

Case Report

Post-EBV Infection Liver Failure in Geriatrics: A Case Report

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Summary

Acute liver failure is a clinical syndrome that can have multiple origins, the most common causes being acetaminophen overdose, drug reactions and viral infections. The associated mortality is high and shows the seriousness of the condition. Rare cases of infection with the Epstein-Barr Virus (EBV), which is a virus affecting almost all people under the age of 40, have led to liver failure. Here we describe the case of a 77-year-old female patient who presented with liver failure following an EBV infection.

Keywords: EBV; Gériatrie; Insuffisance hépatique

Points of Interest

Liver involvement secondary to EBV infection is known, but liver failure due to EBV is rarer, especially in an elderly person.

Introduction

Acute liver failure is a known clinical syndrome with a high mortality rate and can have multiple etiologies, most frequently due to acetaminophen overdose, followed by drug reactions and then viral infections, notably hepatitis B. There are also other toxic, vascular, metabolic, autoimmune and indeterminate causes. Primary EBV infection, on the other hand, is common in the general population and often asymptomatic. Nearly 90 to 95% of adults have this infection before the age of 20. However, a severe primary systemic infection can rarely be associated with hepatitis and jaundice, leading to acute liver failure. In our case, it involves a 77-year-old patient with a geriatric profile, admitted for a clinical picture of liver failure secondary to EBV infection.

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Case Description

77-year-old patient, admitted from the emergency department at the request of her primary care physician, presenting with a clinical picture characterized by liver failure and acute kidney failure, discovered incidentally through lab tests in the emergency department. The patient presented with jaundice and itching for the past 4 days, accompanied by nocturnal sweating, nausea without vomiting and poor appetite. The systematic anamnesis was nearly normal, with complaints of dysuria but no associated hematuria or fever, and other parameters were reassuring.

On clinical examination, the patient was predominantly jaundiced, with a distended abdomen and slight tenderness in the right upper quadrant and hypogastric region. In the blood test, there is a severe impairment of liver and kidney function, as well as significant hyponatremia and hyperkalemia:

Parameter	Value
CRP (C-Reactive Protein)	24 mg/dl
White Blood Cells	11,340
Neutrophils	9,540
Lymphocytes	1,110
Creatinine	2.76 mg/dl
Glomerular Filtration Rate	17
Total Bilirubin	19.83 mg/dl
Direct Bilirubin	13.61 mg/dl
SGOT	561 U/L
SGPT	456 U/L
GGT (Gamma Glutamyl Transferase)	3,831 U/L
Alkaline Phosphatase	2,686 U/L
Alpha-Fetoprotein	4.2
Ferritin	2,478 µg/L
Sodium	119 mmol/l
Potassium	7.2 mmol/L
Chloride	87 mmol/L
INR (International Normalized Ratio)	2.27
APTT	40.7 seconds
Prothrombin	33%
Triglycerides	356 mg/dl
Total Cholesterol	628 mg/dl
HDL	52 mg/dl
LDL	504.4 mg/dl
Non-HDL	576 mg/dl
Blood Ammonia	217 µg NH3/dl

It is important to mention that the patient was recently discharged from a geriatric hospitalization 10 days ago to a nursing home for care after a long hospital stay of 3 months where she went through the services of pulmonology, cardiology, and finally geriatrics due to

pneumonia and cardiac decompensation from rapid atrial fibrillation. In fact, she received numerous treatments with antibiotics such as amoxicillin-clavulanic acid, cefepime, piperacillin-tazobactam and trimethoprim-sulfamethoxazole. At the time of hospital discharge, her blood test results showed a CRP of 37.75 mg/dl, a total bilirubin of 2 mg/dl with 1.62 mg/dl direct bilirubin and marked cytolysis and cholestasis: AST 406 U/L, ALT 236 U/L, alkaline phosphatase 747 U/L, and gamma-glutamyl transferase 195 U/L.

Her home treatment included: Bisoprolol 2.5 mg morning and evening, bumetanide 2 mg in the morning, vitamin D in the afternoon, acetaminophen 1g if there is pain, trimethoprim-sulfamethoxazole 800/160 mg morning and evening which would not be administered in the nursing home, iron gluconate 695 mg at noon, pantoprazole 40 mg on an empty stomach, escitalopram = 10 mg in the morning, spironolactone 50 mg in the morning, trazodone 50 mg in the evening, ipratropium bromide inhalation 0.25 mg four times a day, tiotropium/olodaterol inhaler 2.5 mcg with two puffs in the morning, edoxaban 60 mg in the morning, letrozole 2.5 mg in the morning, chronic oxygen at a rate of 1l/min.

Her medical history included Chronic Obstructive Pulmonary Disease (COPD) Gold II, asthma, long-term former smoking, arterial hypertension, and a right mastectomy in 2019 due to breast cancer. The suspected etiology of the underlying liver failure was possible by a hidden alcoholism and underlying medication toxicity. The hepatotoxic medications were discontinued: Dafalgan, Eusaprim, Losferron, Pantomed, Sipralaxa, Trazodone, Lixiana. Treatment with Lactulose was initiated.

The complementary examinations performed included:

- Urinalysis and blood cultures: Negative.
- ECG and chest X-ray: No abnormalities.
- Abdominal ultrasound and abdominal CT: No obstructive phenomenon, focal hepatic or pancreatic lesion, or biliary dilation were found, but they reported hepatomegaly in the context of a congested liver.
- Influenza A/B antigens and SARS-CoV-2 PCR: Negative.
- Autoimmune analysis: ANA positive with a titer of 1/160 and a finely granular pattern, but no specific antibodies were identified.
- CMV and Herpes zoster serologies: Normal.

Hepatic viral analysis: Normal. Anti-EBV (VCA) IgM: Elevated at 60 U/ml, while anti-EBV (VCA) IgG were measured at over 750 U/ml. However, the PCR for EBV viral load was not performed during hospitalization. Biological results improved rapidly within a few days, and no invasive procedures or liver biopsy were necessary. The diagnostic hypothesis is a potential EBV infection leading to subfulminant hepatitis in a context of toxic medication. Control anti-EBV (VCA) IgM antibodies measured three weeks after discharge were 0.1, while anti-EBV (VCA) IgG antibodies were 71.8.

Discussion

The pathogenesis of EBV-related hepatitis is explained by hepatic damage mediated by EBV-infected T lymphocytes through soluble products of the immune response. It has been demonstrated that the severity of the damage is related to a high viral load and, therefore,

to the number of infected CD8 T lymphocytes. Currently, there are no guidelines for the treatment of severe EBV hepatitis; however, Acyclovir, Valganciclovir, and corticosteroids are mentioned, though there is no evidence of a clear effect against EBV [1]. A case report shows how the only effective treatment strategy for eradicating EBV-infected T- or NK cells is allogeneic stem cell transplantation. EBV-specific T-cells from a third-party donor followed by alloHSCT with an alpha-beta T cell depleted and EBV-specific T-cell enriched peripheral stem cell graft from an EBV seropositive donor served as an effective and safe therapeutic approach in EBV infection. This approach could be an option in robust geriatric patients [2].

A systematic review conducted in 2020 aimed to determine the respective contributions of different viruses to the etiology of acute liver failure of viral origin. According to this review, the prevalence of liver failure induced by EBV was estimated at 6%. It also found that the combined mortality rate associated with viral liver failure in high-income countries was 29%, with a combined liver transplantation rate of 25% and a kidney transplantation rate of 18% [3].

Another case series study determined the median age of individuals with EBV-induced liver failure to be 30 years, with 75% being men and 25% being immunocompromised. Among these patients, 50% had heterophile antibodies IgM VCA (viral capsid antigen), 100% had heterophile antibodies IgG VCA, and 100% had positive EBV PCR results. They considered the diagnosis “probable” if either serological or tissue studies, but not both, confirmed EBV. It was considered “definite” if both studies were concordant. The histopathological results of liver biopsy mentioned extensive sinusoidal and lobular lymphocytosis in most patients, with some even showing signs of massive hepatic necrosis, with complete EBER staining in 75% of cases and positive in 50%. Half of the patients died. The study also mentions that due to a lack of serological data, it is difficult to distinguish between primary EBV infection and reactivation in patients [4].

In the literature review, there are few geriatric cases described with severe liver failure due to EBV infection, and only a few cases have been presented. Additionally, early diagnosis of hepatitis associated with this virus is challenging because reliable diagnostic criteria are not clearly defined. EBV DNA PCR is frequently used, as well as EBV serologies with VCA-IgM, VCA-IgG, EA-D, and EBNA to detect a recent, past, or reactive infection, but these tests are not reliable, especially in older individuals, as they have higher serum titers compared to young adults. Diagnosing this infection earlier could help avoid some cases of liver transplants [5].

Clinical manifestations vary between older adults and younger individuals. It has been found that patients over 40 years of age with EBV infection less frequently present with peripheral lymphadenopathy, pharyngitis, and splenomegaly. In contrast, they experience a more prolonged fever as well as more pronounced hepatic involvement and jaundice. Symptoms are often misleading and can delay diagnosis. It is important to keep this condition in mind to prevent, especially in older patients, the use of inappropriate invasive diagnostic procedures [6]. As chronic EBV infection impacts the innate and adapted immune system, a pro-inflammatory situation occurs. This situation is aggravated by ageing and this could be a reason of some weight as to why individuals with similar characteristics are fragile or not [7].

There are several EBV vaccine prototypes with encouraging results due to the obtention of significant humoral response and blocked

infection in humanized mouse models. This may lead to a reduction of both rare but severe cases such as the one in this article and, for example, neoplastic complications resulting from chronic EBV infection [8].

Conclusion

Severe acute EBV infection is a rare cause of liver failure and is primarily found in young individuals, which is particularly surprising and less described in geriatric patients. The treatment is not yet clearly established and requires further studies. It is important to conduct all viral serologies in cases of liver failure of undetermined origin to detect possible EBV involvement. EBV infection or reactivation may be under-recognized in cases of liver failure at present.

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