Medical Memoranda

Haemorrhagic Fever with Raised Serum Amylase

COMMENT

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The clinical features of haemorrhagic fever have been well described by Chew *et al.* (1961) and Hanam (1964). One of the features is abdominal pain, but Chew *et al.* (1961) noted that it was rare in the Singapore type, in contrast to the related Korean (Barbero *et al.*, 1953), Thai (Nelson, 1960), and Filipino (Quintos, 1954) variants, in all of which pain was a common symptom. Goldsmith *et al.* (1965) noted that there has been a changing pattern in the clinical presentation of the condition in this part of the world. They observed that abdominal pain was present in 26% of cases. So far no explanation has been advanced for the cause of this symptom, though Oliver thought that mild pancreatitis could occur in classical endemic dengue (de Langen and Lichtenstein, 1936).

As far as we are aware this is the first report of a case of haemorrhagic fever with severe abdominal pain in which a raised serum amylase was recorded. This was probably due to acute pancreatitis.

CASE REPORT

A 21-year-old Chinese housewife was admitted to hospital on 11 September 1967 for complaints of fever and abdominal pain. After a fever of two days' duration she developed severe pain in the epigastrium and left hypochondrium. The pain was constant and non-radiating, and was so severe that she vomited yellowish fluid on two occasions. There was no history of similar pains.

Examination showed her to be febrile, with a temperature of 99° F. (37.2° C.). She was not in shock. Her whole body appeared flushed and there were petechial haemorrhages. The blood pressure was 110/70 mm. Hg. There was marked tenderness over the epigastrium and the back. Hess's test was positive. No other abnormalities were noted in the other systems.

On investigation the haemoglobin was 15 g./100 ml., white cell count 3,000/cu. mm., platelets 90,000/cu. mm., serum amylase 256 Wohlgemuth units (normal, 0-16 units), and urinary diastase more than 2,048 Wohlgemuth units (normal, 8-32 units).

She was treated with intravenous fluids, gastric aspiration, and antispasmodics. Her condition improved the next day, though her temperature was 101° F. (38.3° C.), and there was less tenderness in the epigastrium. The white cell count was 3,500/cu. mm. (69% polymorphonuclears, 22% lymphocytes, 3% monocytes, 2% eosinophils, and 4% atypical mononuclear cells), platelets 120,000/cu. mm., serum amylase 128 units, and urinary diastase 256 units.

By the third day in hospital the epigastric tenderness had subsided, and by the fifth day she was afebrile and well, and the haemorrhagic rash had begun to clear up. The white count rose to 7,300 and the platelets to 215,000/cu. mm., but the serum amylase fell to 10 units and urinary diastase to 54 units. Paired sera showed high sustained complement fixation antibodies with titres of more than 512 when tested against three group B arboviruses—Japanese encephalitis, dengue 1, and dengue 2. The method used was that described by Lim *et al.* (1964). Two months later a cholecystogram showed good function of the gall bladder and no stones.

This patient had all the clinical features of haemorrhagic fever—namely, fever, purpuric rash, thrombocytopenia, and positive serology. The unusual feature in this case was the severe abdominal pain almost simulating an acute abdomen. This led us to a study of the serum amylase, which was markedly raised and subsequently fell to normal as the patient's condition improved. A raised serum amylase level is seen in many conditions—namely, acute pancreatitis, mumps, infective parotitis, gall-bladder disease, and macroamylasaemia.

It is unlikely that disease of salivary glands was responsible for the raised amylasaemia, as there was not evidence of parotid gland enlargement. Gall-bladder disease could also be excluded, as there was no clinical evidence to suggest such a condition. Furthermore, a cholecystogram did not show any abnormality in the biliary system.

Macroamylasaemia has been described by Berk *et al.* (1967). It is characterized by persistently raised serum amylase associated with very little clinical symptomatology. This has been postulated to be due to the amylase being attached to large protein molecules which cannot be excreted in the urine. In this case the fall of serum amylase during convalescence excluded the presence of macroamylasaemia.

In our opinion the most likely cause of the abdominal pain was acute pancreatitis. Aw *et al.* (1967) has shown that iso-amylase estimation is of great value in diagnosing pancreatitis. Unfortunately this was not carried out. Had this been done the final diagnosis of acute pancreatitis would have been made with more certainty. It is tempting to speculate that the cause of acute pancreatitis in this case was due to petechial haemorrhages in the pancreas as part of the same disease process.

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