Severe Tubulointerstitial Nephritis from Acute Epstein-Barr Virus Infection

Craig A. Mackaness DO
Lehigh Valley Health Network, Craig_A.Mackaness@lvhn.org

Follow this and additional works at: http://scholarlyworks.lvhn.org/medicine
🔗 Part of the Medical Sciences Commons, and the Nephrology Commons

Published In/Presented At

This Poster is brought to you for free and open access by LVHN Scholarly Works. It has been accepted for inclusion in LVHN Scholarly Works by an authorized administrator. For more information, please contact LibraryServices@lvhn.org.
Severe Tubulointerstitial Nephritis from Acute Epstein-Barr Virus Infection

Craig A. Mackaness, DO
Lehigh Valley Health Network, Allentown, PA

Introduction

Epstein-Barr Virus (EBV) infection presents with diverse clinical manifestations along a broad spectrum of disease severity. The classic triad of EBV infection is fever, pharyngitis, and lymphadenopathy. Acute EBV infection may also manifest as hemolytic anemia, granulocytopenia, encephalitis, pericarditis, hepatitis, and other solid organ involvement.

First described in 1988, Acute renal failure from EBV-infectious mononucleosis is a rare disease complication, but subtle abnormalities in urinalysis, such as hematuria and proteinuria have been noted in as many as 15% of patients.

Renal involvement of EBV typically includes a focal interstitial mononuclear infiltrate. Glomerular involvement is uncommon, though both minimal change lesion and as well as mesangial dysplasia have been reported. The diagnosis of EBV involvement in the kidney has been demonstrated using both in situ hybridization as well as PCR assays.

The pathogenesis of EBV induced renal involvement and its rare occurrence remain unclear. T-cell activation, and direct injury due to EBV have been proposed mechanisms of renal injury.

A previously healthy, 21-year-old Caucasian woman presents to our center with a complaints of fevers recorded as high as 104°F, nausea with vomiting, malaise, and sore throat. Symptoms had been progressive over the course of the prior four weeks. She had been evaluated by her primary care provider 10 days prior to hospitalization, and had received Azithromycin for a diagnosis of left otitis media.

She denies any difficulties with urination – no dysuria, flank pain, hematuria, frothy urine or change in quantity. Oral intake has been maintained.

The patient is an undergraduate student at a local urban university. In addition to the symptoms noted above, she had evidence of bilateral lymphadenopathy was present. A temperature of 40°C was noted at the time of admission. Her heart rate was 117 per minute, blood pressure was 117/75. She had evidence of bilateral swelling of the supraventricular lymph nodes. On examination, her lungs were clear to auscultation, and there was no evidence of rales, rhonchi, or wheezes. She had a stable heart rate and respiratory rate. Her abdomen was soft and non-tender to palpation. No masses, organomegaly, or positive findings for peripheral edema were noted.

Acute Epstein Barr virus infection resulting in severe renal impairment is rare and is typically characterized by acute tubulointerstitial disease. Our case contrasts with previously reported cases as the majority of lymphocytes in the interstitium were CD4+ T-lymphocytes. Consistent with prior studies, the glomeruli were unremarkable.

EBV-encoding region (EBER) in situ hybridization is the methodology of choice for the detection of the Epstein-Barr virus (EBV) in tissue sections. Epstein-Barr Encoding Regions (EBERs) are secreted from EBV-infected cells and are recognized by toll-like receptor (TLR)3, leading to induction of type-I Interferons and inflammatory cytokines, and subsequent immune activation.

This case was complicated by evidence of a recent strep infection (elevated ASO titer) but no evidence of deposits suggestive of post-infectious glomerulonephritis were noted on electron microscopy. Prior cases of EBV induced renal failure have been preceded by streptococcal infection.

In addition, the patient had a recent/concurrent Mycoplasma infection, and it is unclear what role, if any, this had in her renal insufficiency. Therapy is supportive, with anecdotal evidence supporting parenteral corticosteroids as well as acyclovir for the viral infection.

The patient was treated supportively in regard to her renal manifestations, not requiring renal replacement therapy. Corticosteroids were initiated for severe tonsilar swelling, at that time her creatinine had already begun down-trending. She reached a nadir serum creatinine of 0.64 mg/dL in follow up four months following the acute illness.

This case underscores the spectrum of EBV disease beyond the classic manifestations, the difficulty in identifying the specific pathogenesis of the mechanism of renal dysfunction, and the heterogeneity of host immunologic responses.

References:


Acknowledgements: For additional information, please contact webmaster@lvmh.org.