and then develop a management plan that is appropriate to his or her own practice and rehabilitation facility.

**OPERATIVE TREATMENT**

It is desirable to have a surgical plan that will be effective and yet as safe as possible. One must carefully consider three aspects of the surgical plan: management of the skin, management of the fascia, and management of the joints.

**Management of the Skin**

Choices in the management of the skin include skin incision and skin excision. With regard to skin incisions (Fig. 3), longitudinal incisions (multiple Y-to-V advancement flaps, Bruner zigzag incision, and midline longitudinal incision closed with Z-plasties) are more popular and have the advantages of progressive flexible exposure and addressing the skin shortage secondarily to the contracture. These incisions should not cross joint creases unless broken up by Z-plasties. These incisions may be modified by either Z-plasty or by multiple Y-to-V advancement before closure to gain length in the presence of skin shortage. Transverse incisions (long palmar incision and short digital incisions) are advantageous in that they are less likely to be a pathway for subsequent scar contracture. The main disadvantage is the higher likelihood of hematoma formation within the palm. Transverse skin incisions that are not amenable to primary closure may be managed by full-thickness skin graft closure or by allowing the wound to heal secondarily, known as
Dermofasciectomy, a technique popularized by Hueston,\textsuperscript{35,36} consists of excision of diseased fascia and involved skin followed by full-thickness skin grafting of the defect. This technique is useful for replacing skin shortage, producing a “firebreak,” or replacing dermis that has been infiltrated by myofibroblasts.\textsuperscript{35,36} Dermofasciectomy should be considered with recurrent disease or in Dupuytren’s diathesis, in which patients have a high likelihood of recurrent disease.\textsuperscript{37}

Management of the Fascia

General approaches to the management of the diseased fascia include fasciotomy and varying degrees of fasciectomy. Percutaneous fasciotomy\textsuperscript{38} is intended to release the tension in the fascia without necessarily removing the diseased fascia. This procedure is associated with recurrent contracture and should be reserved for the few patients who cannot tolerate excision of the diseased fascia.

Moermans\textsuperscript{39} described a limited fasciectomy, in which only short portions of fascia are removed. The advantage of limited fasciectomy is the minimal associated morbidity; however, as the disease progresses recurrent contracture tends to be a problem. Regional fasciectomy is the most commonly performed operation and entails removing all involved fascia in the palm and digit by a progressive, longitudinal dissection. Extensive or radical fasciectomy removes all involved fascia with the additional removal of uninvolved fascia to try to prevent disease progression or recurrence.\textsuperscript{39,40} This procedure is usually reserved for patients who have extensive disease or an increased diathesis.

Management of the Joints

The metacarpal phalangeal joint contracture can almost always be released by fasciectomy alone. This joint will tolerate prolonged immobilization in flexion, and metacarpal phalangeal flexion is not an urgent indication for surgery unless there is functional limitation. Conversely, the proximal interphalangeal joint presents a difficult problem, and there is little agreement about the methods of management of residual contracture of this joint. Distal interphalangeal joint involvement is a relatively rare finding in Dupuytren’s disease.

When addressing proximal interphalangeal joint contractures it is helpful to differentiate between primary and secondary deformities. According to McFarlane’s concept, primary flexion contractures are directly related to fas-
cial involvement and secondary flexion contractures are caused by either periarticular adhesions or secondary effects on the surrounding structures.\textsuperscript{17} Crowley and Tonkin\textsuperscript{41} describe a number of structures that may contribute to secondary joint contractures, including shortening of the palmar skin, contraction of the flexor sheath, shortening of the flexor muscles, adhesion of the palmar plate, adhesion of the retinacular ligament, contracture or adhesion of the collateral and accessory collateral ligaments, and intraarticular changes of the joint itself.

Recommendations for the treatment of proximal interphalangeal joint flexion contractures include fasciectomy alone, gentle passive manipulation, and varying degrees of capsuloligamentous release. Furthermore, the importance of the flexor tendon sheath and the central slip of the extensor mechanism have been described.

McFarlane\textsuperscript{17} reported that only one out of 64 of his patients required more than just systematic excision of all of the diseased fascia within the finger to correct proximal interphalangeal joint contractures. He stated that capsulotomy leads to further joint scarring and contracture within the capsule and should not be performed routinely. Weinzweig et al.\textsuperscript{42} found no significant improvement in correction of proximal interphalangeal flexion deformities in joints undergoing fasciectomy and capsuloligamentous release when compared with joints undergoing fasciectomy alone. Furthermore, complications occurred more often in patients who underwent capsuloligamentous release.

Breed and Smith\textsuperscript{43} described gentle passive manipulation followed by splinting of the proximal interphalangeal joint, comparing this with the more aggressive surgical interventions. With the metacarpal phalangeal joint flexed, they performed up to 2 minutes of gentle, sustained stretching of the proximal interphalangeal joint contracture with only enough force to rupture periarticular adhesions. They reported an 84 percent improvement of proximal interphalangeal joint contractures with fewer complications and less chance of recurrence than surgical approaches to this joint.

Varying degrees of capsuloligamentous release have been described. These maneuvers include release of the checkrein ligaments of the volar plate, the accessory collateral ligaments, and the proper collateral ligaments—on one or both sides of the joint. On the basis of their experience with checkrein ligament release, Watson et al.\textsuperscript{44} stated that correction of flexion contractures is possible without further capsuloligamentous release. They asserted that violation of the joint by capsulotomy or collateral ligament release is almost never required.

Tonkin et al.\textsuperscript{45} stressed the importance of the flexor tendon sheath. They found that the mobile portion of the sheath between the A2 and A4 pulleys contracts and shortens, contributing to the flexion deformity. They recommended excision of a window from the tendon sheath at this mobile portion and stated that it is required to provide an adequate joint release. Smith and Breed\textsuperscript{46} stressed the importance of central slip attenuation in the management of long-standing Dupuytren’s contractures. They suggested that this may account for recurrence of proximal interphalangeal joint contracture despite full correction at the time of operation. The diagnosis is made at the time of operation by performing a tenodesis test. If central slip attenuation is confirmed, a postoperative regimen of 3 weeks of static splinting followed by 3 weeks of dynamic splinting is instituted. With this regime, they report a 70 percent improvement in contractures associated with central slip attenuation.

Crowley and Tonkin\textsuperscript{41} provided a reasonable approach to treating the proximal interphalangeal joint in Dupuytren’s disease. They first performed precise excision of all diseased fascia and searched for an isolated digital cord. Correction to 30 degrees is considered satisfactory inasmuch as most proximal interphalangeal joints undergoing release eventually return to this position early in the postoperative period. If correction to 30 degrees is not obtained by fasciectomy, gentle passive manipulation is performed. Only if the joint contracture cannot be corrected to 30 degrees with gentle passive manipulation are checkrein ligament release and subsequent capsuloligamentous release performed.

\textbf{Postoperative Rehabilitation}

Postoperative rehabilitation is a very important component in the management of Dupuytren’s disease. It has been stated that 50 percent of operative results depend on effective postoperative management.\textsuperscript{18} Through an effective splinting and exercise program, the surgical outcome can be enhanced. Treatment should be directed toward restoring hand func-
tion and monitoring development of complications that could compromise the outcome.

Postoperative rehabilitation should be initiated after the early inflammatory phase (3 to 5 days), when the wound can tolerate active motion without reactive inflammation. Improved hand function may be achieved through a variety of techniques, including range-of-motion exercises, progressive splinting, scar management, edema control, and early treatment of reflex sympathetic dystrophy.47,48

The initial rehabilitation program consists of range-of-motion exercises, including active flexion and extension of all digital joints without placing undue tension on the wound. This exercise program should be performed for short periods of time, with 10 to 15 repetitions, repeated 4 to 6 times each day.47,48

Splinting is an important part of the postoperative management. Static splints may be used if full proximal interphalangeal joint extension is achieved at the time of operation. The initial splint positioning should provide slight metacarpal phalangeal joint flexion of 10 to 20 degrees with proximal interphalangeal joint extension to allow maximal elongation of the wound. As wound healing progresses, metacarpal phalangeal joint extension can be increased to neutral. The distal interphalangeal joint should generally be left free from splints to enable range of motion and improve tendon excursion. When proximal interphalangeal joint correction is incomplete, achievement of maximum extension may require progressive dynamic splinting. Initially, splints should be worn at all times and removed only for wound care and exercises. Splinting can then be modified as needed to address individual needs and may be continued for 8 to 10 weeks.47,48

After wound healing and suture removal, scar management is initiated to soften the scar. The hand therapist first performs massage therapy and then the patient is instructed in a home massage program to be performed 2 to 3 times a day. Other adjuvant scar management modalities include volar pressure splinting, moist heat, and ultrasonographic therapy.47,48

COMPLICATIONS

Sound surgical judgment does not prevent the development of complications. The incidence of complications in the treatment of Dupuytren's disease is high, being reported by McFarlane and McGrouther49 to be as great as 17 to 19 percent overall. Complications tend to be predictable in that they occur most often in those patients with severe disease who require extensive surgery.49,50

Intraoperative complications include nerve injury, loss of digital circulation, and "buttonhole" through a skin flap. Nerve injury can be avoided by early identification of the nerve in the palm, followed by a proximal-to-distal dissection to the proximal interphalangeal joint where digital nerve displacement is anticipated. Loss of digital circulation may be caused by digital artery spasm secondary to stretch or may result directly from vessel injury. Therefore, it is important to assess digital circulation at the conclusion of the operative intervention. Nerve and arterial injury has been reported by McFarlane to occur in 3 percent of patients, with arterial injury occurring more often. "Buttonhole" through a skin flap can occur when attempting to separate the skin from the underlying diseased fascia, which can be very difficult in cases of dermal invasion.49,50

Postoperative complications include loss of flexion, hematoma, skin loss, infection, edema, wound dehiscence, Dupuytren's flare, and reflex sympathetic dystrophy. Loss of proximal interphalangeal joint flexion is the most common postoperative complication, occurring in more than 6 percent of patients. Hematoma, skin loss, and infection are a triad of associated postoperative complications that often present in sequence and occur in 3 percent of patients. Hematoma may be prevented by liberal use of bipolar cautery, utilization of loupe magnification, and releasing the tourniquet before wound closure. If a hematoma develops, operative evacuation is promptly undertaken because necrosis of the surrounding tissue or infection may follow.50 The risk of postoperative infection is increased in patients with underlying diabetes mellitus or peripheral vascular disease.

Postoperative edema can be detrimental to wound healing and should be minimized by immediate elevation of the hand after surgery. Persistent postoperative edema may be controlled with compressive wrappings such as Coban wraps (Medical Product/3M, St. Paul, Minn.) or elasticized gloves.48 If wound dehiscence or skin loss occurs, the underlying structures are evaluated to determine whether wound closure is necessary. Full-thickness skin grafts may be required to cover larger defects, and if tendon is exposed, cross-finger flap, flag flap, or other flap coverage may be indicated.50
Postoperative pain syndromes include Dupuytren’s “flare” and reflex sympathetic dystrophy and tend to present more commonly in patients with aggressive or early disease. “Flare reaction,” as described by Howard, is an inflammatory reaction, occurring 2 to 3 weeks after surgery. It presents as redness, pain, edema, and stiffness. Zeigel found this reaction to present more often in women, occurring in over 20 percent. Although the cause of flare reactions is unknown, some suggest that acute carpal tunnel syndrome may play a role, and if this is suspected, carpal tunnel release should be performed. If a specific cause cannot be found, symptomatic treatment including sympathetic blockade, hand therapy emphasizing stress-loading techniques, and oral medications (steroids or carbamazepine) may be instituted.

Reflex sympathetic dystrophy is one of the most difficult complications that may follow surgery for Dupuytren’s disease. It normally occurs after the healing process is complete and is reported in as many as 5 percent of patients. It is also more common in women, occurring nearly twice as often. Reflex sympathetic dystrophy usually presents with excessive pain, edema, stiffness, and associated vasomotor disorders of the hand. Once the diagnosis is established, the usual postoperative program should be abandoned and treatment should be focused on the reflex sympathetic dystrophy. Treatment is similar to that for Dupuytren’s flare. It is important to maintain joint mobility by active exercises until the condition resolves.

Multiple factors may predispose to recurrent disease in an area of previous excision. Recurrent disease may be viewed as a late postoperative complication if it is caused by incomplete excision rather than actual extension. Recurrence rates after surgery range from 26 to 80 percent, depending on the operative technique. Recurrence has been associated with initial contracture severity, Dupuytren’s diathesis, advanced disease stage, and the presence of associated diseases such as diabetes. If disease recurs despite adequate initial operation, dermofasciectomy should be considered.

Conclusions

Since its description in 1614, Dupuytren’s contracture has been the topic of many publications. Even though new knowledge has improved our understanding of the basic science and clinical presentation of this malady, we do not yet truly understand the nature of this disease. The answers to two questions, the cause and the cure, continue to elude us. Research continues so that we may not only better control, but also perhaps cure, this affliction. The fascination with this disease will undoubtedly continue.

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1. A HIGHER INCIDENCE OF DUPUYTREN’S DISEASE HAS BEEN LINKED TO ALL OF THE FOLLOWING FACTORS EXCEPT
A) Alcohol
B) Tobacco
C) Diabetes mellitus
D) Epilepsy
E) Rheumatoid arthritis

2. WHICH OF THE FOLLOWING HAS BEEN SHOWN TO INHIBIT FIBROBLAST CONTRACTION IN VITRO?
A) Prostaglandin F
B) Lysophosphatidic acid
C) Prostaglandin E
D) Gamma-interferon
E) Platelet-derived growth factor

3. WHICH OF THE FOLLOWING HAS BEEN IMPLICATED IN THE PATHOGENESIS OF DUPUYTREN’S DISEASE THROUGH MYOFIBROBLAST PROLIFERATION?
A) Basic fibroblast growth factor
B) Platelet-derived growth factor
C) Transforming growth factor-beta
D) All of the above
E) None of the above

4. IN DUPUYTREN’S DISEASE, ADDUCTION CONTRACTURES OF THE DIGITS AND INABILITY TO ABDUCT THE FINGERS IS CAUSED BY WHICH OF THE FOLLOWING CORDS?
A) Pretendinous
B) Natatory
C) Spiral
D) Lateral
E) Retrovascular

5. DISPLACEMENT OF THE DIGITAL NERVE TOWARD THE MIDLINE IS MOST LIKELY THE RESULT OF WHICH CORD OF DUPUYTREN’S DISEASE?
A) Pretendinous
B) Natatory
C) Spiral
D) Lateral
E) Retrovascular

6. THE SPIRAL CORD IS COMPOSED OF WHICH OF THE FOLLOWING STRUCTURES?
A) Pretendinous band, natatory ligament, lateral digital sheet, and Cleland’s ligament
B) Spiral band, longitudinal fibers, lateral digital sheet, and Grayson’s ligament
C) Spiral band, lateral digital sheet, natatory ligament, and Grayson’s ligament
D) Pretendinous band, spiral band, lateral digital sheet, and Grayson’s ligament
E) Pretendinous band and spiral band

7. AFTER METICULOUS FASCIECTOMY FOR DUPUYTREN’S CONTRACTURE, A 45-DEGREE PROXIMAL INTERPHALANGEAL JOINT FLEXION CONTRACTURE REMAINS DESPITE GENTLE PASSIVE MANIPULATION. WHAT IS THE NEXT REASONABLE STEP TO ACHIEVE IMPROVED EXTENSION?
A) Release of the checkrein ligaments
B) Release of the collateral ligaments
C) Release of the accessory collateral ligaments
D) Release of the transverse retinacular ligaments
E) Release of the oblique retinacular ligaments

8. THE MOST FREquent COMPLICATION AFTER SURGERY FOR DUPUYTREN’S DISEASE IS:
   A) Recurrence of deformity
   B) Injury to the digital nerve
   C) Injury to the digital artery
   D) Loss of proximal interphalangeal joint flexion
   E) Triad of hematoma, skin loss, and infection

To complete the examination for CME credit, turn to page 250 for instructions and the response form.