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Case Report

Primary Epstein-Barr Virus Infection Causing Severe Acute Hepatocellular Liver Injury With Evolving Cholestatic Features in a Young Adult: A Case Report and Contextual Review of Published Adult Cases

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ABSTRACT

Primary Epstein-Barr virus (EBV) infection usually causes mild aminotransferase elevation. Severe jaundiced hepatitis without encephalopathy is uncommon in adults.
We describe a 20-year-old woman with primary EBV-associated acute liver injury and summarize a March 2026 PubMed-only English-language contextual review of selected adult case reports/case series. Severe acute liver injury was defined descriptively as acute hepatic injury with INR ≥ 1.5 without encephalopathy. Injury pattern was classified with the R-ratio at presentation and peak jaundice.
The patient presented with headache, nausea, and vomiting and had AST 1,942 U/L, ALT 2,032 U/L, total/direct bilirubin 4.0/1.7 mg/dL, alkaline phosphatase 140 U/L, gamma-glutamyltransferase 73 U/L, and INR 1.1, with atypical lymphocytosis and hepatosplenomegaly without biliary obstruction. Heterophile testing, EBV serology, and plasma EBV DNA supported primary infection; alternative infectious, autoimmune, metabolic, and selected toxic causes were not identified. The R-ratio was 54.7 at presentation and 27.2 at peak jaundice, indicating a hepatocellular pattern at both time points despite evolving cholestatic features. INR peaked at 1.6, but no encephalopathy developed. With supportive care alone, jaundice resolved clinically by 1 month and liver tests and INR normalized by 2 months.
Primary EBV infection should be considered in young adults with marked acute liver injury and jaundice even without classic mononucleosis features. This case illustrates severe hepatocellular liver injury with evolving cholestatic features, transient coagulopathy without acute liver failure, and biochemical normalization by 2 months.

1. Introduction

Primary EBV infection is nearly ubiquitous and is usually acquired by adulthood. In adolescents and young adults, it commonly manifests as infectious mononucleosis, whereas younger children are often asymptomatic [1]. Mild aminotransferase elevation occurs in most symptomatic cases, but clinically significant jaundice is uncommon, and fulminant hepatic failure is rare [1–5].

Most published adult reports emphasize jaundice, cholestatic hepatitis, or acute liver failure, but the chronology of marked hepatocellular injury accompanied by later rises in alkaline phosphatase, direct bilirubin, and gamma-glutamyltransferase without encephalopathy is less clearly described. We therefore report a young adult with hepatitis most consistent with primary EBV-associated acute liver injury, classify the biochemical pattern with R-ratio at presentation and peak jaundice, and provide a limited contextual review of published adult cases to support diagnostic awareness rather than formal comparative inference.

2. Methods

We describe the clinical presentation, diagnostic evaluation, hospital course, and short-term follow-up of a young adult with hepatitis most consistent with primary EBV-associated acute liver injury. Severe acute liver injury was defined descriptively as acute hepatic injury with INR >=1.5 in the absence of encephalopathy; acute liver failure would have required encephalopathy in addition to coagulopathy [6, 7]. Injury pattern was classified using the R-ratio, calculated as (ALT / upper limit of normal for ALT) / (alkaline phosphatase / upper limit of normal for alkaline phosphatase) at presentation and peak jaundice. Institutional upper limits of normal were ALT 39 U/L and alkaline phosphatase 147 U/L. Relevant clinical data were abstracted from the medical record and are presented in de-identified form.

To place the present case within a limited clinical context, we conducted a PubMed review in March 2026. The search string was ((("Epstein-Barr virus" OR EBV) AND (hepatitis OR "cholestatic hepatitis" OR jaundice OR "acute liver injury" OR "acute liver failure") AND (adult OR "young adult") AND ("case report" OR "case series")). We limited inclusion to English-language adult case

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reports or case series describing primary EBV infection with severe acute liver injury, cholestatic hepatitis, or acute liver failure and extracted host status, peak bilirubin, peak INR, encephalopathy, liver transplantation requirement, and outcome. We excluded pediatric reports, immunocompromised-only reports, coinfection or alternative primary hepatic diagnoses, hemophagocytic lymphohistiocytosis-dominant presentations, and biopsy-only or pathology-focused reports without clinically extractable severity data. Seventeen potentially relevant full texts were reviewed manually, and 11 published reports or series met prespecified eligibility criteria for the descriptive contextual summary in Table 3. This component was intended as a narrative contextual review rather than a systematic review or a basis for direct phenotype comparison.

3. Results

Clinical presentation and initial evaluation A 20-year-old woman with a history of asthma presented with one week of headache, progressive nausea, and vomiting. On the day of presentation, she reported three episodes of emesis without improvement. She denied abdominal pain, diarrhea, sore throat, rhinorrhea, myalgias, recent travel, sick contacts, intravenous drug use, blood transfusions, prior liver disease, and a family history of liver disease. There was no family history of Wilson disease, autoimmune hepatitis, or hemochromatosis. She reported no alcohol use, no recent antibiotic or over-the-counter use, no acetaminophen-containing cold remedies, no herbal or workout supplements, no recent medication dose changes, and no antecedent hypotension, sepsis, hypoxemia, cardiac event, major bleeding, or recent surgery. Her chronic home medications included inhaled asthma therapies, omalizumab, antihistamines, and an oral contraceptive without recent initiation or dose escalation. She was sexually active with a male partner.

On admission, she was afebrile and hemodynamically stable, with an oxygen saturation of 98% on room air. Initial examination showed no acute distress, clear lungs, and a soft, nontender abdomen without guarding or rebound. Neurologic examination was normal, with no asterixis or encephalopathic features. Initial laboratory studies demonstrated marked hepatocellular injury, with AST 1,942 U/L, ALT 2,032 U/L, total bilirubin 4.0 mg/dL, direct bilirubin 1.7 mg/dL, alkaline phosphatase 140 U/L, and gamma-glutamyltransferase 73 U/L. Albumin was 4.1 g/dL, glucose was 65 mg/dL, and creatinine was 0.74 mg/dL. Platelet count was 148 x 10^3/uL, and the INR at presentation was 1.1. Using institutional upper limits of normal of ALT 39 U/L and alkaline phosphatase 147 U/L, the admission R-ratio was 54.7, consistent with a hepatocellular pattern. Complete blood count showed a total white blood cell count of 8.3 x 10^3/uL, with absolute lymphocytosis of 5.40 x 10^3/uL, comprising 65.6% of leukocytes. Manual differential later demonstrated 65% atypical lymphocytes. Pregnancy testing was negative.

Radiologic and diagnostic findings Right upper quadrant ultrasound showed normal liver echotexture, no common bile duct dilation, and normal gallbladder wall thickness. Computed tomography of the abdomen and pelvis demonstrated hepatomegaly, mild splenomegaly, and mildly enlarged mesenteric and retroperitoneal lymph nodes, without biliary ductal dilation or other acute intra-abdominal pathology (**Figure 1**).

The diagnostic evaluation was notable for a positive heterophile antibody (Monospot) test and EBV serologies consistent with acute primary infection, including positive VCA IgM and VCA IgG with negative EBNA IgG and EA IgG. Plasma EBV DNA quantitative polymerase chain reaction was 47,010 copies/mL (4.67 log copies/mL). Additional testing did not identify a more likely infectious, autoimmune, metabolic, or selected toxicologic cause: hepatitis A IgM, hepatitis B surface antigen and core IgM, hepatitis C antibody, hepatitis E testing, human immunodeficiency virus testing, cytomegalovirus serologies, COVID-19 testing, and herpes simplex virus types 1 and 2 polymerase chain reaction were negative; serum ethanol and acetaminophen levels were undetectable; antinuclear antibody, anti – smooth muscle antibody, and anti – liver kidney microsome type 1 were negative; total IgG was 8.1 g/L; serum ceruloplasmin was 31 mg/dL; 24-hour urinary copper was 34 µg/24 h; and slit-lamp examination showed no Kayser-Fleischer rings (**Table 1**).

Table 1: Diagnostic evaluation for infectious, autoimmune, metabolic, and selected toxic causes of acute liver injury

Test	Result
Heterophile antibody (Monospot)	Positive
EBV VCA IgM	Positive
EBV VCA IgG	Positive
EBV EBNA IgG	Negative
EBV EA IgG	Negative
EBV DNA quantitative PCR	47,010 copies/mL (4.67 log copies/mL)
Hepatitis A IgM	Negative
Hepatitis B surface antigen	Negative
Hepatitis B core IgM	Negative
Hepatitis C antibody	Negative
Hepatitis E testing	Negative
Human immunodeficiency virus testing	Negative
Cytomegalovirus serologies	Negative
COVID-19 testing	Negative
Herpes simplex virus types 1 and 2 PCR	Negative
Herpes simplex virus types 1 and 2 IgG	Positive; consistent with prior exposure
Serum ethanol	Undetectable
Acetaminophen level	Undetectable
Serum ceruloplasmin	31 mg/dL
24-hour urinary copper	34 µg/24 h (within normal range)
Slit-lamp examination for Kayser-Fleischer rings	Absent
Antinuclear antibody (ANA)	Negative
Anti–smooth muscle antibody (ASMA)	Negative
Anti–liver kidney microsome type 1 (anti-LKM1)	Negative
Total IgG	8.1 g/L (reference range 7–16 g/L)

EBNA, Epstein-Barr nuclear antigen; EA, early antigen; EBV, Epstein-Barr virus; PCR, polymerase chain reaction; VCA, viral capsid antigen.

Hospital course and follow-up She was admitted for close monitoring and supportive care, including intravenous fluids, antiemetics, serial glucose monitoring, and frequent laboratory reassessment. Gastroenterology and Infectious Diseases were consulted. During



Figure 1: Axial contrast-enhanced CT image of the abdomen showing hepatomegaly (left red arrow) and mild splenomegaly (right red arrow).

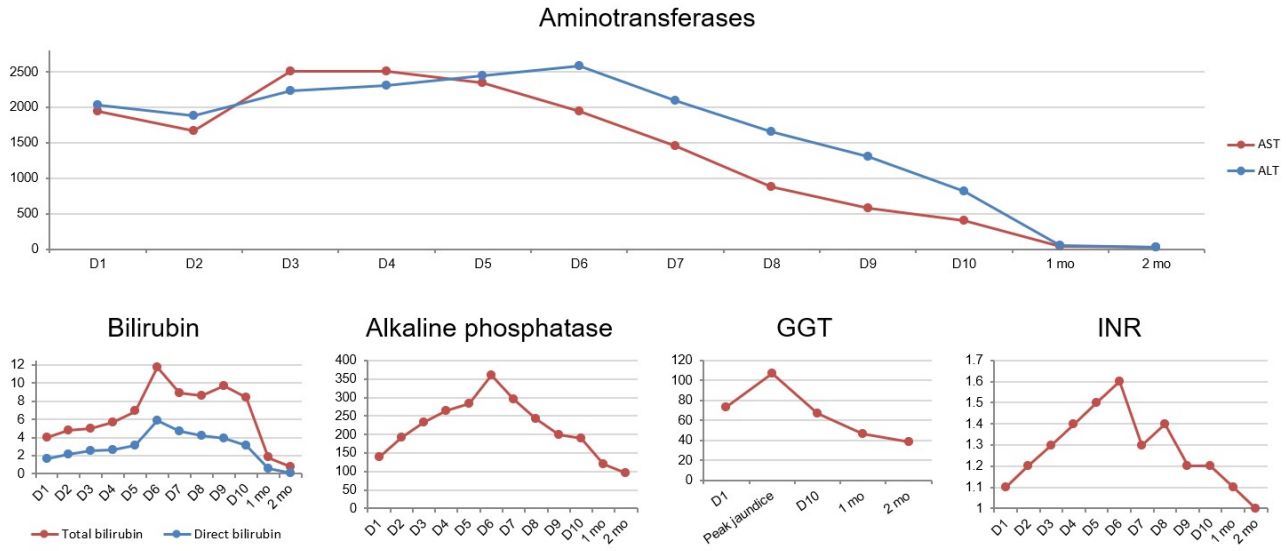


Figure 2: Key laboratory trends during hospitalization and at 1- and 2-month follow-up. The figure summarizes AST, ALT, total/direct bilirubin, alkaline phosphatase, gamma-glutamyltransferase, and INR. The R-ratio remained hepatocellular at presentation and peak jaundice despite evolving cholestatic features.

hospitalization, she developed progressive scleral icterus, generalized jaundice, and worsening biochemical liver injury. Peak AST was 2,508 U/L, peak ALT was 2,589 U/L, peak total bilirubin was 11.8 mg/dL, peak direct bilirubin was 5.9 mg/dL, peak alkaline phosphatase was 359 U/L, peak gamma-glutamyltransferase was 107 U/L, and peak INR was 1.6. At the peak of jaundice, the R-ratio remained 27.2, indicating that the biochemical pattern remained hepatocellular despite evolving cholestatic features. She also had a single elevated ammonia value of 88.3 µmol/L; in the absence of encephalopathy, this did not alter management beyond continued neurologic monitoring. Despite worsening coagulopathy and hyperbilirubinemia, she remained alert and fully oriented, without asterixis or other signs or symptoms of hepatic encephalopathy throughout hospitalization and at discharge.

Table 2: CARE-style timeline of major events, laboratory changes, injury-pattern classification, and management decisions

Time point	Symptoms/findings	Key laboratory data / Management decisions
Week before admission	Progressive headache, nausea, vomiting; no sore throat, rhinorrhea, myalgias, or abdominal pain	No outpatient laboratory data available. Presented for evaluation after persistent emesis
Hospital day 1 (admission)	Hemodynamically stable; no encephalopathy or asterixis; hepatocellular pattern of injury	AST 1,942 U/L; ALT 2,032 U/L; total/direct bilirubin 4.0/1.7 mg/dL; ALP 140 U/L; GGT 73 U/L; INR 1.1; R-ratio 54.7. Admitted for supportive care, serial neurologic examinations, and diagnostic workup
Early hospitalization	Progressive scleral icterus and generalized jaundice; mental status remained normal	EBV heterophile positive; VCA IgM/VCA IgG positive; EBNA IgG and EA IgG negative; EBV DNA 47,010 copies/mL. Gastroenterology and Infectious Diseases consulted; broad alternative-cause evaluation was unrevealing
Peak jaundice/peak hospitalization values	Worsening jaundice without encephalopathy	Peak AST 2,508 U/L; peak ALT 2,589 U/L; peak total/direct bilirubin 11.8/5.9 mg/dL; peak ALP 359 U/L; peak GGT 107 U/L; peak INR 1.6; single ammonia peak 88.3 µmol/L; R-ratio 27.2. Case discussed with tertiary hepatology/transplant center as precautionary management step
Hospital day 10 (discharge)	Clinically improved; tolerating oral intake; no encephalopathy	AST 408 U/L; ALT 813 U/L; total/direct bilirubin 8.4/3.1 mg/dL; ALP 190 U/L; GGT 67 U/L; INR 1.2. Discharged with close gastroenterology follow-up and serial outpatient laboratory monitoring
1-month follow-up	Jaundice resolved clinically	AST 37 U/L; ALT 51 U/L; total/direct bilirubin 1.8/0.6 mg/dL; ALP 121 U/L; GGT 47 U/L; INR 1.1. Early clinical recovery with substantial biochemical improvement
2-month follow-up	Clinically well	AST 25 U/L; ALT 31 U/L; total/direct bilirubin 0.8/0.1 mg/dL; ALP 95 U/L; GGT 39 U/L; INR 1.0. Biochemical normalization documented

ALP, alkaline phosphatase; ALT, alanine aminotransferase; AST, aspartate aminotransferase; EBV, Epstein-Barr virus; GGT, gamma-glutamyltransferase; INR, international normalized ratio; R-ratio calculated using ALT 39 U/L and ALP 147 U/L as institutional upper limits of normal.

Because of worsening coagulopathy and progressive hyperbilirubinemia, the case was discussed with hepatology at a tertiary transplant center. This discussion was used as a precautionary management step rather than as an objective severity marker, and transfer was deferred after serial neurologic examinations remained normal and aminotransferases began to improve.

By hospital day 10, AST had declined to 408 U/L and ALT to 813 U/L, while alkaline phosphatase had declined to 190 U/L, gamma-glutamyltransferase to 67 U/L, and INR improved to 1.2. Total bilirubin remained elevated at 8.4 mg/dL and direct bilirubin at 3.1 mg/dL. She remained clinically stable, tolerated oral intake, and had no evidence of encephalopathy. She was discharged home on hospital day 10 with close gastroenterology follow-up and instructions for serial outpatient monitoring of liver tests and INR. At 1-month follow-up, AST was 37 U/L, ALT was 51 U/L, total bilirubin was 1.8 mg/dL, direct bilirubin was 0.6 mg/dL, alkaline phosphatase was 121 U/L, gamma-glutamyltransferase was 47 U/L, and INR was 1.1, with clinical resolution of jaundice. At 2-month follow-up, AST was 25 U/L, ALT was 31 U/L, total bilirubin was 0.8 mg/dL, direct bilirubin was 0.1 mg/dL, alkaline phosphatase was 95 U/L, gamma-glutamyltransferase was 39 U/L, and INR was 1.0, consistent with biochemical normalization. Selected laboratory trends are shown in **(Figure 2)**, and a concise clinical timeline is provided in **(Table 2)**.

Contextual review findings Across the 10 individual adult case reports included in the contextual table, 9 patients described as immunocompetent or otherwise without evident major immunosuppression improved with supportive care alone, and 1 elderly patient underwent liver transplantation; the separate Acute Liver Failure Study Group series captured a different, higher-risk acute liver failure phenotype [3, 8–17]. Because the published reports were heterogeneous and incompletely reported, **(Table 3)** is presented as descriptive context only and is not intended for direct phenotype comparison. Within that limited literature, the present case serves as an additional example of marked hepatocellular injury with evolving cholestatic features and coagulopathy without encephalopathy.

4. Discussion

This case is notable for marked hepatocellular injury accompanied by progressive jaundice, rising direct bilirubin, alkaline phosphatase, and gamma-glutamyltransferase, transient coagulopathy, and the absence of hepatic encephalopathy despite worsening laboratory results. Formal injury-pattern classification supports this nuance. Using the R-ratio, the pattern was hepatocellular at presentation (54.7). It remained hepatocellular at peak jaundice (27.2), even as alkaline phosphatase rose from 140 to 359 U/L, direct bilirubin from 1.7 to 5.9 mg/dL, and gamma-glutamyltransferase from 73 to 107 U/L. We therefore no longer describe a full biochemical transition to cholestatic injury; instead, the course is best characterized as severe hepatocellular injury with evolving cholestatic features and delayed resolution of cholestatic markers. Although elevated aminotransferases are common in EBV infection, clinically apparent jaundice remains uncommon, and severe jaundiced presentations are infrequently reported [1–5, 8–10, 13, 14, 16, 17]. The patient also lacked the classic triad of fever, pharyngitis, and cervical lymphadenopathy, underscoring that EBV hepatitis may present predominantly with gastrointestinal symptoms and abnormal liver tests [4, 8–10, 13, 14, 16].

The overall presentation was most consistent with primary EBV-associated hepatitis based on atypical lymphocytosis, hepatomegaly and splenomegaly, a positive heterophile test, and the serologic profile of VCA IgM positivity, VCA IgG positivity, and EBNA IgG negativity, with detectable plasma EBV DNA providing additional supportive evidence [1, 18]. Biliary imaging and the broader diagnostic evaluation argued against a more likely obstructive,

Table 3: Descriptive contextual summary of selected adult EBV hepatitis case reports/case series meeting prespecified eligibility criteria

Case/study	Clinical & demographic data	Key severity markers	Outcome / contextual note
Present case (current report)	20/F; no known major immunodeficiency; omalizumab use	Peak total bilirubin 11.8 mg/dL; INR 1.6; encephalopathy No; LT No	Supportive care; biochemical normalization by 2 months; hepatocellular pattern with evolving cholestatic features
Khoo 2016 [9]	59/F; immunocompetent	Peak bilirubin 9.1 mg/dL; INR NR; encephalopathy No; LT No	Supportive care; recovered
Moniri 2017 [10]	23/F; immunocompetent	Peak bilirubin 15.9 mg/dL; INR 1.11; encephalopathy No; LT No	Recovered; normal liver tests during follow-up
Shah 2020 [14]	25/M; immunocompetent	Peak bilirubin 9.1 mg/dL; INR 1.4; encephalopathy No; LT No	Improved and discharged; transplant evaluation considered
Theodory 2023 [16]	42/M; immunocompetent	Peak bilirubin 3.6 mg/dL; INR NR; encephalopathy No; LT No	Recovered; normal labs 1 week after discharge
Njoku 2025 [12]	50/M; immunocompetent	Peak bilirubin 5.4 mg/dL; INR NR; encephalopathy No; LT No	Ambulatory supportive care; recovered
Nishioka 2025 [11]	25/F; immunocompetent	Peak bilirubin 5.1 mg/dL; INR NR; encephalopathy No; LT No	Supportive care; clinical/laboratory improvement
Tak 2025 [15]	56/M; immunocompetent	Peak bilirubin 11.9 mg/dL; INR 1.1; encephalopathy No; LT No	Conservative management; biochemical normalization on follow-up
Joshi 2024 [8]	20/F; immunocompetent	Peak bilirubin 4.4 mg/dL; INR NR; encephalopathy No; LT No	Improved with supportive care
Pamala 2024 [13]	24/M; immunocompetent	Peak bilirubin 6.9 mg/dL; INR normal; encephalopathy No; LT No	Conservative management; recovered
Zhang 2016 [17]	67/F; immunocompetent	INR 3.7; encephalopathy Yes; LT Yes	Alive at 6 months after liver transplantation
Mellinger 2014 (ALFSG, n=4) [3]	Median age 30 y; 75% male; 75% immunocompetent	Median peak bilirubin 21 mg/dL; median INR 3.5; encephalopathy Yes	Aggregate acute liver failure series: 1 spontaneous survivor, 1 LT survivor, 2 deaths

ALFSG, Acute Liver Failure Study Group; LT, liver transplantation; NR, not reported.

autoimmune, metabolic, or competing infectious cause. Even so, causal attribution remains inferential rather than absolute.

Wilson's disease and autoimmune hepatitis were specifically evaluated and considered unlikely. Wilson's disease was ruled out by normal serum ceruloplasmin, normal 24-hour urinary copper excretion, and the absence of Kayser-Fleischer rings on slit-lamp examination. Autoimmune hepatitis was likewise disfavored by negative antinuclear antibody, anti – smooth muscle antibody, and anti – liver kidney microsome type 1 testing together with a normal total IgG level.

Residual diagnostic uncertainty nonetheless remains. Chronic home medications included omalizumab, inhaled asthma therapies, antihistamines, and an oral contraceptive. Still, there was no recent initiation, dose escalation, supplement exposure, use of an acetaminophen-containing product, or ischemic event to suggest a stronger alternative explanation. No formal RUCAM or other drug-induced liver injury causality instrument was prospectively applied, so drug-induced liver injury cannot be excluded with complete certainty.

The original draft described the patient as immunocompetent. Because she was receiving omalizumab, we have softened that wording. She had no known primary immunodeficiency, HIV infection, transplant history, malignancy-related immunosuppression, systemic corticosteroid use, or other conventional immunosuppressive therapy. Still, we now describe her more cautiously as a young adult without a known major immunodeficiency.

This case also illustrates the practical distinction between severe acute liver injury and acute liver failure. The INR rose to 1.6, but the patient never developed encephalopathy, asterixis, or altered mentation; she therefore met the descriptive framework for severe acute liver injury rather than acute liver failure [6, 7]. The single ammonia elevation should likewise be interpreted cautiously. In the absence of encephalopathy, it did not define acute liver failure and did not independently drive management beyond continued neurologic surveillance.

Transplant hepatology discussion is included here as a contextual management detail rather than as a reproducible disease characteristic. In a single case report, such discussion should not be interpreted as proof of objective severity beyond the reported clinical and laboratory findings.

The literature component of this manuscript is intentionally limited. Because the contextual review was PubMed-only, English-language, manually selected, and based largely on heterogeneous case reports, it should not be interpreted as systematic or exhaustive. For the same reason, the present case is better framed as an additional example within a sparsely reported clinical spectrum rather than a definitive expansion of that spectrum.

Follow-up is still limited to 2 months, but it is now more informative. At 1 month, jaundice had resolved clinically while bilirubin remained mildly elevated. By 2 months, AST, ALT, total bilirubin, direct bilirubin, alkaline phosphatase, gamma-glutamyltransferase, and INR had normalized. Accordingly, we describe the outcome as clinical resolution of jaundice by 1 month with biochemical normalization documented by 2 months.

Management of EBV hepatitis remains largely supportive. Routine antivirals and corticosteroids are not recommended for uncomplicated infectious mononucleosis, and evidence for a specific antiviral benefit in EBV hepatitis remains limited [1]. In the present case, careful monitoring, supportive care, and serial neurologic assessment were appropriate because mental status remained normal and laboratory values subsequently improved.

5. Conclusion

Primary EBV infection should be included in the differential diagnosis of marked acute liver injury and jaundice in young adults, even when sore throat and lymphadenopathy are absent. This case illustrates severe hepatocellular injury with evolving cholestatic features, transient coagulopathy without acute liver failure, and biochemical normalization by 2 months. Given the limited case-based literature, the clinical message is confined to diagnostic awareness, cautious interpretation of severity markers, and careful serial monitoring.

Conflicts of Interest

The authors declare that they have no known competing financial interests or personal relationships that could have appeared to influence the work reported in this paper.

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Informed Consent

Written informed consent was obtained from the patient for publication of this case report and accompanying images.

Large Language Model

Grammarly AI was used during manuscript revision for language editing and organizational assistance. All scientific content, data interpretation, and final wording were reviewed and approved by the authors.

Author Contributions

HA handled conceptualization, investigation, data curation, and wrote the original draft. MA conducted the investigation, performed the formal analysis, and contributed to writing, review, and editing. KA worked on the literature review and validation and contributed to writing, reviewing, and editing. RAA managed data curation, created the visualizations, and contributed to writing, review, and editing. AHG supervised the work, contributed to conceptualization and project administration, and participated in writing, review, and editing.

Data Availability

All data relevant to the case and the contextual review are included in the article. Additional patient-level information is withheld to protect patient privacy and confidentiality.

References

1. Dunmire SK, Verghese PS, Balfour J H H. Primary Epstein-Barr virus infection. J Clin Virol. 2018;102:84-92. [PMID: 29525635, https://doi.org/10.1016/j.jcv.2018.03.001].
2. Kang MJ, Kim TH, Shim KN, Jung SA, Cho MS, Yoo K, et al. Infectious mononucleosis hepatitis in young adults: two case reports. Korean J Intern Med. 2009;24(4):381-7. [PMID: 19949739, PMCID: PMC2784984, https://doi.org/10.3904/kjim.2009.24.4.381].
3. Mellinger JL, Rossaro L, Naugler WE, Nadig SN, Appelman H, Lee WM, et al. Epstein-Barr virus (EBV) related acute liver failure: a case series from the US Acute Liver Failure Study Group. Dig Dis Sci. 2014;59(7):1630-7. [PMID: 24464209, PMCID: PMC4250929, https://doi.org/10.1007/s10620-014-3029-2].
4. Vine LJ, Shepherd K, Hunter JG, Madden R, Thornton C, Ellis V, et al. Characteristics of Epstein-Barr virus hepatitis among patients with jaundice or acute hepatitis. Aliment Pharmacol Ther. 2012;36(1):16-21. [PMID: 22554291, https://doi.org/10.1111/j.1365-2036.2012.05122.x].
5. Yang SI, Geong JH, Kim JY. Clinical characteristics of primary Epstein Barr virus hepatitis with elevation of alkaline phosphatase and gamma-glutamyltransferase in children. Yonsei Med J. 2014;55(1):107-12. [PMID: 24339294, PMCID: PMC3874904, https://doi.org/10.3349/ymj.2014.55.1.107].
6. Fernandez J, Bassegoda O, Toapanta D, Bernal W. Acute liver failure: A practical update. JHEP Rep. 2024;6(9):101131. [PMID: 39170946, PMCID: PMC11337735, https://doi.org/10.1016/j.jhepr.2024.101131].
7. Shingina A, Mukhtar N, Wakim-Fleming J, Alqahtani S, Wong RJ, Limketkai BN, et al. Acute Liver Failure Guidelines. Am J Gastroenterol. 2023;118(7):1128-53. [PMID: 37377263, https://doi.org/10.14309/ajg.0000000000002340].
8. Joshi A, Jha D, Wari E, Saeed M, Hussain M, Hiatt TK. Cholestatic hepatitis in acute Epstein-Barr virus infection: A case report. Clin Case Rep. 2024;12(8):e9357. [PMID: 39176101, PMCID: PMC11338836, https://doi.org/10.1002/ccr3.9357].
9. Khoo A. Acute cholestatic hepatitis induced by Epstein-Barr virus infection in an adult: a case report. J Med Case Rep. 2016;10:75. [PMID: 27037083, PMCID: PMC4818405, https://doi.org/10.1186/s13256-016-0859-x].
10. Moniri A, Tabarsi P, Marjani M, Doosti Z. Acute Epstein - Barr virus hepatitis without mononucleosis syndrome: a case report. Gastroenterol Hepatol Bed Bench. 2017;10(2):147-9. [PMID: 28702140, PMCID: PMC5495904].
11. Nishioka H, Kitagawa N. Epstein-Barr Virus-Related Infectious Mononucleosis Accompanied by Cholestatic Hepatitis. Cureus. 2025;17(7):e88392. [PMID: 40842806, PMCID: PMC12367204, https://doi.org/10.7759/cureus.88392].
12. 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 (Lond). 2025;86(4):1-11. [PMID: 40265533, https://doi.org/10.12968/hmed.2024.0919].
13. Pamala C, Orabi M, Avinash S, Kamel I. Atypical Presentation of Epstein-Barr Virus Infectious Mononucleosis With Cholestatic Hepatitis and Hyperbilirubinemia in a Young Adult: A Case Report. Cureus. 2024;16(10):e71066. [PMID: 39380778, PMCID: PMC11459418, https://doi.org/10.7759/cureus.71066].
14. Shah J, Lingiah V, Pyrsopoulos N, Galan M. Acute Liver Injury due to Severe Epstein-Barr Virus Infection. ACG Case Rep J. 2020;7(2):e00325. [PMID: 32309513, PMCID: PMC7145184, https://doi.org/10.14309/crj.0000000000000325].
15. Tak MB, Li Kam Wa A, Simkin L, Abdelnabi M, Tasneem A. Primary Epstein-Barr Virus Infection Presenting as Cholestatic Hepatitis Without Infectious Mononucleosis in an Immunocompetent Middle-Aged Adult: A Case Report. Cureus. 2025;17(11):e96907. [PMID: 41409949, PMCID: PMC12706383, https://doi.org/10.7759/cureus.96907].
16. Theodory B, Dopp M, Swisher AR, Flores RM, Robb PM. Epstein-Barr virus induced acute hepatitis with hyperferritinemia: A rare presentation. IDCases. 2023;33:e01872. [PMID: 37609447, PMCID: PMC10440503, https://doi.org/10.1016/j.idcr.2023.e01872].
17. Zhang W, Chen B, Chen Y, Chamberland R, Fider-Whyte A, Craig J, et al. Epstein-Barr Virus-Associated Acute Liver Failure Present

in a 67-Year-Old Immunocompetent Female. Gastroenterology Res. 2016;9(4-5):74-8. [PMID: 27785330, PMCID: PMC5040549, https://doi.org/10.14740/gr718e].

18. De Paschale M, Clerici P. Serological diagnosis of Epstein-Barr virus infection: Problems and solutions. World J Virol. 2012;1(1):31-43. [PMID: 24175209, PMCID: PMC3782265, https://doi.org/10.5501/wjv.v1.i1.31].