Unusual Presentation of a Pyogenic Liver Abscess - Case Report

Pages with reference to book, From 269 To 270

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Abstract

A middle aged male presented with severe upper gastrointestinal bleeding. Laparotomy was performed and the source was determined to be a spurting vessel in the cavitv of a Pyogenic Liver Abscess which had perforated into the duodenum (JPMA 32 259, 1982).

Case Report

A fifty year old male presented in the Emergency department with right upper quadrant abdominal pain of a 4 weeks duration and 2 bouts of haematemesis and melena occurring twenty days apart. The pain was not related to meals and there was no history of chronic dyspepsia or ulcerogenic drug intake. He was admitted to another unit and transfused 5 units of blood. The bleeding was controlled. He was readmitted on 20.4.1982 with further episodes of bleeding. Blood transfusions were given. Physical examination disclosed severe anaemia, temperature of 101°F and pulse rate of 120/minute. Systemic examination revealed tender hepatomegaly (5cm) and basal rales on the right side of the chest. The Hb was 6 G% with red cell hypochromia and microcytesis, PCV 22, WBC 15,000/mm3, ESR 122mm at 1st hour (Estergren). Bilirubin and Transaminases were normal but Alkaline Phosphatase was elevated at 1201 U./L. Gastroscopy revealed a normal oesophageal and gastric mucosa. Bleeding was seen from the second of the duodenum, but no ulcer was detected. A conservative management for duodenal ulcer was instituted. This was followed by recurrent bleeding and passage of fresh blood pei rectum. A sigmoidoscopic examination was done which ruled out a rectal lesion. Failure to control the bleeding led to an emergency laparotomy on 4.5.1982.

The operative findings were clear ascitic fluid, normal stomach and dense adhesions between duodenum, liver and gall bladder. The gall bladder was thick walled. On separation of duodenal adhesions a perforation was observed communicating duodenum with the liver abscess in the caudate lobe posteromedially to the gall bladder. The abscess contained blood clot and pus as well as a spurting vessel in its wall which was evidently the source of upper gastrointestinal bleeding.

A cholecystectomy was performed and duodenal perforation was closed in two layers. Haemostasis was achieved by underrunning the spurting vessel. An operative diagnosis of Acalculous empyma of gall bladder associated with a pyogenic liver abscess perforating into duodenum was made.

The gall bladder and edges of the duodenal perforation were sent for histopathological examination. Report revealed chronic inflammation of both these strucures.

Discussion

Pyogenic liver abscess is an uncommon disease and is rarely diagnosed at the time of presentation. Sabbaj et al. (1972) found a delay in the diagnosis averaging 50 days in his study involving 55 patients. In the liver unit at King's Hospital not more than 2-3 cases are seen every year. The incidence may be much higher in Asia as Wong et al. (1953) saw 20 cases in a 3 years period from 1948 to 1951. Pyogenic liver disease may be solitary or multiple. The solitary abscesses are common and are usually situated in the right lobe. All ages are susceptible ranging from 9 months-78 years (mean 46 years). In a series of 28 patients 75% were males, 25% were females (Young, 1976). Incidence is high in diabetics (Holt and Spry, 1966). Most cases are cryptogenic (59.5%) (Oschner et al., 1938). Some are related to

known foci of infection. Portal of entry to the liver is via the portal vien from appendicitis, diverticulitis, infected haemorrhoids, simple or malignant ulcerative diseases of the gastrointestinal tract and actinomycosis of the caecum, via the bi iary tree following cholangi is. Ascaris lumbriccides has been known to invade the bile duct and result in pyogenic liver abscess (Wong et al., 1953) via the hepatic artery following septicaemia e.g. from osteomyeli is, perinephric abscess, lung abscess, bronchiectasis and endocarditis. Organisms may also enter the liver following penetrating trauma and direct extension from adjacent infective processes e.g. cholecystitis, subphrenic abscess and pyelonephritis.

The commonly isolated organisms are E. Coli and anaerobes. Sabbaj et al. (1972) observed that anaerobes were cultured from 45% of their 55 cases of liver abscess and that failure to detect anaerobes may reflect inadequate culture facilit ies. The organisms reported by Sabbaj et al. (1972) are, Aerobes, E. Coli, Streptococci (haemolyticus, Viridans, faecalis), Proteus mirabilis, Pseudomonas aeuro-ginosa and Klebsiella. Anaerobes, Anaerobic Streptococci, Microphilic streptococci, Actinomycosis naeshiadu, Bacteroides fragilis and Bacteroides melaninogenicus, Staphylococci were cultured by Wong et al. (1953).

In one study of 30 patients by Northover et al. (1982) the common clinical manifestations were:

Symptoms	Sings
Malaise	87%
Fever	83%
Anorexia	77%
Hepatomegaly	67%
Subcostal	subcosta
Pain	63%
Tenderness	57%
Rigors	63%
Jaundice	50%
Weight loss	57%
Vomiting	27%

In another study by Young (1976) involving 28 patients nocturnal sweating was prominent in 43% patients.

The abnormal laboratory investigations as shown by Young's study are:

Raised ESR	95%
Leucocytosis	82%
Elevated Alkaline Phosphatase	79%
Reduced Hb	75%

Northover et al. (1982) reported hypoal-buminaemia in 20 patients, (80%) of his 30 cases of pyogenic liver abscess.

A raised level of serum vitamin B12 was seen in many young patients with aver abscesses and was attributed to a release of this vitamin into the circulation following destruction of hepatoevtes (Butler and McCarthy, 1969).

In a series of 1500 patients reported by Cuaron and Gordon (1970) hepatic scanning was reliable in 95% of cases. Scans should be obtained in both AP and lateral views. Other sophisticated diagnostic measures are ultrasound scans, Coeliac arteriography and Splenoportography.

Most cases of pyogenic liver abscess (upto 60%) are diagnosed at autopsy (Pyrtek and Bartus, 1965). Rarity of this condition and a poor index of suspicion on part of the clinician and consequent failure to request the pertinent investigations may be responsible for difficulty in diagnosis.

As far as is known pyogenic liver abscesses presenting as upper gastrointestinal haemorrhage has not been reported.

Earlier diagnosis and a reduction in mortality may be achieved by an awareness of this condition.

Unusual presentation such as upper gastrointestinal bleeding as in our case should be kept in mind.

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