SCIENTIFIC ARTICLE

Eosinophilic Fasciitis: Case Report

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A 16-year-old healthy boy presented with an acute claw hand and signs of flexor tenosynovitis or possible necrotizing fasciitis of all 4 fingers in 1 hand. After an operative incision and drainage and treatment with antibiotic, the patient improved; cultures were negative. He relapsed shortly after completing the 10-day course of antibiotics. A second incision and drainage was performed. Cultures from the second operation also revealed no infectious agent. The pathological report indicated a diagnosis of eosinophilic fasciitis. Our patient improved on antibiotics and nonsteroidal anti-inflammatory medication and has since had no further relapse. Eosinophilic fasciitis should be considered in the setting of acute claw hand and physical examination findings consistent with necrotizing fasciitis in which no infection is identified. One should consider biopsies as well as cultures during operative intervention in cases that clinically mimic serious infection but do not have purulence. *(J Hand Surg 2013;38A:2204–2207.*

Key word Eosinophilic fasciitis.

A HEALTHY, RIGHT-HANDED, HIGH school boy developed acute left hand and forearm swelling and erythema suggestive of a rapidly spreading infection. Intraoperative cultures were negative. Biopsy indicated a diagnosis of eosinophilic fasciitis. Eosinophilic fasciitis can mimic dramatic infections such as flexor tenosynovitis or necrotizing fasciitis.

CASE REPORT

A 16-year-old boy developed a swollen left little finger proximal interphalangeal joint with no discrete trauma on the first day of football practice. He subsequently presented to the emergency department with a 1-day history of considerable pain, swelling, and ascending erythema and cellulitis volarly from the left hand to the midforearm. He described median nerve distribution paresthesias distally. The patient exhibited clawing of all digits with fusiform swelling and tenderness over the flexor tendon sheaths extending into the palm and volar forearm. He had decreased active and passive range of motion of the wrist and fingers secondary to pain with any motion, especially on extension. The clinical presentation appeared consistent with flexor tenosynovitis. Although the patient was afebrile and stable, there was also concern for necrotizing fasciitis given the rapid progression. His white blood cell (WBC) count was 9.9×10^9/L (normal, $4.0–10.0 \times 10^9$/L) with 2% eosinophils (normal, 1%–4%). His erythrocyte sedimentation rate was 11 mm/h (normal, 0–20 mm/h), and his C-reactive protein was 23.5 mg/L (normal, 0.1–3.0 mg/L).

The patient underwent arthrotomy of the left little finger proximal interphalangeal joint, which was thought to be the source of the infection, debridement of the distal forearm and flexor tendon sheaths on the ulnar side of the hand, and a carpal tunnel release. We encountered no purulence. However, upon deeper dissection, we noted thickening of the flexor digitorum profundus tendon tenosynovium as well as copious edema in the forearm musculature and in the carpal tunnel. The median nerve was hyperemic.

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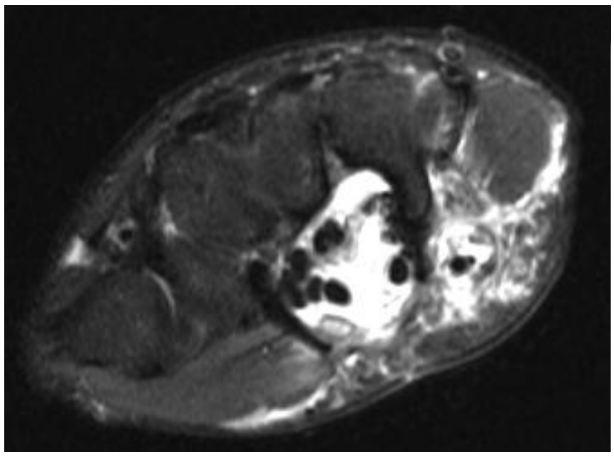

FIGURE 1: T2-weighted MRI scan shows marked edema within the carpal tunnel as well as in the overlying palmar surface.

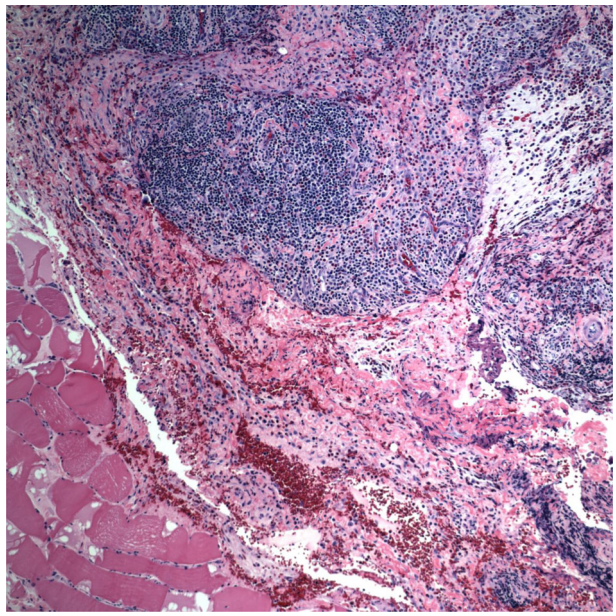

FIGURE 2: Histological slide at × 10 shows numerous cells, including lymphocytes, histiocytes, and eosinophils.

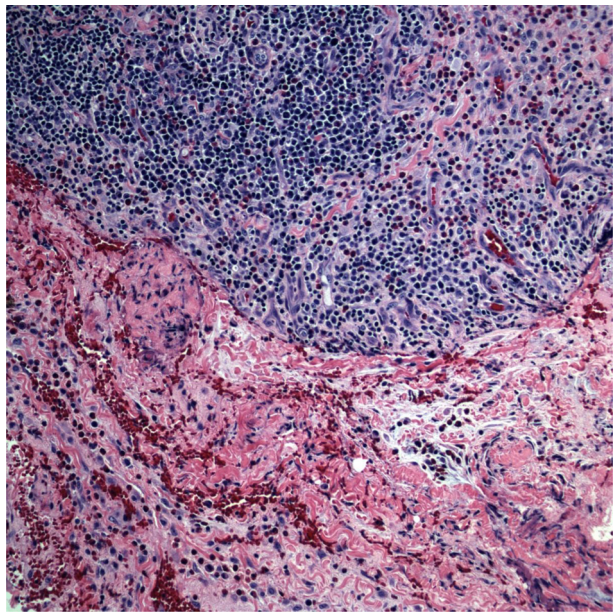

FIGURE 3: The same histological slide at × 20 shows many lymphocytes and eosinophils.

After surgery, the patient received vancomycin and then clindamycin intravenously and then clindamycin orally after marked improvement in pain, swelling, and median nerve symptoms. He was discharged with a presumptive diagnosis of infectious flexor tenosynovitis on the third postoperative day and was prescribed oral clindamycin for 10 days. The multiple intraoperative cultures from the proximal interphalangeal joint, forearm, and carpal tunnel remained negative. No biopsies were sent for pathology.

The patient completed 10 days of oral clindamycin. Four days after completing the antibiotic, he suddenly developed a complete recurrence of his earlier symptoms. His WBC count was 10.9×10^9/L with 6% eosinophils, the erythrocyte sedimentation rate was 10, and the C-reactive protein was 0.9. Radiographs showed only soft tissue swelling. A magnetic resonance imaging (MRI) study displayed extensive deep fascial and muscle edema involving the superficial and deep flexor muscles and tendons suggestive of a deep fascial infection and necrotizing fasciitis (Fig. 1). He was admitted to the hospital for repeat incision with debridement, re-release of the carpal tunnel, and flexor tenosynovectomy. We sent multiple cultures as well as biopsies. The patient gradually improved. After infectious disease consultation, he was placed on vancomycin and piperacillin/tazobactam and then transitioned to clindamycin. All cultures remained negative for bacteria, acid-fast bacteria, and fungi. However, the pathology report revealed an infiltrate of lymphocytes, abundant histiocytes, and numerous eosinophils, consistent with eosinophilic fasciitis (Figs. 2 and 3).

The patient underwent 6 weeks of oral nonsteroidal anti-inflammatory medication and clindamycin without relapse. Nine months later, he remained symptom free on no medications.

DISCUSSION

In 1974, Shulman[1,2] described an uncommon condition similar to scleroderma. Rodnan et al[3] designated it eosinophilic fasciitis at a meeting of the American Rheumatism Association the same year. Both Shulman and Rodnan et al had patients presenting with