MEDICAL MEMORANDA

Unusual Presentation of Seat-belt Syndrome

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Seat-belt injuries are being increasingly recognized as the use of seat belts gains in popularity. Only a small proportion of the injuries involve intra-abdominal organs (Garrett and Braunstein, 1962), the small bowel and mesentery being most often damaged (Macleod and Nicholson, 1969), whereas solid organs are relatively infrequently affected. Only one case of pancreatic injury was reported among the 41 cases reviewed by Macleod and Nicholson (1969). We report a case of seat-belt pancreatic injury presenting with subcutaneous fat necrosis.

Case History

A 27-year-old woman was a front-seat passenger in a car which was travelling at about 50 miles (80 km) an hour when it collided with another vehicle. She was wearing a combined lap strap and diagonal seat belt but this was lying rather high up on her abdomen as the seat was fully reclined. She lost consciousness briefly after the accident and was admitted to the regional hospital with chest pain. A fractured left ninth rib was the only injury found and she was discharged after nine days in hospital. A week later crops of reddish, tender nodules appeared on her legs and, later, also on her abdomen and right breast. These nodules became darker and somewhat indurated and resolved leaving a brownish plaque in the skin. They came and went in cycles lasting about two weeks. Biopsy of two lumps showed simple fat necrosis.

The nodules ceased to appear eight weeks after the accident. She then noticed painless, progressive distension of her abdomen over a period of three weeks and was admitted to the Manchester Royal Infirmary. Her general condition was excellent and apart from moderate ascites physical examination showed nothing abnormal. White cell count was 7,900/mm³, haemoglobin 12.6 g/100 ml, E.S.R. 44 mm in the first hour, serum albumin 3.5 g and globulin 3.7 g/100 ml, and serum electrolytes, calcium, inorganic phosphorus, and fasting blood sugar were normal. Serum amylase was 2,300 units/100 ml (normal 40-140 units/100 ml) and serum lipase 5.5 units/ml (normal less than 1.5 units/ml). A peritoneal tap yielded 3.5 l. of straw-coloured fluid with a protein content of 2.5 g/100 ml and an amylase content of greater than 30,000 units/100 ml. A diagnosis of pancreatic injury was made and she was put on a "drip-and-suck" regimen pending laparotomy. At operation the pancreas was found to be transected for three-quarters of its width in the neck, and the head and body were minimally thickened. There was a small cyst in communication with the injured region. A few tiny areas of fat necrosis were seen and 2 l. of clear, straw-coloured fluid was removed. The pancreas

distal to the rupture was removed together with the spleen. Post-operative recovery was uneventful. She was well when last seen on 19 April 14 months after the operation, having no abdominal pain or steatorrhoea, and her random blood sugar remained within the normal range.

Comment

This case presents several unusual features. To our knowledge it is the first reported case of the seat-belt syndrome in which the pancreas was the only abdominal organ injured. Systemic fat necrosis is a well-documented feature of acute pancreatitis and has been reported in association with pancreatic carcinoma (Belsky and Cornell, 1955) and pancreatic calculi (Lucas and Owen, 1962). It is believed to result from activation of circulating pancreatic enzymes, and a common feature is a high serum amylase level, usually over 600 Somogyi units/100 ml. Virtually all reported cases of pancreatitis with systemic fat necrosis as a prominent feature have also shown other acute symptoms and signs of pancreatitis. It is unusual, as in our patient, for the condition to present with subcutaneous fat necrosis without acute symptoms of pancreatic damage.

It is possible that the pancreatic duct was injured and became quickly sealed off as a result of oedema and other post-traumatic tissue reactions. A significant leakage of pancreatic enzymes into the abdominal cavity was thus prevented, thereby explaining the absence of acute symptoms. Popper and Necheles (1940) showed in animals that obstruction of the pancreatic duct leads to an excess of amylase and lipase in the portal vein and thoracic duct. A similar mechanism would explain the high enzyme levels in our patient, in whom the distal portion of the severed pancreas presumably continued to secrete enzymes. The development of ascites could be explained by recannulation of branches of the pancreatic duct, some of which probably communicated with and secreted enzymes slowly into the abdominal cavity. Another route by which pancreatic enzymes could have reached the abdominal cavity was a leak from the cyst.

It is obvious from the history of this patient and the experience of other workers (Williams *et al.*, 1966; Sube *et al.*, 1967; Towne and Coe, 1971) that the absence of symptoms is no guarantee against the presence of serious abdominal injuries. Patients who have had a sudden and unexpected impact against the car seat belt should be closely watched for the development of any symptoms indicating visceral damage.

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