

Case Report

Surgical management of Dupuytren contracture of the ring finger: a case report

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ABSTRACT

Dupuytren's disease, also called Dupuytren's contracture or palmar fibromatosis, is a condition in which the connective tissue under the skin of the palm contracts and toughens over time. The gold standard treatment for Dupuytren's contracture is surgery. We present a case of Dupuytren's contracture treated with Bruner incision which resulted in good functional outcome. A 79 year old male presented to our Orthopaedic Clinic RSUP Sanglah Denpasar with the complaints of pain and stiffness on his left ring finger since 5 years ago. Patient had same history with his right hand and had operation 20 years ago. The patient underwent release of contracture using Bruner incision and had no complaint in 6 month follow-up. Extension deficit was $<5^\circ$, reduction of contracture was $>50\%$, the Patient and Observer Scar Assessment Scale (POSAS) gave overall opinion of the scar being minimal. The Dupuytren's disease etiology remains uncertain, with genetics perceived as most probable factor. Despite short-term success, there is a high rate of recurrent contracture with additional comorbidities such as wound-healing complications and neurovascular injury. In this case, the patient was treated using fasciotomy with Bruner incision and had satisfying outcome. Fasciotomy is known to have less complication in terms of recurrence. Dupuytren disease is characterized by abnormal thickening of the palmar fascia beneath the skin and the gold standard of treatment is surgery. This case presented surgical treatment using Bruner incision which had satisfying outcome in 6 month follow-up.

Keywords: Bruner incision, Contracture, Dupuytren, Fasciotomi

INTRODUCTION

Dupuytren's disease predominantly affects older men. Men are six times more likely than women to present with the condition, and children are rarely affected. Bilateral disease is also more common in men, affecting 59 percent of men versus 43 percent of women. The incidence rises sharply for men in their fifth decade and for women in their sixth decade. By the ninth decade, the prevalence across genders is equal. Disease recurrence is equal in both sexes.¹ Basic pathophysiology involves fibroblast proliferation and collagen deposition leading to

contractures of the palmar fascia. The most preponderant cytokine in Dupuytren's fasciae is interleukine-1 (IL-1). It stimulates platelets and macrophages giving rise to the secretion of growth factors, in particular TGF- β , fibroblast growth factor FGF and platelet-derived growth factor PDGF. This process occurs along with direct stimulation of fibroblast proliferation and activation of Langerhans cells in the epidermis.^{2,3}

Therefore, it seems reasonable to believe that immunologic response plays an important role in the pathogenesis of Dupuytren's contracture. Increased

secretion of IL-1, TGF- β and fibronectin in Dupuytren's contracture elevates the number of inflammatory cells that can also themselves stimulate production of cytokines and growth factors. In order to allow these events to take place, apoptosis in the tissues affected by Dupuytren's contracture should be either completely abrogated or partially deregulated.³⁻⁵

One of the major histological features of Dupuytren's contracture, differentiation of fibroblasts into myofibroblasts, is considered to be responsible for the development of typical disease symptoms, and this process is driven by TGF- β (tumor growth factor- β). Furthermore, Meek et al. suggested that sustained myofibroblast proliferation might be the consequence of reduced rate of apoptosis of inflammatory cells present in Dupuytren's tissue.⁴

In Dupuytren's disease, the normal anatomy of the hand becomes distorted as the normal palmar fascial bands become diseased cords. Most cords occur in the palm and cause metacarpophalangeal joint flexion deformity.⁵

The central cord is an extension of the pretendinous cord into the finger. It forms along the midline between the neurovascular bundles, which are rarely displaced, and attaches to the flexor tendon sheath or the periosteum of the middle phalanx. The spiral cord consists of a pretendinous band, a spiral band, the lateral digital sheet, and Grayson's ligament. The cord begins as a pretendinous cord in the palm and passes deep to the neurovascular bundle just distal to the metacarpophalangeal joint. The cord then runs lateral to the neurovascular bundle as it starts to involve the lateral digital sheet and then runs superficial to the neurovascular bundle as it begins to fuse with Grayson's ligament.^{5,6}

As the contracture becomes more severe, the cord straightens and the neurovascular bundle begins to spiral around the cord, which causes it to be displaced toward the midline and closer to the surface of the palm. Because of its superficial displacement, this nerve is now at risk during surgery. One should expect to see a spiral cord when proximal interphalangeal joint contracture and an interdigital soft-tissue mass are present.^{1,2}

The lateral cord is formed from the lateral digital sheet and is often present with the pretendinous cord and natatory cord. It is attached to the skin or tendon sheath through Grayson's ligament, resulting in contracture of the proximal interphalangeal joint and possibly the distal interphalangeal joint, and can cause displacement of the neurovascular bundle toward the midline. Four different cords can form near the thumb and radial side of the hand.^{1,3,4}

The proximal commissural cord originates from the proximal commissural ligament, and the distal commissural cord is an extension of the distal commissural ligament, with both causing first web contractures. The thumb pretendinous cord is similar to other pretendinous cords and causes metacarpophalangeal

contracture. Lastly, the thenar cord is a product of the thickening of the thenar fascia and is associated with the thumb pretendinous cord.^{2,4}

Treatment is indicated if the deformity is a nuisance or rapidly progressing. Once a 30 degree angle is reached at the PIP or MCP, the patient usually cannot get the hand flat on the table (the 'table-top test') and starts to be aware of functional problems. Most treatments are effective at this earlier stage but, in particular, PIP joint contractures can become irreversible if left too long. The aim is reasonable, but not always complete, correction. Several non-operative treatments, including radiotherapy, splinting, steroids, and topical vitamin A, have been tried and tested, w4 w5 but none has gained popularity. Radiotherapy is an option for a very small group with painful widespread proliferative disease. Dry skin is a common side effect. Percutaneous needle fasciotomy is an isolated discrete cord across the front of the MCP joint can be divided by scratching several times through the skin with a bevelled needle. This can be very gratifying for selected cords with a very quick recovery. However, no fibrous matrix is removed and the altered cells remain; recurrence is very high.^{3,4}

The gold-standard treatment for Dupuytren's contracture has involved surgery, ranging from percutaneous release to dermatofasciectomy. Despite short-term success, there is a high rate of recurrent contracture with additional comorbidities such as wound-healing complications and neurovascular injury. The optimal form of treatment for Dupuytren's contracture should allow correction of the deformity with minimal complications and rehabilitation, maintain the correction with the passage of time, and be cost-effective.⁵

CASE REPORT

A 79 year old male presented to our Orthopaedic Clinic RSUP Sanglah Denpasar with the complaints of pain and stiffness on his left ring finger since 5 years ago. Patient also complained about stiffness on index finger, but he was still able to flex, extend, and snapped his finger. No history of trauma before. Patient had same history with his right hand and had operation 20 years ago. Everyday patient works as a Fisherman. The patient underwent release of contracture (Figure 1).



Figure 1: Contracture release procedure for Dupuytren's Disease.

Six months after the surgery, patient had no complaint. Extension deficit was <5 and reduction of contracture

was $>50^\circ$. The wound was dry and range of motion of ring finger was good.

DISCUSSION

The Dupuytren's disease or Dupuytren's Contracture (DC) etiology remains uncertain. Today, the hereditary factors are most accepted. It is believed that this may be associated to a genetic predisposition through dominant chromosome. Diseased tissue originates in longitudinally oriented fascial structures. Early proliferative phase is characterized by high cell concentrations of immature fibroblasts and myofibroblasts in a whorled pattern; this early hypercellular structure is often referred to as a histologic nodule. In the involutinal phase, fibroblasts align along tension lines and produce more collagen. The final residual phase is relatively acellular and features contracted, collagen-laden tissue more characteristic of scar formation.^{1,3,5}

More recently, research has focused on potential treatments targeting the molecular processes underlying DC. Fibrogenic cytokines, which may be capable of inducing the growth of fibroblasts, are involved in the molecular mechanisms underlying DC. Aberrant growth factors expression, e.g. of TGF- β and TGF- α , known to be an extremely important event in all types of fibrosis, is believed to drive at least two molecular processes in Dupuytren's contracture: A) proliferation of fibroblasts and their differentiation into myofibroblasts, and B) production of dense extracellular matrix containing elevated levels of fibronectin, type III collagen and proteoglycans.^{3,4}

However, there is no current evidence proving the association between the common TGF- β 1 and TGF- β 2 polymorphisms and Dupuytren's contracture. Several authors have reported that the expression of unstable form of zinc-finger protein 9 (Zf9) could predispose patients for development of Dupuytren's contracture as this protein seems to be directly responsible for increased synthesis of TGF- β 1 and 2 and their receptors in serum and tissue.^{4,5}

The gene coding for this transcription factor therefore represents a potential candidate for investigating genetic susceptibility to this disease. Indeed, Bayat et al. genotyped a novel single nucleotide polymorphism in the 3' untranslated region of the Zf9 gene in the study following up a cohort of 138 patients with Dupuytren's contracture. The results indicated that the presence of the G allele versus the A allele is associated with an increased risk of developing Dupuytren's contracture.³⁻⁵

Myofibroblast is cellular contractile culprit of Dupuytren contracture. The myofibroblast differs from the fibroblast in that it has actual bundles of contractile actin microfilaments arranged parallel to the long axis of the cell. Adjacent myofibroblasts connect via extracellular fibrils of fibronectin and act together to generate the contracted tissue seen in Dupuytren contracture.^{3,5}

Intervention is traditionally considered for patients with functional impairment and contractures of the metacarpophalangeal (MCP) joint more than 30° or any degree of proximal interphalangeal (PIP) joint contracture. Options for treatment are numerous and include percutaneous and open fasciotomy, injection of collagenase *Clostridium histolyticum*, radiation therapy, and subtotal or complete palmar fasciectomy. Each treatment option has utility and may be used for specific patients depending on their degree of contracture and the functional requirements for treatment.⁶ The optimal form of treatment for Dupuytren's contracture should allow correction of the deformity with minimal complications and rehabilitation, maintain the correction with the passage of time, and be cost-effective.⁶

Aside from growth factors and their respective receptors, several molecular factors, including, transcriptional factors, metalloproteinase enzymes and their inhibitors, extracellular matrix components, immune system components and oxidative stress response elements are involved in the DC development. Dense extracellular matrix over-produced by myofibroblasts and containing fibronectin, laminin, collagen and tenascin as major constituents, represents one of the major biochemical features of the palmar fascia affected by Dupuytren's contracture.^{5,6}

Normal palmar fascia contains mainly type-I collagen. On contrary, type-III collagen, which is virtually absent from normal adult palmar fascia, predominates in the tissue of patients with Dupuytren's contracture. After quick synthesis of immature, normal-length type-III collagen, contractile force of myofibroblasts causes collagen structure of tissues to shorten, leading to an increase of contractile force and loss of hand function.^{2,3}

This process is believed to be induced by increased density of fibroblasts, stimulation of fibroblast growth by growth factors, decreased rate of apoptosis (programmed cell death) and disproportion between the amounts of collagenase (also known as matrix metalloproteinase) and matrix metalloproteinase inhibitors (tissue inhibitors of metalloproteinase, TIMP). Besides type-III collagen, Dupuytren's contracture tissues also contain abundant amounts of fibronectins, glycoproteins ubiquitously found in connective tissue produced by fibroblasts, platelets and keratinocytes.^{1,3,4}

In this case, we performed fasciotomy with Bruner incision due to lower complications. Previous study reported a lower risk of recurrence with open fasciectomy versus percutaneous fasciotomy. However, needle fasciotomy appears to confer a lower risk of digital nerve injury compared with fasciectomy, ranging from 1% to 4%. To avoid inadvertent nerve injury during Dupuytren surgery, it is imperative to understand the anatomic displacement of the neurovascular structures that can occur with development of Dupuytren cords, particularly abductor digiti minimi cords, lateral cords, and spiral cords. It is advisable to identify the neurovascular

structures proximally and trace them distally (or vice versa) to avoid nerve injury during surgery.⁶

Dupuytren disease is characterized by abnormal thickening of the palmar fascia beneath the skin. Thickened fascia features high concentrations of fibroblasts and contractile myofibroblasts, resulting in eventual fascial contracture (hence the term Dupuytren contracture). The specific treatment of Dupuytren's disease will be determined taking into account the following factors: Age of the patient, Clinical history, Disease stage, Tolerance to certain medications, procedures or therapies, Expectations regarding the disease, Opinion and patient preferences. After evaluating all these factors, the clinician should adopt the best therapy for each case. Treatment for this case is operative treatment.

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REFERENCES

1. Rayan GM. Dupuytren disease: anatomy, pathology, presentation, and treatment. JBJS. 2007 Jan 1;89(1):189-98.
2. Townley WA, Baker R, Sheppard N, Grobbelaar AO. Dupuytren's contracture unfolded. BMJ. 2006 Feb 16;332(7538):397-400.
3. Hart MG, Hooper G. Clinical associations of Dupuytren's disease. Postg Medi J. 2005 Jul 1;81(957):425-8.
4. Shaw Jr RB, Chong AK, Zhang A, Hentz VR, Chang J. Dupuytren's disease: history, diagnosis, and treatment. Plastic Reconst Surg. 2007 Sep 1;120(3):44e-54e.
5. Rodrigo JJ, Niebauer JJ, Brown RL, Doyle JR. Treatment of Dupuytren's contracture. Long-term results after. J Bone Joint Surg Am. 1976;58:380-7.
6. Eberlin KR, Mudgal CS. Complications of treatment for Dupuytren disease. Hand Clin. 2018 Aug 1;34(3):387-94.

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