Jaundice and Multiple Tender Nodules on the Lower Legs in a 54-year-old Woman

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CASE REPORT

A 54 year-old woman presented with tender subcutaneous nodules on lower extremities for 2 weeks. One week before the eruption, she also developed jaundice and mild abdominal pain. Physical examination revealed multiple red to brownish nodules on the lower legs without skin necrosis or ulceration (Fig. 1). A skin biopsy was performed and the specimen was submitted for hematoxylin-eosin stain (Fig. 2). Abnormal laboratory results included hemoglobin 11.1 g/dl (12-16), amylase 445 U/l (27-137), lipase 4292 U/l (<190), and total serum bilirubin 9.6 mg/dl (0-1.3).
Diagnosis: Pancreatic Panniculitis Associated with Pancreatic Carcinoma

Microscopic findings and clinical course

Hematoxylin and eosin stain revealed loci of fat necrosis with neutrophil infiltrates, shadowy basophilic and necrotic adipocytes (ghost-like cells), and microcyst formation.

On admission, a computed tomography scan of the abdomen showed an ill-defined heterogeneous mass at the pancreatic head measuring 6 cm in diameter. A biopsy specimen of the small intestine taken with ERCP (endoscopic retrograde cholangio-pancreatography) showed poorly-differentiated carcinoma. The patient expired due to severe biliary tract infection with septic shock.

Disseminated fat necrosis is a characteristic cutaneous manifestation of pancreatic disorders. In a review of 873 patients with various pancreatic disorders, only one patient associated with pancreatic panniculitis was identified. Classic clinical features are single or multiple red to brown, occasionally painful and ill-defined nodules that predate or coincide with other manifestations of underlying pancreatic diseases. Nodules typically occur on lower extremities, and cases involving buttocks, upper extremities, and trunk had been reported. The lesions usually involute after 2-8 weeks with sequela of lipoatrophy and hyperpigmentation. In severe cases, the lesions may become fluctuant and ulcerate with oily and yellow discharge. Other systemic complications of pancreatic panniculitis included fever, mild eosinophilia, abdominal pain, vomiting, and arthritis.

Histopathology of pancreatic panniculitis is pathognomonic and fat necrosis, ghost-like cells, calcification, microcyst formation. The pathogenesis of pancreatic panniculitis remains obscure. There is considerable evidence to suggest that pancreatic enzyme release plays a key role in the fat necrosis. In experimental study had also documented the fat necrosis is correlated with the amylase level in pancreatitis. However, other factors may be involved in the pathogenesis because only a minority of patients with pancreatic disorders with high level of pancreatic enzyme in the serum developed the distant fat necrosis.

Pancreatic panniculitis is usually associated with major morbidity and mortality. In a review of 27 cases of pancreatic panniculitis, 8 patients developed pancreatic carcinoma and 8 (42%) of the 19 patients with pancreatitis died of their disease.

Treatment of pancreatic panniculitis is mainly supportive and the condition usually subsides if the underlying pancreatic disorder is corrected. Plasmapheresis, cholecystectomy, pancreaticoduodenectomy and placement of pancreatic duct stent were reported modalities of treatment of pancreatic panniculitis.

In conclusion, disseminated fat necrosis is a well-established complication of pancreatic disease. The knowledge of its specific clinical and pathological features facilitates early and rapid diagnosis without other manifestations of underlying diseases. We reported our first experience and reviewed the literature herein.

Reference

Symmetric Erythema in A Diabetic Patient

Jung-Yi Chan, Chia-Yu Chu

CASE REPORT

A 54-year-old female patient, who has been diagnosed as having diabetes mellitus for more than 10 years without regular medical control, was transferred to our hospital due to active upper gastrointestinal (UGI) bleeding and impending hypovolemic shock. Asymptomatic skin rashes developed symmetrically on the four extremities on admission. She had been treated with ampicillin/subactam for 4 days in another hospital for symptomatic pyuria. Erysipelas or cellulitis was suspected by the internal medicine doctors. On physical examination, she was afebrile. Several well-defined, palm-sized, erythematous patches distributed symmetrically on the forearms, dorsal hands, and legs with swelling were noted. There were also purpura and bulla formation on both shins (Fig. 1, 2). The peripheral pulsations were all intact.

The hemogram revealed WBC: 6300/mm³. The erythrocyte sedimentation rate (ESR) level was within normal limit. The X-ray studies of the feet and hands did not reveal any destructive bone lesions.

A skin biopsy taken from the erythematous lesion on her left forearm (Fig. 3) revealed no remarkable epidermal change with solar elastosis and sparse inflammatory cells in the dermis microscopically. Many ectatic vessels were also noted in the dermis (Fig. 4). The skin lesions were gradually resolved two weeks later without active treatments.

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Accept for publication: February 19, 2003

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DIAGNOSIS: Erysipelas-like erythema in diabetes mellitus (EEDM)

DISCUSSION

Erysipelas-like erythema, one of the cutaneous manifestations due to small vessel insufficiency in diabetes mellitus was first described by Lithner. The erythema is well-dermacated, painless with the size of a child's palm or larger and is mainly located on the legs and feet of diabetic patients. It is not associated with pyrexia, elevated ESR, or leukocytosis. In most of the patients in the Lithner's original report, their average age was 73 and the average duration of diabetes was 5.4 years. The erythema appeared to be precipitated by cardiac decompensation with or without leg edema, or by venous thrombosis with resulting unilateral edema. It was obvious that swelling always preceded the development of erythema and that the latter receded when swelling subsided. The skin lesions were usually reversible. It is characteristic that the skin lesions may recur after once having healed, may be at a site other than that at which they were first observed. It was also noted that increased percentage of roentgenologic evidence of bone destruction in involved feet of EEDM. They considered this erysipelas-like erythema to be incipient gangrene and the bony destruction to be the result of an underlying microangiopathy.

Histopathologically, there are many ecstatic vessels in the dermis. The mechanism is attributed to the compensatory increased peripheral microcirculation caused by decreased perfusion of large vessels in diabetic complications.

The most important differential diagnosis of EEDM is erysipelas or cellulites. Lack of systemic manifestations such as fever, leukocytosis and increased ESR in addition to the symmetrical distribution of diffuse erythema and neither local heat nor pain, all suggested a diagnosis of EEDM.

REFERENCES