

Commentary: Dupuytren's Disease vs Non-Dupuytren's Contracture

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Gabiani and Majno¹ described in 1972 a cell that is encountered in Dupuytren's nodules and named it the *myofibroblast* because it shares morphologic features with fibroblasts and smooth muscle cells. They proposed that the clinical contracture in Dupuytren's disease (DD) is dependent on cell contraction. Laboratory studies concurred with these findings and documented the importance of this cell in the pathophysiology of DD; factor(s) in bovine serum can modulate normal fibroblasts into myofibroblasts, which are crucial for this active cellular process.² Although genetic predisposition is important in the etiology of DD environmental factors such as social habits and systemic disease seem to play a role in influencing the course of the disease.³

The debate about trauma and its potential role in the etiology of DD began when Dupuytren described the case of Demarteau the coachman who developed the disease from whipping horses and Goyrand who described the case of Chainé the hospital manager who developed the disease but had never done one day of hard work.⁴ This issue was not resolved and the debate continues. Larsen and Posch⁵ described hemosiderin deposits in some Dupuytren's diseased fascia and suggested that prior hemorrhage may be a causative factor. They also produced rupture of the palmar fascia in monkeys and created a lesion that was histologically similar to DD without digital contracture.⁶ Scores of reports and clinical observations for more than a century confirmed that trauma often preceded the development of Dupuytren's contracture. The issue that often was overlooked on examination of these reports was that most of these patients did not progress to typical DD; similar to Larsen's monkeys, they did not have digital contractures. The presence of blood factor(s) adjacent to a normal palmar fascia can modulate normal fibroblasts into contractile myofibroblasts that will manufacture abnormal, immature type III collagen with subsequent fascial proliferation and thickening. This happens regardless of the genetic make-up of the patient.

Palmar fascial proliferation therefore has 2 distinct clinical types, the typical DD and the atypical non-Dupuytren's contracture.⁷ McFarlane et al⁸ described the typical patient with DD as a white male of northern European origin, averaging 57 years of age, with bilateral hand and multiple digital involvements. The disease progresses over time but at variable rates. Heuston⁹ described patients with DD as expressing disease diathesis and those with strong diathesis invariably have family history and ectopic sites of fibromatosis such as the planter fascia (Ledderhose's contracture), male genitals (Peyronie's disease) or dorsal digital cutaneous disease (Garrod's nodes). This typical DD is believed to be transmitted as an autosomal dominant pattern of inheritance with variable penetrance.

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No benefits in any form have been received or will be received from a commercial party related directly or indirectly to the subject of this article.

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0363-5023/05/30A05-0021\$30.00/0

doi:10.1016/j.jhsa.2005.03.015

Non-Dupuytren's contracture is a nonprogressive palmar fascial proliferation that is distinguishable from DD by the following: the patients have no family history and there is ethnic diversity without gender predilection. The condition is nonprogressive and sometimes regressive. It is unilateral, is confined to the hand without ectopic manifestations, involves the palm, and may be in line with a single digit but without digital contracture. Nongenetic factors such as trauma (antecedent wrist or hand fracture), previous surgery (carpal tunnel or trigger finger release), and systemic conditions (diabetes, alcohol consumption) play a role in its pathogenesis. Patients with early typical DD may present as the non-Dupuytren's variety but as the condition progresses it becomes apparent that it belongs to the typical category.

The study by Reilly et al¹⁰ clearly introduced a novel concept and used a rational approach for understanding the progression of Dupuytren's nodules. Findings of interest in this investigation are: gender distribution, low rate of family history, ethnic diversity, and nodules not affecting the digits; furthermore, some patients' nodules did not progress but rather regressed. Undoubtedly with their clinical acumen these researchers included patients with typical disease in their cohort. Is it possible that some of their patients are of the non-Dupuytren's variety? This is difficult to answer because their patients did not have history of trauma. This study, however, is important because it brings this issue to the forefront and may serve as a reminder that future epidemiologic, natural history, and outcome studies pertaining to DD should differentiate between these 2 clinical entities if sound and accurate conclusions are to be drawn. Is it feasible to substantiate the clinical observations regarding the types of palmar fascial contracture? Only future genetic studies can elucidate this debate and reaffirm or refute the validity of these clinical observations about this enigmatic and fascinating disease.

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