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Case Report

Shigella gastroenteritis with myocarditis and acute kidney injury: a case report

Salini N. R.*, Srikantan S., Hitha Sebastian

Department of General Medicine, Government Medical College, Thiruvananthapuram, Kerala, India

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***Correspondence:**
Dr. Salini N. R.,
E-mail: salininora@gmail.com

ABSTRACT

Shigella are a well-known cause of acute gastrointestinal infections but sometimes it can present with extraintestinal manifestations. Extra intestinal complications of shigella includes seizures, encephalopathy, reactive arthritis, acute kidney injury of Haemolytic-Uremic Syndrome (HUS). Other severe manifestations can involve the myocarditis. severe hyponatremia, bacteremia, and a leukemoid reaction. Shigella myocarditis refers to a very rare complication during or after a Shigella infection. Viral infections are more common cause of acute myocarditis. Bacterial myocarditis is very rare but only few cases shigella myocarditis have been reported in the past. This article presents a 38year old male presenting with a case of shigella gastroenteritis complicated by myocarditis and acute kidney injury. He was presented with dysentery and chest pain with electrocardiogram showing ST-T changes, his echo showed global hypokinesia with low ejection fraction. Stool examination revealed shigella species. This event was resolved with treatment of antibiotics.

Keywords: Shigella, Myocarditis, Rhabdomyolysis, Hemolytic uremic syndrome

INTRODUCTION

Shigella is a non-spore forming gram negative bacterium belongs to the enterobacter family. It mainly affects the intestine and causes bloody diarrhoea, abdominal pain, and fever. There are four types of Shigella bacteria that affect humans — Shigella sonnei, Shigella flexneri, Shigella boydii, and Shigella dysenteriae. The bacteria, after entering the body attacks the epithelial lining of the colon, triggering inflammation of the cells. A combination of direct bacterial invasion and a massive inflamatory response cause severe damage to the colonic mucosa.

Shiga toxins are type 2 ribosome-inactivating proteins; that consist of an A subunit surrounded by 5 identical B subunits, which are responsible for the toxin's ability to enter target cells.[1] Shigella dysenteriae produces its toxin via activation of the chromosomally located Stx gene; but the EHEC Stx-1 and Stx-2 are produced via lysogenic incorporation of genes carried into the bacterium by lambdoid bacteriophages, followed by the lytic cycle that allows for toxin release.[1] Extra-intestinal manifestations include bacteraemia, reactive arthritis metabolic abnormalities, acute kidney injury, haemolytic uremic syndrome and encephalopathy. Cardiac manifestations are very rare in shigellosis.[2]

We present the case of a patient admitted in our hospital with simultaneous gastroenteritis and myocarditis associated with an infection by shigella.

CASE REPORT

A 38-year-old male with no significant past medical history presented to the emergency room with fever, abdominal pain and bloody diarrhoea of 3 days duration. Upon further inquiry, he mentioned that he had food intake from an outside restaurant before the diarrhoea started. After admission, he started experiencing intermittent pressure-like chest pain, which was mid-sternal and

associated with diaphoresis and breathlessness. He had signs of dehydration.

On admission his vitals pulse 98/min blood pressure 90/60 mm of Hg, SpO2-99%. His first electrocardiogram (ECG) taken outside the hospital showed tachycardia (Figure 1). The patient was not on any medication. His breathlessness got worsened, and he developed severe hypotension with reduced urine output. An ECG taken four hours after admission showed inverted T waves in leads I, II, III, AVF, and V4 to V6 (Figure 2). An emergency echo done showed global LV hypokinesia with a low ejection fraction of 37% and moderate LV dysfunction. (Figures 3 and 4). His lab values shown in the table 1. His cardiac troponin I level was high. His blood investigations revealed mild anaemia, thrombocytopenia and acute kidney injury. The platelet count was 86,000/µl, and the haemoglobin concentration was 10.3 g/dl. Despite having no prior kidney disease, his serum creatinine increased to 5.7 mg/dL and potassium to 5.4 mmol/l. His creatinephospho kinase and urine myoglobin values were high at the time of admission. Abdominal ultrasonography revealed normal echogenicity in both kidneys, with no findings suggestive of chronic kidney disease. Arterial blood gas test results revealed metabolic acidosis. His peripheral smear showed toxic granules with mild thrombocytopenia and anaemia. Stool PCR was positive for Shigella spp. in this patient and stool culture yielded S. sonnei.

In view of persistent hypotension with shock he was treated with an injection of noradrenaline infusion along with IV fluids. Empirical antimicrobial therapy with injection of piperacillin tazobactum, and ciprofloxacin were given for 7 days. In view of worsened renal function test and oliguria hemodialysis was performed. After three days of treatment, the patient's condition improved. His fever and diarrhoea were subsided, and urine output returned to normal. Renal function tests and CPK values normalized subsequently. He was discharged on day 10 of hospitalization with normalization of ECG findings. He responded well to treatment with troponin-I level decreasing to normal. At the time of discharge his platelet count improved to 1.3 lakh, and the haemoglobin concentration was 12.6 g/dl.

At one-month follow-up, the patient remained asymptomatic, with a normal left ventricular ejection fraction (LVEF) and a normal ECG.

This patient was presented with bloody diarrhoea, shigella positive in stool studies, with high troponin levels, ECHO findings suggestive of global LV hypokinesia, these changes were reverted with treatment so most probably diagnosis is shigella gastroenteritis complicated by myocarditis. He also had oliguria with elevated urea, creatinine and creatine phosphokinase with positive urine myoglobin which was improved with dialysis so a provisional diagnosis of shigella dysentery with prerenal acute kidney injury secondary to rhabdomyolysis was made.

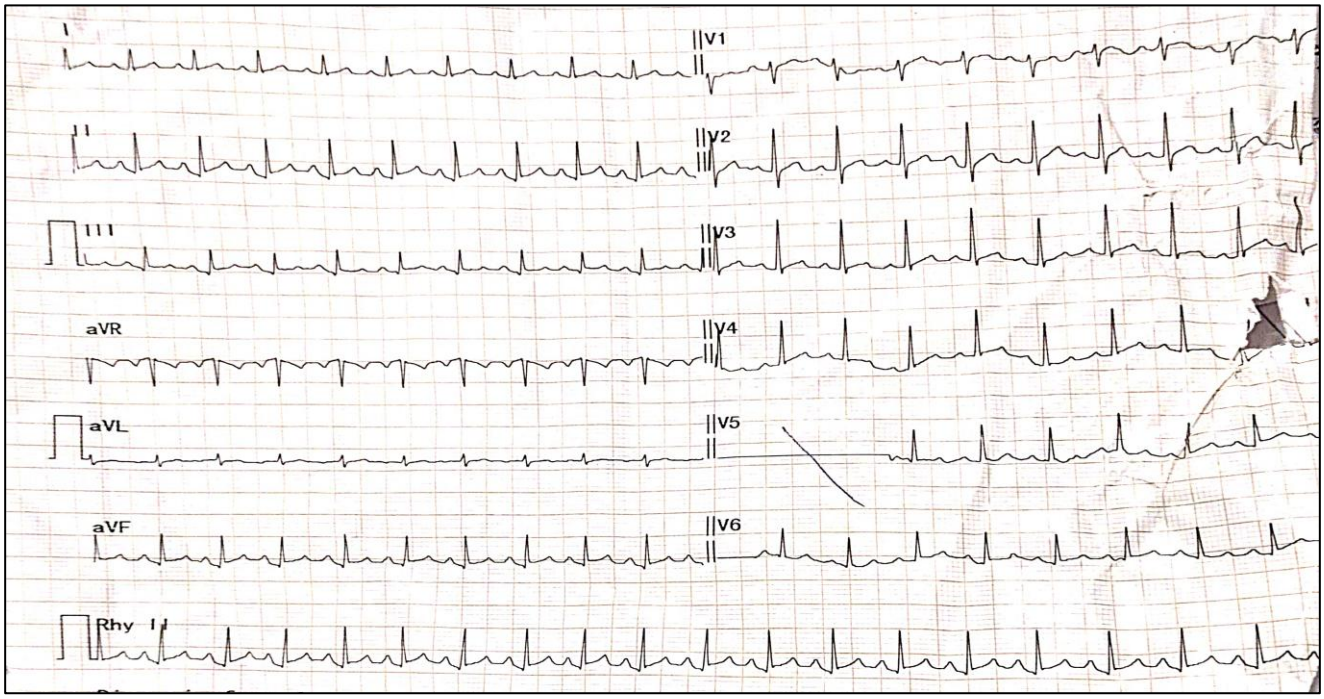

Figure 1: ECG showing tachycardia-first ECG of the patient.

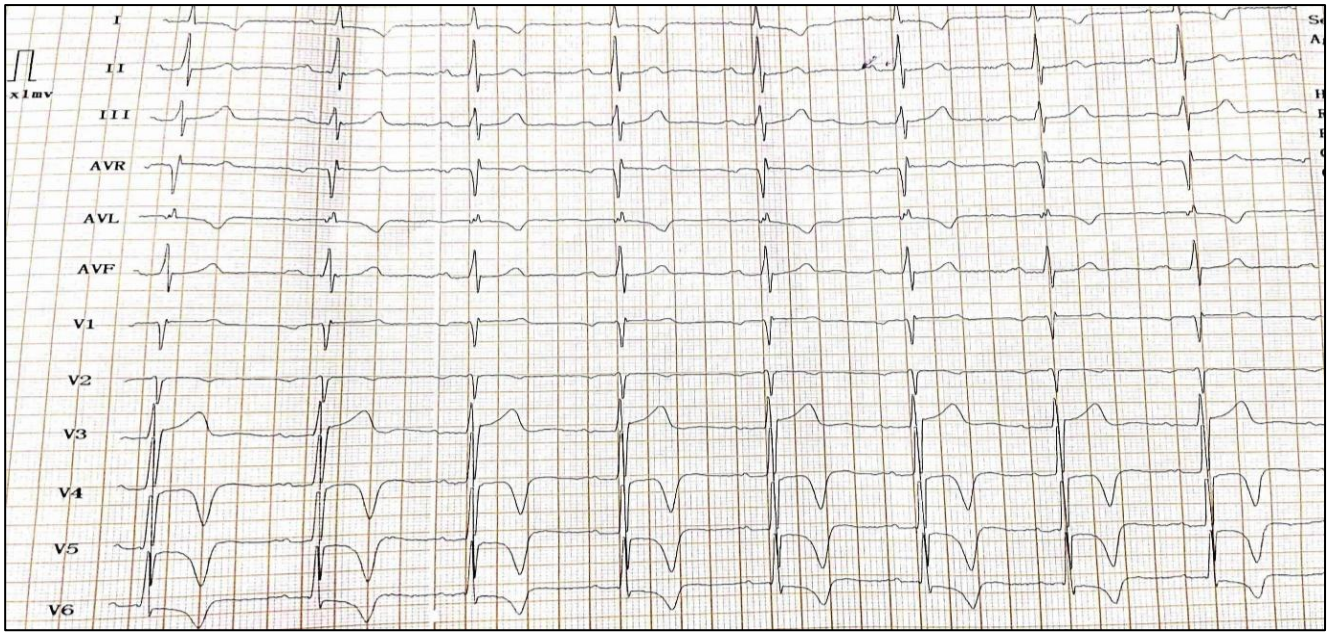

Figure 2: ECG showed inverted T waves in leads I, II, III, AVL, AVF, and V4 to V6.

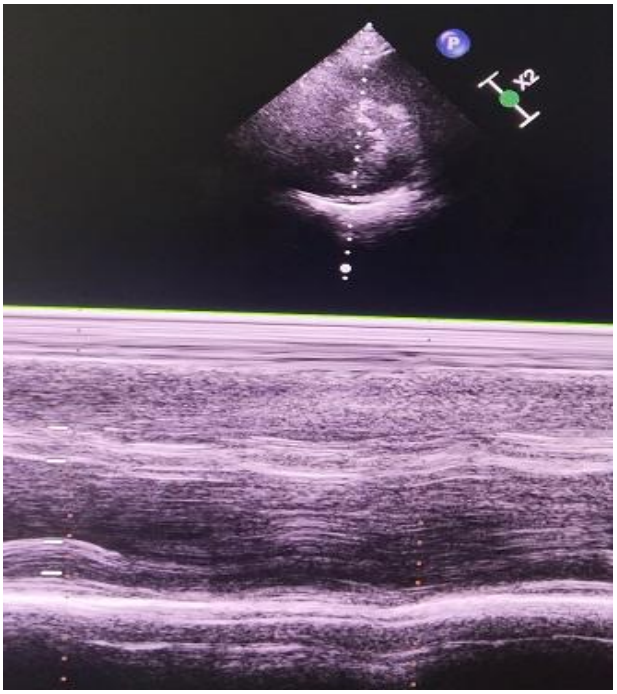

Figure 3: ECHO showing moderate LV dysfunction and global LV hypokinesia.

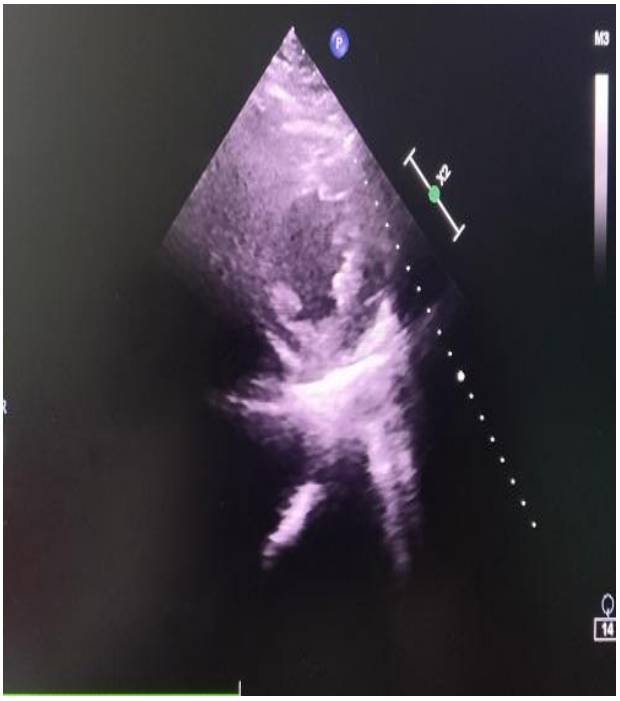

Figure 4: ECHO showing global LV hypokinesia.

Table 1: Lab values of the patient.

	On admission	Day of discharge
Hb	10.3	12.6
Platelet count	86000	1.3 lakh
Total count	4600	10800
PCV	48	28
ESR/CRP	64/8	18

Continued.

	On admission	Day of discharge
RBS	123	112
Total bilirubin/direct bilirubin	2/0.5	1.8/1
SGOT/SGPT	41/27	43/38
B urea/s creatinine	139/5.72	27/1.1
Na/k	131/5.4	134/3.9
Stool culture	Shigella	
Fever profile **IgM lepto** **IgM scrub** **Malarial parasite** **Widal tests**	Negative	
TSH	2.64	
S. calcium	9.30	9.20
S. magnesium	1.88	1.98
Troponin I	1.55	0.02
CK MB	54.3	
CPK	4410	220
LDH	1337	150
Lipase/amylase	372/150	

Normal laboratory values: Hb:13-17 g/dl, TC: 4000-10,000 cells/mm^3, Platelets:1.5-4.0 L cells/mm^3, ESR: ≤20 mm/hr, CRP: 0.3-1mg/l, RBS: <140 mg/dl, TB: 0.1-1.2 mg/dL, SGOT: 5-40 U/l, SGPT: 5-40 U/l, Serum creatinine: 0.8-1.3 mg/dl, Blood urea: 5-20 mg/dl.

Total protein: 6-8.3 g/dl, albumin-3.5-5.5 g/dl. scalcium: 8.5-10.5 mg/dl. sphosphorus: 3.4-4.5 mg/dl. INR: 0.8-1.1, TSH: 0.4-4mIU/l, CKMB: 0-24IU/l, CPK-20-170 IU/l, LDH-140-280 U/l, troponin I:<0.04 ng/ml.

Hb-haemoglobin, TC-total count, ESR-erythrocyte sedimentation rate, SGOT: Serum glutamic oxalo acetic transaminase, SGPT: Serum glutamic pyruvic transaminase, CK MB-creatine kinase-Myoglobin binding, TSH-thyroid stimulating hormone, CPK-Creatine phosphokinase, LDH-lactate dehydrogenase.

DISCUSSION

Shigella infection typically causes a watery diarrhea; but the development of hemorrhagic colitis indicates toxin production and risk for extra-intestinal disease, like HUS.[3] Once the Shiga toxins enter in to the circulation, they target organs by binding to Gb3 receptors located in the kidney and brain.[3] This causes Shiga toxin entrance into renal tissues, followed by inflammation, lysis, and destruction of those cells. Destruction of glomerular cells by toxin can cause complications like hematuria, and hemolytic uremic syndrome.[3]

The most common cause of myocarditis in developed countries is a viral infection. Coxsackie B viruses, enteroviruses, adenovirus are commonly associated with myocarditis. Acute bacterial myocarditis due to Shigella sonnei gastroenteritis is a rare phenomenon.[2] Only few cases were reported in literature. The mechanism of Shigella sonnei toxin induced myocardial damage could be similar to that of the diphtheria toxin, that works by reducing protein synthesis.[3] Other mechanisms include direct invasion of myocardium or immunologically mediated myocardial damage as occurs with other extra-intestinal manifestations of shigellosis, such as reactive arthritis.[4] Carco et al was reported case of 19-year-old young adult with myocarditis due to shigellosis in the form of acute EKG changes and a murmur that resolved in 5 days.[5] Sherif Eltawansy et al reported acute myocarditis secondary to shigella sonnei gastroenteritis.[2] Jayakrishnan et al reported children presented with cardiac manifestation and ECG EC changes due to shigella infection.[6] Vieira et al reported case of myocarditis hasalso been reported secondary to infection with Shigellaboydii.[7]

The gold standard test for detection of myocarditis needed an endomyocardial biopsy, but this invasive examination is only recommended in rare circumstances.[2] Medical management of myocarditis is usually dependent on the haemodynamic status of the patient and treating complications.[2] Shigella gastroenteritis is not commonly associated with rhabdomyolysis; but severe infection can lead to complications including acute kidney injury although specific co-occurrence with rhabdomyolysis is rare. Goldfarb et al reported a case of shigella infection with AKI (acute kidney injury) and rhabdomyolysis.[8]

The gold standard for the diagnosis of shigella is the isolation and identification of the pathogen from faecal material. Shigellosis is generally a self-limiting disease, and the decision to prescribe antibiotics is predicated on the severity of disease, the age of the patient, and the likelihood of further transmission of the infection.[9]

Effective antibiotic treatment reduces the average duration of illness from approximately 5–7 days to approximately 3 days and also reduces the period of Shigella excretion after symptoms subside.[9] Ciprofloxacin (1 g/day for 3 days) is recommended as first line treatment. Other antibiotics recommended included ceftriaxone, Azithromycin, pivmecillinam and some fifth-generation fluoroquinolones.[10]

In summery we are reporting a case of a 38-year-old male with shigella gastroenteritis who developed chest pain, and positive troponins with ECG and ECHO findings more in favour of shigella myocarditis. This patient had oliguria with elevated s creatinine, CPK and which was improved with dialysis, so a diagnosis of shigella infection with prerenal acute kidney injury secondary to rhabdomyolysis was made.

CONCLUSION

Myocarditis secondary to shigella gastroenteritis is rare, that requires prompt recognition and treatment to improve outcomes. This article emphasizes the need for physicians to consider bacterial gastroenteritis as a cause of myocarditis, especially in patient with non-responding gastroenteritis, to prevent severe cardio vascular compromise.

Shigella infection causing rhabdomyolysis and acute kidney injury is rare. Dehydration and severe hypovolemia can cause this complication. Fluid resuscitation and antibiotics are the better treatment option to avoid complications.

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