Open Access Library Journal
2023, Volume 10, e9997
ISSN Online: 2333-9721
ISSN Print: 2333-9705

Myocarditis and Medullary Aplasia Post Parvovirus B19: A Case Report

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How to cite this paper: Boudarbala, H., Anane, S., Bouhafs, N., Babakhouya, A., Ghanam, A., Elouali, A., Rkain, M. and Benajiba, N. (2023) Myocarditis and Medullary Aplasia Post Parvovirus B19: A Case Report. *Open Access Library Journal*, **10**: e9997.
https://doi.org/10.4236/oalib.1109997

Received: March 12, 2023
Accepted: April 21, 2023
Published: April 24, 2023

Abstract

Infection with parvovirus B19 is common in children and typically causes mild illness. However, it is occasionally associated with severe diseases such as acute myocarditis and medullar aplasia. We report a rare case of an 11-year-old female patient with severe medullary aplasia induced by parvovirus B19 and complicated by myocarditis with a fatal evolution.

Subject Areas

Pediatrics

Keywords

Medullar Aplasia, Pancytopenia, Myocarditis, Parvovirus B19

1. Introduction

Parvovirus B19 (PVB19) infections are common in childhood. They usually cause mild illness especially erythema infectiosum also known as the fifth disease [1]. However, it can be formidable because of the occurrence of serious cardiac, hepatic and hematological complications which condition the vital prognosis: fulminant hepatitis, fulminant cardiomyopathy, and severe medullar aplasia [2].

We report the case of a patient with severe bone marrow aplasia induced by parvovirus B19, complicated by myocarditis.

2. Case Report

This is a female child, 11 years old, with no previous pathological history, who presented to the pediatric emergency with signs of modular insufficiency.

Clinical examination on admission found a girl, very asthenic, with genera-

lized skin pallor and ecchymotic spots on the lower limbs and per-orbital (Figure 1), without tumor syndrome. The initial biological showed a regenerative pancytopenia with moderate hepatic cytolysis, the blood smear was negative for blasts, the myelogram showed a deserted marrow, and the BOM confirmed the diagnosis of medullary aplasia. A complete etiological workup was performed, which showed a positive parvovirus B19 serology. The aplasia was immediately classified as severe with a PNN count of less than 500.

The patient received transfusion support while awaiting marrow transplantation. The evolution was marked by an increase in transfusion requirements and the appearance of prolonged fever, arthralgia, abdominal pain and a striking biological inflammatory syndrome.

A trans-thoracic ultrasound as part of the pre-transplant workup showed decreased myocardial contractility with mitral leakage and low ejection fraction: a finding suggestive of parvovirus B19-induced myocarditis (Figure 2).

Later, the patient died following an acute cardiac insufficiency.

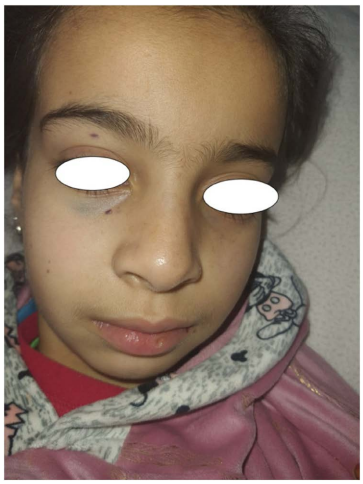

Figure 1. Periorbital ecchymotic spot.

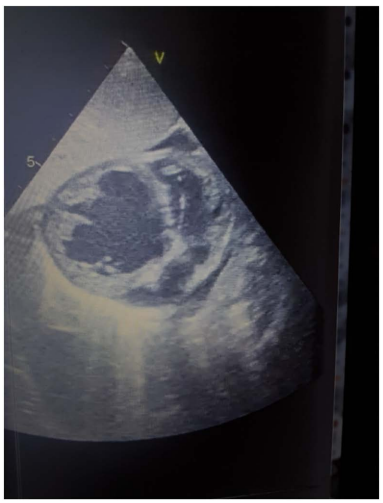

Figure 2. Dilatation of the left ventricle.

3. Discussion

Parvovirus B19 is a single-stranded DNA virus belonging to the Parvoviridae family. Transmission of parvovirus B19 occurs via the respiratory route, blood products, and vertically through the placenta from mother to fetus [3] [4]. Clinical manifestations of parvovirus B19 infections in children range from erythema infectiosum in healthy children to aplastic crisis in patients with hematologic disorders (such as sickle cell disease) and immunocompromised patients [5]. The clinical manifestations of parvovirus B19 infections in children range from erythema infectiosum in healthy children to aplastic crisis in patients with hematologic disorders (such as sickle cell disease) and immunocompromised patients [5].

There are few reports of parvovirus B19 infections with a prolonged course and unexpected complications, such as hepatitis and meningoencephalitis [6] [7]. Rarely, parvovirus B19 infection can trigger a clinical course similar to systemic lupus erythematosus with nephritis, arthritis, vasculitis and nephrotic syndrome [8].

Similar to other infections, parvovirus B19 infection can sometimes trigger a transient autoantibody response, including ANA and rheumatoid factor.

Parvovirus B19 infection can also lead to myocarditis, which is a potentially life-threatening condition in pediatric patients (as in our patient's case).

Parvovirus B19 is known to have a tropism for erythroid progenitor cells.

Parvovirus B19 is cytotoxic to host cells, resulting in hemolysis and suppression of erythropoiesis. Their entry into erythroid progenitor cells is via the cellular P antigen receptor, which is present in the liver, placenta, coronary artery endothelial cells, and myocardial cells. The mechanisms that contribute to heart failure, due to myocarditis and liver dysfunction, are unclear and may be related to P-receptor-mediated viral endocytosis of the primary infection or to autoimmune inflammation [9] [10].

Clinical presentation spectrum of myocarditis is wide, ranging from the absence of any symptoms to fulminant cardiogenic shock and sudden death [2].

La résonance magnétique cardiovasculaire avec l'utilisation complémentaire de gadolinium est un moyen utile de diagnostiquer la myocardite sans avoir recours à une biopsie. L'infection induite par le PVB19 est associée de manière caractéristique à un rehaussement tardif de la paroi latérale [2].

Patients with acute myocarditis require, often, standard heart failure therapy including diuretics, angiotensin converting enzyme inhibitors or angiotensin receptor antagonists [11]. b-blockers are introduced once the patients are clinically stable [11]. Patients with left ventricular enlargement should receive anticoagulation therapy to prevent thrombus formation in the left ventricle. In some cases with severely impaired left ventricle function, mechanical assist devices may be required waiting for a heart transplant.

Before the development of mechanical assist devices, a previous study described that most of patients with fulminant myocarditis die at the acute phase

because of rapidly progressive cardiac decompensation [12]. Some medical treatments, like IVIG were tried for viral myocarditis. However, the role of this therapy remains controversial. IVIG has both antiviral and immunomodulation effects, suggesting that it might be an effective therapy in acute viral myocarditis [11]. There are no randomized controlled trials investigating the use of IVIG to treat children with acute myocarditis, but several case series have shown that the use of high-dose IVIG to treat acute myocarditis in this population leads to improved recovery of left ventricular function and better survival [13]. In most children, PVB19 myocarditis causes significant mortality and morbidity [11].

4. Conclusion

Parvovirus B19 infection is a benign condition, however, it can cause serious complications: cardiac (cardiomyopathy), hematological (severe bone marrow aplasia), hepatic and others. In this case, we report the association of two complications of parvovirus B19: medullary aplasia and myocarditis.

Conflicts of Interest

The authors declare no conflicts of interest.

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