Multiple liver abscesses caused by Yersinia enterocolitica

A case report

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Summary

Yersinia enterocolitica is an important cause of multiple liver abscesses in iron-overloaded or debilitated patients. The condition has a high mortality rate and causes diagnostic and therapeutic problems. The successful management of an iron-overloaded patient presenting with jaundice and chronic renal failure is described.

Yersinia enterocolitica is a Gram-negative rod-shaped organism belonging to the family Enterobacteriaceae. A fascinating feature of this bacterium is that it is one of the most iron-dependent of all microbes. A case of Y. enterocolitica septicaemia in a patient with iron overload and chronic renal disease is reported. The history suggested cholangitis but investigations revealed multiple small liver abscesses.

Case report

A 65-year-old black man was admitted to Baragwanath Hospital, Johannesburg, with a 2-week history of anorexia, weight loss, crampy abdominal pains, vomiting, rigors and night sweats. There was no history of diarrhoea. The urine was dark but the stool was normal and there was no pruritus. The patient was a heavy imbiber of traditional beer. He had a history of mild chronic renal disease of unknown cause, but was not on dialysis.

On examination, the patient was obviously ill. The blood pressure was 95/60 mmHg and the pulse rate 120/min. He was afebrile and jaundiced and his skin was hyperpigmented. Abdominal examination revealed a 10 cm tender hepatomegaly and a 6 cm splenomegaly.

Laboratory test results were: haemoglobin 11 g/dl; white blood cell count 18,9 x 10^9/l with predominant neutrophilia; erythrocyte sedimentation rate 120 mm/1st h (Westergren); serum sodium level 135 mmol/l, potassium 6,8 mmol/l, chloride 109 mmol/l, urea 68,5 mmol/l, creatinine 1.155 mmol/l, total protein 79 g/l (normal 53-84 g/l), albumin 31 g/l (normal 20-45 g/l), total bilirubin 101 µmol/l (normal 0-5 µmol/l), indirect bilirubin 29 µmol/l (normal 0-17 µmol/l), alkaline phosphatase 316 U/l (normal 90-283 U/l), aspartate aminotransferase 143 U/l (normal 6-38 U/l), alanine aminotransferase 69 U/l (normal 6/40 U/l), γ-glutamyltransferase 109 U/l (normal 7-50 U/l), amylase 362 U/l (normal 70-300 U/l) and serum ferritin 2000 ng/ml (normal 12-400 ng/ml). Blood and urine cultures were negative but stool culture revealed a growth of Shigella sonnei sensitive to ampicillin and trimethoprim.

Ultrasoundography of the abdomen confirmed the hepatomegaly and in addition revealed multiple echolucencies (0.25-0.5 cm in diameter) in the liver. Both kidneys were small with a few simple cysts.

Computed tomography (CT) of the abdomen showed generalised increase in density of the liver and spleen on pre-contrast scan suggestive of haemochromatosis (Fig. 1) and multiple low-density areas within the liver, which did not enhance with intravenous contrast material (Fig. 2). The features were suggestive of micro-abscesses of bacterial or fungal origin, cysts or multiple metastases.

Needle biopsy obtained a core of liver tissue with a small amount of thick yellow pus. Histologically there was severe haemosiderosis but no cirrhosis. Culture grew Y. enterocolitica, which was resistant to ampicillin but sensitive to trimethoprim-sulphamethoxazole, tetracycline, chloramphenicol, cefoxitin, cephradine, cefotaxime, piperacillin, tobramycin and amikacin.

Serological investigation for Y. enterocolitica agglutinin yielded a titre of 1:50 for the 0.3 antigen, negative for H antigen initially. This rose to a titre of 1:1000 for 0.3 antigen and 1:25 for H antigen 2 weeks later.

The patient was initially treated with ampicillin and peritoneal dialysis. Therapy was then changed to a combination of trimethoprim 90 mg-sulphamethoxazole 400 mg 2 tablets twice a day for 3 weeks. He recovered and was discharged 2 weeks later.

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Discussion

Y. enterocolitica usually produces a benign infection confined to the intestinal tract. The two major clinical presentations of septicaemia, i.e. the acute septicaemia form mimicking typhoid fever and the subacute localising form characterised by hepatosplenic abscesses, are rare. In South Africa the incidence may be increased because of the occurrence of haemosiderosis in the local black population. Other predisposing factors are: age (greater in the first decade of life and in the elderly), alcoholism, cirrhosis, diabetes mellitus, thalassaemia, aplastic anaemia, leukaemia, sickle-cell anaemia, haemodialysis, peritoneal dialysis and immunosuppressive treatment. Twenty-two per cent of patients have no predisposing illness.

Early diagnosis of pyogenic liver abscess is particularly important because a delay in therapy carries a high mortality rate. Although this patient had multiple liver abscesses, he remained apyrexial. According to some authors this is very unusual. The tender liver and neutrophilia were compatible with pyogenic abscess.

CT demonstrated multiple micro-abscesses scattered throughout the entire liver; such patients frequently have an obvious source of infection, e.g. appendixitis, cholecystitis or diverticulitis, but micro-abscesses are now frequently seen in immunosuppressed patients who develop fungal infection of the liver — usually caused by Candida albicans. The present case would therefore further reinforce the observation that not all cases of multiple liver abscesses are related to surgical conditions. The splenomegaly was probably related to the underlying haemosiderosis but multiple splenic abscesses have been described in this condition.

An important differential diagnosis in the septicaemic form is amoebic liver abscess; however, jaundice and splenomegaly are uncommon in amoebiasis and investigations demonstrate a few large abscesses rather than multiple small lesions. Our patient was initially diagnosed as having cholangitis because of the history of jaundice, rigors and abdominal pain.

Surgeons should be aware that Y. enterocolitica infection may resemble choledochitis and be accompanied by elevated levels of aspartate aminotransferase, lactate dehydrogenase, alkaline phosphatase and bilirubin.

The isolation of S. sonnet from the patient's stool is also interesting. Such cases have been encountered especially in regions where enteritis with a mixed aetiology is common. Y. enterocolitica could be either a co-pathogen or a secondary invader in the setting of established infection. The Shigella organism may cause mucosal damage and stimulate the invasion of Y. enterocolitica, resulting in septicaemia. That enteropathogens other than Y. enterocolitica may act synergistically with the latter organism was shown in experimental studies when mice fed intragastrically with a mixture of Salmonella typhimurium and Y. enterocolitica developed systemic infection more readily and had a significantly higher mortality rate than when fed with one of these enteropathogens at a time.

The therapeutic regimen for systemic Y. enterocolitica infection has been a problem. All pathogenic strains are resistant to ampicillin by virtue of the production of a β-lactamase. Many antibiotics have been tried, including tetracycline, amoxicillin, chloramphenicol and cephalosporin, with variable results. Trimethoprim-sulphamethoxazole has also been used. Despite adequate treatment with any of these agents, the mortality rate is very high, but there may be some cases of spontaneous cure of Y. enterocolitica septicaemia. Our patient was severely ill and was given the appropriate treatment only 2 weeks after admission but responded well with complete resolution of the lesions on CT 4 weeks after the start of therapy.

REFERENCES