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CASE REPORT: MYOCARDITIS, A RARE PRESENTATION OF ENTERIC FEVER

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Abstract:
Enteric fever is a disease which predominantly presents with features of high grade fever, abdominal pain, constipation and headaches. Enteric fever is generally thought of as an infection which usually affects the gastrointestinal system of the body. It has certain cardiovascular complications as well which albeit rare, but still very important to consider regarding the severity of the condition. There have only been so far only a few case reports regarding this rare complication of enteric fever. We present a case of enteric fever induced myocarditis in a young male patient. The presentation is that of a cardiomyopathy of any other cause which makes it a challenging case to diagnose and it makes it basically a diagnosis of exclusion. Cardiac MR (CMR) forms the main diagnostic tool for its diagnosis with supporting evidence from echo and troponin levels. The main management is the same as any other cardiomyopathy with ACE inhibitors, beta blockers and diuretics forming the main treatment strategy. Our patient recovered completely from the myocarditis with no long lasting cardiovascular outcomes. The purpose of our study is to add to the much deficient knowledge regarding the cardiovascular complications of enteric fever and point to the clinicians that they have to vary of such a presentation when they come across enteric fever patients.

Keywords: *Myocarditis, Enteric fever, Cardiac MR (CMR), Cardiac Function, Outcome*

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INTRODUCTION:

Enteric fever is a disease which is caused by a bacteria, Salmonella typhi, and is still a major disease burden in developing and under-developed countries. The clinical presentation of enteric fever varies from a mild illness which presents with features of low grade fever, headache, fatigue, malaise, loss of appetite, cough, constipation and rose spots to some cases in which it can result in fatal complications such as intestinal perforations, gastrointestinal hemorrhages, encephalitis, cranial neuritis and rarely myocarditis[1]. Myocarditis typically presents with non-specific clinical symptoms, and can easily be missed in the absence of a high index of clinical suspicion[1]. Myocarditis is usually caused by viruses such as cocksackie virus or entero viruses and in immunocompetent individuals it is very rarely caused by a bacteria[1]. *Salmonella* generally causes enteric fever but it can also be a rare cause of myocarditis even in healthy individuals[2].

Patients having enteric myocarditis will present just as any other case of myocarditis. Patient will have shortness of breath, chest pain and hypoxemia[3]. The resulting inflammation will result in worsening of the systolic function of heart and as a result there will be signs and symptoms of heart failure[3]. Several new diagnostic methods, such as cardiac magnetic resonance imaging (CMR), are useful for diagnosing myocarditis. Echo and ECG can also be done and they perform an ancillary role in diagnosis and their findings are generally considered as non-specefic[3]. Endomyocardial biopsy may be used for patients with acute dilated cardiomyopathy associated with hemodynamic compromise, those with life-threatening arrhythmia, and those whose condition does not respond to conventional supportive therapy[3]. The diagnosis is based on exclusion of the more common causes of myocarditis like viral infections[3]. Treatment is mainly via antibiotics which will depend on the culture and sensitivity and antibiotic resistance pattern of the local area and routine cardiac failure medications in the form of diuretics, ACE inhibitors or ARBs and beta blockers[9]. The outcome of enteric induced myocarditis varies from case to case but it is generally considered to have a favorable outcome and majority of the patients are able to regain back their baseline cardiac function[10].

CASE PRESENTATION:

We put forward a case of a young 26 years old male that initially presented with complaints of high grade fever, abdominal pain and constipation. He had multiple visits to his local hospital for which he received oral ciprofloxacin 500 mg BD for 7 days but he didn't improve. His fever was peaking up-to 104.6 F and he was very toxic on arrival to hospital. On examination he had a fever of 103.8 F with a pulse rate of 68 bpm and a blood pressure of 100/65 mm Hg. On his abdominal palpation there was mild tenderness in the left upper quadrant and there was palpable splenomegaly. His chest auscultation showed normal vesicular breath sounds with no evidence of any respiratory infection. His cardiovascular examination did not reveal any significant findings. His lab reports are given below:-

S.No	Lab investigation	Unit	Patient Value	Reference Value
1	WBC	Cells/mm^3	3700	4500-11000
2	Hemoglobin	g/dl	9.6	12-16
3	Platelets	Cells/mm^3	98000	150000-450000
4	LDH	U/L	672	140-280
5	Urea	mg/dl	53	10-50
6	Creatinine	mg/dl	1.2	0.6-1.2
7	ALT	U/L	78	10-50
8	ALP	U/L	128	40-129
9	Na	mEq/L	147	135-150
10	K	mEq/L	3.6	3.5-5.0
11	Coxsackie virus serology		Negative	
12	HIV serology		Negative	
13	Epstein Barr virus serology		Negative	
14	HSV serology		Negative	

For his abdominal pain an abdominal ultrasound was done which showed an enlarged spleen measuring 14 cm. His blood cultures were sent which came out to be positive for Salmonella typhi, indicating a diagnosis of enteric fever. He was initially started on Inj Ceftriaxone 500mg BD for 7 days but patient fever and symptoms did not improve. As a result antibiotic regimen was changed to Tab Azithromycin 500mg BD and Inj Meropenem 500mg TDS based on the culture and sensitivity report. He was started on dual antibiotics bases on the local antibiotic guidelines as the enteric fever had become very resistant in our domestic setup. Patient fever responded well to antibiotics. However over a couple of days he started having symptoms of shortness of breath, chest pain and orthopnea. His chest auscultation now revealed fine inspiratory crepitations bilaterally. His cardiovascular examination did not show any significant findings. He had a blood pressure of 100/60 mm Hg and a pulse of 110 bpm. He had also developed moderate pedal edema which was not present before. Due to his cardiac symptoms an echo was done which showed an ejection fraction of 36% with moderately impaired systolic function. Troponin levels were done which came out to be 2.6 (0-0.6). He had no such previous cardiac history. His ECG was done which showed sinus tachycardia with a rate of 105 bpm, normal axis, no bundle branch block and non-specific ST-T changes. All the important and common causes of myocarditis were ruled out and hence there was a strong suspicion of enteric induced myocarditis and a cardiac MR was planned which showed an increased wall thickness, patchy areas or regions of high T2 signal intensity indicating focal or regional edema and Increased myocardial early gadolinium enhancement ratio. These findings were suggestive of myocarditis and thus patient was labelled as having enteric induced myocarditis. For his heart failure symptoms he was also started on Inj furosemide 40mg BD (1mg/kg), ACE inhibitors (Tab Ramipril 2.5mg OD) and beta blockers (Tab Bisoprolol 5mg OD) and patient responded well to treatment. He was followed up regularly as on outpatient and his cardiac functions came back to baseline on his follow-up echo with his ejection fraction returning back to 62 % after a period of 3-4 months.

DISCUSSION:

Enteric fever is a systemic illness and it can affect a variety of organ systems in the body. Most of the complications occur in the third week of the illness and the most common of them is intestinal perforation[4]. Other organ systems affected include pulmonary, rheumatological, central nervous system, and hepatobiliary problems[4]. Cardiovascular complications occurred in 1%-5% of patients and include a spectrum of myocarditis and endocarditis as the main complications, and pericarditis and arteritis, which occur less often[4]. In the younger age group, myocarditis can have similar clinical and electrocardiographical features as that of ischemia[5]. Any apparent ischemia associated with myocarditis may be due to localized inflammation or coronary spasm. Myocarditis is often assumed by exclusion of other causes of increased cardiac enzymes and electrocardiographic changes. Cardiac MR may show myocardial edema and myocyte damage[5].

Myocarditis is an unusual association of enteric and has only a few reported cases, the first one having been reported in 1884. It is an inflammatory disorder affecting the cardiac muscle and may mimic myocardial ischemia or be totally asymptomatic during its course and then abruptly causing nonspecific hemodynamic instability and symptoms[6]. Because of the non-specificity of symptoms, it can be easily missed in the absence of high clinical suspicion[6]. It is important to note that myocarditis can be acute or fulminant and the mode of presentation and prognosis may vary depending on the presentation. Acute myocarditis presents as an indistinct illness leading to gradual hemodynamic alterations and can end up as dilatation and impaired contractility of the ventricles, which is often a bad outcome in the long-term prognosis[7]. On the other hand, fulminant myocarditis presents as a distinct clinical entity with rapid and progressive hemodynamic deterioration but shows a substantial improvement in ventricular function on initiating treatment[7]. Echocardiography can be utilized to differentiate between the two conditions. Patients with fulminant myocarditis had near normal left ventricular diastolic dimensions but increased septal thickness at presentation, while those with acute myocarditis had increased diastolic dimensions but normal septal thickness[7]. In our case the patient was having fulminant myocarditis as the onset of his symptoms were very abrupt and severe. His echo also showed normal diastolic ventricular dimensions and had an increased septal thickness. Upon initiation of his treatment he quickly responded well, highlighting the need of prompt treatment initiation and follow up.

Enteric myocarditis will lead to cardiovascular insufficiency which is the most common cause of death in the second week of illness[8]. It has been postulated that the pathogenesis of Salmonella typhi is dependent on the amount of inoculum size, virulence,

immune-host response, history of previous exposure, and local protective factors[8].

The treatment of enteric myocarditis and associated cardiomyopathy is no different from other variants of myocarditis[9]. Effective treatment of enteric myocarditis with supportive management and antibiotics should be initiated; some studies also show beneficial use of dexamethasone[9]. Non-steroidal inflammatory drugs (NSAIDs) should be best avoided to prevent disruption in the healing of the myocardium[9]. The systolic dysfunction can be encountered by blocking the neurohormonal cycle leading to heart failure by initiating angiotensin-converting enzyme (ACE) inhibitors, beta-blockers, and diuretics[9]. In our case we started the patient on Inj furosemide 40mg BD (1mg/kg), ACE inhibitors (Tab Ramipril 2.5mg OD) and beta blockers (Tab Bisoprolol 5mg OD). Patients should also be followed up in the long-term to document ventricular recovery with noninvasive imaging[10]. On his follow up echo after 30 days of treatment initiation, he had an ejection fraction of 62% returning back to its baseline showing completely recovery back to pre-infection state.

CONCLUSION:

Enteric fever has a multitude of presentations and enteric myocarditis is a rare yet an important diagnosis to consider as its timely management is of utmost importance. Due to the rarity of myocarditis in enteric fever it can easily be missed so a very high index of suspicion is needed. Troponin and echo findings can give a supportive diagnosis of enteric myocarditis and cardiac MR can confirm the diagnosis. Prompt initiation of treatment is very important as it can lead to a rapid recovery and save the permanent myocardial damage. Diuretics, ACE inhibitors or ARBs, beta blockers and appropriate antibiotics based on culture and sensitivity reports forms the mainstay of therapy. Serial follow-up with echo should also be done as to monitor the ventricular function and should be carried out until the ventricular function returns back to normal. In majority of the cases, cardiac functions will return back to baseline as shown in our study which signifies that enteric fever myocarditis has got a favorable outcome with no permanent cardiovascular disability.

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