RESEARCH ARTICLE

PARVOVIRUS B19 INDUCED MYOCARDITIS, SIMULATING ACUTE MYOCARDIAL INFARCTION

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Manuscript Info

Abstract

Myocarditis is an inflammation of the myocardium associated with mechanical or electrical dysfunctions that usually lead to inappropriate ventricular dilatation or hypertrophy. A number of inciting factors are known to cause myocarditis- genetic defects, viruses, bacteria, parasites, granulomatous inflammations, collagen vascular disorders, chemotherapeutic agents- to name a few. Among the viruses, Adenovirus, Coxsackie virus, Human Herpes virus six and Parvovirus B19 are the encountered pathogens. We hereby present a case of a 14 years old boy who presented with typical cardiac chest pain associated with electrocardiographic changes of ST segment elevation MI in the inferior leads. Cardiac biomarkers were elevated, adding to the diagnostic confusion of MI. On subjecting the patient to a coronary angiography, his arteries were found to be patent, directing us towards the diagnosis of myocarditis. After getting a cardiac MRI and serum tests, the diagnosis of fulminant myocarditis induced by Parvovirus B19 was made. Fortunately, the patient survived on inotropes and other supportive therapy which helped him overcome the cardiac failure.

Introduction:-

Myocarditis is an inflammation of the myocardium associated with mechanical or electrical dysfunctions that usually lead to inappropriate ventricular dilatation or hypertrophy. Clinically, patients can present with a range of symptoms, including absent cardiac symptoms but with abnormal ECG and cardiac biomarkers, to fulminant myocarditis with heart failure, chest pain and significant hemodynamic compromise.

Among the viral causes of cardiomyopathy, Parvovirus B19 happens to be very rare, but fulminant. The virus enters the body via the respiratory tract and once inside the myocardium, activates the host’s tyrosine kinase. This subsequently leads to an immune reaction that stimulates matrix metalloproteinase which disrupts collagen and elastin, leading to dilation of the cardiac chambers.

Cardiac MRI, Immunohistochemistry, endomyocardial biopsy and virus-specific quantitative PCR tests are invaluable tools for the diagnosis of viral myocarditis.

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Treatment consists of supportive care and management of heart failure, along with maintaining hemodynamic soundness. Antiviral agents or immunosuppressive agents have not been found to contribute significantly to the treatment of this condition.

**Case Report:**
A 14 year old boy was brought to the emergency room, with the complaints of acute onset left sided, severe chest pain, associated with breathlessness, since 2 hours.

On enquiry with the relatives, it was found that the patient had been experiencing fever, cold and cough since the past seven days for which he was on treatment with anti histaminic.

The patient had no past medical or surgical history.

Pulse rate was 92 beats per minute, with a blood pressure of 90/50 mm hg. Spo2 was 98 percent on room air.

The ECG showed ST segment elevation in leads II, III and aVF and v2 to v5. His cardiac biomarkers were ordered and horrifyingly showed Troponin I value of 99.2 ng/ml and NT pro BNP of 855 pg/ml. Patient was started on intravenous Dobutamine (10 microgram/kg/min) along with fluid resuscitation, with intense monitoring.

Echocardiography demonstrated Global left ventricular hypokinesia which was more marked in the posterior wall, with preserved thickness. The ejection fraction of his left ventricle was down to 30 percent, indicating severe LV systolic dysfunction. Mild pericardial effusion was present. Right ventricular systolic function was normal.

CT coronary angiogram was obtained to rule out coronary artery disease. The calcium score of the patient was 0, with all vessels being normal in course and caliber, without any stenosis.

Owing to the diagnostic perplexity, a cardiac MRI was done which revealed abnormal patchy hyper intensities on STIR, suggestive of myocardial edema. On late gadolinium enhancement (LGE) there were abnormal hyper intensities in the lateral, inferior, anterior and septal portions, which involved more than 75 percent of the myocardial thickness. The abnormal myocardial areas were not confined to any particular arterial territory. The LVEF was 31 percent.

**Features were suggestive of myocarditis.**
Serum investigations for the causative organism tested positive for Parvovirus B19 (57750 copies per ml). Reports for other cardio tropic viruses were negative.

The patient was started on intense supportive care and gradually got better.

![Twelve lead electrocardiogram showing ST elevation in II, III, aVF and v2 to v5.](image-url)
Figure 2: CT coronary angiography.

Figure 3: Cardiac MRI showing diffuse edema, detected by increased signal intensity during late gadolinium enhancement.
Figure 4: ECG at the time of discharge. Absence of ST elevation, without q waves.

Discussion:
Parvovirus B 19 causes devastating myocarditis in children, leading to life threatening clinical situations. This case highlights the close similarities in the clinical, electrocardiographical and laboratory findings of Myocardial Infarction and viral myocarditis. The usefulness of Cardiac MRI in making a definitive diagnosis of myocarditis is worth acknowledging.

The etiologies of myocarditis are numerous, and the clinical presentation is varied. This creates a major diagnostic challenge. When coronary artery disease or vasospasm is ruled out as a cause of cardiac illness, targeted search for other conditions causing cardiomyopathy should be embarked upon.

To diagnose viral myocarditis, detection of viral genomes by molecular methods and evaluation of myocardial inflammation by immunohistochemistry on endomyocardial biopsy samples is undertaken. Immunohistochemistry will reveal the lymphocytic subtypes and HLA antigens.

Treatment of viral myocarditis consists primarily of hemodynamic supportive therapy which has been the most beneficial line of management. The importance of immunosuppressive therapy and antiviral drugs during the acute stage of myocarditis, is under study.

References:
1. Sinagra G, Anzini M, Pereira NL, Bussani R, Finocchiaro G, Bartunek J, Merlo M. Myocarditis in clinical practice. Mayo Clin Proc. 2016;91(9):1256–1266. doi: 10.1016/j.mayocp.2016.05.013. [PubMed] [CrossRef] [Google Scholar]
2. Fung G, Luo H, Qiu Y, Yang D, McManus B. Myocarditis. Circ Res. 2016;118(3):496–514. doi: 10.1161/CircRes.115.306573. [PubMed] [CrossRef] [Google Scholar]
3. Schultz JC, Hilliard AA, Cooper LT Jr, Rihal CS. Diagnosis and treatment of viral myocarditis. Mayo Clin Proc. 2009;84(11):1001–1009. doi: 10.1016/S0025-6196(11)60670-8. [PMC free article] [PubMed] [CrossRef] [Google Scholar]
4. Basic D, Gupta S, Kwong RY. Parvovirus b19-induced myocarditis mimicking acute myocardial infarction: clarification of diagnosis by cardiac magnetic resonance imaging. Circulation. 2010;121(7):e40–42. doi: 10.1161/CIR.0b013e3181d310ea. [PubMed] [CrossRef] [Google Scholar]
5. Mahrholdt H, Goedecke C, Wagner A, Meinhardt G, Athanasiadis A, Vogelsberg H, Fritz P. et al. Cardiovascular magnetic resonance assessment of human myocarditis: a comparison to histology and molecular pathology. Circulation. 2004;109(10):1250–1258. doi: 10.1161/01.CIR.0000118493.13323.81. [PubMed] [CrossRef] [Google Scholar]
6. Cooper LT, Baughman KL, Feldman AM, Frustaci A, Jessup M, Kuhl U, Levine GN. et al. The role of endomyocardial biopsy in the management of cardiovascular disease: a scientific statement from the American Heart Association, the American College of Cardiology, and the European Society of Cardiology. Endorsed by the Heart Failure Society of America and the Heart Failure Association of the European Society of Cardiology. J Am Coll Cardiol. 2007;50(19):1914–1931. doi: 10.1016/j.jacc.2007.09.008. [PubMed] [CrossRef] [Google Scholar]

7. Stiermaier T, Fohrenbach F, Klingel K, Kandolf R, Boudriot E, Sandri M, Linke A. et al. Biventricular endomyocardial biopsy in patients with suspected myocarditis: Feasibility, complication rate and additional diagnostic value. Int J Cardiol. 2017;230:364–370. doi: 10.1016/j.ijcard.2016.12.103. [PubMed] [CrossRef] [Google Scholar]

8. Besler C, Schuler G, Lurz P. Myocarditis in the differential diagnosis of cardiomyopathies. Endomyocardial biopsy or MRI? Herz. 2015;40(4):607–615. doi: 10.1007/s00059-015-4229-z. [PubMed] [CrossRef] [Google Scholar]

9. Mahrholdt H, Wagner A, Judd RM, Sechtem U, Kim RJ. Delayed enhancement cardiovascular magnetic resonance assessment of non-ischaemic cardiomyopathies. Eur Heart J. 2005;26(15):1461–1474. doi: 10.1093/eurheartj/ehi258. [PubMed] [CrossRef] [Google Scholar]

10. Mahrholdt H, Wagner A, Deluigi CC, Kispert E, Hager S, Meinhardt G, Vogelsberg H. et al. Presentation, patterns of myocardial damage, and clinical course of viral myocarditis. Circulation. 2006;114(15):1581–1590. doi: 10.1161/CIRCULATIONAHA.105.606509. [PubMed] [CrossRef] [Google Scholar]

11. Mavrogeni S, Bratis K, Terrovitis J, Tsagalou E, Nanas J. Fulminant myocarditis. Can cardiac magnetic resonance predict evolution to heart failure? Int J Cardiol. 2012;159(2):e37–38. doi: 10.1016/j.ijcard.2011.11.053. [PubMed] [CrossRef] [Google Scholar].