

Spontaneous Splenic Infarction Associated with Cytomegalovirus Viremia in an Immunocompromised Renal Transplant Recipient

Sarah Abu Kamal¹, Sarah Farnand¹, Caroline Pinkerman¹, and Abdelrahman Mansour^{2*}

¹Midwestern University

²Franciscan Alliance - Olympia Fields

*Corresponding author

Abdelrahman Mansour, Franciscan Alliance - Olympia Fields, IL 60461, United States.

Received: September 11, 2026; Accepted: September 17, 2026; Published: September 25, 2026

Abstract

Splenic infarction is a rare but serious complication of cytomegalovirus (CMV) infection that can present with multiple and often non-specific manifestations. We present a case of splenic infarction in a 34-year-old kidney transplant recipient receiving immunosuppressive therapy who presented with fatigue, abdominal pain, and diarrhea. Imaging revealed multiple splenic hypodensities initially concerning for lacerations but ultimately consistent with nontraumatic subacute splenic infarcts.

Laboratory evaluation showed a positive CMV polymerase chain reaction (PCR) of peripheral blood (25,361 IU/mL). A broad infectious, cardioembolic, and hereditary hypercoagulable workup was unrevealing, supporting a diagnosis of CMV-associated hypercoagulopathy leading to thrombus formation and splenic infarction in an immunocompromised host. The patient improved with supportive care, anticoagulation, and valganciclovir and was discharged with multidisciplinary follow-up; he was subsequently lost to follow-up, precluding confirmation of radiographic or virologic resolution.

Background

Cytomegalovirus (CMV) is a double-stranded DNA virus of the Betaherpesvirinae subfamily and is globally prevalent, with an estimated seroprevalence of 83% in the general population [1]. CMV infection, which is evidence of viral replication regardless of symptoms, is distinguished from CMV disease, which requires infection plus attributable clinical or laboratory findings; tissue-invasive disease specifically requires histopathologic or immunohistochemical confirmation of viral presence within affected tissue. Immunocompetent patients with CMV infection most often range from asymptomatic presentation to infectious mononucleosis, whereas immunocompromised individuals more often develop CMV disease with more serious end-organ manifestations [2]. Primary CMV infection; as opposed to reactivation of latent virus occurring more than three years after solid organ transplantation most commonly manifests as gastrointestinal disease, a pattern also described among primary CMV infections in immunocompetent hosts [3]. Thrombotic complications represent another significant, though less recognized, manifestation of CMV disease. Splenic infarction, splanchnic vein thrombosis, and venous thromboembolism (VTE) are the most prevalent thromboses associated with acute CMV infection. VTE has been more commonly reported among immunocompromised patients, whereas splanchnic vein

thrombosis has been more often described in immunocompetent patients [4]. Splenic infarcts also predominantly affect younger individuals, with a mean age of approximately 38 years [4]. Our case, a 34-year-old immunocompromised patient with splenic infarcts, underscores the variable thrombotic manifestations of CMV disease and the importance of considering CMV-associated hypercoagulopathy in the differential diagnosis of unexplained visceral infarction.

Case Report

A 34-year-old male with a history of end-stage renal disease secondary to autosomal dominant polycystic kidney disease, treated with two years of dialysis prior to deceased donor kidney transplantation in 2022, presented to the emergency department with a two-week history of progressive fatigue, mild shortness of breath, decreased appetite, body aches, chills, night sweats, and vague periumbilical abdominal pain. He reported four to five loose bowel movements daily at the peak of his illness approximately one week prior to admission, which had improved by the time of presentation. He denied fever, abdominal trauma, and any prior history of deep vein thrombosis or pulmonary embolism. He was maintained on immunosuppressive therapy with prednisone, tacrolimus, and mycophenolate, with a baseline serum creatinine of approximately 1.5 mg/dL. His post-

Citation: Sarah Abu Kamal, Sarah Farnand, Caroline Pinkerman, Abdelrahman Mansour. Spontaneous Splenic Infarction Associated with Cytomegalovirus Viremia in an Immunocompromised Renal Transplant Recipient. *J Infect Dis Treat*. 2026. 4(3): 1-5. DOI: doi.org/10.61440/JIDT.2026.v4.88

transplant care, including tacrolimus monitoring, was managed by the University of Indiana, and he mostly received his post-transplant medical care out of state but was visiting family in Illinois. He worked in a nursing home with known ill contacts and had been prescribed amoxicillin-clavulanate five days prior to presentation for an odontogenic abscess.



Figure 1: Contrast-Enhanced CT of the Abdomen and Pelvis Demonstrating Splenic Hypodensity.

On presentation, vital signs were within normal limits. Laboratory evaluation revealed leukocytosis (white blood cell count $13.2 \times 10^9/L$), hyponatremia (129 mmol/L), elevated liver enzymes (alkaline phosphatase 444 U/L, aspartate aminotransferase [AST] 142 U/L, alanine aminotransferase [ALT] 231 U/L), and a serum creatinine of 1.9 mg/dL, mildly elevated from baseline. Computed tomography (CT) of the abdomen and pelvis demonstrated mild splenomegaly with three splenic hypodensities, initially interpreted as a grade III splenic injury without active hemorrhage, and a transplanted kidney in the right lower quadrant without hydronephrosis. General surgery was consulted; in the absence of a trauma history, the lesions were reinterpreted as infarcts rather than lacerations, and no operative intervention was recommended.

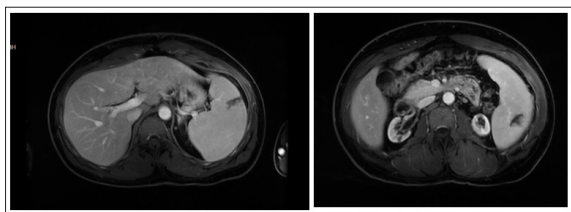


Figure 2: MRI Abdomen Confirming Subacute Splenic Infarcts. Magnetic resonance imaging (MRI) of the abdomen demonstrating multiple irregular peripheral splenic lesions corresponding to hypodensities identified on CT. Lesions demonstrate isointense T1 signal with peripheral T2 hypointensity and mild central T2 hyperintensity, without post-contrast enhancement or abnormal diffusion restriction. An additional lesion in the peripheral mid-anterior spleen and far lower peripheral spleen demonstrated questionable peripheral nodular post-contrast enhancement.

The patient received intravenous fluid resuscitation and was admitted. By hospital day one, abdominal pain, diarrhea, and nausea had fully resolved. Physical examination revealed a nontoxic-appearing, alert, and oriented patient with conjunctival injection and no jaundice. Repeat laboratory studies demonstrated improving leukocytosis and stable renal function, though transaminases remained elevated.

Further workup was undertaken given the clinical picture of splenic hypodensities in an immunocompromised patient without a trauma history. Magnetic resonance imaging (MRI) of the abdomen revealed multiple irregular peripheral splenic lesions consistent with subacute splenic infarcts, without post-contrast enhancement or abnormal diffusion restriction. Liver ultrasound confirmed a patent splenic vein; splenic artery patency was also confirmed.

Transthoracic echocardiogram showed no intracardiac thrombus, and a cardiac event monitor showed no evidence of atrial fibrillation, arguing against a cardioembolic source. Chest radiograph showed no active pulmonary disease.

Laboratory workup was broadened to evaluate infectious, hematologic, and hypercoagulable etiologies (Table 1). CMV IgM and IgG were positive, with a peripheral blood CMV PCR viral load of 25,361 IU/mL, Epstein-Barr virus (EBV) IgM was negative with positive IgG. HIV, hepatitis panel, and viral respiratory panel were all negative. Peripheral blood differential showed no left shift or blasts, arguing against an underlying lymphoproliferative or myeloproliferative disorder.

Table 1: Diagnostic Laboratory Findings

Test	Result	Reference Range
Complete Blood Count		
White blood cell count	$13.2 \times 10^9/L \uparrow$	$4.5-11.0 \times 10^9/L$
Differential	No left shift or blasts	—
Platelet count	$167 \times 10^9/L$	$150-450 \times 10^9/L$
Comprehensive Metabolic Panel		
Sodium	129 mmol/L $\downarrow$	136–145 mmol/L
Creatinine (admission)	1.9 mg/dL $\uparrow$	0.7–1.3 mg/dL
Creatinine (baseline)	1.5 mg/dL	—
Alkaline phosphatase	444 U/L $\uparrow$	44–147 U/L
AST	142 U/L $\uparrow$	10–40 U/L
ALT	231 U/L $\uparrow$	7–56 U/L
Inflammatory / Tumor Markers		
LDH	441 U/L $\uparrow$	140–280 U/L
Uric acid	8.8 mg/dL $\uparrow$	3.5–7.2 mg/dL
Viral Serologies, PCR, and Transplant Status		
CMV IgM	Positive	Negative
CMV IgG	Positive	Negative
CMV PCR, peripheral blood (viral load)	25,361 IU/mL	Undetectable
Donor/recipient CMV serostatus (D/R)	D-/R-	—
Post-transplant CMV prophylaxis	None	—
EBV IgM	Negative	Negative
EBV IgG	Positive	Negative
HIV	Negative	Negative
Hepatitis panel	Negative	Negative
Viral respiratory panel	Negative	Negative

Hypercoagulable Workup		
Antiphospholipid panel	Unremarkable	—
Factor V Leiden mutation	Negative	Negative
Prothrombin gene mutation	Negative	Negative

Cardiac event monitor	No evidence of atrial fibrillation
-----------------------	------------------------------------

Antiphospholipid antibody panel, Factor V Leiden mutation, and prothrombin gene mutation panel were unremarkable, excluding the most common hereditary and acquired hypercoagulable states.

Lactate dehydrogenase (LDH) was elevated at 441 U/L and uric acid at 8.8 mg/dL.

Table 2: Imaging and Cardiac Studies

Study	Finding
CT abdomen and pelvis	Mildly enlarged spleen with 3 splenic hypodensities; initially interpreted as grade III splenic injury without active hemorrhage; transplanted kidney in right lower quadrant without hydronephrosis; incidental 4.4-cm exophytic lesion at anterior left iliac crest consistent with osteochondroma
Liver ultrasound	Patent splenic vein; no splenic lesions on prior abdominal ultrasound
MRI abdomen	Multiple irregular peripheral splenic lesions; isointense T1 signal with peripheral T2 hypointensity and mild central T2 hyperintensity; no post-contrast enhancement; no abnormal diffusion restriction — most consistent with subacute splenic infarcts
Chest radiograph	No evidence of active pulmonary disease
Transthoracic echocardiogram	No intracardiac thrombus identified

The differential diagnosis included infectious etiologies (CMV, EBV, infective endocarditis), hematologic malignancy (lymphoma, leukemia, myeloproliferative disorder), inherited hypercoagulability, cardioembolic disease, and sickle cell disease. Given confirmed CMV viremia with a high viral load, subacute splenic infarcts on imaging without an identifiable arterial, venous-structural, cardioembolic, or hereditary hypercoagulable cause, and an immunocompromised state, the findings were attributed to CMV-associated hypercoagulopathy as a diagnosis of exclusion.

Given the competing risk of graft rejection, immunosuppressive therapy was continued rather than reduced, per nephrology's recommendation. Short-term anticoagulation was initiated with intravenous heparin, and the patient was discharged on a one-month course of apixaban and valganciclovir with outpatient follow-up arranged with hematology, cardiology, and infectious disease. The patient was subsequently lost to follow-up, precluding repeat imaging or CMV viral load monitoring to confirm resolution of splenic infarcts or viremia following antiviral therapy.

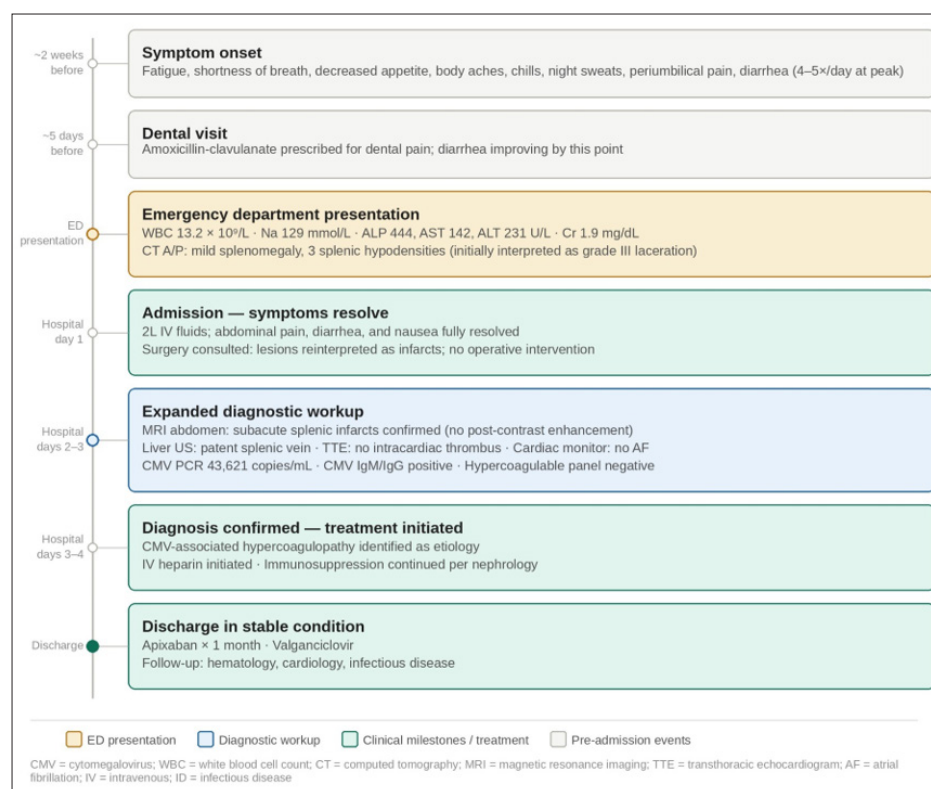


Figure 3: Clinical Timeline of Presentation, Diagnostic Workup, and Management. Chronological summary of key events from symptom onset through hospital discharge in a 34-year-old immunocompromised renal transplant recipient with cytomegalovirus-

associated splenic infarction.

Amber node indicates emergency department presentation; blue node indicates expanded diagnostic workup; teal nodes indicate clinical milestones and treatment decisions. CMV = cytomegalovirus; WBC = white blood cell count; CT = computed tomography; MRI = magnetic resonance imaging; TTE = transthoracic echocardiogram; AF = atrial fibrillation; IV = intravenous; ID = infectious disease.

Discussion

The clinical presentation of CMV lies on a spectrum ranging from asymptomatic infection to a mild mononucleosis-like syndrome to overt visceral organ disease, with CMV disease reserved for infection accompanied by attributable clinical or histopathologic findings [5].

The CMV viral load is significantly greater in patients with viremia-induced visceral organ disease compared with viremia alone [6]. An adequate immune response can limit viral replication, preventing viremia significant enough to cause end-organ disease for that reason, visceral organ disease is most common in immunocompromised individuals [5]. Even when immunocompetent patients develop visceral organ disease, they still present with lower viral loads compared with immunocompromised patients who have similar manifestations [7]. Our patient, a young, immunocompromised individual with splenic infarction, had a peripheral blood CMV viral load of 43,621 copies/mL (25,361 IU/mL); existing literature describing immunocompetent individuals with CMV-associated splenic infarction reports viral loads between 900 and 3,100 copies/mL [8,9]. While this comparison does not establish causation, the substantially higher viral burden in our patient is consistent with a dose-dependent relationship between CMV replication and end-organ thrombotic disease.

Thrombotic manifestations of CMV are believed to be largely driven by a virally induced hypercoagulable state. Signs of endothelial cell activation have been demonstrated early in CMV infection, evidenced by leukocyte adhesion and endothelial ballooning [10].

Increased expression of von Willebrand factor, ICAM-1, and VCAM-1 in virally infected cells promotes platelet aggregation and adhesion, contributing to thrombus formation [11,12]. Other proposed mechanisms include activation of factor X, increased circulating levels of factor VII, and transient appearance of antiphospholipid antibodies [12].

While the predominant mechanism may vary by clinical scenario, existing evidence supports CMV infection as a pathologic trigger for thrombosis and subsequent infarction of visceral organs.

In our patient, this diagnosis was reached by exclusion: a cardioembolic source was excluded by transthoracic echocardiogram and cardiac event monitoring; a venous-structural etiology was excluded by a patent splenic vein on ultrasound; and the most common hereditary and acquired hypercoagulable states were excluded by negative Factor V Leiden mutation, prothrombin gene mutation, and antiphospholipid antibody panel results. An underlying hematologic malignancy was not

supported by peripheral blood findings, though bone marrow evaluation was not performed. This exclusionary approach is consistent with the recommendation that CMV-associated thrombosis be considered only after other, more common causes have been ruled out. We were unable to confirm tissue-invasive disease, as no splenic tissue was sampled for immunostaining or tissue PCR; we therefore describe this case as CMV viremia with associated splenic infarction rather than confirmed tissue-invasive disease.

Notably, immunosuppressive therapy was continued rather than reduced during treatment, per nephrology's assessment of allograft rejection risk. Current guidance for CMV disease in solid organ transplant recipients generally recommends reduction of immunosuppression in addition to antiviral therapy; our patient's management therefore represents a deviation from this standard, reflecting a clinical judgment that balanced infection control against graft preservation. The impact of this trade-off on the course of CMV-associated thrombosis could not be assessed, given the patient's subsequent loss to follow-up.

CMV infection should be considered in patients presenting with thrombosis without an obvious cause or identifiable risk factor. While several case reports describe infarcts in immunocompetent patients, there is limited discussion of splenic infarction specifically in immunocompromised patients in recent literature. Splenic infarction in CMV patients is more frequently documented among immunocompetent patients (Bertoni 2018); we propose this reflects ascertainment bias rather than a true difference in incidence. Immunocompromised CMV patients are typically monitored closely for more classic end-organ disease, such as colitis, pneumonitis, or retinitis, and a thrombotic event in this population may be attributed to other causes common after transplantation (e.g., catheter-related thrombosis, immobility) rather than specifically investigated as CMV-related. Immunocompetent patients, by contrast, may have more clinically apparent and therefore more reportable thrombotic manifestations, precisely because thrombosis is unexpected in that population and prompts more thorough investigation. For these reasons, we suspect the true incidence of thrombosis among CMV patients; including immunocompromised patients, is likely higher than published literature suggests [13].

This case has several limitations. Definitive proof of tissue-invasive CMV disease was not obtained, as no splenic tissue was available for histopathologic confirmation. Whether the infarcts were arterial/embolic or venous in origin could not be definitively established on imaging. Malignancy was not excluded with certainty beyond peripheral blood findings and short-term clinical follow-up.

Most importantly, the patient was lost to follow-up before repeat imaging or CMV viral load monitoring could confirm resolution of both splenic infarcts and viremia following antiviral therapy, precluding assessment of the full natural history and treatment response in this case.

References

1. Zuhair M, Smit GSA, Wallis G, Jabbar F, Smith C, et al. Estimation of the worldwide seroprevalence of cytomegalovirus: A systematic review and meta-analysis.

- Reviews in Medical Virology. 2019. 29: e2034.
2. Kana T, Mehjabeen S, Kawamj A, Patel N, Shamim Z. Splenic infarction in acute cytomegalovirus and Epstein-Barr virus concomitant infection. *Cureus*. 2023. 15: e46235.
 3. Roy S, Kaul A, Bhadhuria DS. Clinico-epidemiological characteristics of early- versus late-onset cytomegalovirus disease among renal transplant recipients. *Transplant Immunology*. 2024. 84: 102040.
 4. Bertoni M, Squizzato A, Foretic M, Zanieri S, Di Natale ME. Cytomegalovirus-associated splanchnic vein thrombosis in immunocompetent patients: A systematic review. *Thrombosis Research*. 2018. 168: 104-113.
 5. Griffiths P, Reeves M. Pathogenesis of human cytomegalovirus in the immunocompromised host. *Nature Reviews Microbiology*. 2021. 19: 759-773.
 6. Saltzman RL, Quirk MR, Jordan MC. High levels of circulating cytomegalovirus DNA reflect visceral organ disease in viremic immunosuppressed patients other than marrow recipients. *Journal of Clinical Investigation*. 1992. 90: 1832-1838.
 7. Chaemsupaphan T, Limsrivilai J, Thongdee C. Patient characteristics, clinical manifestations, prognosis, and factors associated with gastrointestinal cytomegalovirus infection in immunocompetent patients. *BMC Gastroenterology*. 2020. 20: 22.
 8. O'Brien, J, Feller E, Shah S. Splenic infarction due to CMV infection. *The American Journal of Gastroenterology*. 2010. 105: 709.
 9. Pakkiyaretnam, M. Cytomegalovirus-associated splenic infarction in a young female patient. *Cureus*. 2020.
 10. Persoons MC, Stals FS, van Dam-Mieras MC, Bruggeman CA. Multiple organ involvement during experimental cytomegalovirus infection is associated with disseminated vascular pathology. *The Journal of Pathology*. 1998. 184: 103-109.
 11. Rahbar A, Söderberg-Nauclér C. Human cytomegalovirus infection of endothelial cells triggers platelet adhesion and aggregation. *Journal of Virology*. 2005. 79: 2211-2220.
 12. Sherman S, Eytan O, Justo D. Thrombosis associated with acute cytomegalovirus infection: A narrative review. *Archives of Medical Science*. 2014. 10: 1186-1190.
 13. Justo D, Finn T, Atzmony L, Guy N, Steinvil A. Thrombosis associated with acute cytomegalovirus infection: a meta-analysis. *European Journal of Internal Medicine*. 2011. 22: 195-199.