An Unusual Presentation of Acute Abdomen

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Background:

Acute fulminant hepatic failure to present as acute abdominal pain is rare. Acute abdominal pain is the UK's most common surgical emergency, it accounts for

- 1% of all hospital admissions
- 6% of referrals to A&E
- 18% of admissions to surgical wards

For a senior house officer to miss a diagnosis like acute fulminant hepatic failure is very easy as most of the patients present with following diagnosis.

For patients in A&E or on surgical wards these include:	For women these include:
• Perforated ulcer • Pancreatitis • Cholecystitis • Renal Colic • Bowel Obstruction • Dyspepsia	• Appendicitis • Non Specific • Abdominal pain (NSAP) • Pelvic Inflammatory Disease (PID) • Urinary Tract Infection (UTI) • Ectopic Pregnancy • Ovarian cyst • Incomplete abortion

INTRODUCTION:

A 39-year-old lady presented to A & E with sudden onset of generalized abdominal pain since morning and vomiting for the last 2 days. She vomited out whatever she ate or drank. she had a generalized abdominal pain radiating to her back. Earlier she had absolute constipation for 2 days but later she developed diarrhoea. She also complained of burning micturition. She had a break up with her husband last week and since then she had not eaten properly. There is no history of overdose of any medication. She only drinks alcohol moderately. She has no past significant medical or surgical history. Vital sign were Temp 37.2 , Pulse 142, BP 130/80 , oxygen saturation 97%.

On general physical examination she was mildly jaundiced and had generalized abdominal tenderness with rigidity. Bowel sounds were absent. Rest of the systemic examination was normal.

Blood gases showed pH 7.32 oxygen saturation 96%, po2 9.2, pco2 5.0 mild hypoxic picture. Erect chest x-ray was normal but abdominal x-ray showed cut off sign as picked up by an A&E registrar. Blood investigations were requested at that stage for full haematological, biochemical and toxicological screen.

Patient was referred to surgical team with suspicion of acute pancreatitis. Later on laboratory informed us that she had markedly deranged LFT's with serum bilirubin 98, ALT 13267 serum LDH was in thousands, all other investigations including serun amylase,paracetamol , salicylate levels were normal.

She was immediately shifted to medical HDU and treated as a case of acute fulminant hepatitis /hepatic failure. Later on her serological screening for viral infection proved to be negative for antibodies to hepatitits A, hep B surface antigen, hep B core antigen, and hep C virus. Serum titres for smooth muscle

antibodies and nuclear and mitochondrial antibodies (ANA, AMA) were negative. No paraprotein was detected in serum electrophoresis. Only positive finding was detection of igG antibodies of CMV and Epstein barr virus. Her blood cultures were negative for any bacterial growth and abdominal ultra sound was normal.

She was treated in medical high dependency under c/o GI team of RHH in liaison with liver unit of QE hospital for acute fulminant hepatic failure of unknown etiology. After three weeks of intense treatment on tazosin and fluconazole her liver function tests improved dramatically in three weeks.Later ALT was 178 when she was discharged .

She was offered follow up as an outpatient in clinic.

Discussion:

Acute liver failure or fulminant hepatitis is a rare but potentially fatal disease. Mortality without supportive management and/or liver transplantation is in excess of 70%. Adult definitions of fulminant hepatic failure, which include the development of hepatic necrosis and encephalopathy within 8 weeks of onset of liver disease do not apply to acute liver failure, in children particularly if secondary to autoimmune or metabolic liver disease

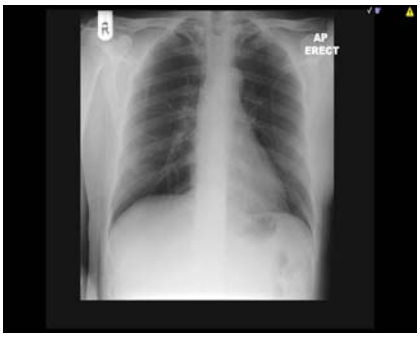
In our case clinical presentation was look like acute abdomen initial abdominal Xray blood gases picture were all misleading us towards the acute pancreatitis. But laboratory findings of markedly deranged LFTs made the diagnosis of fulminant hepatic failure. Among clinical signs jaundice was the only positive clue towards any sort of liver disorder. In our case , serum transaminases and LDH were very high but patients didn't develop any signs of hepatic encephalopathy fortunately and recover remarkably on coservative management.

Summary:

This case emphasizes that Fulminant hepatic failure should be suspected in all the cases with acute abdomen. Avoiding getting caught in diagnostic traps a detailed biochemical screeing and histology to make correct diagnosis.

Among various manifestation of the fulminat hepatic failure this is the most uncommon presentation. Left unrecognised and with out proper therapy , in time the disease rapidly progresses to death.

Chest X-ray



Abdominal X-ray