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ANASARCA SECONDARY TO CYTOMEGALOVIRUS VIREMIA IN A POST-LIVER TRANSPLANT PATIENT

Noor Albusta

Department of Internal Medicine, Lahey Hospital and Medical Center, Burlington, USA

Corresponding author's e-mail: noor.albusta@lahey.org

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ABSTRACT

Introduction: Cytomegalovirus (CMV) infection is a common complication following solid organ transplantation and is associated with significant morbidity. While CMV classically presents with systemic symptoms, bone marrow suppression or gastrointestinal disease, atypical manifestations may occur. Anasarca and refractory ascites in liver transplant recipients are more commonly attributed to graft dysfunction, vascular complications, cardiac disease or renal pathology, and CMV is not often considered as a primary aetiology.

Case description: A 63-year-old woman with a history of alcohol-related cirrhosis underwent deceased donor liver transplantation and later presented with progressive abdominal distension and bilateral lower extremity oedema. Evaluation demonstrated tense ascites and anasarca without evidence of graft dysfunction, acute rejection, portal vascular complications, cardiac disease or nephrotic syndrome. Liver function tests were normal, transthoracic echocardiography revealed preserved systolic function, and urinalysis was negative for proteinuria. Further workup identified CMV viremia, and the patient was treated with valganciclovir per infectious disease recommendations, along with supportive measures including diuresis and large-volume paracentesis. Serial monitoring demonstrated improvement in CMV viral load with corresponding gradual resolution of fluid retention.

Conclusion: This case highlights CMV viremia as an important and potentially reversible cause of anasarca in post-liver transplant patients, likely mediated by CMV-induced endothelial dysfunction. Recognition of CMV-related fluid retention is essential to avoid unnecessary invasive procedures and to guide timely antiviral therapy in immunosuppressed individuals.

KEYWORDS

Cytomegalovirus infection, liver transplantation, anasarca, ascites, immunosuppression

LEARNING POINTS

- Cytomegalovirus (CMV) infection should be considered in the differential diagnosis of unexplained anasarca and refractory ascites in liver transplant recipients, even in the absence of graft dysfunction or classic CMV symptoms.
- A systematic evaluation to exclude cardiac, renal, vascular and graft-related causes of fluid overload is essential before attributing post-transplant anasarca to non-infectious aetiologies.
- Early recognition and treatment of CMV viremia can lead to clinical improvement and optimise outcomes in immunosuppressed patients.

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INTRODUCTION

Cytomegalovirus (CMV) infection remains one of the most common infectious complications following solid organ transplantation and continues to be a major contributor to post-transplant morbidity[1,2]. Liver transplant recipients are particularly susceptible due to immunosuppressive therapy, donor–recipient serostatus mismatch and the early post-transplant inflammatory milieu. CMV infection may present with a wide spectrum of manifestations, ranging from asymptomatic viremia to tissue-invasive disease involving the gastrointestinal tract, bone marrow or allograft[3].

Classic clinical features of CMV infection include fever, malaise, cytopaenias and gastrointestinal symptoms; however, atypical or indirect presentations are increasingly recognised[4]. CMV has been implicated in systemic inflammatory responses, endothelial dysfunction and immune-mediated effects that may occur in the absence of overt organ involvement[5]. These indirect effects can complicate the post-transplant course and mimic other transplant-related complications.

Anasarca and refractory ascites in liver transplant recipients typically prompt evaluation for graft dysfunction, acute or chronic rejection, portal or hepatic vascular complications, cardiac disease, renal dysfunction or recurrence of portal hypertension[6]. Infectious aetiologies are often considered later in the diagnostic process, particularly when liver biochemistry and imaging studies are unremarkable. As a result, patients may undergo repeated invasive procedures before an underlying infectious cause is identified. While CMV-induced endothelial dysfunction is recognised in the literature, anasarca as a predominant presenting feature remains underreported[3].

We report a case of severe anasarca and ascites in a post-liver transplant patient in whom CMV viremia was identified as a likely contributing factor after exclusion of more common aetiologies. This case highlights the importance of considering CMV infection in the differential diagnosis of unexplained fluid overload in immunosuppressed patients.

CASE DESCRIPTION

A 63-year-old woman with a history of alcohol-related cirrhosis underwent a deceased donor liver transplant in January 2025. Her early post-transplant course was uncomplicated, and she was maintained on tacrolimus-based immunosuppression. Several months later, she presented with progressive abdominal distension, bilateral lower extremity oedema and generalised anasarca. She reported early satiety and discomfort related to fluid accumulation but denied fever, chest pain, dyspnoea, diarrhoea, gastrointestinal bleeding or changes in mental status. She noted that her symptoms resembled episodes of refractory ascites she had experienced prior to transplantation, during which she required frequent therapeutic paracentesis. Pre-transplant CMV serostatus demonstrated a donor-positive/recipient-negative (D^+/R^-) mismatch, placing her at high risk for CMV disease in the post-transplant period. She received standard valganciclovir prophylaxis following transplantation, which was discontinued after completion of a six-month prophylactic course in accordance with institutional protocol. Maintenance immunosuppression consisted of tacrolimus-based therapy with adjunctive mycophenolate mofetil and prednisone. She had not experienced any episodes of acute rejection requiring augmented immunosuppression prior to this presentation.

On examination, she was afebrile and haemodynamically stable. Physical findings included marked abdominal distension with shifting dullness, bilateral 2+ pitting oedema of the lower extremities and generalised fluid retention. There were no stigmata of acute graft dysfunction or hepatic encephalopathy. Cardiopulmonary examination was unremarkable.

Initial laboratory evaluation demonstrated preserved graft function, with aspartate aminotransferase 24 U/l (normal 10–40 U/l), alanine aminotransferase 28 U/l (normal 7–56 U/l), alkaline phosphatase 92 U/l (normal 44–147 U/l), total bilirubin 0.8 mg/dl (normal 0.2–1.2 mg/dl) and an international normalised ratio of 1.1, with no biochemical evidence of acute rejection. Serum creatinine was 0.9 mg/dl (baseline 0.9 mg/dl), and serum albumin was 3.6 g/dl (normal 3.5–5 g/dl). Urinalysis was negative for proteinuria, with a urine protein-to-creatinine ratio of 0.08 g/g (normal <0.2 g/g), making nephrotic syndrome unlikely. Complete blood count revealed a white blood cell count of 6.1 ×10^9/l.

Given concern for graft-related complications and alternative causes of fluid overload, abdominal ultrasound with Doppler evaluation was performed. The examination was partially limited by body habitus and bowel gas. The liver graft demonstrated mildly echogenic parenchyma without focal lesions. The biliary tree showed no intrahepatic or extrahepatic ductal dilatation, with the proximal common bile duct measuring 0.4 cm. Moderate ascites was present, most pronounced in the lower

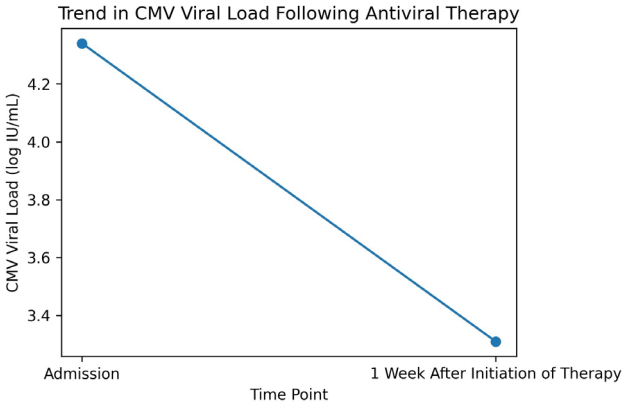


Figure 1. CMV viral load measured by PCR at admission and at one-after initiation of antiviral therapy. A marked decline in CMV viral load was observed following initiation of valganciclovir therapy, corresponding with clinical improvement in anasarca and ascites.

abdomen and pelvis, increased compared with prior imaging. The spleen measured 15.4 cm, consistent with stable splenomegaly. Hepatic vasculature was patent with normal hepatopaetal flow in the main, right and left portal veins, and waveforms in the hepatic arteries and hepatic veins were normal; no evidence of portal or hepatic vein thrombosis was identified. Mild fullness of the right renal collecting system was noted, unchanged from prior imaging. No pleural effusions were visualised.

Transthoracic echocardiography demonstrated preserved left ventricular systolic function with an ejection fraction of 60%, without evidence of systolic or diastolic dysfunction, making a primary cardiac aetiology of anasarca unlikely.

Given the patient's immunosuppressed state, an extensive infectious evaluation was pursued. CMV polymerase chain reaction (PCR) testing revealed significant viremia, with an initial viral load of 21,800 IU/ml (log 4.34). Epstein–Barr virus PCR testing was negative; hepatitis B serologies and BK virus PCR testing were also negative.

The patient was diagnosed with CMV viremia and initiated on valganciclovir 900 mg twice daily per infectious disease recommendations. She was also treated with intravenous diuresis, transitioned to oral furosemide 40 mg daily, and counselled on adherence to a low-sodium diet. A large-volume paracentesis was performed for symptomatic relief. Constipation and nausea were managed with polyethylene glycol and a proton pump inhibitor.

During hospitalisation, urine output gradually improved and no further accumulation of ascites was noted. Repeat CMV PCR obtained one week after initiation of antiviral therapy demonstrated a marked decline in viral load to 2,060 IU/ml (log 3.31), representing a 91% reduction from baseline *(Fig. 1)*. Given clinical improvement and a down trending viral burden, she was discharged with plans for weekly CMV PCR monitoring and repeat laboratory testing including liver profile, complete blood count, chemistry panel and tacrolimus level.

At outpatient follow-up, the patient reported significant improvement in abdominal distension and lower extremity oedema. Laboratory studies demonstrated continued decline in CMV viral load and stable graft function. In the absence of vascular, cardiac, renal or rejection-related aetiologies, her anasarca and ascites were attributed to CMV viremia in the post-liver transplant setting.

DISCUSSION

CMV infection remains a major source of morbidity in solid organ transplant recipients and is most commonly associated with systemic symptoms, cytopaenias, gastrointestinal disease and allograft dysfunction[1–3]. In liver transplant patients, post-transplant ascites and generalised oedema are typically attributed to graft dysfunction, portal or hepatic vascular complications, cardiac disease, renal pathology or recurrence of portal hypertension[4]. Consequently, diagnostic evaluation is often directed toward these aetiologies, particularly when fluid overload resembles the patient's pre-transplant clinical course. Infectious causes such as CMV are frequently considered later, especially in the absence of classic CMV syndrome.

In this case, a comprehensive evaluation excluded the more common causes of post-transplant anasarca. Liver biochemistry remained within normal limits, arguing against acute rejection or graft dysfunction. Doppler ultrasound demonstrated patent hepatic vasculature without evidence of portal vein thrombosis or hepatic outflow obstruction. Transthoracic echocardiography revealed preserved systolic function, and urinalysis was negative for proteinuria, making cardiac failure and nephrotic syndrome unlikely. The absence of these conditions prompted further investigation, ultimately revealing significant CMV viremia.

Although CMV infection is frequently encountered after transplantation, its presentation as predominant anasarca without overt tissue-invasive disease is less commonly described. However, a growing body of evidence supports a mechanistic link between CMV infection and systemic fluid retention through endothelial dysfunction. CMV directly infects endothelial cells, inducing inflammatory cytokine production, upregulation of adhesion molecules such as VCAM-1 and fractalkine, and recruitment of inflammatory cells resulting in progressive endothelial injury[5–7]. Additionally, CMV impairs endothelial nitric oxide synthase activity through increased production of asymmetric dimethylarginine, an endogenous nitric oxide synthase inhibitor, leading to vasoconstriction and endothelial dysfunction[8]. Viral infection also induces oxidative stress with increased superoxide production, further compromising endothelial barrier integrity[8].

These processes culminate in increased capillary permeability to plasma proteins, allowing protein-rich fluid to extravasate from the intravascular space into interstitial and third spaces – the fundamental pathophysiology underlying capillary leak and generalised oedema[9,10]. In transplant recipients with active CMV infection, markers of endothelial injury such as von Willebrand factor and soluble VCAM-1 have been shown to rise significantly and correlate with the presence of circulating endothelial cells, supporting a direct role for CMV-mediated endothelial damage[6,9]. Notably, the combination of CMV infection and heightened immune activation confers increased susceptibility to endothelial injury even in the absence of overt organ-invasive disease.

Reports in the literature further support CMV's capacity to cause systemic fluid accumulation. CMV-associated protein-losing enteropathy has been described in paediatric and immunocompromised populations, presenting with generalised oedema and hypoalbuminaemia that resolve following antiviral therapy[6]. CMV-related ascites and massive oedema have also been reported in neonates and immunocompetent adults, attributed to microvascular injury and increased vascular permeability rather than structural organ disease[8]. These observations parallel the

presentation in our patient and provide additional biological plausibility for CMV-induced anasarca.

Atypical and late presentations of CMV disease in solid organ transplant recipients are increasingly recognised, particularly in the era of antiviral prophylaxis and delayed-onset disease. Such presentations may lack fever, leukocytosis or thrombocytopaenia and instead manifest with indirect or systemic effects of infection[10]. Our patient's presentation aligns with this pattern as she was afebrile without laboratory evidence of graft dysfunction, and her predominant manifestation was fluid overload rather than classic CMV syndrome.

While paracentesis and diuretic therapy provided immediate symptomatic relief, sustained resolution of anasarca coincided temporally with declining CMV viral load following initiation of valganciclovir therapy, with CMV PCR decreasing from 21,800 IU/ml to 2,060 IU/ml within one week. Importantly, the patient had experienced refractory ascites prior to transplantation that required frequent paracentesis, yet her post-transplant fluid retention improved only after treatment of CMV viremia, despite similar supportive measures. This temporal relationship, combined with systematic exclusion of alternative aetiologies and the biological plausibility of CMV-induced endothelial dysfunction, supports CMV viremia as a primary contributor to her presentation rather than an incidental finding.

This case highlights an underrecognized manifestation of CMV infection in liver transplant recipients and underscores the importance of maintaining a broad differential diagnosis when evaluating post-transplant anasarca. Early identification of CMV-related fluid retention may prevent unnecessary invasive procedures, reduce hospital length of stay and allow for timely initiation of targeted antiviral therapy. For internists and transplant clinicians, this case reinforces that CMV infection should be considered even when graft function appears preserved and classic CMV symptoms are absent[1,9].

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