ACUTE HAEMORRHAGIC PANCREATITIS SIMULATING RUPTURED ECTOPIC PREGNANCY (2 CASE REPORTS)

PIKU CHAUDHURI • GIRIN DAS • SANKAR KUMAR DAS

SUMMARY

Two cases of Acute Haemorrhagic Pancreatitis simulating ruptured ectopic pregnancy are reported. The cases were admitted with signs and symptoms of shock and haemoperitoneum, pain and tenderness in abdomen and pouch of Douglas, anaemia and pallor. Laparotomy was performed in both the cases. The diagnosis of Haemorrhagic Pancreatitis was established at laparotomy by the presence of haemoperitoneum, the finding of a firm pancreas and of omentum with necrotic foci characteristic of Haemorrhagic Pancreatitis and with no evidence of ectopic pregnancy in the abdomen and pelvis. Histological study of the omental biopsy showing fat necrosis was also very suggestive.

Introduction

Acute Haemorrhagic Pancreatitis is a complication of acute pancreatitis. Pancreatitis is caused essentially by the digestion of the pancreas by its own enzyme. Enzyme trypsin is the key offender which activates the majority of the pro-enzyme participating in autodigestion. Trypsin by indirectly activating clotting and complement systems and releasing the mediators contributes to local inflammation, thrombosis, tissue damage and haemorrhage. Elastase present in inactive form in the pancreatic juice is activated by trypsin and this active form causes destruction of the elastic fibres of blood vessels leading to haemorrhage.

In Britain about 50% of cases (John Macleod, 1981) are associated with biliary disease and about 20% with alcoholism, while in about 9-50% of cases (Robbins Cotran, 1981) no cause can be identified. About one - third of the cases are recognised as perforated peptic ulcer or acute appendicitis or cholecystitis. A hereditary predisposition to pancreatitis has been identified in some families transmitted as autosomal dominant trait. However such familial diseases more commonly take the form of chronic pancreatitis rather than acute pancreatitis (Robbin & Cotran, 1981).

Dept. of Obstetrics & Gynaecology, Calcutta National Medical College & Hospitals
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Acute Pancreatitis is a recognised complication of renal transplanation, maybe caused by corticosteriod and oral contraceptive. Patients on estrogen therapy who develop hyperlipidaemia have recurrent attacks of Pancratitis. It occurs occasionally after abdominal trauma and rarely in mumps, hyperparathyroidism, hyperlipidaemia, hypothermia.

Case History

Case - I

Mrs. J.A., 21 years, muslim female of 42/1/B Samsul Hauda Road, Calcutta - 17, married for 1′ years, Para 1+0, had a normal delivery 6 months ago, L.M.P. : 15.4.86 just overdue without definite amenorrhoea. (Overdue of the period was not very unusual in her previous menstrual history), weight 55 kgs, was attended on 17/7/1987, Registration No.1733/CMC/86 with acute pain in the abdomen, anaemia and pallor. She was immediately admitted to Elliot Nursing Home, Calcutta with a provisional diagnosis of Ruptured Ectopic Pregnancy. There was no history suggestive of Alcoholism, Cholecystitis, corticosteriod therapy. She had a history of intake of oral contraceptives pills after marriage but flatulance for last 1-2 years without exacerabation and not completely relieved after treatment.

General Surgey and Examination

Examination of the patient revealed the patient to be anaemic and pale in appearance with signs and symptoms of shock. Blood Pressure was 100/60 mm of Hg, Pulse 120/min, Respiration rate was 24-26/min and extremities were cold. On palpation of the abdomen, there was acute widespread tenderness and muscles guarded. Chest was auscultated and found to be normal. No jaundice was detected.

Investigations

Hb% : 9.2 gm%
P.P. Sugar :
1st post operative day - 122 mgs/100 ml
8th post operative day - 110 mgs/100 ml
Serum Amylase estimation
1st post operative day - 600 units.

EUA, D&C and Needling of the pouch of Douglas was done on 17/5/86. Per vaginal examination revealed a normal size uterus, but the fornices and pouch of Douglas were full and very tender. This was followed by immediate laparotomy. Laparotomy revealed haemoperitoneum, uterus and adnexae were found normal. Pancreas was felt firm. Omentum was found thick and presented with opaque yellowish white hard necrotic areas. Biopsy was taken from the omental tissues. Both omental biopsy tissue and endometrial curettage were sent for histopathological examination. No blood was transfused post operatively. Patient started vaginal bleeding resembling normal menstruation from the day of the operation.

Histopathology of omental tissue

This revealed fat necrosis of the omental tissues which is characteristic of Acute Haemorrhagic Pancreatitis.

Histopathology of the Endometrial tissue

This revealed late secretory phase of the menstrual cycle.

Case - II

Mrs. S.B., 30 years, muslim female of 43, Phool Bagan Road, Calcutta - 14, Para 3+0, L.C.B - 1′ years back, was admitted to Chittaranjan Hospital on 2/5/86, Registration No.2233 with pain in the abdomen, fever (101°F) and vomiting and a provisional diagnosis of ruptured ectopic

pregnancy. There was no history of any amenorrhoea and her last normal period was 26 days ago.

General Survey and Examination

On examination patient revealed severe anaemia and pallor with signs and symptoms of shock. Blood Pressure was 90/70 mm of Hg, Pulse 130/min, with cold and clammy extremeties. Abdomen on palpitation was tender all over and chest was normal on auscultation.

Per Vaginal Examination

This revealed a normal size uterus but the fornices and pouch of Douglas were full and very tender.

Investigation

Hb - 8 gms%,
P.P. sugar - 128 gms/100 ml.
ABO group and Rh type :
Group 'B' RhD +ve
Serum Amylase Estimation : Not done.

Laparotomy was performed on 2/5/86 which revealed Haemoperitoneum. Uterus and adnexa were found normal. Pancreas was palpated to be firm and omentum presented with hard yellowish white necrotic foci. Biopsy from omental tissue was not taken. Patient received two bottles of blood post operatively.

Conclusion

Prognosis of Acute Pancreatitis depends upon severity of the attack. Overall mortality os 20% and in Acute Haemorrhagic Pancratitis it is over 50% (John Macleod, 1981). If the patient survives, active necrotising damage may slowly resolve and be replaced by diffuse or focal parenchymal or stromal fibrosis, calcification and irregular ductal dilation.

Fortunately both the cases reported survived and had uneventful post operative recovery.

References

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2. *Robbins Stanely, L., Cotran Ramzi, S. (1981): Pathologic Basis of Disease, 2nd Edition, W.B. Saunders Company, 1097, 1098, 1100.*