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CASE REPORT

A case of acute infectious mononucleosis presenting with very high ferritin

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Abstract

Hepatitis is an important but uncommon manifestation of acute Epstein Barr infection. Infectious mononucleosis is usually a disease of young adults. We report a case of infectious mononucleosis in a 72-year old jaundiced gentleman with ferritin level of 2438 that normalised on clinical improvement.

Key words: Epstein Barr virus; Infectious mononucleosis; Ferritin; Jaundice; Liver function tests

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INTRODUCTION

Hepatitis is an important but uncommon manifestation of acute Epstein Barr infection. Infectious mononucleosis is usually a disease of young adults. We report a case of infectious mononucleosis in a 72-year old jaundiced gentleman with ferritin level of 2438 that normalised on clinical improvement.

CASE REPORT

A 72-year old retired farmer was admitted with a three-week history of feeling generally unwell. He gave a history of loss of appetite with stable weight. He denied any history of fever. Having a long history of prostatism, he was found to be in urinary retention at the same time for which he was catheterised. He was not started on any new medications and denied taking any over the counter medications. He said his urine was dark and sometimes had dysuria. He denied any gastro-intestinal symptoms.

His past history included osteoarthritis, previous myocardial infarction in 1993, gastro-oesophageal reflux disease and laparoscopic herniorrhaphy,

His medications were 75 mg aspirin, 10 mg atorvastatin, 300 mg quinine sulphate, 20 mg omeprazole and 5 mg amlodipine once daily. He said he was allergic to codeine phosphate. He was living on his own and his wife had recently passed away. His alcohol intake was negligible. There was no family history of any clinical significance.

On clinical examination he was noticed to be jaundiced, afebrile, haemodynamically stable. There were no stigmata of chronic liver disease. His abdomen was soft with mild tenderness elicited in the right hypochondrium. No organomegaly or masses were noted. Examinations of cardiovascular and respiratory systems were normal.

His blood tests were as follows: Hb = 14.7 g/dL (13.5-16.9), WBC = 6.1 (4.5-13.0), platelets = 161 (150-400), MCV = 92 fL (84-99.0), neutrophils = 1.65 (2.0-7.5), lymphocytes = 3.97 (1.5-4.0). A blood film showed a moderate number of atypical lymphocytes.

Serum ferritin was high at 2438 μg/L (25-400). Haemochromatosis screen was negative. PT, APTT and fibrinogen were normal. Sodium was 135 mmol/L (135-145), potassium 4.2 mmol/L (3.5-5.3), urea 6.1 mmol/L (3-7) and creatinine 102 μmol/L (53-115) (Table 1).

Bilirubin was 50 μmol/L (0-20), alanine aminotransferase (ALT) 254 U/L (0-37), alkaline phosphatase (ALP) 677 U/L (39-128), gamma glutamic transpeptidase(GT) 817, albumin 34 g/dL (35-52), calcium 2.18 mmol/L (2.1-2.6), C reactive protein (CRP) 21 mg/L (0-8), alfa- fetoprotein 3.5 (0-5.8), C3 and C4 (complements) were normal; IgM was 3.73 g/L (0.5-2).

Blood and urine cultures were negative. Hepatitis A, B, C and CMV serology were negative. Anti Epstein Barr virus (EBV) capsid antigens IgG and IgM were detected, Anti EBV nuclear antigen IgG was also detected.

Anti nuclear antibody (ANA) was negative. Anti neutrophil cytoplasmic antibody (ANCA), antimitochondrial, smooth muscle, liver kidney microsomal antibodies were all negative.

Chest and abdominal X-rays showed no radiological abnormalities. He had an abdominal CT showing moderate

Table 1 Abnormal blood tests in patient

1	Ferritin 2438 (25-400)
2	Bilirubin 50 (0-30)
3	ALT 254 (0-30)
4	ALP 677
5	Gamma GT 817
6	CRP 21 (0-8)
7	IgM 3.73 (0.5-2)
8	Anti EBV nuclear IgG detected
9	Anti EBV capsid antigen IgM and IgG detected
10	Atypical lymphocytes

splenomegaly and liver changes that were suggested as possibly secondary to hepatitis.

He was managed conservatively, his LFTs improved. He remained very well and was discharged after 2 wk of hospitalisation.

He was seen in Outpatient Follow-up Clinic after 4 mo, he was well and his weight was stable. His ferritin and LFT at that time became normal.

DISCUSSION

Infectious mononucleosis (IM) was first described in 1920. It was not until 1968 that EBV infection was described as a causative agent of IM. Incubation period is 25-50 d. It is usually a self-limiting disease.

There is a mortality rate of 0.1% associated with IM. Ninety percent of patients have asymptomatic deranged liver function tests, but jaundice is rare. EBV hepatitis is an important cause of viral hepatitis.

Fulminant hepatitis is rare but has been reported[1]. Severe hepatitis is rare, although it has been reported. Hepatomegaly is frequently present. Jaundice is rare. Occasionally, jaundice may be due to autoimmune haemolysis. Cholestasis can be noticed[2]. Splenomegaly is common and 50%-75% of patients develop it. Dommerby *et al*[3] reported that all patients have ultrasonic splenomegaly but that is palpable in only a few. Splenic rupture should be ruled out in patients who present with severe abdomen pain. Rupture can be precipitated by sports activity or can occur spontaneously and 0.1%- 0.5% of cases can result in rupture[4]. Spleen is most vulnerable to rupture in the 2nd and 3rd week of infection. Preparation with amoxicillin can result in rash frequently. Fever can be noted in 90 % of cases, usually low grade. Maculo- popular rash can be noticed in 5%-10% of cases, but is more common in children.

Malaise and fatigue may persist for weeks or months after IM. Persistent EBV infection is not a cause of chronic fatigue syndrome (CFS). High titres of antibodies to EBV may be noticed in patients with CFS that are identical to healthy EBV- sero positive adults.

Table 2 Gastrointestinal manifestations of EBV infection

1	Asymptomatic deranged LFTs
2	Viral hepatitis, usually self limiting
3	Fulminant hepatitis
4	Cholestasis
5	Auto-immune haemolysis causing jaundice
6	Splenomegaly
7	Splenic rupture (rare)

Duncan syndrome, a rare X linked recessive disorder, was reported by Purtilo *et al* in 1975 which is characterised by a defect in the immune response against EBV resulting failure to prevent EBV replication and hence leading to fatal complications[5]. Patients with combined immunodeficiency, Wiskott- Aldrich syndrome and Ataxia telangiectasia develop severe IM[6]. Likewise, patients with acquired immunodeficiency states like patients with AIDS or those receiving immunosuppressive agents develop severe IM and malignant lymphoma[7].

Treatment for IM is supportive with analgesic as required and rest. Due to the risk of splenic rupture physical activity in excess is to be avoided. There is no place for oral glucocorticoids in uncomplicated IM. In patients with severe tonsillar enlargement that might predispose to airway obstruction, severe thrombocytopenia or haemolytic anaemia, Prednisolone 40 to 60 mg once daily for 2-3 d with tapering over 3 wk is adviced.

In conclusion, acute EBV hepatitis should be ruled out in acutely jaundiced patients with high ferritin at any age group (Table 2).

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