



CYTOMEGALOVIRUS-ASSOCIATED MULTIFOCAL ARTERIAL THROMBOEMBOLISM IN AN IMMUNOCOMPETENT PATIENT: A CASE REPORT AND REVIEW OF THE LITERATURE

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Department of Virology, Infectious and Tropical Diseases, Mohammed V Military Teaching Hospital, Rabat, Morocco. DOI: <https://doi.org/10.5281/zenodo.23117193>



How to cite this Article: Youssef Aadi^{1*}, A'mr Nachite¹, Youssef Sefsafi¹, Yassine Benaissi¹, Ahmed Reggad¹, Khalid Ennibi¹ (2026). Cytomegalovirus-Associated Multifocal Arterial Thromboembolism In An Immunocompetent Patient: A Case Report And Review Of The Literature. European Journal of Biomedical and Pharmaceutical Sciences, 13(10), 354-359. This work is licensed under Creative Commons Attribution 4.0 International license.

Article Received on 05/09/2026

Article Revised on 25/09/2026

Article Published on 01/10/2026

ABSTRACT

Introduction: Arterial thromboembolic complications of cytomegalovirus (CMV) infection are exceedingly rare, accounting for fewer than five of the ~113 cases documented in the world literature. We report an extensive case of CMV-associated multifocal arterial thromboembolism combined with CMV encephalitis in an immunocompetent patient. **Case Presentation:** A 46-year-old woman with insulin-treated type 2 diabetes (HbA1c 7.6%) presented with sequential thromboses of the left anterior descending coronary artery (03/01/2017), both renal arteries, the splenic, left iliac, and left popliteal arteries, a right sylvian ischemic stroke, and periventricular white matter changes consistent with CMV encephalitis. CMV IgM and IgG were positive with a plasma viral load of 4.01 log copies/mL. Concurrent bacteraemia was ruled out, and infective endocarditis was formally excluded by Modified Duke's criteria. Skin biopsy demonstrated leukocytoclastic vasculitis. Treatment included renal-function-adapted intravenous ganciclovir for six weeks, corticosteroids, anticoagulation, and clopidogrel. Follow-up at six months showed renal stabilization (creatinine 21 mg/L) and recovery of left ventricular ejection fraction from 40% to 54%. **Conclusion:** This case illustrates CMV's capacity to induce severe, multifocal arterial thromboembolism in immunocompetent patients. CMV serology and PCR should be systematically included in the workup of any unexplained febrile arterial thrombosis.

KEYWORDS: cytomegalovirus; arterial thrombosis; encephalitis; thromboinflammation; immunocompetent; ganciclovir.

1. INTRODUCTION

Cytomegalovirus (CMV), a member of the Herpesviridae family (HHV-5), is a ubiquitous pathogen with a global seroprevalence estimated at 83%, ranging from 39% to 96% across countries.^[1] In immunocompetent adults, primary infection is most often asymptomatic or presents as a self-limiting mononucleosis-like illness. In immunocompromised hosts, CMV can cause life-threatening end-organ disease.

The association between CMV and thromboembolism is rare. A 2011 meta-analysis identified 97 cases worldwide, updated to ~113 in 2013.^[2,3] The predominant pattern is venous; arterial thromboses represent only four of the reported cases.^[2] CMV is also the only human herpesvirus whose plasma viral load is

significantly elevated in patients with acute coronary syndromes, correlating positively with systemic inflammation markers.^[4,5]

We report a case of simultaneous multifocal arterial thromboembolism with CMV encephalitis in an immunocompetent patient with insulin-treated type 2 diabetes, with comprehensive longitudinal follow-up, and review the literature on pathophysiology, diagnosis, and management.

Ethics statement: Written informed consent was obtained from the patient for publication of this case report. This work was conducted in accordance with the Declaration of Helsinki.

2. CASE PRESENTATION

2.1. Patient history and risk factors

The patient was a 46-year-old married woman with two children. She had been followed for type 2 diabetes mellitus for ten years, initially diagnosed in the context of gestational diabetes, and managed with insulin therapy (Lantus 6 UI nightly + Actrapid 11 UI/day in three divided doses). Her HbA1c at presentation was 7.6%. She had a documented allergy to aspirin. She had no hypertension, dyslipidemia, tobacco use, obesity, or personal or family history of thrombosis or thrombophilia, and was not taking hormonal contraception.

2.2. Chronological timeline of events

03/01/2017 — Index coronary event (external clinic)

The patient presented with an acute myocardial infarction secondary to thrombosis of the left anterior descending (LAD) artery, requiring coronary artery bypass grafting, stenting of the LAD, and aspiration of a left ventricular thrombus. Post-operative echocardiography demonstrated moderate left ventricular systolic dysfunction with an ejection fraction of 40%.

30/01/2017 — Acute kidney injury and nephrology admission

Progressive oliguria necessitated transfer to the nephrology ward of our institution, where hemodialysis was initiated. Laboratory workup at this stage showed: creatinine 108 mg/L, urea 2.42 g/L, hyperuricemia (87.5 mg/L), severe hyponatremia (119 meq/L), hypocalcemia, hyperphosphatemia, and 24-hour proteinuria of 0.814 g/day. CT angiography of the aorta and lower limbs revealed bilateral filiforme renal arteries with renal cortical infarctions, splenic artery thrombosis with splenic infarction, left internal iliac artery thrombosis, and left popliteal artery thrombosis. A bone infarct was also noted on the right fibula, confirmed on bone scintigraphy performed on 27/02/2017. Patient presented fever during hospitalization, motivating her transfer to infectious diseases department.

01/02/2017 — Transfer to Infectious Diseases

On admission to our department, the patient was acutely unwell: hypotensive (BP 87/58 mmHg), tachycardic (HR 101 bpm), tachypneic (respiratory rate 26/min), and oxygen saturation at 96% on room air. She was afebrile at 37.5°C at the time of examination (antipyretics administered). Cardiovascular examination revealed no murmur; heart sounds B1 and B2 were well perceived at all four foci. Peripheral pulses were present but diminished. Cutaneous examination was notable for multiple erythematous, indurated, deeply infiltrated inflammatory nodules over the right lower limb; extensive sacral pressure ulcer extending to the left buttock and intergluteal fold; squamous lesions of the plantar surface of the feet; and necrotizing ischemic lesions of the right hallux and left heel. There were no signs of phlebitis, lymphadenopathy, hepatomegaly, or splenomegaly.

14/02/2017 — CMV diagnosis and treatment initiation

CMV serology returned positive for both IgM and IgG (IgG titre: 7,250 AU/mL). Quantitative plasma CMV PCR confirmed active viral replication at 102,580 copies/mL (4.01 log copies/mL). Peripheral blood smear documented enlarged lymphocytes with abundant basophilic cytoplasm (atypical mononuclear cells), consistent with a mononucleosis syndrome. Ganciclovir was initiated on 14/02/2017 at a dose adapted to the patient's renal function (1.25 mg/kg/day), with the dose subsequently divided into two daily administrations.

15/02/2017 — Brain MRI

Brain MRI with angiography performed on 15/02/2017 demonstrated a right sylvian ischemic stroke with periventricular white matter signal abnormalities (T2 and diffusion-weighted hyperintensities), suggestive of CMV encephalitis. Duplex ultrasound of the supra-aortic trunks was normal.

2.3. Investigations

Laboratory findings and serial evolution

Initial blood tests (01/02/2017) showed CRP 72.6 mg/L, white blood cell count 17,300/mm³ (lymphocytes 6,750/mm³; neutrophils 9,000/mm³), hemoglobin 9 g/dL, and platelets 420,000/mm³. By 08/02/2017, leukocytosis had risen to 23,400/mm³ with increasing lymphocytosis (8,100/mm³), while CRP trended downward at 44.2 mg/L. By 21/02/2017, white cells were 8,700/mm³ with a marked lymphocytic predominance (lymphocytes 6,080/mm³; neutrophils 2,410/mm³), CRP 58 mg/L. Albumin was consistently low at 26–27 g/L throughout the admission. Serum protein electrophoresis demonstrated a polyclonal hypergammaglobulinemia with elevated alpha-1 and alpha-2 fractions, reflecting the ongoing systemic inflammatory and infectious process. Renal function progressively improved: creatinine 53 mg/L (08/02) → 40 mg/L (09/02) → 28 mg/L (27/02) with GFR rising from 9 to 18 mL/min.

Cardiac workup

ECG showed sinus rhythm. A 24-hour Holter ECG excluded atrial fibrillation as a cardioembolic source. Transthoracic echocardiography (31/01/2017) showed features of ischemic cardiomyopathy with moderate left ventricular systolic dysfunction (EF 40%), no suspected mobile element on the ventricular aspect of the mitral valve and left atrial dilatation without thrombus. Transesophageal echocardiography (01/02/2017) demonstrated septo-apical akinesia without any vegetation.

Microbiological workup

Blood cultures drawn on admission to our department returned negative. Infective endocarditis was formally excluded by Modified Duke's criteria, yielding a rejected classification.

Urinalysis identified *Candida non-albicans* candiduria on two consecutive urine cultures, treated with fluconazole (400 mg loading dose, then 200 mg three times weekly). Serum hepatic enzymes were normal on 09/02/2017 (no cytotoxicity or cholestasis). Stool culture was sterile. Serological screening for HIV-1/2, HBV (HBsAg), HCV and syphilis (TPHA/VDRL) were negative.

CMV serology was positive as mentioned before (IgG = 7250 and IgM positive). Quantitative plasma CMV PCR confirmed active viral replication at 102,580 copies/mL (4.01 log copies/mL).

Thrombophilia and autoimmune workup

A comprehensive etiological screen returned entirely negative: antinuclear antibodies, ANCA (anti-myeloperoxidase and anti-proteinase 3), anticardiolipin antibodies, anti- β 2-glycoprotein I antibodies (negative on two occasions twelve weeks apart), lupus anticoagulant, complement C3 and C4 (normal at 1.02 and 0.2 g/L respectively), cryoglobulinemia, and JAK2 V617F mutation. Protein C, protein S, and antithrombin III levels were normal. CH50 was 61.7% (normal).

Factor V Leiden, prothrombin G20210A, homocysteine, and MTHFR testing were not performed due to unavailability at our institution and financial constraints. These represent acknowledged limitations of the workup.

CMV end-organ disease screening

Fundoscopy and slit-lamp examination (08/02/2017 and 10/02/2017) were unremarkable, with no evidence of chorioretinitis or diabetic retinopathy. There was no clinical or radiological evidence of gastrointestinal or pulmonary CMV involvement.

Brain MRI (15/02/2017) revealed a right sylvian ischemic stroke with periventricular T2 and diffusion-weighted white matter signal abnormalities consistent with CMV encephalitis. Duplex ultrasound of the supra-aortic trunks was normal. CSF analysis including CMV PCR was not performed, representing an acknowledged diagnostic limitation.

Skin biopsy

Biopsy of a right lower limb inflammatory nodule demonstrated leukocytoclastic vasculitis of small vessels. Immunohistochemical staining for CMV was not performed, as IHC is not available at our institution.

Immunological workup

Basic immunological assessment was performed: normal C3, C4, CH50 (61.7%), negative HIV serology and hypergammaglobulinemia with elevated alpha-1 and alpha-2 globulins on protein electrophoresis. Lymphocyte subset analysis (CD4/CD8 counts), immunoglobulin levels, and lymphocyte proliferation studies were requested and pending at the time of initial discharge. Advanced immunological testing was not subsequently completed. The patient's complete clinical

recovery without recurrence and without ongoing immunosuppression makes a significant underlying primary immunodeficiency unlikely.

2.4. Anticoagulation management

Anticoagulation was initiated with acenocoumarol (2 mg/day) and low-molecular-weight heparin (enoxaparin 0.6 cc/12h). On 13/02/2017, the patient developed an episode of epistaxis; INR was found at 8.82 (TP 8%), requiring temporary suspension of acenocoumarol, which was reintroduced at a reduced dose (1 mg/day) with INR normalization within 48 hours. Enoxaparin was subsequently discontinued once therapeutic INR was re-achieved. INR was maintained in the therapeutic range (target 2–3) for the duration of hospitalization. Clopidogrel 75 mg/day was prescribed as antiplatelet therapy instead of aspirin because of patient's known history of allergy.

2.5. Diagnosis, treatment, and outcome

The final diagnosis retained was CMV-associated multifocal arterial thromboembolism with CMV encephalitis, after exclusion of competing etiologies and formal rejection of infective endocarditis by Modified Duke's criteria. Treatment comprised.

- Ganciclovir IV at 1.25 mg/kg/day (renal-function-adapted), initiated 14/02/2017, administered over six weeks;
- Corticosteroids at 1 mg/kg/day, maintained at full dose for two months then tapered;
- Anticoagulation with acenocoumarol (target INR 2–3);
- Clopidogrel 75 mg/day. Concomitant medications included: IEC, beta-blocker, statin, diuretic (furosemide), and insulin therapy.
- Fluconazole 400mg the first day, followed by 200 mg/day for 3 weeks was administered for *Candida non-albicans* candiduria.
- The sacral pressure ulcer was managed, with assistance of plastic surgery consultation, with anti-decubitus mattress, and wound care.

Clinical course was favorable: ischemic phenomena progressively resolved, inflammatory markers normalized, hemodialysis was discontinued on 07/02/2017 after seven sessions, and renal function stabilized with a serum creatinine of 21 mg/L and GFR ~25 mL/min at discharge. The antiviral treatment was discontinued at six weeks; the end-of-treatment plasma CMV PCR was not repeated due to logistical constraints.

A significant late complication arose: the patient developed a septic arthritis of the left knee with bone infarct, confirmed on X-ray, bone scintigraphy (27/02/2017), and MRI. Synovial fluid culture was positive for *Pseudomonas aeruginosa*, treated with ciprofloxacin 750 mg orally twice a day for three months. This complication occurred in the context of knee osteoarthritis with bone infarction and prolonged immobility related to the overall disease course.

At six-month follow-up (04/07/2017), the patient was clinically well and hemodynamically stable, ambulating with crutches due to residual left knee impairment. Laboratory results showed CRP 4.2 mg/L, creatinine 21 mg/L, GFR ~25 mL/min, hemoglobin 9.2 g/dL, and fasting glycemia 0.70 g/L. Follow-up echocardiography demonstrated remarkable recovery of left ventricular ejection fraction from 40% to 54%. Corticosteroids were tapered to 20 mg/day at this visit.

3. DISCUSSION

3.1. Epidemiology of CMV-associated thrombosis in the literature

CMV-associated thrombosis is rare. The 2011 meta-analysis by Justo et al. identified 97 cases, updated to ~113 by 2013. Two-thirds occurred in immunocompetent patients. Arterial thromboses accounted for only four cases in the entire series.^[2,3]

A community cohort of >90,000 patients confirmed an association with venous thromboembolism (OR 2.49; 95% CI 1.53–4.06; $p < 0.001$) but found no significant association with arterial thrombosis.^[6]

The retrospective series by Lecomte et al. described only two arterial events among ten CMV-related thrombosis.^[7]

The simultaneous involvement of five arterial territories with cerebral complications and a confirmed ischemic bone infarct in our patient places it among the most extensive descriptions of CMV-associated arterial thromboembolism in the literature.

3.2. Differential diagnosis: CMV versus infective endocarditis

The central diagnostic challenge was distinguishing CMV-associated arterial thromboembolism from septic embolism secondary to infective endocarditis. Four converging arguments support the rejection of IE.

- (1) the chronological sequence — the coronary and renal thromboses occurred in January 2017, at least three weeks before the fever was identified on 01/02/2017, and blood cultures drawn at the time of established thromboses were sterile.
- (2) formal exclusion by Modified Duke's criteria.
- (3) clinical recovery occurred specifically under ganciclovir with arrest of vascular events.
- (4) the bilateral and simultaneous nature of the renal artery thromboses is mechanistically inconsistent with sequential septic embolization from a single cardiac source: Septic embolization from a cardiac source (such as infective endocarditis) involves the periodic shedding of bacterial vegetations into the systemic circulation. These events are stochastic and happen over a timeline, typically resulting in sequential, asymmetrical, and multi-organ infarctions rather than an identical, simultaneous occlusion of both main renal arteries.

3.4. Pathophysiology: thromboinflammation driven by CMV

Infections are recognized as potent triggers of thromboembolic events, with a risk magnitude comparable to major surgery, a phenomenon termed “thromboinflammation”.^[8] CMV drives this process through four converging mechanisms.

- (1) the research by Bentz et al. show that CMV infection results in increased endothelial cells proliferation, motility, and capillary tube formation, resulting in dysregulated angiogenesis and driving neovascularization as observed in atherosclerotic disease.^[9]
- (2) Dam-Mieras et al. showed that CMV infection caused the appearance of procoagulant activity on the endothelial cells.^[10]
- (3) transient induction of aPL through molecular mimicry.^[11,12]
- (4) stimulation of vascular smooth muscle cell proliferation.^[5,13]

In our patient, the leukocytoclastic vasculitis on skin biopsy provides in vivo histological evidence of vascular inflammation. Although CMV immunohistochemistry was not performed (unavailable at our institution), the complete absence of serological or clinical evidence of primary systemic vasculitis, combined with the high viral load and documented CMV infection, supports CMV-associated vasculopathy.

The addition of corticosteroids at 1 mg/kg/day to the antiviral and anticoagulant regimen was motivated by the vasculitic and encephalitic components of the presentation. While evidence-based guidelines for corticosteroid use in CMV-associated thromboinflammation do not exist, their use in CMV vasculitis has been described in isolated reports, and the favorable clinical evolution in our patient supports the rationale of combining immunomodulatory therapy with direct antiviral treatment in severe cases.

3.5. CMV and acute coronary syndromes

Among all eight human herpesviruses, only CMV displays a significantly elevated plasma viral load in acute coronary syndrome (ACS) patients vs. healthy controls ($p < 0.05$), correlating positively with high-sensitivity CRP.^[4] A 2018 systematic review confirmed CMV's role in atherosclerotic coronary lesions, including direct detection of viral DNA in atheromatous plaques.^[13] A parallel phenomenon is observed in cardiac transplantation, where CMV infection accelerates allograft vasculopathy.^[14,15] In our patient, the remarkable recovery of LVEF from 40% to 54% at six months, despite the severity of the initial ischemic cardiomyopathy, may in part reflect resolution of the CMV-driven inflammatory cardiomyopathic component alongside the structural post-infarct recovery.

3.6. CMV neurological involvement in immunocompetent patients

CMV encephalitis in immunocompetent hosts is exceedingly rare.^[16–18] In our patient, the neurological involvement was a right sylvian ischemic stroke of embolic origin combined with periventricular T2/DWI white matter signal abnormalities consistent with direct CMV encephalitis. This dual neurovirulence mechanism (ischemic vascular injury and direct viral parenchymal invasion) has rarely been described.^[19–21] CSF CMV PCR, the gold standard for confirming direct cerebral involvement, was not performed in our case, representing an acknowledged limitation.

3.7. Diagnosis: distinguishing primary infection from reactivation

Active CMV infection was confirmed by the combination of positive CMV IgM, positive CMV IgG (titre 7,250 AU/mL), and a plasma viral load of 4.01 log copies/mL. The simultaneous positivity of IgM and IgG is consistent with either a recent primary infection with early IgG seroconversion, or reactivation of a latent infection. IgG avidity testing, which would have distinguished these scenarios (low avidity = primary infection; high avidity = reactivation), was not available at our institution, representing a diagnostic limitation. The magnitude of viremia (>4 log copies/mL), the mononucleosis syndrome with documented atypical lymphocytosis, and the pattern of multiorgan involvement are more consistent with a significant primary infection than with incidental critical illness-related CMV reactivation, which typically produces low-level viremia without overt disease.

3.8. Treatment and management considerations

Three distinctive features of our therapeutic management deserve emphasis.

First, ganciclovir was dosed at 1.25 mg/kg/day, adapted to the patient's significantly impaired renal function (GFR ~9–12 mL/min at treatment initiation), rather than the standard 5 mg/kg/12h used in patients with normal renal function. This renal-adapted dosing is important as standard ganciclovir doses carry substantial nephrotoxic risk in patients with pre-existing acute kidney injury.

Second, corticosteroids at 1 mg/kg/day were added given the vasculitic and encephalitic presentation, maintained at full dose for two months then tapered to 20 mg/day at six months.

Third, an anticoagulation complication occurred: a significant over-anticoagulation episode (INR 8.82, epistaxis) on 13/02/2017, requiring temporary suspension and dose readjustment, highlighting the complexity of AVK management in this polyathological context.

Secondary prophylaxis with oral valganciclovir was not instituted: current evidence-based guidelines do not recommend secondary antiviral prophylaxis in

immunocompetent patients following CMV disease in the absence of ongoing immunosuppression.

4. CONCLUSION

This case represents one of the most extensively documented examples of CMV-associated multifocal arterial thromboembolism in an immunocompetent patient in the published literature, with a complete longitudinal clinical record spanning the index event in January 2017 through six-month follow-up in July 2017.

CMV should be systematically included in the differential diagnosis of any unexplained febrile multifocal arterial thrombosis, even in immunocompetent patients. CMV serology and quantitative PCR, combined with rigorous exclusion of competing etiologies using validated criteria such as Modified Duke's criteria for endocarditis, should be incorporated into the standard workup. Skin biopsy of associated cutaneous lesions and, where available, CMV immunohistochemistry should not be overlooked.

Prospective multicenter studies are needed to clarify the optimal antiviral dosing, the role of corticosteroids, the duration of anticoagulation, and the long-term vascular and immunological outcomes of CMV-associated arterial thrombosis in immunocompetent patients.

CONFLICTS OF INTEREST

The authors declare no conflicts of interest.

AUTHOR CONTRIBUTIONS

Y. Aadi: Conceptualization, investigation, writing original draft and editing.

A. Nachite, Y. Sefsafi, I. Roufik, F. Amraoui, Y. Benaissi, Y. Aouni: Clinical management, data collection, review.

A. Reggad, M. Qatni, K. Ennibi: Supervision, critical revision, final approval.

All authors read and approved the final version of the manuscript.

ACKNOWLEDGEMENTS

The authors thank the patient for her consent and the multidisciplinary team at Mohammed V Military Teaching Hospital for their collaborative care.

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