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ACUTE PARVOVIRUS B19 INFECTION ASSOCIATED WITH MULTI-ORGAN INVOLVEMENT IN AN IMMUNOCOMPETENT ADULT

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ABSTRACT

Introduction: Human parvovirus B19 is a highly prevalent single-stranded deoxyribonucleic acid virus that infects a large proportion of the global population. It can involve multiple organ systems, leading to a broad spectrum of clinical manifestations. While most infections in immunocompetent individuals are mild and self-limiting, parvovirus B19 can occasionally cause severe and diverse complications.

Case description: We present a case of a 44-year-old, immunocompetent woman, who presented acutely with a severe acute inflammatory response associated with hepatomegaly, oedema, and cardiac and renal involvement. Early suspicion of atypical infections including parvovirus B19 was considered given the multi-organ involvement and non-specific aetiology. Parvovirus B19 infection was confirmed alongside the development of the typical rash seen in acute infection and the patient improved with supportive therapy with full resolution.

Conclusion: This case underscores the potential severity of parvovirus B19 infection. Early testing and a high index of suspicion is required to accurately diagnoses these patients and may improve clinical outcomes whilst the mainstay of management is supportive. A potential underlying pathophysiology is suggested in acute parvovirus B19 infection with multi-organ involvement highlighting the link between infection and cytokine-driven inflammation and endothelial dysfunction.

KEYWORDS

Parvovirus B19, acute kidney injury, acute infection, cytokine-driven inflammation, endothelial dysfunction

LEARNING POINTS

- Early testing for parvovirus B19 should be considered in immunocompetent adults with features of acute hepatitis of unknown aetiology, especially when there is early haematological and joint involvement.
- In patients with acute infection with parvovirus B19, cytokine-driven inflammation and endothelial dysfunction can result in extremis and multi-organ involvement.
- Supportive treatment and close monitoring is the mainstay of treatment for patients identified and early escalation to intensive care may be required.

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INTRODUCTION

Parvovirus B19 is a single-stranded deoxyribonucleic acid (DNA) virus belonging to the *Parvoviridae* family[1,2]. In children a classical "slapped-cheek" rash or erythema infectiosum (fifth's disease) often is recorded alongside flu-like symptoms. In the majority of immunocompetent adults, infection with parvovirus B19 is asymptomatic or results in mild symptoms such as a flu-like illness followed by acute arthralgia or arthritis[1,2]. These presentations are self-limiting to 5-10 days. Parvovirus B19 can however cause more severe complications such as transient aplastic anaemia, especially in those with underlying haematological conditions[1,2] and case reports have associated the infection with myocarditis[3], hepatitis[4-6] and renal complications[7,8].

We present a case demonstrating an acute parvovirus B19 infection resulting in a unique constellation of acute onset anaemia, hepatitis, acute kidney injury (AKI) and cardiopulmonary impairment with generalised oedema.

We discuss an underlying mechanism of cytokine-driven inflammation and endothelial dysfunction resulting in extreme presentations of patients with acute parvovirus B19 infection.

CASE DESCRIPTION

A 44-year-old female patient, with no comorbidities, presented to her local hospital with a 7-day history of lower back and abdominal pain. Fevers and arthralgia had developed over the last 24 hours. Salient details from her medical history included recent commencement on an anti-depressant and significant alcohol consumption ongoing for the last 2 months, estimating 3-4 litres of vodka a week. She was recently widowed and lived with her 2 children and worked in a primary school. On examination she was found to have tender hepatomegaly, alongside general arthralgia affecting her large joints. She had bilateral basal crepitations and peripheral oedema in her lower limbs. Bloods taken on admission demonstrated significant derangement with anaemia (haemoglobin 91 g/l, normal range 115-155 g/l), an acute inflammatory response with raised C-reactive protein (CRP) (616 mg/l, normal range <5 mg/l), AKI (serum creatinine 249 µmol/l, normal range 45-84 µmol/l), and a marked transaminitis (alkaline phosphatase (ALP) 583 IU/l, normal range 30-130; alanine aminotransferase (ALT) 100 IU/l, normal range 7-35 IU/L), raised gamma-glutamyltransferase (246 IU/l, normal range 5-40 IU/l) with a normal bilirubin (7 umol/l, normal range 0-21 umol/l). Urinalysis performed after catheterisation was unremarkable.

Due to the acuity and severity of her presentation and the concerning blood results, a computerised tomography (CT) scan of her abdomen and pelvis was performed. The results of this showed hepatosplenomegaly, splenic infarction and small bilateral pleural effusions *(Fig. 1)*. Of note there was no abscess or thrombosis demonstrated in the hepatic or portal vasculature and no obstruction in the kidneys, ureters or bladder which may have been contributing to the severity of her AKI. To further investigate the causes of significant multi-organ involvement, a full septic screen, brain natriuretic peptide (BNP), non-invasive liver screen, renal screen and blood film were performed. Given the patient's arthralgia, hepatomegaly, general malaise, and recent contact with unwell young children, parvovirus B19 serological testing was performed.



Figure 1. *Computed tomography scan: transverse cross-section demonstrating hepatomegaly with mottled enhancement and dilated hepatic veins suggestive of congestive hepatomegaly.*

Antinuclear antibody, anti-double stranded DNA, myeloperoxidase and proteinase 3 anti-neutrophil cytoplasmic antibodies and extractable nuclear antigen were all negative. Alpha-1-antitrypsin, ceruloplasmin, serum copper immunoglobulins and complement (C3 and C4) also returned normal. Serological studies for hepatitis A, B, C and E, human immunodeficiency virus, Epstein-Barr virus and cytomegalovirus were negative.

The blood film reported evidence of anisocytosis. Of note, there no evidence of haemolysis, or evidence of an underlying haematological condition. There was a reticulocytopenia (15.2 10^9/l, normal range 23 -8110^9/l).

An echocardiogram was performed in view of the markedly elevated BNP (3263 pg/ml; normal <300 pg/ml) to assess for cardiac involvement, including myocarditis or infective endocarditis. This demonstrated a preserved left ventricular ejection fraction (60-65%) with elevated right-sided pressures, consistent with right heart strain and suggestive of significant pulmonary hypertension. Troponin levels sent on admission and repeated during admission were not elevated.

A CT pulmonary angiogram (CTPA) was organised after consultation with the local respiratory teams to investigate causes of pulmonary hypertension. The results showed small pleural effusions and changes in keeping with congestive hepatomegaly but no evidence of embolism, fibrosis or interstitial changes *(Fig. 2)*.

The patient reported symptomatic improvement with

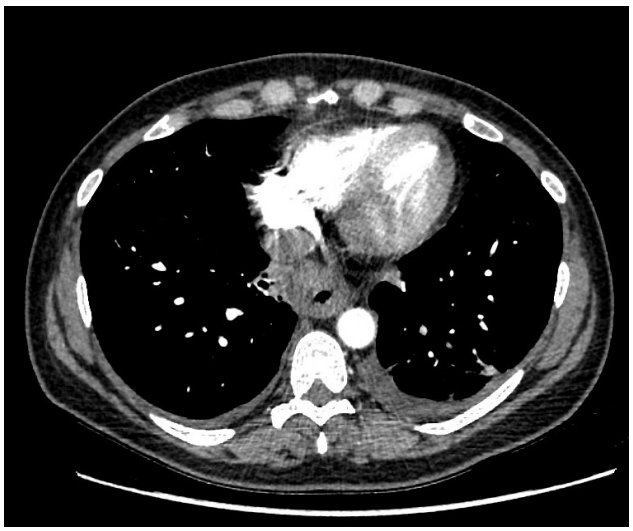


Figure 2. Computed tomography scan, transverse cross-section showing normal pulmonary arterial enhancement, with evidence of small bilateral pleural effusions.

supportive therapy, reflected clinically by resolution of arthralgia and biochemically by improving ALP, ALT, CRP, and renal function. Three days into admission the patient noticed a reticular, pink rash which had develop across her torso, associated with mild pruritus *(Fig. 3)*. She denied any prior drug allergies or systemic symptoms, and given her overall improving clinical trajectory, a conservative approach with close monitoring of the rash was adopted.

Five days into her admission, the parvovirus B19 panel returned positive for immunoglobulin G and immunoglobulin M antibodies, indicating an acute infection. By this time her symptoms had largely resolved, and she was discharged the following day.

Follow-up 4 weeks after admission confirmed complete symptomatic recovery, including resolution of hepatomegaly, rash, and arthralgia. Bloods tests performed at this time confirmed full biochemical resolution of hepatitis, AKI, and anaemia, with normal inflammatory markers. Follow-up chest radiography showed no evidence of pleural effusions.

DISCUSSION

Hepatic involvement in parvovirus B19 infection is a recognised manifestation and, in isolation, is not unexpected. Adult cases have described a spectrum ranging from mild, transient transaminitis to acute hepatitis and, rarely, fulminant liver failure[1,4,9]. The underlying mechanism is thought to be predominantly immune mediated, as parvovirus B19 does not primarily replicate within hepatocytes[2,4,9]. Proposed mechanisms include cytokine-driven inflammation, immune complex deposition, and endothelial dysfunction affecting the hepatic microvasculature[4]. Biochemically, parvovirus B19–associated hepatitis often demonstrates a cholestatic or mixed pattern with a normal or minimally elevated bilirubin[5,6], consistent with the findings in this patient. The presence of hepatomegaly and biochemical resolution following supportive management further support a reversible inflammatory process rather than primary toxic, autoimmune, or obstructive liver disease. Although this patient had a history of recent excessive alcohol consumption, the acute presentation, marked inflammatory response, and complete resolution within weeks argue strongly against alcohol-related liver injury as the principal aetiology.

What distinguishes this case is the concurrent involvement of multiple organ systems, including haematological, renal, hepatic, and cardiopulmonary dysfunction, in an immunocompetent adult. We propose that this constellation is best explained by a severe, transient systemic inflammatory response to acute parvovirus B19 infection. Parvovirus B19 is known to infect endothelial cells and provoke a robust cytokine response, even in individuals without underlying immunodeficiency[10]. The markedly elevated inflammatory markers in this case suggest widespread endothelial activation and dysfunction, resulting in increased vascular permeability and capillary leak. This provides a unifying pathophysiological explanation for the generalised oedema, pleural effusions, congestive hepatomegaly, and AKI observed.

The echocardiographic finding of elevated right-sided pressures with preserved left ventricular systolic function is most plausibly explained by acute, reversible pulmonary hypertension secondary to systemic inflammation. Cytokine-mediated pulmonary vasoconstriction and endothelial dysfunction within the pulmonary circulation have been described in severe inflammatory states and may transiently increase pulmonary vascular resistance[11]. This process was likely compounded by intravascular volume expansion related to AKI and capillary leak, increasing right ventricular preload and afterload. The absence of pulmonary embolism on CTPA, normal serial troponins, preserved left ventricular function, and complete clinical resolution argue strongly against myocarditis or primary cardiopulmonary disease. The markedly elevated BNP is

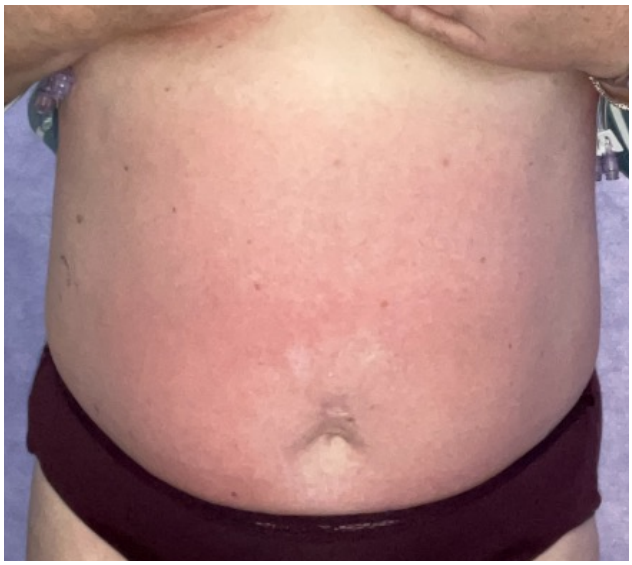


Figure 3. Reticular, pink rash on patient's torso.

consistent with acute right ventricular strain and hepatic congestion, both of which improved in parallel with clinical recovery.

Renal involvement in parvovirus B19 infection has been described and is thought to result from systemic inflammation, altered renal perfusion, immune complex deposition, or endothelial injury rather than direct viral cytotoxicity[7,8]. The bland urinalysis and complete recovery of renal function in this case support a functional, reversible process.

Anaemia is a well-recognised feature of parvovirus B19 infection, typically manifesting as a transient aplastic crisis in patients who are immunosuppressed or have high red cell turnover, such as sickle cell disease[1]. The blood film in this case showed no evidence of an underlying haematological disorder or haemolysis. We propose that the patient's chronic alcohol consumption, a known cause of bone marrow suppression, alongside the acute parvovirus B19 infection precipitated a transient aplastic crisis, evidenced by the observed reticulocytopenia. The subsequent spontaneous resolution, supports the diagnosis of a self-limiting viral-induced erythroid suppression, likely exacerbated by haemodilution from acute fluid shifts.

CONCLUSION

Overall, this case illustrates a systemic inflammatory phenotype of acute parvovirus B19 infection, resulting in transient multiorgan dysfunction with full recovery following supportive care. Early consideration of parvovirus B19 in adults presenting with acute hepatitis and unexplained multisystem involvement may facilitate timely diagnosis and avoid unnecessary invasive investigations. A prudent history and examination including travel and occupational history is required to exclude emergency pathology and to ensure that common causes are considered and investigated early.

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