

## CASE REPORT

**Parvo B 19 Myocarditis in Immunocompetent Patient**Asif Nadeem<sup>1</sup>, Abidullah Khan<sup>2</sup>, Muhammad Farooq<sup>3</sup>, Mumtaz Malik<sup>4</sup>, Zulifiqar Congo<sup>5</sup>**ABSTRACT**

Parvo B 19 virus can cause different diseases in human. It can cause myocarditis which if not treated in time can prove fatal. Here we are presenting a case of 43 years old immune-competent male who was found to be infected with Parvo B 19 virus, which was diagnosed by positive serology and PCR technique. He was successfully treated and is on regular follow up. Every clinician should consider the possibility of PVB 19 in any patient presenting with acute myocarditis.

**Key Words:** *Myocarditis, Parvo B 19 Virus.*

**Introduction**

Parvo B 19 is the only member of family parvoviridae, discovered in 1974 and is pathogenic in humans.<sup>1-3</sup> It can cause fatal myocarditis leading to heart failure if not treated in time.<sup>4-6</sup> We are presenting a case of parvo B 19 myocarditis who presented with acute myocarditis and was successfully treated with antiviral therapy and inotropic support.

**Case Report**

A 43 years old serving soldier presented with one week history of flu like illness, dry cough, low grade intermittent fever with myalgia. He took treatment from some local general practitioner but his symptoms did not resolve. After about 3 days, he also developed signs of acute heart failure which were progressive and found to be NHYA Class IV accompanied by orthopnea and frothy sputum.

His past, personal, social, drug and family history were insignificant. Physical examination revealed pulse of 90/minute which was irregularly irregular, low volume. BP was 90/75 mmHg with no postural hypotension, temperature 99.0 F and respiratory rate of 20/minute. Chest auscultation revealed bilateral few basal crepitations. ECG showed AF, LAD, LBBB, T wave inversion in I, II, III, aVL, aVF and V3 to V6. Blood

complete picture showed lymphocytosis. Blood Culture and Sensitivity, CRP, Sputum AFB, Urine R/E and C/S, USG Abdomen, Trop T, CK MB, AST, LDH, RFT, LFT, Serology for EBV, CMV, adenovirus and enterovirus and angiography were all normal. 2 D Echo showed EF 20% with dilated LA, LV and severe LV systolic dysfunction, global hyperkinesia with no effusion. Parvo B 19 virus IgM was positive and was further confirmed by PCR. Cardiac MRI showed increased signal intensity at septal wall on T2 weighted image. Myocardial perfusion SPECT with Tl-201 after physiological stress and at rest revealed fixed perfusion defect (partial thickness MI/scarred myocardium of inferior wall) and multiple areas of moderate to severe fixed defects scattered all over left ventricular myocardium plus severe global hyperkinesia with 20% EF and poor LV function.

He was admitted in intensive care unit and was treated with IV diuretics and inotropic support. He remained admitted in hospital and became stable in one week. His EF improved to 55% after one week of treatment. He was discharged after ten days of hospitalization and PVB 19 IgM was repeated at the time of discharge, which was negative with negative PCR.

At the time of discharge he was advised ACE inhibitors, digoxin, beta blockers, and low dose aspirin. He was properly counseled regarding his illness and follow up.

**Discussion**

Acute myocarditis is an inflammatory condition of myocardium due to various pathogens.<sup>1</sup> Patient can present with different clinical features which may be fulminant or non-fulminant. Different infections, drugs, toxins and systemic diseases have been found to cause myocarditis.<sup>2</sup> Parvo B19 virus, human herpes

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virus 6 and enterovirus are some of the most common viral causes of acute myocarditis. PVB19 can cause fatal myocarditis because it affects myocardial endothelial cells.<sup>3</sup> Clinical features can vary from simple flu like illness to heart failure, arrhythmias or pseudo myocardial infarction.<sup>4</sup> Myocarditis is found in 42% of cases with unexplained deaths in individuals under 35 years of age. Exact incidence and prevalence of fulminant myocarditis is not known but it is seen in 10 % of biopsy proven myocarditis.<sup>1,5-8</sup>

An accurate and rapid diagnostic approach is very crucial in the management of viral myocarditis. Serology, Cardiac MRI, Angiography and Cardiac biopsy are some of the crucial diagnostic investigations.<sup>6</sup> Additional viral PCR (quantitative) and for viral genome and immunohistochemistry for cardiac inflammation and necrosis also help in diagnosis.<sup>6-8</sup> This will help in accurate diagnosis and targeted treatment. When diagnosis is confirmed, treatment of underlying cause is the main life saving step. In addition to specific treatment of the underlying cause, intense hemodynamic support like inotropic support, intra-aortic balloon pumps and ventricular assistance devices can also be used to save life of the patient depending upon the clinical condition of the patient. Heart transplantation is the last option in developed countries where facilities are available.<sup>7,8</sup>

Seven percent of the patients presenting with fulminant myocarditis will have fatal outcome.<sup>2,5-7</sup> Mortality rates are different due to various patient

risk factors.<sup>5,7-8</sup> After acute phase of treatment, patient should be managed with standard heart failure medications with beta blockers, Angiotensin-converting enzyme inhibitors, Angiotensin receptor blockers (ARBs), Calcium channel blockers (CCB), and Digoxin. Regular follow up is very necessary as recurrence has been reported.<sup>5-7</sup>

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