Resolution of transient, marked eosinophilia, hyperimmunoglobulinemia E, and refractory eczematous dermatitis after the treatment of pancreatic pseudocysts

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Eosinophilia, defined as greater than 450 eosinophils/µL, can occur in various disease processes, including infectious, allergic, neoplastic, and primary hematologic disorders.1 Marked eosinophilia (>1500 eosinophils/µL) can be a diagnostic dilemma because blood eosinophilia rarely exceeds 1500/µL in atopic/allergic diseases.1 Hyperimmunoglobulinemia E is seen in allergic, parasitic, and primary immunodeficiency diseases.2 Although a hallmark of allergic disease is the infiltration of tissues with increased numbers of eosinophils, there is little evidence for immunoglobulin E–dependent function of eosinophils.2 Primary immunodeficiency disease, autosomal dominant hyperimmunoglobulinemia E syndrome or Job syndrome, is caused by mutations of STAT3 gene, and shows greater than 2000 IU/mL of a peak serum immunoglobulin E, eosinophilia, and eczematous dermatitis.3 Thus, T-cell immunodeficiency also causes eosinophilia, hyperimmunoglobulinemia E, and eczematous dermatitis.

Pancreatic diseases occasionally cause eosinophilia.4,5 Although both eosinophilia and hyperimmunoglobulinemia E are linked with eczematous dermatitis, skin manifestations associated with pancreatic diseases such as pancreatic panniculitis are uncommon.6 We present a case of a 60-year-old Japanese man with pancreatitis and refractory eczematous dermatitis accompanied with transient, marked eosinophilia and hyperimmunoglobulinemia E. Eczematous dermatitis completely resolved with treatment of his pancreatic pseudocysts.

CASE REPORT

A previously healthy 60-year-old Japanese man presented to the division of internal medicine with dyspnea and left-sided chest pain. His past medical history was only significant for excessive alcohol consumption. On examination, he was tachycardic and had slightly low pulse oximetry saturation with a normal blood pressure range and body temperature. Baseline laboratory results were as follows: while blood cell count 9290/µL (increased), serum total protein 7.1 g/dL, serum albumin 3.8 g/dL, aspartate aminotransferase 28 IU/mL, alanine aminotransferase 19 IU/L, alkaline phosphatase 285 IU/L, γ-GTP 53 IU/L (increased), and C-reactive protein 11.60 mg/dL (increased). Chest radiograph showed infiltrates in the left lower lobe of the lung. He was admitted to our division of internal medicine for potential pneumonia.

His major symptom of chest pain resolved with oral tosufloxacin (antibacterial) and diclofenac sodium. The initial computed tomography (CT) scan showed ascites and an intraperitoneal abscess in the left upper quadrant, which raised the potential diagnosis of peritonitis. After resolution of chest pain, he reported diffuse pruritus. Eosinophilia and hyperimmunoglobulinemia E rapidly increased to...
55.3% (6420/\text{L}) and 3713 \text{IU/mL}, respectively (Fig 1). Repeat CT scan showed that the abscess in the left upper quadrant was diminished in size, but an accumulation of fluid appeared in the tail of the pancreas. Because his pruritus and skin manifestations were worsening, he was referred to the dermatology division.

On his first visit to the dermatology division, he exhibited lichenified papules and plaques with excoriations on his back and legs (Fig 2) and scaly erythema on the scalp. A third CT scan confirmed pancreatic pseudocysts. These CT findings suggested that the causal factor of his disease was pancreatic inflammation. Although he strongly preferred supportive care for pancreatic pseudocysts, the cysts were becoming larger (Fig 3) with increasing amylase levels. In addition, his pruritus and eczematous dermatitis were refractory to oral olopatadine hydrochloride (antihistamine) and topical betamethasone butyrate propionate. Finally, ultrasound-guided percutaneous drainage (US-PD) was performed to treat the cysts.

The patient’s pruritus temporally exacerbated shortly after US-PD, but he began to respond to treatment and the symptom improved. Approximately 75% of his skin lesions resolved 6 months after US-PD combined with an improvement of pancreatic pseudocysts (Fig 4). Currently abstinent from alcohol, he remains free of eczematous dermatitis with quiescent pancreatic pseudocysts.

**DISCUSSION**

Pancreatic pseudocysts are a delayed complication of acute or chronic pancreatitis that consist of fluids containing pancreatic enzymes. Most pseudocysts resolve spontaneously with supportive care, but symptomatic ones often require therapeutic
intervention such as endoscopic drainage, US-PD, or open surgery.\textsuperscript{7}

Chronic pancreatitis was first reported to be associated with eosinophilia in 1946.\textsuperscript{8} The incidence of eosinophilia is between 15.6\% (28 of 180 patients)\textsuperscript{4} and 17.2\% (21 of 122 patients)\textsuperscript{5} during the course of chronic pancreatitis. However, marked peripheral blood eosinophilia is rarely associated with chronic pancreatitis.\textsuperscript{1}

In our case, we propose that pancreatic inflammation may have caused skin manifestations for the following reasons: (1) the patient did not have any atopic/allergic diseases; (2) both eczematous dermatitis and pruritus were exacerbated with pancreatic inflammation, including elevated serum amylase levels (Fig 1); (3) after the patient’s pseudocysts were treated with US-PD, the skin manifestations and pancreatic pseudocysts improved; and (4) eczematous dermatitis resolved spontaneously with quiescence of pancreatic pseudocysts as a result of abstinence from alcohol. Taken together, these data suggest that severity of skin manifestations may be correlated with the extent of pancreatic pseudocysts.

In conclusion, pancreatic diseases, including pancreatic pseudocysts, should be considered in the differential diagnosis of refractory dermatitis with eosinophilia and hyperimmunoglobulinemia E.

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