

Delayed diagnosis of cytomegalovirus in a patient with splenectomy: A case report

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Abstract

Human Cytomegalovirus (CMV) is a common flu-like virus that is largely benign but has the potential to cause serious complications in certain patient groups.

We present the case of 43-year-old Male with a previous history of splenectomy for Immune Thrombocytopenia, who presented with non-specific symptoms of headache and recurrent fevers. Eventual detection of acute CMV infection was found after ongoing fevers and a significant leucocytosis.

CMV infection can present atypically in patients with splenectomy, increasing the risk of it being overlooked as a differential and leading to delays in diagnosis and treatment.

There is a lack of literature exploring the role of asplenia in viral infections and their complications.

Keywords: Cytomegalovirus; Reactive leucocytosis; Asplenia; Immunoglobulin G; Immunoglobulin M.

Abbreviations: CMV: Cytomegalovirus; CRP: C-Reactive Protein; LP: Lumbar Puncture; CSF: Cerebrospinal Fluid; IgG: Immunoglobulin G; IgM: Immunoglobulin M.

Introduction

Human Cytomegalovirus (CMV) is a common virus belonging to the *Herpesviridae* family, most commonly presenting asymptotically or with flu-like symptoms in the immunocompetent individual [1]. In those with a compromised immune system, the virus can cause direct viral-mediated damage to numerous organ systems. Within this subgroup, complications commonly include CMV oesophagitis and CMV colitis.

Having an estimated seroprevalence of 24-100% globally, CMV can be transmitted through exchange of bodily fluids, solid organ transplantation, or hematopoietic stem cell transplantation [1]. Active replication

of the virus within tissue, blood, or other body fluids defines a CMV infection. The virus establishes lifelong latency after acute - or primary - infection, becoming dormant within monocytes [2] and can reactivate in significant immunosuppression.

Case Presentation

A 43-year-old man with background of previous splenectomy for Immune Thrombocytopenia in his 20s, was referred to medical Same Day Emergency Care from his GP with a 24-hr history of «worst ever» headache. He had been feeling unwell with fevers for the past 3 days. There was mild associated nausea but no rash, neck stiffness, photophobia, new neurology, or other specific infective symptoms. He denied any recent travel, ingestion of uncooked foods, or unwell contacts.

His regular medications included amoxicillin (prophylactic antibiotic post splenectomy), indapamide (hyperkalaemia), and sertraline (anxiety). He reported formerly smoking, drinking alcohol rarely, and denied any recreational drug use.

On arrival, his National Early Warning Score (NEWS) was 4 (mild tachycardia at 112, tachypnoea at 23) with a low-grade pyrexia of 37.8°C. Examination, including neurology, was unremarkable.

Table 1: Inpatient investigation results.

Test	Reference range	Day 1 of admission	Day 3 of admission	Day 6 of admission	Day 15 of admission	On Discharge
Sodium (mmol/L)	133 - 146	134		131 (L)	132 (L)	136
Potassium (mmol/L)	3.5 - 5.3	4.0		3.7	4.1	5.2
eGFR (mL/min/1.73m ²)	>60	62		73	69	66
CRP (mg/L)	<5	31 (H)		68	33	6 (H)
Haemoglobin (g/L)	115 - 165	183 (H)		174 (H)	155	157
Platelets (10 ⁹ /L)	140 - 400	123 (L)		67 (L)	203	380
WBC (10 ⁹ /L)	3.6 - 11.0	9.0		16.1 (H)	41.0 (H)	22.3 (H)
Neutrophils (10 ⁹ /L)	1.80 - 7.50	5.47		7.75 (H)	10.55 (H)	4.33
Lymphocytes (10 ⁹ /L)	1.00 - 4.00	2.29		6.84 (H)	27.68 (H)	16.34 (H)
Monocytes (10 ⁹ /L)	0.20 - 0.80	0.72		1.14 (H)	2.23 (H)	1.10 (H)
Eosinophils (10 ⁹ /L)	0.10 - 0.40	0.39		0.24	0.25	0.34
Basophils (10 ⁹ /L)	0.02 - 0.10	0.10		0.13 (H)	0.29 (H)	0.23 (H)
ALP (U/L)	30 - 130	169 (H)		224 (H)		203 (H)
ALT (U/L)	<35	96 (H)		208 (H)		77 (H)
Bilirubin (umol/L)	<21	11		12		10
Blood Cultures		Negative				
CSF			Clear, colourless			
CSF RBC Count Bottle 3 (10 ⁶ /L)			355			
CSF Culture			No growth			
Stool C. diff				Not detected		
Ferritin (ug/L)	11 - 310				2942 (H)	

eGFR: Estimated Glomerular Filtration Rate; CRP: C-Reactive Protein; WBC: White Blood Cells; ALP: Alkanine Phosphatase; ALT: Alanine Aminotransferase; CSF: Cerebrospinal Fluid; RBC: Red Blood Cell.

Table 1 shows his initial and subsequent investigation results. A CT head was performed with normal findings.

In light of reassuring initial investigations, a plan was made to discharge the patient.

However, he continued to consistently spike a temperature $>38^{\circ}\text{C}$, despite regular paracetamol and IV antibiotics. He continued to describe flu-like symptoms including generalised myalgia with ongoing severe headache. Over the next few days, he developed abdominal bloating, discomfort, and loose stools.

On day 6 of admission, with ongoing pyrexia, differentials included viral infection including blood borne viruses, malignant lymphoproliferative disorder, and hemophagocytic lymphohistiocytosis. CT abdo-pelvis showed gastrohepatic lymphadenopathy and post-splenectomy changes only. A plan was agreed with haematology to rule out all possible infective causes before considering bone marrow biopsy. The patient also had an echocardiogram to exclude infective endocarditis.

Extended viral and BBV testing including hepatitis and HIV came back as negative. However, there was some difficulty around the testing of suspected CMV infection including what clinical information was required for the laboratory to process the sample. This meant serological tests needed to be repeated multiple times.

On day 14 of admission, the microbiology lab confirmed positive tests for CMV IgG and IgM and treatment with IV ganciclovir was commenced, with symptoms resolving within 48 hrs of treatment initiation. Subsequent avidity results were reported as low; indicating acute infection as opposed to re-activation.

Discussion/Conclusion

Viral replication of CMV is controlled predominantly by cytotoxic T-cell immunity, specifically CD4+ and CD8+ T cells, whose responses tend to be dysfunctional in immunosuppression. This crucial role played by T lymphocytes accounts for the common laboratory finding of leucocytosis in acute CMV infection [3]. Complications of CMV, such as CMV associated pneumonitis or gastrointestinal CMV, are largely driven by lymphocytic and cytokine proliferation, causing tissue inflammation, impaired function, and necrosis [4].

In this case, the patient had previously undergone a splenectomy. The spleen plays an important role in the body's immune response. Evidence around the significance of asplenia in bacterial and parasitic infections is well documented, however, other than a handful of case reports, there remains limited literature describing the relationship between splenectomy and viral infections [5,6].

Not only did the splenectomy likely contribute to the patient's exaggerated leucocytic response, complicating the clinical picture and raising suspicion of an underlying haematological disorder, it potentially prolonged the viral illness and increased the risk of complication if left untreated.

Treatment of acute CMV infection in immunocompetent individuals is typically limited to reassurance,

so long as the infection remains self-limiting. UK national guidelines; The BMJ Best Practice, describes treatment of CMV with anti-virals in patients with complications of CMV, Acquired Immunodeficiency Syndrome (AIDS), or who are solid organ transplant recipients, but there is no mention of patients with splenectomy [3].

Due to the self-limiting nature of CMV in fit and well patients, testing is not always useful and as such may be rejected by the laboratory unless relevant clinical information is provided. As well as any history of organ transplant, HIV/AIDS infection, or other immunosuppression, any signs or symptoms of complications of CMV should also be outlined as this would warrant active treatment.

This case highlights the importance of considering CMV infection as a differential for acute leucocytosis in patients having undergone splenectomy presenting with recurrent fever and unspecific symptoms. By increasing awareness in unscheduled areas of care, delays in treatment and unnecessary investigations can help be avoided, reducing risk of complications as well as patient anxiety. The authors also feel further evidence around the role asplenia plays in viral infections is needed.

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Manuscript Information: Received: July 07, 2026; Accepted: August 24, 2026; Published: August 31, 2026

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Citation: Beange D. Delayed diagnosis of cytomegalovirus in a patient with splenectomy: A case report. Open J Clin Med Case Rep. 2026; 2429.

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