CASE REPORT

Aplastic Anaemia Due to Parvovirus B19 in An Immunocompetent Adult: A Case Report

Perera D[1], Thirumavalavan K[2], Costa YJ[3], Somaratne I[4]

[1]Senior Lecturer in Medicine and Consultant Physician, General Sir John Kotelawala Defence University, Sri Lanka

[2]Senior Consultant Physician, Colombo North Teaching Hospital, Ragama, Sri Lanka

[3]Consultant Haematologist, Colombo North Teaching Hospital, Ragama, Sri Lanka

[4]Consultant Clinical Haematologist and Senior Lecturer in Pathology, General Sir John Kotelawala Defence University, Sri Lanka

Article Information

Corresponding Author
Dr. D. Perera

Email: neomal.p@kdu.ac.lk

https://orcid.org/0009-0009-9075-0692

https://doi.org/10.4038/sljms1.v2i2.75

Received 31 March 2026
Accepted 17 May 2026

Declaration of conflicts of interest: The authors declare no competing interests.

Declaration of patient's consent: Consent obtained from patient's next of kin for publication as a case report.

Funding: No external funding was received.

Abstract

Aplastic anaemia (AA) is a rare but potentially fatal disorder of the bone marrow characterised by severe pancytopenia and hypocellular marrow. Viral infections are recognised secondary causes of AA. Parvovirus B19 is more commonly associated with transient aplastic crisis or pure red cell aplasia rather than true aplastic anaemia. We report a case of severe aplastic anaemia in a previously healthy 29-year-old male, attributed to acute Parvovirus B19 infection. He presented with symptoms of anaemia, fever, and painful oral ulcers and was found to have severe pancytopenia with reticulocytopenia and hypocellular bone marrow. Extensive evaluation excluded autoimmune, malignant, and other infectious causes. Parvovirus B19 serology confirmed recent infection. Despite supportive care and immunosuppressive therapy with anti-thymocyte globulin (ATG), the patient developed refractory neutropenic sepsis and succumbed to complications. This case highlights parvovirus B19 as a rare but important cause of acquired aplastic anaemia, as well as the challenges clinicians face in managing AA in resource-limited settings.

Keywords: aplastic anaemia, parvovirus B19, anti-thymocyte globulin (ATG)

Background

Aplastic anaemia is characterized by peripheral pancytopenia and a hypocellular bone marrow, in the absence of marrow infiltration or fibrosis, causing bone marrow failure. Acquired aplastic anaemia is most commonly immune mediated. Secondary causes include drugs, toxins, radiation, autoimmune diseases, and viral infections. Aplastic anaemia secondary to

Cite this article as: Perera D, *et al.* Aplastic Anaemia Due to Parvovirus B19 in An Immunocompetent Adult: A Case Report. SLJMS 2025; 2(2): 93-96

This is an open-access article distributed under the terms of the Creative Commons Attribution 4.0 International License, which permits unrestricted use, distribution and reproduction in any medium provided the original author and source are credited.

viral infections is well recognised, particularly following hepatitis, but parvovirus B19 is an uncommon and under-reported cause of true aplastic anaemia.

Parvovirus B19 has a predilection for erythroid progenitor cells, and is classically associated with transient aplastic crisis and pure red cell aplasia, especially in individuals with chronic haemolytic disorders or immunodeficiency. Involvement of all three haematopoietic lineages resulting in aplastic anaemia is rare. Recognition of this association is important, as it has implications for investigation, management, and prognosis.

Case presentation

A 29-year-old previously healthy male presented with symptoms of anaemia for three weeks, accompanied by intermittent fever with chills and rigors and painful oral ulcers for three days. There was no history of jaundice, dark-coloured urine, spontaneous bruising, overt bleeding, or recurrent infections. There were no features suggestive of a connective tissue disorder. He admitted to heroin use via inhalation and had a family history of myelodysplasia. On examination, he was pale but not icteric, with no lymphadenopathy or hepatosplenomegaly. Multiple superficial tender oral ulcers without necrosis were noted. He had multiple tattoos, with no evidence of injection marks or superficial thrombophlebitis. There were no peripheral stigmata of infective endocarditis. He was haemodynamically stable with no cardiac murmurs. Fundoscopic examination revealed bilateral fundal haemorrhages, with Roth spots in the left eye, attributed to severe anaemia. There was no papilloedema.

Initial investigations demonstrated severe pancytopenia with reticulocytopenia: haemoglobin 6.7 g/dL, white blood cell count 2.41×10^9/L (neutrophils 8%, lymphocytes 88%, eosinophils 1%, monocytes 3%), and platelet count 21×10^9/L. Peripheral blood smear showed no blasts or dysplastic features. There were no features consistent with haemolysis. Serum bilirubin and Lactate Dehydrogenase level (LDH) were normal. Direct Coombs Test (DAT) was negative. Bone marrow aspiration and trephine biopsy revealed a markedly hypocellular marrow (without any infiltration) consistent with aplastic anaemia.

Further evaluation showed negative antinuclear antibody, anti-double-stranded DNA antibody, rheumatoid factor, hepatitis B and C, Cytomegalovirus (CMV), and Epstein-Barr virus (EBV) serology. Retroviral screening was negative. Flow cytometry of peripheral blood did not identify a paroxysmal nocturnal haemoglobinuria (PNH) clone. Urine haemosiderin was not detected. Multiple blood cultures and a trans-oesophageal echocardiogram (TOE) excluded infective endocarditis.

Parvovirus B19 serology subsequently became positive for both IgM and IgG antibodies, confirming recent infection and identifying the likely aetiology of the aplastic anaemia.

During the hospital stay, the patient required multiple red cell and platelet transfusions. Haematopoietic stem cell transplantation was not feasible due to financial constraints. Following correction of severe thrombocytopenia, immunosuppressive therapy with anti-thymocyte globulin (ATG) was initiated. After the second cycle of ATG, he developed persistent fever due to neutropenic sepsis, requiring admission to the intensive care unit. He was managed according to strict neutropenic sepsis protocols. Despite maximal supportive care, he succumbed to complications of overwhelming sepsis.

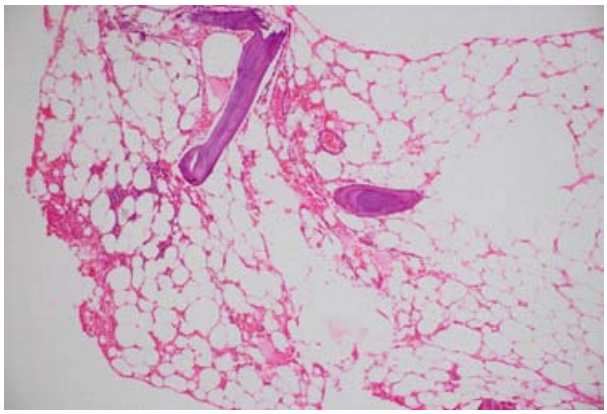

Figure 1. Bone marrow trephine biopsy, Haematoxylin and eosin stain, × 100.

Discussion

This case report describes development of severe aplastic anaemia in an immunocompetent patient without prior haematological disorders, following acute parvovirus B19 infection, which is a rare occurrence.

Aplastic anaemia is defined by the presence of bone marrow hypocellularity accompanied by peripheral pancytopenia.[1,2] Diagnostic criteria include bone marrow cellularity below 25%, or 25-50% with less than 30% residual haematopoietic cells, along with at least two cytopenias.[1,2] Disease severity is classified as non-severe, severe, or very severe based primarily on neutrophil count and marrow cellularity, as these correlate with infection risk and prognosis.[1,2]

Parvovirus B19 is a single-stranded DNA virus that infects erythroid precursor cells via the P antigen.[3] The virus is transmitted primarily from an infected person through respiratory secretions. Transmission through blood products and vertical transmission during pregnancy have

been reported.[4] Symptoms vary according to the age, immunity, and underlying comorbidities of the affected host. Immunocompetent individuals present with nonspecific, self-limiting "flu-like" symptoms.[5] Erythema Infectiosum (EI), with its characteristic facial rash described as a "slapped cheek appearance", also known as "fifth disease"[5], is commonly seen among children infected with Parvovirus B19, although it may rarely be seen among adults. In pregnancy, the virus can result in miscarriages due to hydrops fetalis.[5] Parvovirus B19 infection typically causes pure red cell aplasia due to selective destruction of erythroid progenitor cells, and in immunocompetent patients this effect is transient even though severe persistent symptoms can be seen in patients with haematological disorders like haemolytic anaemia.[5,6] In this case presented, Parvovirus B19 was responsible for severe aplasia of the bone marrow in an immunocompetent patient with no prior history of hematological disorders, thus indicating an unusual and rare presentation.

The proposed mechanisms for development of aplastic anaemia include direct cytotoxicity to hematopoietic progenitors[3], immune-mediated destruction of stem cells triggered by viral antigens[7], and cytokine-mediated suppression of marrow function.[7] Persistence of viral DNA within the bone marrow may trigger immune activation, resulting in severe persistent pancytopenia irrespective of the immune status of the affected individual.[7]

Secondary causes of acquired aplastic anaemia include medications (such as chloramphenicol and antithyroid medications), toxins, autoimmune diseases, pregnancy, and infections. Many viruses have been implicated as acquired causes of aplastic anaemia. A systematic exclusion of these conditions is essential before diagnosing idiopathic aplastic anaemia. The clinical assessment and extensive investigations done in this patient exclude other possible causes for aplastic anaemia.

Management of AA depends on severity, age, and donor availability. Supportive care includes blood product transfusions and infection prophylaxis. Immunosuppressive therapy with ATG and cyclosporine remains first-line treatment for severe aplastic anaemia[8] in patients without a suitable donor or in resource-limited settings where stem cell transplantation facilities are not available. The addition of eltrombopag has improved haematological response rates in recent studies.[9] Allogeneic hematopoietic stem cell transplantation offers curative potential,[9,10] particularly in younger patients, but accessibility remains limited in many settings, as in our patient.

In Parvovirus B19-associated marrow failure, intravenous immunoglobulin has shown benefit in selected cases,[9,11] especially in immunocompromised patients, although evidence remains limited.

Table 1. Causes of aplastic anaemia

Congenital	Dyskeratosis congenita
	Fanconi anaemia
	Shwachman-Diamond syndrome
Acquired	Idiopathic immune mediated (70-80 %)
	Paroxysmal nocturnal haemoglobinuria -10%
	Viruses – CMV, HIV, EBV, Parvovirus B19, Seronegative (non-A through-G) hepatitis
	Connective tissue disorders – SLE, Rheumatoid arthritis
	Occupational exposure – Benzene, pesticides
	Medications - Antibiotics – Chloramphenicol, sulphonamides
	• Anti-inflammatory – naproxen, indomethacin, diclofenac
	• Anti-rheumatics – penicillamine, gold
	• Antithyroid – carbimazole, methimazole
	• Anticonvulsants – carbamazepine, phenytoin
	• Cytotoxic drugs and radiation

With contemporary management, five-year survival rates exceed 80% in severe aplastic anaemia.[10] However, prognosis is adversely affected by severe neutropenia, delayed diagnosis, high susceptibility to infections, and limited access to definitive therapy, as illustrated in this case. In our patient, HSCT was not a feasible option. Therefore, he was treated with ATG. However, the outcome of the treatment could not be assessed since he succumbed to sepsis irrespective of best supportive care.

Conclusions

Parvovirus B19 should be recognised as a rare but important cause of acquired aplastic anaemia in adults, even in the absence of underlying chronic haemolytic disorders, although pure red cell aplasia is more common. Early identification through targeted viral testing is essential, as delayed diagnosis may lead to fatal complications. This case highlights the importance of recognizing parvovirus B19 as a cause of aplastic anaemia in immunocompetent patients, and the challenges faced during the management of aplastic anaemia in resource-limited settings.

Authors' contributions

Concept and design: DP

Literature review: DP, IS

Compilation of manuscript: DP, IS

Manuscript editing and proofreading: DP, KT, YJC, IS

References

1. Young NS. Aplastic anemia. *N Engl J Med.* 2018; **379**(17): 1643-56. doi:10.1056/NEJMra1413485
2. Killick SB, Bown N, Cavenagh J, *et al*. Guidelines for the diagnosis and management of adult aplastic anaemia. *Br J Haematol.* 2016; **172**(2): 187-207. doi:10.1111/bjh.13853
3. Simões FO, Goncalves H, Sá R, Campos BF, Domingues RM, Oliveira N, Pimentel T. Parvovirus B19-induced pancytopenia: a case report of aplastic crisis. *Cureus.* 2024; **16**(12): e76660. doi:10.7759/cureus.76660.
4. Dittmer FP, Guimarães C de M, Peixoto AB, *et al*. Parvovirus B19 Infection and Pregnancy: Review of the Current Knowledge. *J Pers Med.* 2024; **14**(2): 139. doi: 10.3390/jpm14020139
5. Servey JT, Reamy BV, Hodge J. Clinical presentations of Parvovirus B19 infection. *Am Fam Physician.* 2007; **75**(3): 373-8.
6. Ameur O, Zahid H, Elkochri S, Tagajdid R, Elannaz H, Hassine S, *et al*. Aplastic anemia induced by Parvovirus B19 infection in an immunocompetent adult: a case report and literature review. *J Community Med Health Solut.* 2025; **6**(1): 50-2. doi:10.29328/journal.jcmhs.1001058.
7. Servant-Delmas A, Lefrère JJ, Morinet F, Pillet S. Advances in human B19 erythrovirus biology. *J Virol.* 2010; **84**(19): 9658-65. doi:10.1128/JVI.00603-10
8. Scheinberg P, Young NS. How I treat aplastic anemia. *Blood.* 2012; **120**(6): 1185-96. doi:10.1182/blood-2011-12-292078
9. Townsley DM, Scheinberg P, Winkler T, *et al*. Eltrombopag added to standard immunosuppression for aplastic anemia. *N Engl J Med.* 2017; **376**(16): 1540-50. doi:10.1056/NEJMoa1613878
10. Bacigalupo A. How I treat acquired aplastic anemia. *Blood.* 2017; **129**(11): 1428-36. doi:10.1182/blood-2016-08-693481
11. Osaki Y, Matsubara K, Matsubara Y, *et al.* Severe aplastic anemia associated with human parvovirus B19 infection in an immunocompetent adult. *Intern Med.* 2011; **50**(7): 733-7. doi:10.2169/internalmedicine.50.4606