BRITISH JOURNAL OF HOSPITAL MEDICINE

CASE REPORT

Acute Epstein-Barr Virus Hepatitis With Cholestatic Jaundice and Hyperferritinaemia: A Case Report

Paul Njoku[1,*], Dorette Ngemoh[1], Shahriar Amin[2], Amit K J Mandal[1], Constantinos G Missouris[1,3]

[1]Department of Medicine, Wexham Park Hospital, Frimley Health NHS Foundation Trust, Berkshire, UK
[2]Department of Medicine, University Hospitals Coventry & Warwickshire NHS Trust, Coventry, UK
[3]Department of Clinical Cardiology, University of Nicosia Medical School, Nicosia, Cyprus
*Correspondence: paul.njoku1@hotmail.co.uk (Paul Njoku)

Abstract

Epstein-Barr virus (EBV) is a common human pathogen often associated with infectious mononucleosis (IM), a typically self-limiting illness. EBV can affect the liver, with manifestations ranging from asymptomatic hepatitis to acute liver failure, particularly in immunocompromised individuals. Cholestatic jaundice and marked hyperferritinaemia are rare in EBV hepatitis, with ferritin levels correlating with disease severity. We report a case of EBV hepatitis in a 50-year-old immunocompetent man presenting with cholestatic jaundice, pharyngitis, cervical lymphadenopathy, and hyperferritinaemia (ferritin 5384 µg/L). Liver biochemistry showed elevated transaminases (alanine transaminase 190 U/L, aspartate transaminase 310 U/L), hyperbilirubinaemia (92 µmol/L), and cholestasis (alkaline phosphatase 902 U/L, gamma-glutamyl transferase 941 U/L). Atypical lymphocytes were seen on peripheral blood smear. Acute EBV infection was confirmed by the presence of EBV Immunoglobulin M (IgM) antibodies, and the patient was successfully managed in an ambulatory care setting with supportive treatment.

Key words: Epstein-Barr virus; hepatitis; ferritin; cholestatic jaundice; infectious mononucleosis; case report

Submitted: 16 November 2024 Revised: 19 December 2024 Accepted: 30 December 2024

Introduction

Epstein-Barr virus (EBV), a member of the Herpesviridae family, is a ubiquitous pathogen with over 90% of the global population exposed to the virus by adulthood (Rutkowska and Pokorska-Śpiewak, 2023). EBV primarily spreads through saliva, earning its reputation as the "kissing disease". However, it can also be transmitted through organ transplantation, blood transfusion, and close contact with infected individuals. Although EBV infection is often asymptomatic or manifests as infectious mononucleosis in adolescents and young adults, it can occasionally lead to severe complications, including hepatitis (Salva et al, 2013).

EBV-associated hepatitis is an uncommon manifestation and is typically mild and self-limiting. In rare cases, it can present with significant hyperferritinaemia and obstructive jaundice, mimicking more severe hepatobiliary diseases (Crum, 2006; Yang et al, 2014). The pathogenesis of EBV hepatitis involves the virus's ability to infect B lymphocytes via the cluster of differentiation 21 (CD21) receptor and replicate within these cells, triggering a cascade of immune responses (Hatton

How to cite this article:
Njoku P, Ngemoh D, Amin S, Mandal AKJ, Missouris CG. Acute Epstein-Barr Virus Hepatitis With Cholestatic Jaundice and Hyperferritinaemia: A Case Report. Br J Hosp Med. 2025. https://doi.org/10.12968/hmed.2024.0919

et al, 2014). Infected B cells can disseminate the virus to the liver, where it induces hepatocyte injury through cytotoxic T-cell activity and cytokine-mediated inflammation. This case report describes a rare instance of self-limiting EBV hepatitis presenting with profound hyperferritinaemia and obstructive jaundice, highlighting the need for early recognition and management to avoid unnecessary interventions.

Case Report

Initial Presentation

A 50-year-old Caucasian male presented at eleven am in the morning to our same day emergency care (SDEC) unit with a three-week history of yellow discolouration of the eyes, headache, sore throat and lassitude. He received influenza vaccination one week prior to the onset of his symptoms. He denied fever, abdominal pain, diarrhoea or vomiting. There was no recent travel. The patient's past medical history was notable for embolic myocardial infarction with full recovery. Drug history included aspirin, atorvastatin and ramipril with no recent additions, including over the counter medications. He took alcohol rarely, was a non-smoker and denied recreational drug use. Family history was significant for haemochromatosis on the paternal side.

On examination, the patient was afebrile and observations were normal bar a resting tachycardia. There were scleral icterus, cervical lymphadenopathy and erythematous non-exudative tonsils. There was no skin rash. Abdomen was benign with a palpable splenic edge and mild hepatomegaly.

The laboratory investigations and normal ranges are depicted in Table 1. Liver function tests (LFTs) revealed hyperbilirubinaemia at 92 µmol/L, alanine transaminase (ALT) 190 U/L, aspartate transaminase (AST) 310 U/L, alkaline phosphatase (ALP) 902 U/L and gamma-glutamyl transferase (GGT) 941 U/L. Ferritin was 5384 µg/L. Transferrin saturation was 16%. Caeruloplasmin was 0.46 g/L. C-reactive protein was 34 mg/L. Urea and electrolytes showed a mild hyponatraemia with normal kidney function (Table 1). The blood film showed lymphocytosis with reactive and atypical lymphocytes. The CARE checklist has been attached as **Supplementary material 1** associated with this article.

Ultrasound of the liver was arranged on the same day which showed mildly increased echogenicity suggestive of fatty liver disease, normal common bile duct and intrahepatic bile duct diameter and no evidence of gallstones (Fig. 1A,B). The spleen was mildly enlarged at 14.4 cm in length (Fig. 1C). We arranged an urgent outpatient magnetic resonance cholangiopancreatography (MRCP) with follow-up in our ambulatory care unit one week later. Atorvastatin was suspended pending resolution of ALT levels. Viral serologies for possible causes of hepatitis and autoimmune hepatitis screen testing were sent for analysis. This included Epstein-Barr virus (EBV), Cytomegalovirus (CMV), Human Immunodeficiency virus (HIV) and viral hepatitis A and B serologies.

Table 1. Patient's laboratory values at each presentation.

Laboratory value	Day 0	Reference range
White blood cell ($\times 10^3/\mu L$)	16.0	4.0–11.0
Haemoglobin (g/L)	132	130–180
Platelets ($\times 10^9/L$)	267	150–400
Sodium (mmol/L)	131	133–146
Potassium (mmol/L)	3.7	3.5–5.3
Urea (mmol/L)	2.7	2.5–7.8
Creatinine (µmol/L)	58	64–104
Estimated glomerular filtration rate (eGFR – mL/min)	>90	90–120
Aspartate transaminase (U/L)	310	0–34
Alanine transaminase (U/L)	190	0–55
Alkaline phosphatase (U/L)	902	30–130
Gamma-glutamyl transferase (U/L)	941	0–54
Total bilirubin (µmol/L)	92	0–20
Ferritin (µg/L)	5384	30–300
Iron (µmol/L)	8	12–31
Transferrin saturation (%)	16	20–50
Caeruloplasmin (g/L)	0.46	0.2–0.6
C-reactive protein (mg/L)	34	0–5
Blood film	Lymphocytosis. Reactive and atypical lymphocytes	

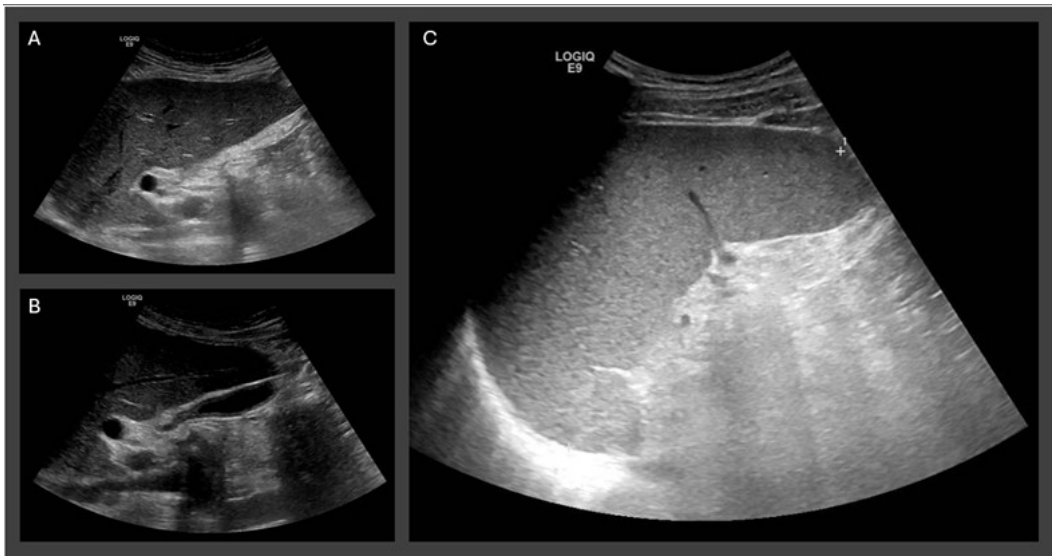

Fig. 1. Abdominal ultrasound assessment of the liver and spleen. Abdominal ultrasound scan showing the liver parenchyma. Image A shows mildly increased echogenicity suggestive of fatty liver disease. Image B shows normal diameter of the biliary tree with no evidence of gallstones. Image C is an ultrasound image of an enlarged spleen.

1st Follow-up Appointment (Day 7 Since Initial Presentation)

At follow-up, ALT levels had reduced to 143 U/L, ALP had risen to 932 U/L, GGT reduced to 901 U/L and bilirubin had halved to 46 µmol/L. C-reactive protein

and Ferritin levels also improved to 10 mg/L and 3066 µg/L respectively. Viral serology was positive for Immunoglobulin M (IgM) EBV, confirming acute EBV infection. Immunoglobulin G (IgG) EBV was non-reactive. EBV viral load was detectable at 2954 IU/mL. Remaining serologies were non-reactive for CMV, HIV 1 & 2, Hepatitis serologies were consistent with previous Hepatitis A and B vaccination (Table 2). Autoimmune haemolysis screen was performed by direct antiglobulin test which was negative. Antinuclear, anti-smooth muscle, anti-liver-kidney microsomal and antimitochondrial antibody were not detected. Alpha-1-antitrypsin levels were within normal range. A preliminary diagnosis of EBV hepatitis was confirmed, pending review of the patient's MRCP findings. We advised rest, oral hydration, time off work and to avoid alcohol consumption and intense strenuous activity until full recovery.

2nd Follow-up Appointment (Day 14 Since Initial Presentation)

The MRCP was performed thirteen days after the initial presentation. This demonstrated patent, non-dilated intrahepatic and extrahepatic bile ducts with mild hepatosplenomegaly and no other pathology (Fig. 2). He was reviewed in our ambulatory clinic for repeat blood tests showing further improvement in his liver function tests (Table 3) and significant improvement of his symptoms. He was referred to the virtual clinic for remote monitoring of his liver function biochemistry.

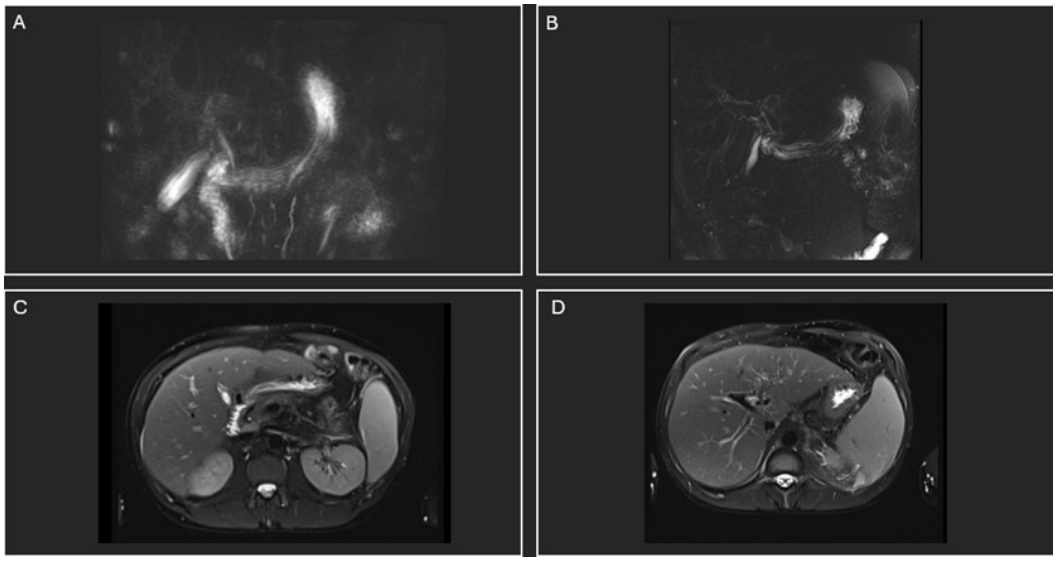

Fig. 2. Magnetic resonance cholangiopancreatography (MRCP) images. (A) and (B) demonstrating a contracted gallbladder and patent intra and extrahepatic bile ducts. Axial plane views of the patient's MRCP (C) and (D) demonstrating hepatosplenomegaly and a patent common bile duct with no evidence of gallstones.

3rd Follow-up Appointment (Day 21 Since Initial Presentation)

The following week the patient was reviewed in our ambulatory virtual clinic. He remained clinically well and reported complete resolution of symptoms. Normalisation of liver function tests (ALT and Bilirubin) were seen with further im-

Table 2. Patient's laboratory values at 1st follow-up appointment 7 days after initial presentation.

Laboratory value	Day 7	Reference range
White blood cell ($\times 10^3/\mu L$)	6.9	4.0–11.0
Haemoglobin (g/L)	133	130–180
Platelets ($\times 10^9/L$)	282	150–400
Sodium (mmol/L)	134	133–146
Potassium (mmol/L)	4.1	3.5–5.3
Urea (mmol/L)	4.2	2.5–7.8
Creatinine (µmol/L)	63	64–104
Estimated glomerular filtration rate (eGFR – mL/min)	>90	90–120
Aspartate transaminase (U/L)	185	0–34
Alanine transaminase (U/L)	143	0–55
Alkaline phosphatase (U/L)	932	30–130
Gamma-glutamyl transferase (U/L)	901	0–54
Total bilirubin (µmol/L)	46	0–20
Ferritin (µg/L)	3066	30–300
C-reactive protein (mg/L)	10	0–5
EBV IgM	Positive	
EBV IgG	Not detected	
EBV viral load (IU/mL)	2954	
CMV IgM	Not detected	
CMV IgG	Not detected	
HIV 1&2 Antibodies	Not detected	
p24 antigen	Not detected	
Hepatitis B surface antigen antibody	Detected	
Hepatitis B surface antigen	Not detected	
Hepatitis B core antigen antibody	Not detected	
Hepatitis B core antigen	Not detected	
Hepatitis A IgG antibody	Detected	
Hepatitis A IgM antibody	Not detected	
Anti-nuclear antibody	Not detected	
Anti-smooth muscle antibody	Not detected	
Anti-liver-kidney-microsomal antibody	Not detected	
Alpha-1-antitrypsin (g/L)	2.1	1.1–2.1
Direct antiglobulin test (DAT)	Negative	
Blood film	Moderate neutropenia, toxic granulation seen, atypical lymphocytes seen, anisochromia	

Abbreviations: EBV, Epstein-Barr virus; CMV, Cytomegalovirus; HIV, Human Immunodeficiency virus; IgM, Immunoglobulin M; IgG, Immunoglobulin G; DAT, direct antiglobulin test.

provement in ferritin, AST, ALP and GGT levels (Table 4). We advised that the patient could resume his statin therapy. Given the reassuring prognosis of EBV hep-

Table 3. Patient's laboratory values at 2nd follow-up appointment 14 days after initial presentation.

Laboratory value	Day 14	Reference range
White blood cell ($\times 10^3/\mu L$)	5.4	4.0–11.0
Haemoglobin (g/L)	130	130–180
Platelets ($\times 10^9/L$)	421	150–400
Sodium (mmol/L)	136	133–146
Potassium (mmol/L)	4.5	3.5–5.3
Urea (mmol/L)	4.7	2.5–7.8
Creatinine (µmol/L)	68	64–104
Estimated glomerular filtration rate (eGFR – mL/min)	>90	90–120
Aspartate transaminase (U/L)	112	0–34
Alanine transaminase (U/L)	89	0–55
Alkaline phosphatase (U/L)	462	30–130
Gamma-glutamyl transferase (U/L)	427	0–54
Total bilirubin (µmol/L)	25	0–20
Ferritin (µg/L)	2023	30–300
C-reactive protein (mg/L)	1.1	0–5

atitis no further follow-up was arranged. Fig. 3 summarises the timeline of events from initial presentation to discharge.

Table 4. Patient's laboratory values at 3rd follow-up appointment 21 days after initial presentation.

Laboratory value	Day 21	Reference range
White blood cell ($\times 10^3/\mu L$)	5.6	4.0–11.0
Haemoglobin (g/L)	130	130–180
Platelets ($\times 10^9/L$)	401	150–400
Sodium (mmol/L)	132	133–146
Potassium (mmol/L)	4.4	3.5–5.3
Urea (mmol/L)	4.4	2.5–7.8
Creatinine (µmol/L)	75	64–104
Estimated glomerular filtration rate (eGFR – mL/min)	>90	90–120
Aspartate transaminase (U/L)	41	0–34
Alanine transaminase (U/L)	52	0–55
Alkaline phosphatase (U/L)	147	30–130
Gamma-glutamyl transferase (U/L)	99	0–54
Total bilirubin (µmol/L)	19	0–20
Ferritin (µg/L)	959	30–300
Iron (µmol/L)	13	12–31
Transferrin saturation (%)	24	20–50
C-reactive protein (mg/L)	<1.0	0–5

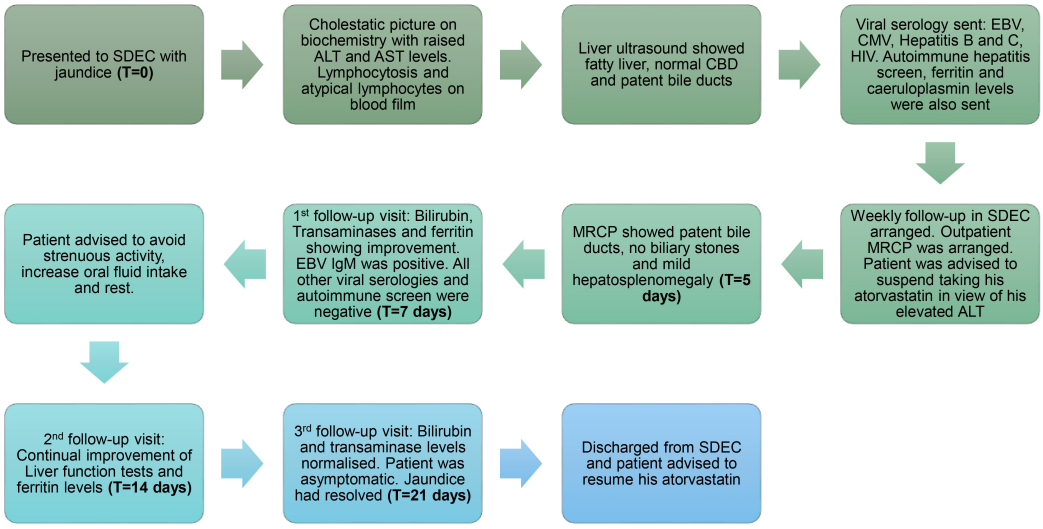

Fig. 3. Timeline from patient's initial presentation to Ambulatory Emergency Care Unit (AECU) to discharge. T, time in days since initial presentation; ALT, alanine transaminase; AST, aspartate transaminase; CBD, common bile duct; SDEC, Same Day Emergency Care.

Discussion

Epstein-Barr virus (EBV) is typically a mild, self-limiting illness and the commonest cause of infectious mononucleosis (IM) (Rutkowska and Pokorska-Śpiewak, 2023; Salva et al, 2013). It usually presents in young adults with the classic triad of pharyngitis, lymphadenopathy and fever (Salva et al, 2013). Other less common manifestations include hepatosplenomegaly, thrombocytopenia and hepatitis (Yang et al, 2014).

Our patient had an atypical presentation of primary EBV infection consisting of cholestatic jaundice and markedly high ferritin levels. Subclinical hepatic involvement is common in primary EBV infection occurring in 80–90% of cases (Crum, 2006). EBV typically causes mild elevations in transaminases, rarely presenting with cholestatic jaundice, symptomatic hepatitis, or marked hyperferritinaemia (Thoufeeq et al, 2007; Vine et al, 2012). Ferritin, a marker of iron storage and an acute-phase reactant, correlates with disease severity and reflects the severity of inflammation and hepatocellular injury in EBV hepatitis (Thoufeeq et al, 2007; van de Veerdonk et al, 2012). Hepatocyte injury is primarily immune-mediated, involving cytokine-driven damage and cytotoxic cluster of differentiation 8 (CD8+) T-cell infiltration (van de Veerdonk et al, 2012). Notably, this patient experienced a self-limiting illness despite significantly elevated ferritin levels. These findings highlight the importance of interpreting ferritin levels within the clinical context.

Alkaline phosphatase (ALP) and gamma-glutamyl transferase (GGT) levels are usually normal or mildly elevated. Significantly elevated levels as in this case with hyperbilirubinaemia, typically point to a cholestatic pathology warranting imaging of the biliary ducts. Cholestasis in EBV hepatitis in the absence of obstructive biliary pathology is uncommon and is postulated to be caused by periportal inflammation and immune-mediated biliary injury although the exact mechanism remains unclear (Uluğ et al, 2010). Ultrasound and Magnetic Resonance Imaging

(MRI) are useful in this setting to eliminate biliary obstruction as a cause. Given our patient's prodromal upper respiratory tract symptoms, a viral cause was high on the list of differentials. Other important differentials we considered included autoimmune hepatitis, haemophagocytic lymphohistiocytosis (HLH) and hereditary haemochromatosis. Autoimmune hepatitis (AIH) has a chronic progressive course which persists without appropriate treatment. AIH was highly unlikely due to the undetectable antinuclear, anti-smooth muscle and liver-kidney-microsomal antibodies and prompt resolution of the illness in this case within weeks. The disproportionately elevated ALP compared to ALT and AST also made AIH very unlikely. HLH is a potentially fatal hyperinflammatory syndrome sometimes triggered by EBV infections leading to exaggerated immune activation, systemic inflammation and multi-organ dysfunction. Whilst the patient met some of the diagnostic criteria for HLH such as increased ferritin levels, splenomegaly and hepatitis, the patient did not fulfil the necessary parameters for diagnosis (Henter et al, 2007). Given the patient's family history of haemochromatosis, this was an important differential to consider which was excluded by the patient's normal transferrin saturation levels.

Ultrasound and MRCP excluded post-hepatic cause for jaundice. Marked lymphocytosis and atypical lymphocytes seen on the blood film supported primary EBV infection as the most likely diagnosis (Salva et al, 2013). On presentation, an acute liver screen panel was performed and the diagnosis was confirmed serologically after positive IgM EBV and high EBV viral load titres.

The non-specific and diverse presentations of EBV infection and the rarity of clinically evident EBV hepatitis (Kang et al, 2009) present a diagnostic challenge. A prompt diagnosis is important to ensure patients receive appropriate management and avoid harmful sequelae such as fulminant hepatic failure, autoimmune hepatitis and chronic EBV infection (Cabibi, 2008; Sánchez et al, 2008). Treatment is largely supportive and rarely warrants hospitalisation. Corticosteroid and antiviral therapy are not routinely given in immunocompetent patients as studies have shown it to be largely ineffective (Gershburg and Pagano, 2005; Mellinger et al, 2014). To date, there is a dearth of randomised controlled trials demonstrating a mortality or significant morbidity benefit of corticosteroid or antiviral therapy for EBV infections. Currently there exist no evidence-backed guidelines recommending the use of antivirals or corticosteroids routinely, however a number of case studies suggest there may be a role for antivirals in severe cases or in high-risk or immunocompromised patients (Adams et al, 2006; Pisapia et al, 2013). Pharmacological therapy in EBV hepatitis remains controversial and there is insufficient evidence to support its routine use (Mellinger et al, 2014).

This patient presented with mild symptoms of a headache, sore throat and lethargy. Despite the markedly raised ferritin levels suggestive of severe infection, he remained clinically stable throughout follow-up in the ambulatory care setting and was managed conservatively. Resolution of clinically evident jaundice was seen by week 3. The patient was advised that should his transaminase or ferritin levels continue to rise, or should he become systemically unwell, we would consider in-patient management.

Serial measurements of transaminases, bilirubin, and ferritin levels were performed weekly in our virtual clinic to ensure resolution of liver function tests, which normalized by the third week without treatment. The prognosis of EBV hepatitis is generally favourable with spontaneous recovery seen in most cases without long-term sequelae. However, patient outcomes vary depending on the severity of hepatic involvement. Complications such as chronic EBV hepatitis, fulminant hepatitis and HLH are rare in immunocompetent patients and long-term surveillance is not advised. High-risk individuals such as the elderly or immunocompromised may have worse outcomes and follow-up with liver function testing and imaging is advised to rule out chronic sequelae. This case describes a 50-year-old immunocompetent male with minimal comorbidities, which conferred a favourable prognosis. Further follow-up was deemed unnecessary after the resolution of his liver enzymes.

Conclusion

Epstein-Barr virus (EBV) hepatitis is typically asymptomatic, with severe cholestatic jaundice and hyperferritinaemia being rare presentations. Potential complications include chronic EBV infection, hepatocellular carcinoma, and fulminant hepatic failure. Prompt diagnosis can prevent unnecessary invasive procedures, such as liver biopsy, and can guide appropriate surveillance and management. Serum ferritin serves as a useful biomarker for disease severity, aiding in decisions regarding inpatient versus outpatient care when interpreted within the clinical context.

Learning Points

- Cholestatic jaundice is an uncommon presentation of EBV hepatitis.
- Hyperferritinaemia in primary EBV infection is rare and correlates with severe infection in the appropriate clinical context. Systemically well patients with hyperferritinaemia may be managed supportively in the outpatient setting.
- EBV hepatitis is usually self-limiting and does not typically require pharmacological treatment.
- There is limited evidence to suggest corticosteroids or antivirals are beneficial in EBV hepatitis in immunocompetent patients.

Availability of Data and Materials

All data generated or analyzed during this study are available from the corresponding author upon reasonable request.

Author Contributions

PN, DN and SA were involved in conceptualisation of this case report. PN, DN and SA drafted the manuscript. PN, AKJM and CGM were involved in data acquisition and analysis. All authors read and approved the final manuscript. All

authors have contributed to important editorial changes in the manuscript. As per the ICMJE guidelines, all authors have participated sufficiently in the work and agreed to be accountable for all aspects of the work.

Ethics Approval and Consent to Participate

Written consent was obtained from the patient. This manuscript complies with the principles of the Declaration of Helsinki. As this is a case report, specific ethical approval was not required. However, all identifying details have been anonymised to ensure patient confidentiality.

Acknowledgement

Not applicable.

Funding

This research received no external funding.

Conflict of Interest

The authors declare no conflict of interest.

Supplementary Material

Supplementary material associated with this article can be found, in the online version, at https://www.magonlinelibrary.com/doi/suppl/10.12968/hmed.2024.0919.

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