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Sepsis Induced Hyperbilirubinemia - A Case Report

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ABSTRACT

The case of a young lady with transient jaundice secondary to abdominal sepsis is reported and the relevant literature briefly reviewed.

KEYWORDS

Peritonitis; Laparotomy; Colon; Gangrene; Cholestasis; Hepatocellular dysfunction

INTRODUCTION

Sepsis is life-threatening organ dysfunction caused by dysregulated host response to infection [1]. The liver has an important role in fighting sepsis, but the liver itself can be affected adversely in sepsis leading to jaundice, either from bacterial products or due to the host response [2,3], with progression to acute liver failure and high mortality [4,5]. There is no clear-cut definition of sepsis induced hyperbilirubinemia, but it is usually considered when a patient's total serum bilirubin is more than 2 mg/dl along with a more than two-fold rise in either alkaline phosphatase or serum aminotransferase [5,6]. It has a wide variation in incidence, being reported in nearly 40 % of sepsis patients [7], and is now well-established as an important prognostic factor [8].

CASE REPORT

A 30-years-old lady presented to our emergency with abdominal pain and vomiting for a duration of one week. On examination, she was afebrile, with normal vitals. Her abdomen was soft, with guarding over a vague tender mass palpable in the right lumbar region. A computerized tomogram scan of the abdomen had already been done, and this showed thrombosis of the superior mesenteric vein as well as ischemic wall thickening of the terminal ileum and right colon with multifocal perforations and a large air containing collection in the right paracolic region. Her routine laboratory investigations were normal, except for deranged liver functions, showing high bilirubin (4.2 mg/dl; conjugated 3.4 mg/dl), mildly elevated alkaline phosphatase (145) and transaminases.

She was taken up for emergency laparotomy. During surgery, it was noted that the posterior wall of the ascending colon and hepatic flexure was gangrenous and sloughed off, with a 2 cm by 2 cm perforation in anterior wall of the colon at the hepatic flexure. The mesocolon had thrombosed vessels, with necrotic lymph nodes. A right hemicolectomy was done, and the ends of the bowel brought out as end ileostomy and mucus fistula.

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Post-operatively the patient remained was well, but her bilirubin continued to rise, going up to 14.4 mg/dl (conjugated 10.8). She was investigated to rule out underlying liver disease, but her viral markers and liver ultrasound were normal. Sequential liver functions showed a falling trend of bilirubin starting on the 6th post-operative day (down to 3.3 mg/dl). She was discharged on the 8th post-operative day with normalized liver function tests.

DISCUSSION

Jaundice in sepsis is usually seen in sepsis arising from the biliary tract, abdominal cavity, urinary system or bloodstream, but has also been reported in pneumonia, meningitis and bacterial endocarditis [3,7]. Infections by aerobic and anaerobic gram-negative, gram-positive bacteria as well as fungi have all been reported to cause jaundice; commonly, gram-negative bacteria are considered to be the predominant cause, but recently, gram-positive bacteria (enterococcus species) were reported to be predominant [7].

The mechanism of occurrence of jaundice in sepsis is complicated, and involves a complex interplay of haematological and cellular events, metabolic changes and immune response that act under the influence of exogenous mediators (antigens, endotoxins, micro-organisms, systemic cytokines, acute phase proteins) to cause liver dysfunction [2,5,9]. A full discussion on these is out of the scope of this short report, but all these pathways translate into a few common mechanisms to cause jaundice - (a) hypoxic hepatitis, and (b) cholestasis. Hypoxic hepatitis occurs due to reduced oxygen delivery or utilization by the liver, and should be suspected when there is an abrupt elevation of serum aminotransferase levels (more than 20 times normal) in the absence of other causes of acute liver injury [2,5]. These usually peak by 24 hours and start falling by the 3rd day, usually to normal by the end of the 2nd week [5]. However, jaundice in sepsis is usually due to cholestasis [10]; this occurs due to the effects of various inflammatory products released in response to infection leading to derangement in the transport of bile acids and organic anions, degradation of membrane proteins, and disturbance of cell membrane fluidity [2-4,9,10]. Hemolysis may contribute to jaundice in sepsis, but it is unlikely to be the principal cause [10].

When jaundice develops in a patient with an established diagnosis of infection, the possibility of sepsis induced hyperbilirubinemia is obvious. The diagnosis is to be considered to when total serum bilirubin is greater than 2 mg/dl with a more than two-fold rise in either alkaline phosphatase or serum aminotransferase [5,6]. Early onset of jaundice (within 72 hours of admission) and total bilirubin levels more than 5 mg/dl are associated with higher mortality [8,11,12]. If the source of sepsis is not obvious, it is important to aggressively evaluate these patients with a full work up including chest x-ray, urine, blood and catheter tips/drain cultures; abdominal ultrasonography should be done and computed tomography may be indicated to locate any covert sources of sepsis [10].

Adequate and timely treatment of the underlying condition usually leads to complete resolution of hepatic dysfunction, with poorer outcomes if they are delayed [10,13]. Once the source of infection is identified, it should be adequately dealt with. There is no specific therapy for jaundice of sepsis, and early, goal directed therapy is the norm [5]. In addition to source control, this includes early and appropriate antibiotic therapy, fluid resuscitation, and vasopressor support [5,13]. Early enteral feeding and ursodeoxycholic acid may be useful in resolving cholestasis; although corticosteroids have immunomodulatory effects, their use in sepsis induced jaundice is not recommended at present [5].

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