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Successful Recovery of Fulminant Myocarditis in Primigravida: A Case Report

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Abstract: Fulminant myocarditis (FM) is a life-threatening disease with a rapid, progressive course of deterioration. The prognosis is favorable with appropriate management in the initial vulnerable stages. Here we report the first occurrence of FM in a healthy primigravid woman. We report the case of a previously healthy 30-year-old woman with FM in whom cardiac function normalized within 4 days with aggressive pharmacological support using positive inotropic drugs, intravenous steroids, high-dose immunoglobulin, and intravenous antibiotics. FM remains a challenging disease for diagnosis and treatment in clinical practice. This case serves to emphasize the importance of FM and its management. Myocardial failure due to FM can be reversible if treated early.

Keywords: Fulminant myocarditis, disease, management, woman.

INTRODUCTION

Myocarditis is an inflammation of the myocardium. Although it occurs in people of all ages, the young are affected most often [1]. Viral infections are the most common causes, such as those by Coxsackie B virus, adenovirus, and parvovirus B-19. Other causes include bacterial and protozoan infections. Myocarditis can also occur in autoimmune and other systemic illnesses, including systemic lupus erythematosus, scleroderma, and sarcoidosis [2]. Symptoms can range from mild fevers, shortness of breath, and palpitations to severe hemodynamic collapse [3].

Fulminant myocarditis (FM) is a clinical diagnosis and is an unusual complication of myocarditis with a rapidly progressive course, resulting in severe acute heart failure and cardiogenic shock [4]. The diagnosis is usually made based on clinical presentation and noninvasive imaging findings. Treatment depends on both the severity and the cause. Most patients respond well to standard heart failure therapy, although in severe cases, mechanical circulatory support or heart transplant may be required [2]. We present a case of a young woman who presented with unusual features and who was treated for ectopic pregnancy, which was later discovered to be FM.

CASE PRESENTATION

On October 28, 2009, a previously healthy 30-year-old woman presented to the emergency room of King Abdulaziz Medical Center (KAMC/NGHA) with epigastric and right-side pain for 3 days, with 2 months amenorrhea. She became clinically unstable and was taken for emergency exploratory laparotomy because of the suspicion of an ectopic pregnancy. The laparotomy report showed a gravida uterus of 2 months, no ectopic pregnancy, and severely inflamed organs, especially the pancreas. The patient was moved to the intensive care unit, where she was intubated and ventilated. She was still febrile (39°C–40°C) and was in poor health due to inotropes and a high central venous pressure of 20. She had a pulse rate of 136 bpm, a respiratory rate of 16 breaths per minute, blood pressure of 102/77, oxygen saturation of 99, white blood cell count of 12.3×10^9, lactate dehydrogenase level of 262 U/L, aspartate aminotransferase level of 87 U/L, creatine phosphokinase (CPK) level of 225 U/L, and a CPK-myocardial bundle level of 16.72 mcg/L. These parameters kept rising, as shown in (Table 1). An electrocardiogram, chest X-ray, and echocardiogram were performed (Figures 1,2,3).

Timeline

Oct 28, 2009	Admission to emergency room and emergency laparotomy for expected ectopic pregnancy.
	After laparotomy, the patient was moved to the intensive care unit, intubated, and ventilated.
Oct 29, 2009	Patient was moved to a medical cardiac intensive care unit and underwent pericardiocentesis.
Nov 02, 2009	After normalization of left ventricle, patient was extubated and transferred to the ward for 2 weeks' observation.
Nov 16, 2009	Patient discharged.
May 2009	Patient delivered a healthy baby with a cleft lip.
Feb 27, 2013	Routine checkup showed normal electrocardiogram.
May 31, 2018	Cardiac echo and magnetic resonance imaging.

Table-1: Laboratory test results

Date Time	TROP I Ug/L	LDH U/L	AST U/L	CKP U/L	MB ng/ml	WBC $\times 10^9$/L	Neutrophils %	Amylase U/L	BNP PMO/L
10/28/09 01:37	5.98	262	87	225	16.72	13	11.1	31	
10/28/09 06:22	6.03	222	78	237	15.94	18.6			1354.6
10/28/09 17:00	7.66	321	57	486	19.04	11.5	7.7		
10/29/09 04:44	10.45	381	57	745	21.38	11		237	
10/29/09 09:22	7.91	352	52	887	13.75				2599.9
10/29/09 13:14	6.84	331	48	876	8.2	11.3	8.4		
10/29/09 18:40	4.87	347	46	874	8.32				
10/30/09 02:14	4.03	323	37	596	6.53	10.3	9.1	89	2353
10/30/09 08:30	3.22	302	34	494	5.6				
10/31/09 08:31	2.16	407	33	448	3.81	10.3	7.4	31	1922.3

TROP I: troponin I; LDH: lactate dehydrogenase; AST: aspartate aminotransferase; CPK: creatine phosphokinase; MB: myocardial bundle; WBC: white blood cells; BNP: brain natriuretic peptide.

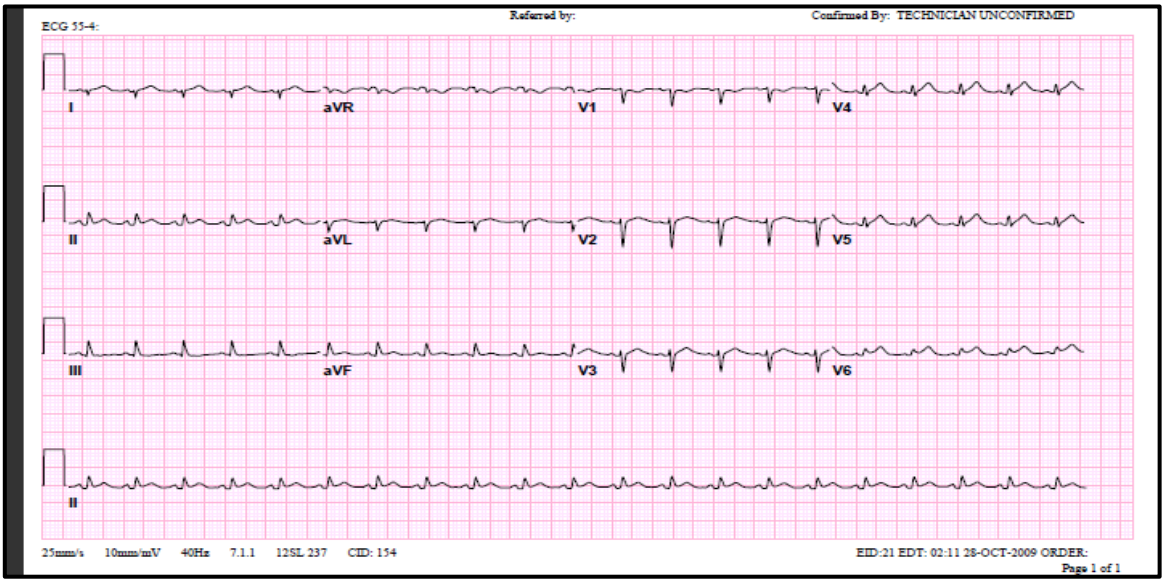

Fig-1: Patient's electrocardiogram, showing right axis deviation and low-voltage diffuse minimal ST elevation

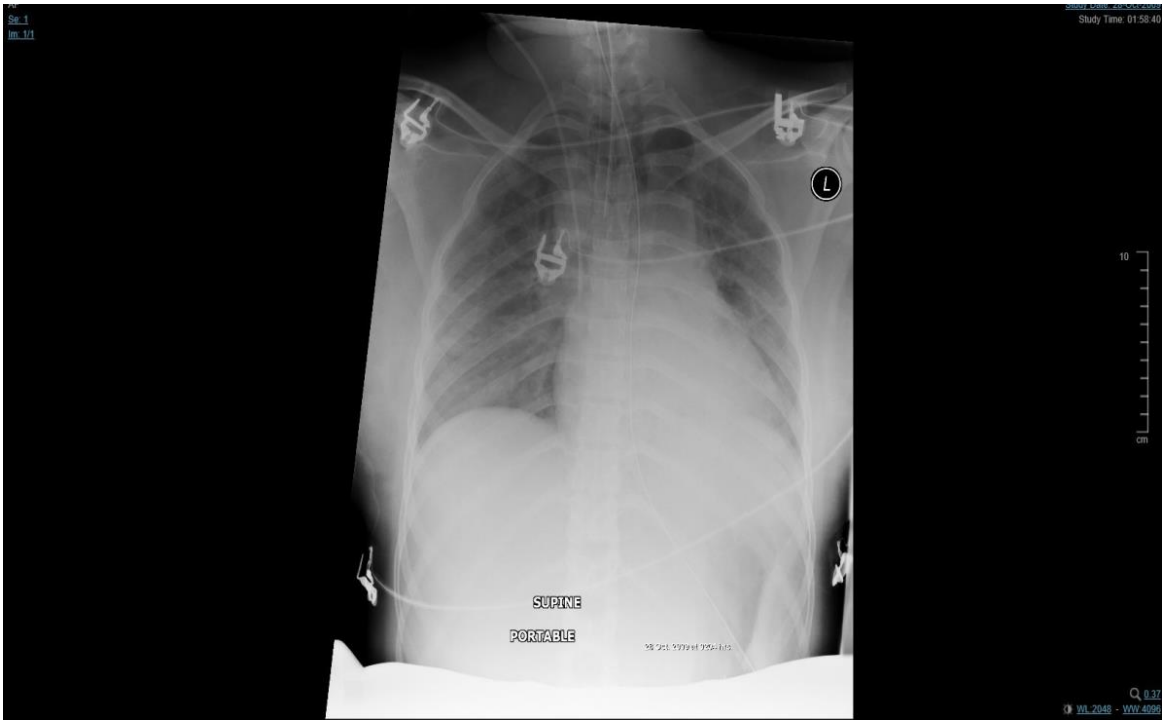

Fig-2: Chest X-ray, showing left mid and lower lung collapse consolidation, with left sided pleural effusion, widening of the superior mediastinum, and some scattered alveolar shadowing

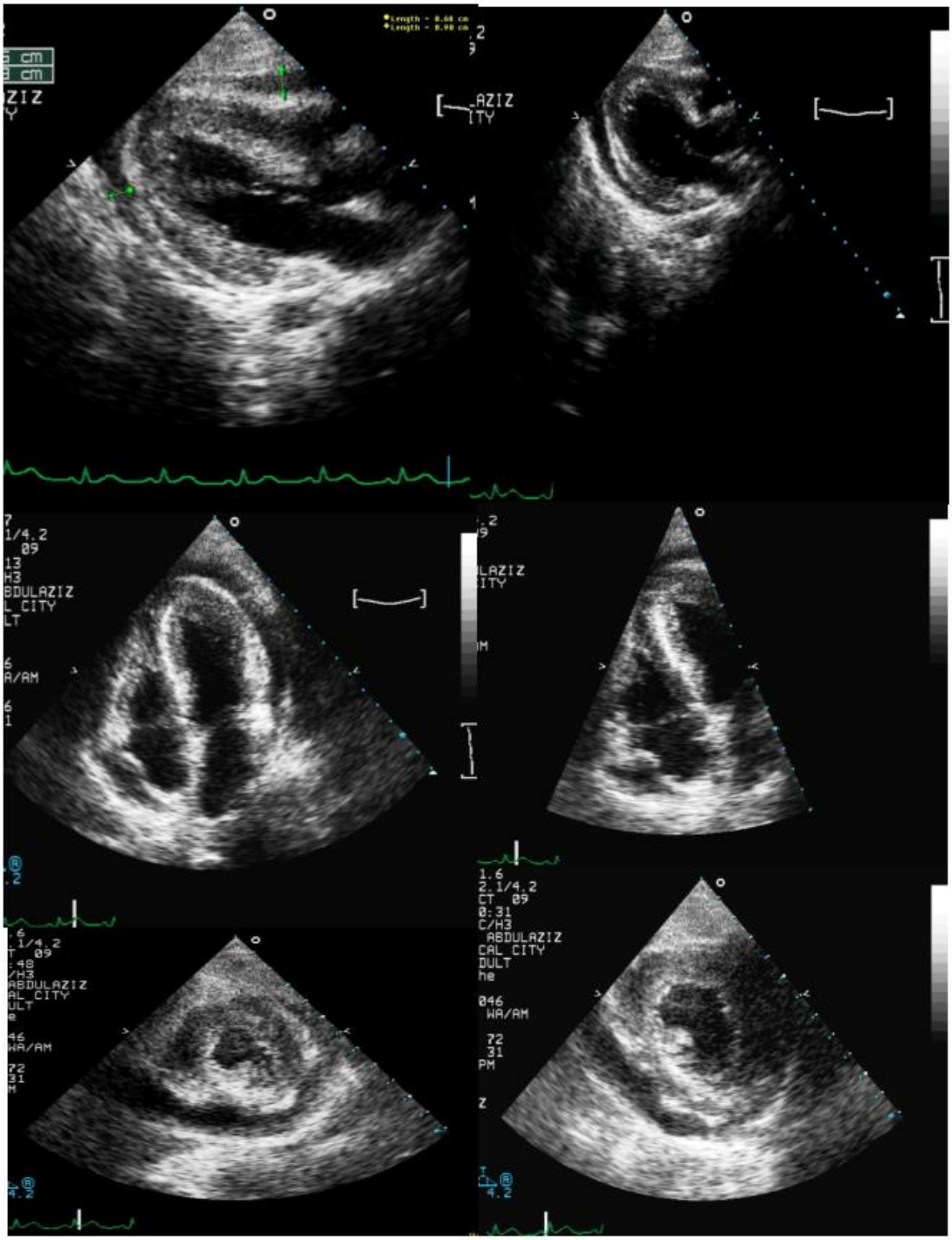

Fig-3: Echocardiogram demonstrating moderate concentric left ventricular hypertrophy (myocardial edema manifested by increased wall thickness); left ventricular systolic function is moderate to severely reduce; ejection fraction is 25%–35%; moderate to severe global hypokinesis of the left ventricle; and moderately sized pericardial effusion, with early signs of cardiac tamponade. The right ventricle is normal in size and function, with normal function and structure of the valves

The patient was moved to the medical cardiac intensive care unit 12 hours after the emergency laparotomy, and pericardiocentesis was ultimately performed to relieve possible pericardial tamponade. The patient was continued on inotropes, and a clinical diagnosis of FM was made by the treatment team. The patient was considered for extracorporeal membrane oxygenation (ECMO), but this was ultimately not performed. She also received intravenous steroids, high-dose polyvalent immunoglobulin, and intravenous antibiotics (oseltamivir, piperacillin/tazobactam, vancomycin, meropenem, and metronidazole). During her stay of 4 days in the medical cardiac intensive care unit, serial echocardiograms were performed until the

last day, when they showed normalization of the left ventricle. The patient was then extubated and transferred to the ward for 2 weeks' observation. She delivered a healthy infant with a cleft lip 7 months later. In October 2018, the patient was seen healthy, with a normal ECG and MRI (Figure 4).

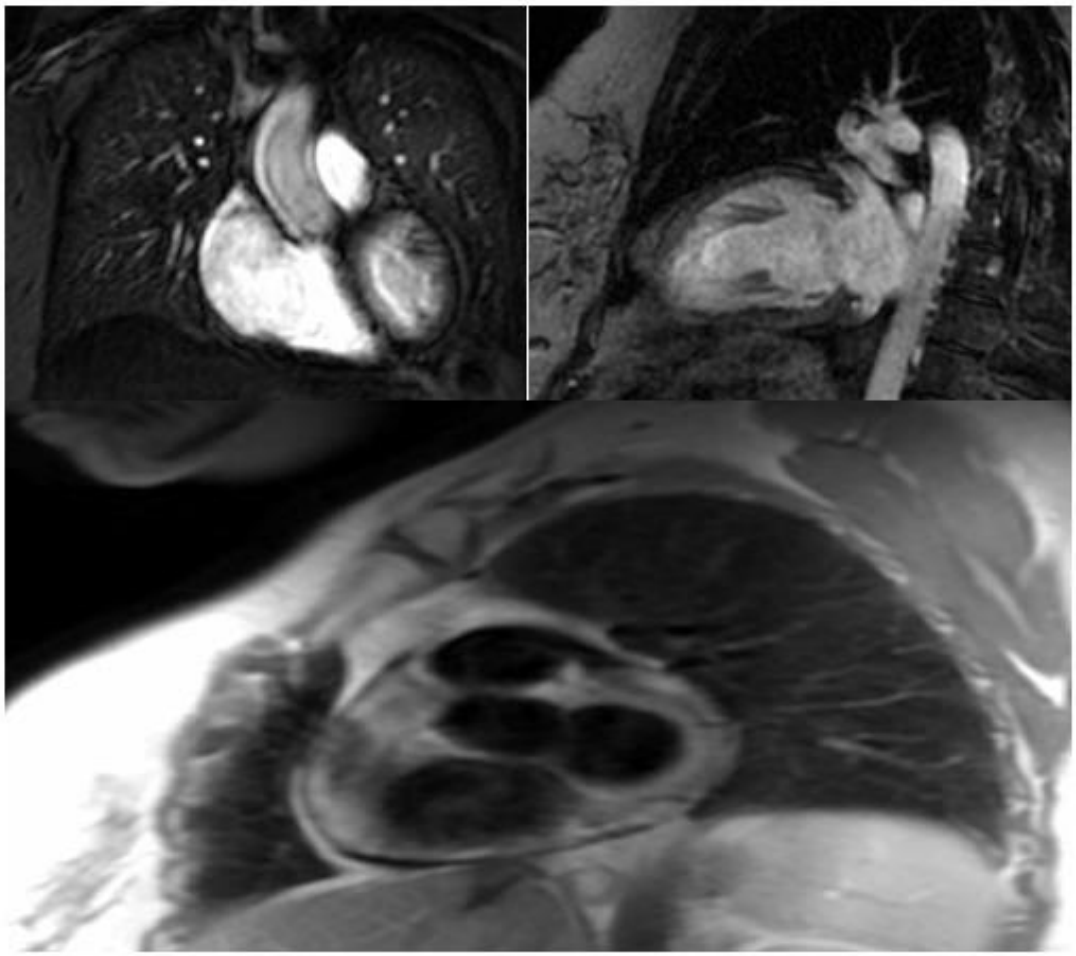

Fig-4: MRI shows Normal LV and RV size and systolic function, no abnormal enhancement of the LV or RV myocardium, mild RA and LA enlargement, any significant valve lesion, pericardium is of normal thickness with no pericardial enhancement

CASE DISCUSSION

FM is an inflammatory process that occurs in the myocardium and causes acute-onset heart failure. The onset of cardiac symptoms is abrupt, and the initial presentation is often cardiogenic shock. Echocardiography is essential for the diagnosis of FM, which features severe systolic dysfunction and increased wall thickness, reflecting myocardial edema, which is similar to the presentation of our patient. However, in acute but nonfulminant myocarditis, the left ventricle is dilated, with a normal wall thickness. Cardiac catheterization and coronary angiography are often necessary to exclude acute ischemia as a cause of acute heart failure [5-7]. The definitive diagnostic technique is percutaneous endomyocardial biopsy (EMB) [8]. EMB should be performed for patients with FM, severe ventricular arrhythmias, or advanced heart block (class I indication) according to the recommendations of the American Heart Association/American College of Cardiology/European Society of Cardiology. EMB is beneficial and effective for the differentiation of lymphocytic myocarditis from giant cell myocarditis and eosinophilic myocarditis [9, 10]. EMB and coronary angiography could not be performed for our patient due to her pregnancy, as well as the fact that EMB should be performed in centers with great experience, proven safety, and availability of appropriate pathology techniques, which were not available at our center at that time. On initial presentation, these patients require aggressive hemodynamic support with positive inotropic drugs, intra-aortic balloon pump, or other mechanical circulatory support, such as ECMO, given that significant improvement in left ventricular function will often occur. The role of immunosuppressive therapy in the treatment of FM remains unclear. A number of randomized clinical trials have assessed the efficacy of immunosuppressive therapies, such as steroids, intravenous immunoglobulins, and interferon, for the resolution of myocarditis. Overall, these trials have

failed to demonstrate a beneficial effect of immunosuppression [11-13]. Currently, the only option for patients with end-stage or irreversible heart failure is supportive care until heart transplantation is performed.
To our knowledge, this is the first case of FM in primigravida from our region.

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