treated with direct arterial anastamosis, $P = 0.86$). Finally, digits replanted with a vein graft survived 53% of the time compared to the 52% survival rate of those replanted without the use of a vein graft, which was also not statistically different ($P = 0.94$).

Summary Points:

- Vein grafts were utilized for arterial repairs in nearly half of all dysvascular digits that underwent revascularization or replantation.
- There was no statistical difference in the survival rate of dysvascular digits treated with a vein graft versus those that underwent direct arterial anastomosis.
- The need for a vein graft for a large zone of injury should not be considered a relative contraindication to perform revascularization or replantation of dysvascular digits.
- If the zone of injury is large, surgeons should have a low threshold to use vein grafts for the revascularization or replantation of digits.

PAPER 62

Clinical Paper Session 9
Vascular/Soft Tissue/Tumor — Saturday, September 9, 2017 • 10:27—10:32 AM
Evaluation/Diagnosis; Treatment; Basic Science

Involvement of Thrombin and Osteopontin in the Pathophysiology of Dupuytren's Contracture

N/A - not a clinical study

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COI: There is no financial information to disclose.

Hypothesis: Pathophysiology of Dupuytren's contracture (DC) remains unclear.[1,2] Thrombin is a multi-functional serine protease and a potent inducer of fibrogenic cytokines in various cells. Osteopontin (OPN), one of ECM proteins, can also modulate a variety of cellular activities associated with various chronic inflammatory disease including myocardial fibrosis after ischemic heart disease, liver cirrhosis and lung fibrosis.[3,4] Additionally, the presence of the thrombin-cleaved form of OPN is well correlated with various inflammatory disease activities.[5] We herein presented that myofibroblast (MF) expressed osteopontin, especially of thrombin-cleaved form, and the administration of thrombin induced differentiation into MF of fibroblast derived from Dupuytren's fascia.

Methods: The study group consisted of 25 patients (4 women and 21 men) who underwent resection of the palmer fascia for DC. The patients' mean age was 69.1 years (range, 58 to 82 years). All patients signed an informed consent document, and the study was approved by the institutional review board. The palmer apponeurosis resected in carpal tunnel release were used as control. Immunohistochemical studies were performed on serial sections with antibody against aSMA, OPN and thrombin cleaved-form of OPN antibody. For the determination of the effect of thrombin on DC, cells isolated from nodules and cords were starved in serum-free medium overnight prior to treatment with thrombin, 1 U/ml. After 24 hours, expression of aSMA and OPN were analyzed in total proteins collected from cells.

Results: Morphometric analysis showed that expression of aSMA was significantly correlated with that of OPN in the nodules of Dupuytren's fascia. In addition, there was expression of OPN and aSMA in 16 (67%) and 5 (20%) of 25 cases, respectively. Furthermore, thrombin-cleaved OPN was also immunelabeled on similar areas with OPN in nodules of Dupuytren's fascia (Fig. 62-1), considered that the majority of OPN's expression was thrombin-cleaved form in the nodules centered in the pathology. After treatment of thrombin, expression of aSMA and OPN were clearly upregulated in the cells from nodules as well as cord in western-blotting (Fig. 62-2), although there were weak expression of these molecules without application of thrombin.

Summary Points: The present study showed that myofibroblast expressed osteopontin (OPN) as well as thrombin-cleaved OPN in the nodules of Dupuytren's contracture (DC). In in vitro study, thrombin induced differentiation into myofibroblast from fibroblasts of both nodules and cords. Thrombin may involve in the pathology of progression and recurrence by direct effect or indirect pathway via cleavage of OPN.

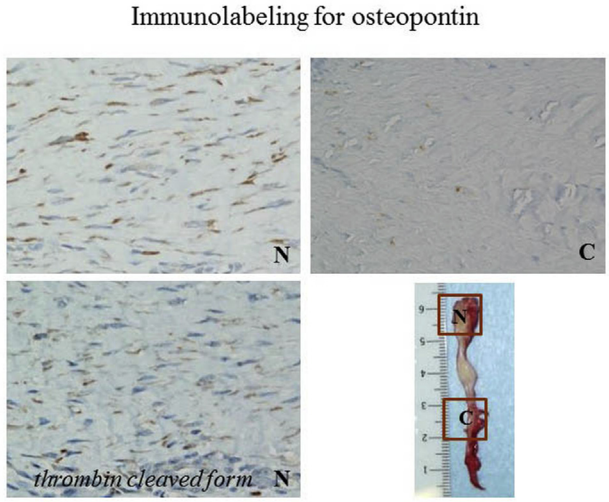

Figure 62-1: Immunolabeling for osteopontin.

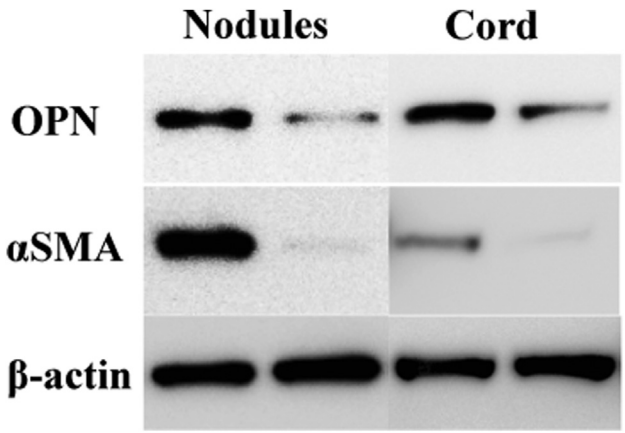

Figure 62-2: Western-blotting after treatment of thrombin.

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