Dupuytren contracture in diabetic hand

Alessandro Geraci,¹ Renzo Bianchi,¹
Antonio Sanfilippo,² Michele D'Arienzo²

¹Orthopaedic and Traumatology
Department, Santa Maria del Prato
Hospital, Feltre;

²Orthopaedic and Traumatology
Department, University of Palermo, Italy

Abstract

Dupuytren's disease (DD) is a fibroproliferative disorder of unknown origin causing palmar nodules and flexion contracture of the digits. About 5% of individuals with DD are diabetic, with an increased prevalence that is proportional to the duration of the diabetes. The association with diabetes mellitus is well recorded, with a reported prevalence of between 3% and 32%. This review considers the evidence behind the association between DD and diabetes mellitus, considering the pathogenesis, clinical manifestations and therapies that can be found in the literature.

Introduction

DD is a benign nodular fibromatosis of the palmar fascia, affecting the hands, named after Baron Guillaume Dupuytren who was the first to operate on the condition.¹ Dupuytren's disease (DD) is a progressive fibro-proliferative disorder resulting in abnormal *scar-like* tissue in the palmar fascia leading to irreversible, permanent, and progressive contracture of the involved digits. DD leads to shortening of the skin anchoring ligaments of the palm (palmar fascia), which may lead to contracture of the digits. The overall incidence of DD in the Northern Europe is around 8%, rising to about 15% in those over 65 years.² DD is commonly bilateral, and "Dupuytren-like" fibrotic tissue can occur on the dorsum of the hand over the knuckles (Garrod's pads), feet (Lederhose's disease), and penis (Peyronies disease). It is usually more severe in one hand, but there is no relation to handedness. The ring finger is the most frequently involved, followed by the little finger, and then middle finger; the index finger and thumb are rarely involved.³ The incidence of DD also increases with concurrent patient clinical conditions or factors such as diabetes, smoking, chronic alcoholism, seizures, and infection.⁴ Although the reason for this association is unclear, microvascular changes in smokers may play a role.⁵

Prevalence in diabetes

About 5% of individuals with DD are diabetic, with an increased prevalence that is proportional to the duration of the diabetes. The association with diabetes mellitus (DM) is well recorded, with a reported prevalence of between 3% and 32% and an average of around 20%.^{6,7} It is equally common in both type 1 and type 2 diabetes, although it occurs at a younger age in patients with type 1 diabetes.^{8,9} DD occurring in diabetic patients is different from the condition in non-diabetic patients. There is a lower incidence of contractures, and fewer cases require surgery.¹⁰ Usually DD is more common in men, but in diabetic patients the sex ratio is equal.¹¹ The exact relation of DD and diabetes has been the focus of much research. However, both conditions are common, and increase in prevalence with age. As a result, DD has been considered to be an early warning sign of diabetes or a late complication.^{12,13}

Pathogenesis in diabetic patients

Histologically, the cords of DD consist of a dense collagenous matrix containing fibroblasts, arranged along the longitudinal lines of stress. Nodules contain myofibroblasts in bundles of collagen. Investigators have proposed several hypotheses for the pathogenesis of DD. One cause may be localized ischemia and subsequent xanthine oxidase-derived free-radical formation from endothelial cells.¹⁴ The basic pathophysiological process in DD is similar to wound healing involving fibroblast proliferation, collagen deposition and myofibroblast contraction driven by various growth factors such as transforming growth factor beta.¹⁵ Research has revealed that lower concentrations of free radicals cause fibroblast proliferation in laboratory cultures. Because active fibroblasts produce free radicals as well, the fibroblasts induce an autocrine positive-feedback effect on themselves, causing further ischemia to the microvasculature. Research has shown that growth factors such as basic fibroblast growth factor (FGF), platelet-derived growth factor (PDGF), and transforming growth factor-beta (TGF- β) may signal the overproduction of the myofibroblasts and/or myofibroblastic activity of the fibroblasts. In addition, high levels of TGF- β may hinder apoptosis, or cell death, of the active myofibroblasts, unlike normal tissue healing.^{16,17} The increased concentration and activity of the myofibroblasts not only increase the total amount of collagen leading to the pathologic nodule but also cause remodeling of the normal collagen matrix and an increase in the ratio of type I collagen to type III collagen. Ultimately, the excess deposition of type III collagen and the formation of cross-links along the lines of tension on the palm and proximal digits result in contractures. The exact relation

Correspondence: Dr. Alessandro Geraci,
via Montegrappa 18/C, Feltre (BL), Italy.
E-mail: geracialessandro@libero.it

Key words: Dupuytren's disease, diabetes mellitus, hand, contracture, fasciectomy.

Contributions: all the authors contributed equally to the present work.

Conflict of interest: the authors report no conflicts of interest.

Received for publication: 17 October 2010.

Revision received: 24 November 2010.

Accepted for publication: 5 January 2010.

This work is licensed under a Creative Commons Attribution 3.0 License (by-nc 3.0).

©Copyright A. Geraci et al., 2011
Licensee PAGEPress, Italy
Endocrinology Studies 2011; 1:e2
doi:10.4081/es.2011.e2

of DD and diabetes has been the focus of much research. However, both conditions are common, and increase in prevalence with age. As a result, DD has been considered to be an early warning sign of diabetes or a late complication. There has been much debate about whether diabetes is an aetiological factor for DD, or whether the two conditions are inherited together. It is unlikely that DM has an important role in the aetiology of DD, as most of those with DM do not get DD. Diabetes may only be a triggering factor. It may be that microvascular changes in DM encourage local hypoxia, and this could elicit DD in those who would otherwise not have been affected.^{18,19} The microvascular changes that have been seen in DD are noted to be similar to those found in DM.

Clinical presentation

The typical patient with DD is aged 50 years or older and presents with a palmar nodule and cord adherent to the skin, as well as with a flexion contracture (Figure 1, 2). DD can be distinguished from other causes of hand contracture because it begins as a nodule and slowly progresses to contracture of the fingers. There is no relation between diabetic control and the severity of contractures, and DD has a generally milder form in diabetic patients. This suggests that diabetes may only be a triggering factor. During the physical examination, physicians should note the site of the nodule and the presence of contractures; bands; and skin pitting, tenderness, and dimpling. If contractures are present, the angles should be noted at the MCP and proximal interphalangeal (PIP) joints. DD must be distinguished from several other conditions that affect the hand,

including trigger finger, stenosing tenosynovitis, a ganglion cyst, or a soft-tissue mass. Unlike Dupuytren's contracture (DC), trigger finger typically involves pain with flexion followed by the inability to extend the affected digit. Stenosing tenosynovitis may be distinguished from DD by pain and a history of overuse or trauma. A small, movable nodule that is tender to palpation at the metacarpophalangeal (MCP) joint is likely a ganglion cyst. A soft-tissue mass must also be excluded from the diagnosis, especially if the patient is significantly younger than the typical patient with DD and if he or she has no other risk factors. Disease progression is classified using a grading system.²⁰ The grading system by Tubiana is the most popular.²⁰ It primarily assesses the extension deficit. Tubiana subdivides the hand into five digito-palmar segments. Each segment consist of a finger including its palmar zone. For scoring purposes, the first interdigital space counts as part of the thumbs segment. For each segment, the palmar and digital lesions are assigned to a particular stage; the score of that particular longitudinal segment also reflects the stage. Each stage corresponds to a stepchange or an extension deficit of 45 degree of a finger or segment. The total deformity or contracture score of each finger is determined as the sum of extension deficit of all three digital joints. The degrees of severity or stages of flexion contracture of the fingers and their palmar segments can be divided into six stages, as follows (Table 1):

- Stage 0: no extension deficit, no lesion (nil points).
- Stage N: the suffix N describes a nodule, but without contracture.
- Stage 1: the sum of extension deficit of all three finger joints is equivalent or less than 45 degrees.
- Stage 2: total contracture of 46-90 degrees.
- Stage 3: extension deficit reaches 91-135 degrees.
- Stage 4: total contracture reaches more than 135 degrees.

Bilateral involvement is common, although one hand is usually more severely involved than the other; the patient's dominant hand is not a predictor of severity.²¹ The fingers most commonly involved (in decreasing order) are the fourth, fifth, third, and second. Soft tissue tumors of the palm and digits may be confused with DD. In patients younger than 50 years, DD tends to progress faster than in older patients.²²

Treatment

In the past, non surgical approaches to DC such as splinting, irradiation, ultrasonography, dimethylsulfoxide, vitamin E therapy, and allopurinol treatment were shown to be ineffective.²³⁻²⁵ Disease initially can be managed expectantly, but injecting the nodule with a

steroid²⁶ can be helpful. Surgery is recommended if function is impaired, contracture is progressing, or severe deformity is disabling. Although the option for surgery in DD is considered on a case-by-case basis. Timing of surgical intervention varies, but surgery is usually performed when the MCP joint contracture exceeds 40 degrees or when the PIP joint contracture exceeds 20 degrees.²⁷ The Hueston tabletop test is a good indication for referral: if a patient is unable to lay his palm flat on the tabletop, the test is positive²⁸ (Figure 3). Surgery is usually successful, but recurrence is common. Many surgical operations of different magnitudes are available. One common surgical approach is a limited fasciectomy removing involved fascia. For less extensive cases, fasciotomy (simple division) of contracted fascia may be performed by an open operation or a closed technique. The closed technique has been advocated using a blade or as percutaneous needle fasciotomy. This last technique should only be performed in selected cases and by a surgeon with detailed knowledge of hand anatomy.²⁹ For more extensive cases or recurrences, both skin and fascia may be removed *en bloc* (dermofasciectomy) and the wound skin grafted. This may reduce disease recurrence but not its extension.³⁰ Usually, a limited fasciectomy of the pretendinous cord is sufficient to establish normal function in the MCP joint. McFarlane favors use of a regional fasciectomy of the pretendinous cord to prevent recurrence of DC.³¹ The evaluation of a PIP joint in DD is different from that of an MCP joint, and the prognoses differ as well. In PIP joint contractures, one should clearly define the method to be used in surgery, as well as discuss with patients their expectations, occupation, and activities that may require use of their hands. Given the difficulty of correcting severe disease, fasciectomy is indicated for any amount of PIP joint contracture. Unfortunately, recurrence is common. The procedures of choice in the PIP joint are dermatofasciectomy or extensive fasciectomy. Extensive fasciectomy prevents recurrence because the entire diseased fascia is removed, along with the central, lateral, spiral, natatory, and retrovascular cords, as well as any normal fascia that may later be affected.

In advanced cases an amputation may be necessary. Amputation is reserved only for cases without a surgical option for the contractures correction. Postoperative complications include loss of flexion, hematoma, skin sloughing, infection, edema, and reflex sympathetic dystrophy. The most common PIP joint postoperative complication is loss of flexion, which occurs in 6% of patients. The triad of hematoma, infection, and skin loss occurs in 3% of patients; Hematomas most often form in the palm, and they may be prevented by meticulous hemostasis, by removing the tourniquet

before the wound is closed, and by rapid evacuation of hematomas, which prevents necrosis of tissue and skin and decreases the risk of infection.³²

Discussion

Diabetes mellitus is a chronic metabolic condition characterized by persistent hyperglycaemia with resultant morbidity and mortality

Figure 1. Hand with Dupuytren's disease with involvement of the 5th finger.

Figure 2. Tubiana's stages (picture provided by Charles Eaton, florida Handcenter).

Figure 3. Tabletop test: if a patient is unable to lay his palm flat on the tabletop, the test is positive.

Table 1. The grading system by Tubiana.

Stage 0: no extension deficit, no lesion (nil points).
Stage N: the suffix N describes a nodule, but without contracture.
Stage 1: the sum of extension deficit of all three finger joints is equivalent or less than 45 degrees.
Stage 2: total contracture of 46-90 degrees.
Stage 3: extension deficit reaches 91-135 degrees.
Stage 4: total contracture reaches more than 135 degrees.

related to its microvascular and macrovascular complications. In 2000, according to the World Health Organization, at least 171 million people worldwide suffer from diabetes, or 2.8% of the population.³³ In addition diabetes is also associated with several musculoskeletal disorders of the hand, that can be debilitating (Table 2).³⁴ Reference to the literature reveals that an association between DC and diabetes has been frequently suggested. A distinct pattern of DD in the diabetic patient came to be recognised. Signs are rare in the little finger and commoner in the middle finger.³⁵ Severe contractures requiring operative treatment are seldom encountered, suggesting that the common diabetic DD is mild and of benign prognosis. The only other factor with which the incidence of DD in diabetes could be associated was age. Severity of diabetes did not correlate with the incidence of signs of DD. It was also interesting to note that the two most serious features of DD, bands and contractures, were more common in diabetic men than women. There was no obvious reason for this other than the many different clinical patterns of DD. The diabetic pattern could be of clinical importance in predicting disease, and an index group of patients with Dupuytren's signs in the fourth and third digital rays is being collected, so that in five years the group can be re-examined for the development of diabetes. In same studies the prevalence of DC in diabetic patients ranges from 3-32%,^{36,37} compared with 13% in the general population.³⁸ Among patients with DC, 13-39% have diabetes.^{39,40} Noble *et al.*⁴¹ in controlled clinical studies of adult diabetics found a 42% incidence of signs of DD. The incidence was highest in the older patients with a longer history of diabetes, but was not related to the severity of the diabetes. The features of DD in the diabetics has a distinctive pattern, being more severe in men than women and, compared with controls, having a radial shift towards the middle finger. The disease was mild and of benign prognosis, rarely needing operation. In a further study, 13% of patients with DD were found to have a raised blood glucose level. Fujinaka⁴² described that limitation of joint mobility and painful disorder associated with inflammation of tendons and sheaths are more frequently observed on hands and shoulders. Most of these complications are not specific for diabetes mellitus, but more often in diabetic populations than in non-diabetic populations. On the other hand, destructive bone change due to diabetic polyneuropathy are commonly observed on lower extremities. Papanas *et al.*⁴³ say that there is evidence that disease of hands are significantly more frequent in patients with diabetes and also that they may be associated with diabetes duration, poor metabolic control and presence of microvascular complications. In a more general sense, though, there are

Table 2. Prevalence of musculoskeletal disorders in patients with or without diabetes.

Musculoskeletal disorder	With diabetes %	Without diabetes %
Adhesive capsulitis	11-30	2-10
Limited joint mobility	8-50	0-26
Dupuytren's contracture	20-63	5-10
Carpal tunnel syndrome	11-16	2-5
Flexor tenosynovitis	10-12	<1
Diffuse idiopathic skeletal hyperostosis	13-50	2-15

other conditions affecting the hands, which also occur more frequently in diabetes. Treatment consists of optimising glycaemic control, physiotherapy, and hand exercises if required, and surgery only if function is severely affected. Several different surgical techniques can be used to remove the thickened fascia and correct the contracture: partial fasciotomy, complete fasciotomy, dermofasciectomy.

Conclusions

The etiology and pathogenesis of DD remains unclear. The literature suggests that the frequency of DD is ten times greater in diabetes than in the general population. DC can be considered a complication of diabetes and of the local neurovascular changes since both are often associated. The contracture is usually not severe in diabetes, is nodular in form, and usually crushes the palmar surface of the long and ring fingers. It is normally well tolerated by the patients and surgery is indicated when the deformity of the hand limit the function of the fingers. These procedures can return function to a disabled hand.

References

1. Dupuytren G. Permanent retraction of the fingers, produced by an affection of the palmar fascia'. *Lancet* 1834;ii:222-5.
2. Gudmundsson KG, Arngrimsson R, Sigfusson N, et al. Epidemiology of Dupuytren's disease: clinical, serological, and social assessment. *The Reykjavik Study. J Clin Epidemiol* 2000;53: 291-6.
3. Bayat A, McGrouther DA. Management of Dupuytren's disease – clear advice for an elusive condition. *Ann R Coll Surg Engl* 2006;88:3-8.
4. Geoghegan JM, Forbes J, Clark DI, et al. Dupuytren's disease risk factors. *J Hand Surg [Br]* 2004;29:423-6.
5. Godtfredsen NS, Lucht H, Prescott E, et al. A prospective study linked both alcohol and

tobacco to Dupuytren's disease. *J Clin Epidemiol* 2004;57:858-63.

6. Hart MG, Hooper G. Clinical associations of Dupuytren's disease. *Postgrad Med J* 2005;81:425-8.
7. Chammas M, Bousquet P, Renard E, et al. Dupuytren's disease, carpal tunnel syndrome, trigger finger, and diabetes mellitus. *J Hand Surg* 1995;20:109-14.
8. Smith LL, Burnet SP, McNeil JD. Musculoskeletal manifestations of diabetes mellitus. *Br J Sports Med* 2003;37:30-5.
9. Revach M, Cabilli C. Dupuytren's contracture and diabetes mellitus. *Isr J Med Sci* 1972;8:774-5.
10. Abe Y, Rokkaku T, Ofuchi S, et al. An objective method to evaluate the risk of recurrence and extension of Dupuytren's disease. *J Hand Surg Br* 2004;29:427-30.
11. Leden I, Schersten B, Svensson B, Svensson M. Locomotor system disorders in diabetes mellitus. Increased prevalence of palmar flexor tenosynovitis. *Scand J Rheumatol* 1983;12:260-2.
12. Heathcote JG, Cohen H, Noble J. Dupuytren's disease and diabetes mellitus. *Lancet* 1981;i:1420.
13. Larkin JG, Frier BM. Limited joint mobility and Dupuytren's contracture in diabetic, hypertensive and normal populations. *BMJ* 1986;292:1494.
14. Murrell GA, Hueston JT. Aetiology of Dupuytren's contracture. *Aust N Z J Surg* 1990;60:247-52.
15. Seyhan H, Kopp J, Schultze-Mosgau S, Horch RE. Increased metabolic activity of fibroblasts derived from cords compared with nodule fibroblasts sampling from patients with Dupuytren's contracture. *Plast Reconstr Surg* 2006;117:1248-52.
16. James WD, Odom RB. The role of the myofibroblast in Dupuytren's contracture. *Arch Dermatol* 1980;116:807-11.
17. Sanders JL, Dodd C, Ghahary A, et al. The effect of interferon-alpha2b on an in vitro model Dupuytren's contracture. *J Hand Surg Am* 1999;3:578-85.
18. Arkkila PE, Kantola IM, Viikari JS. Dupuytren's disease: association with chronic diabetic complications. *J*

- Rheumatol 1997;24:153-9.
19. Arkkila PE, Kantola IM, Viikari JS, et al. Dupuytren's disease in type 1 diabetic patients: a five-year prospective study. *Clin Exp Rheumatol* 1996;14:59-65.
 20. Tubiana R. Prognosis and treatment of Dupuytren's contracture. *J Bone Joint Surg Am.* 1955;37:1155-116.
 21. Hueston JT. Lessons in Dupuytren's disease. *Ann Chir Main Memb Super* 1992; 11:349-54.
 22. Early P. Population studies in Dupuytren's contracture. *J Bone Joint Surg* 1962;44B: 602-13.
 23. Freiberg A, Mulholland RS, Levine R. Non operative treatment of trigger fingers and thumbs. *J Hand Surg* 1989;14A:553-8.
 24. Adamietz B, Keilholz L, Grunert J, Sauer R. Radiotherapy of early stage Dupuytren disease. Long-term results after a median follow-up period of 10 years. *Strahlenther Onkol* 2001;177:604-10.
 25. Murrell GA, Murrell TG, Pilowsky E. A hypothesis for the resolution of Dupuytren's contracture with allopurinol. *Speculations Sci Technol* 1987;10:107-12.
 26. Ketchum LD, Donahue TK. The injection of nodules of Dupuytren's disease with triamcinolone acetonide. *J Hand Surg* 2000; 25:1157-62.
 27. Au-Yong IT, Wildin CJ, Dias JJ, Page RE. A review of common practice in Dupuytren surgery. *Tech Hand Up Extrem Surg* 2005;9:178-87.
 28. Trojjan TH, Chu SM. Dupuytren's disease: diagnosis and treatment. *Am Fam Physician* 2007;76:86-9.
 29. Wilbrand S, Ekblom A, Gerdin B. A cohort study linked increased mortality in patients treated surgically for Dupuytren's contracture. *J Clin Epidemiol* 2005;58:68-74.
 30. Abe Y, Rokkaku T, Ofuchi S, et al. An objective method to evaluate the risk of recurrence and extension of Dupuytren's disease. *J Hand Surg Br* 2004;29:427-30.
 31. McFarlane RM, Jamieson WG. Dupuytren's contracture. The management of one hundred patients. *J.B.F.J.S.* 1966;48:1095-1105.
 32. Leclercq C. Results of surgical treatment. In: Tubiana R, Mackin EJ, Hurst LC, Leclercq C, Badalamente MA. *Dupuytren's Disease.* Taylor & Francis 2000;239-265.
 33. World Health Organisation World Health Organization Department of Noncommunicable Disease Surveillance (1999).
 34. Crispin JC, Alcocer-Varela J. Rheumatologic manifestation of diabetes mellitus. *Am J Med* 2003;114:753-7.
 35. Kapoor A, Sibbit WL Jr. Contractures in diabetes mellitus: the syndrome of limited joint mobility. *Semin Arthritis Rheum* 1989;18:168-80.
 36. Jennings AM, Milner PC, Ward JD. Hand abnormalities are associated with the complications of diabetes in type 2 diabetes. *Diabet Med* 1989;6:43-7.
 37. Forgacs SS. Diabetes mellitus and rheumatic disease. *Clin Rheum Dis* 1986;12: 729-53.
 38. Carson J, Clarke C. Dupuytren's contracture in pensioners at the Royal Hospital Chelsea. *J R Coll Physicians Lond* 1993;27: 25-7.
 39. Lennox IA, Murali SR, Porter R. A study of the repeatability of the diagnosis of Dupuytren's contracture and its prevalence in the grampian region. *J Hand Surg* 1993;18:258-61.
 40. Starkman HS, Gleason RE, Rand LL, et al. Limited joint mobility (LJM). of the hand in patients with diabetes mellitus: relation to chronic complications. *Ann Rheum Dis* 1986;45:130-5.
 41. Noble J, Heathcote JG, Cohen H. Diabetes mellitus in the aetiology of Dupuytren's disease. *J Bone Joint Surg* 1984;66:322-5.
 42. Fujinaka Y. Diabetic osteoarthropathy. *Clin Calcium* 2009;19:1299-303.
 43. Papanas N, Maltezos E. The diabetic Hand: a forgotten complication? *J Diabetic Complications* 2010;24:154-62.