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Grey Turner's sign in acute pancreatitis

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An 83-year-old man, a known alcoholic, complained of a fever and acute epigastric pain radiating to the back. Tenderness on palpation was present in the right flank. On admission, the patient was febrile and had increased abdominal girth with pain. On the third day of hospitalization, distinct ecchymosis developed in the right flank (Grey Turner's sign; Fig 1). Laboratory findings revealed leukocytosis of 24,000/mm3, aspartate aminotransferase 51 U/L, alanine aminotransferase 22 U/L, creatinine phosphokinase 553 U/L, amylase 1071 IU/L, and 1.4 mg/dL total bilirubin. An abdominal noncontrast computed tomography revealed acute pancreatitis with inflammation that had spread to the duodenum and forward right anterior abdominal wall (Fig 2, *A*). Extensive retroperitoneal inflammation from severe acute pancreatitis was present. The site of the Grey Turner's sign correlated with the site of the extrapancreatic inflammation on imaging (Fig 2, *B*). Based on these findings and the clinical history, a diagnosis of acute severe pancreatitis with Grey Turner's sign was made. The patient developed pancreatic ascites and right pancreatic hydrothorax, but was managed conservatively. The patient recovered well despite the poor prognosis associated with Grey Turner's sign.

Grey Turner's sign was first described in 1920 in a patient with acute pancreatitis.[1] Subcutaneous manifestations of severe pancreatitis are often discussed but seldom observed. Grey Turner's sign develops in 3% of patients with acute pancreatitis. Discoloration indicates extravasation of blood in the subcutaneous tissue and resembles ecchymosis.[2] Extension of the effects of pancreatitis may occur through the loose retroperitoneal tissue space.[3] Areas of fat necrosis are common in the transverse mesocolon and omentum.[2] Grey Turner's sign is nonspecific and has been described as bruising of retroperitoneal hemorrhage secondary to

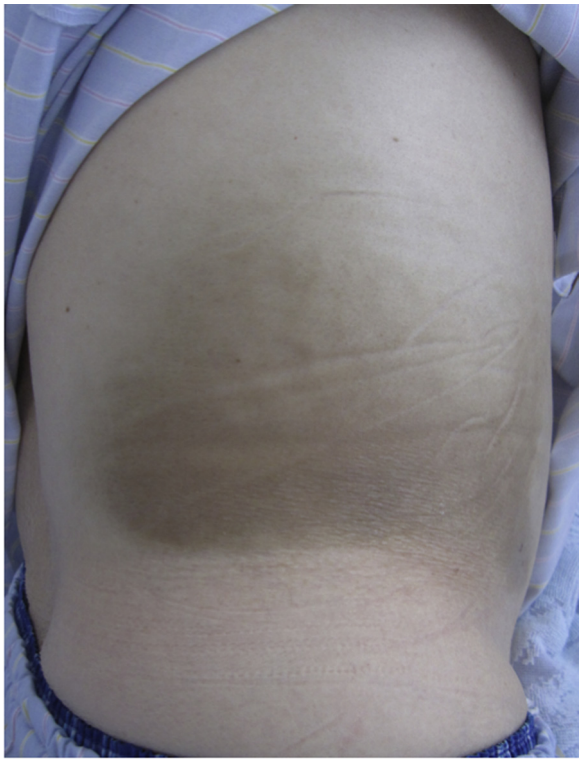

Fig 1. Grey Turner's sign. A dirty green ecchymosis developed in the right flank on the third day of hospitalization.

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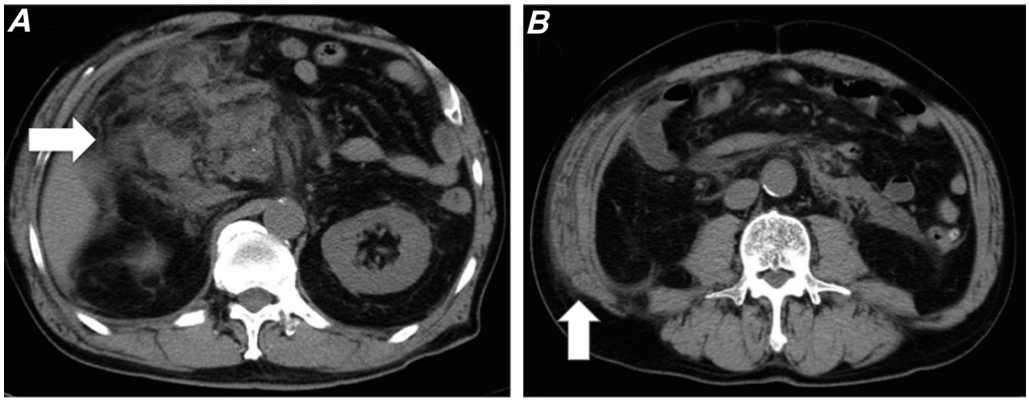

Fig 2. Abdominal noncontrast computed tomography (CT). An abdominal CT revealed an extension of extra pancreatic fluid and reaction into the (*A*) transverse mesocolon and small intestine mesentery, as well as (*B*) thickening of the lateral paries.

hepatocellular carcinoma, perirenal hematoma, trauma, ruptured ectopic, aortic rupture, and portal hypertension.[4] Most cutaneous manifestations are in patients with severe disease who develop major complications. Communication may be established to the posterior pararenal space and to the structures of the flank wall. The lumbar triangle of anatomic weakness on the flank wall may serve as a structural predisposition. Defects in the lumbar musculature in the region result in two sites known as the larger superior triangle, inferior to the 12th rib, the floor of which is formed by the outer third of the quadrant lumborum muscle and transversalis fascia, and the smaller inferior lumbar triangle to the iliac crest, the floor of which is composed of the underlying internal oblique and transversus abdominal muscles.[5] Grey Turner's sign is caused by pancreatitis or the spread of hemorrhagic fluid from the anterior pararenal space, between the posterior renal fascia and the quadrant lumborum muscle, and subsequently, to the subcutaneous tissues via a defect in the fascia of the flank. Since it was first described, skin signs have been considered rare and indicate an unfavorable prognosis in acute severe pancreatitis.

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