



Case report

Pericardial salmonella with cardiac tamponade and ventricular wall rupture: A case report

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HIGHLIGHTS

- Patient with *Salmonella* pericarditis complicated by tamponade and ventricle rupture.
- *Salmonella enteritidis* bacteremia secondary to a cardiac source.
- Promptly diagnose and treat patients with hemodynamic compromise from tamponade.
- Consider a cardiac etiology when faced with *Salmonella* bacteremia.

ARTICLE INFO

Article history:

Received 7 November 2015

Received in revised form

17 March 2016

Accepted 17 March 2016

Keywords:

Salmonella pericarditis

Pericarditis

Tamponade

Ventricle rupture

salmonella enteritidis

ABSTRACT

Introduction: Non-typhoidal Salmonella (NTS) is mostly restricted to gastroenteritis; however, we report a case of Salmonella pericarditis complicated by tamponade and spontaneous ventricular wall rupture. **Case presentation:** A 67-year-old male presents to the Emergency Department with complaints of fevers, chills and body aches. A chest radiograph displayed an infiltrate and an electrocardiogram suggested acute pericarditis. An echocardiogram revealed a small pericardial effusion without tamponade. Broad-spectrum antibiotics were initiated until Salmonella was discovered in blood cultures. The hospital course was complicated by sudden decompensation, and a repeat echocardiogram displayed a large effusion with constrictive physiology. During a pericardial window, the tissue was noted to have a thickened appearance with a complex effusion. The following day, the patient developed increased chest tube drainage, hypotension and acidosis, requiring an emergent sternotomy. The right ventricle was friable and had spontaneously ruptured. After ventricular repair and pericardiectomy, the tissue was sent for cultures and pathology. The specimen revealed Salmonella enteritidis. Treatment with ceftriaxone and ciprofloxacin was initiated. On postoperative day four, the patient was successfully extubated. Repeat blood cultures were negative.

Discussion: In our review of literature, only 19 cases of NTS pericarditis have been reported. Prior to our case, salmonellosis resulting in ventricular rupture has been reported once. Early diagnosis and treatment is crucial in minimizing morbidity and mortality. Clinical suspicion based on electrocardiogram and hemodynamic assessment is critical in suspecting pericardial effusion in a patient with nonspecific symptoms and Salmonella bacteremia. The key to recovery involves aggressive treatment, including pericardiectomy and antibiotic treatment.

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1. Introduction

Salmonella is a non-spore forming, gram-negative, facultatively anaerobic bacillus. Non-typhoidal salmonella (NTS) is mostly restricted to gastroenteritis secondary to food contamination;

however, cardiovascular manifestations may occur though are extremely rare [1,2]. Here, we report a case of *Salmonella* pericarditis complicated by spontaneous cardiac tamponade and ventricular wall rupture. A cardiac tamponade results from an accumulation of fluid around the pericardium, which causes reduced cardiac output and hemodynamic compromise. It is a syndrome that is classified as a medical emergency as it is associated with a high mortality if not addressed in a timely fashion.

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2. Case report

A 67-year-old gentleman presented to the emergency department with complaints of fevers, chills, body aches and productive cough ongoing for five days. He also noted dyspnea at rest and on exertion. He denied any sick contacts or significant pet exposure. He traveled to the Philippines earlier that year.

His past medical history is significant for hypertension, diabetes mellitus, hyperlipidemia, hepatitis B and a previous stroke without any residual deficits. The patient is a retired nurse of thirty-seven years, reporting no tobacco or illicit drug usage and occasional social alcohol use. In the emergency department, initial vital signs yielded a blood pressure of 130/71 mm of Mercury, heart rate of 95 beats per minute, respiratory rate of 20 breaths per minute, temperature of 102.4° Fahrenheit and an oxygen saturation of 95% on room air. Physical examination revealed bibasilar, inspiratory

crackles and decreased air entry but was otherwise unremarkable. Electrocardiogram (ECG) revealed ST-segment elevations in leads II, III, aVF, V5 and V6 without reciprocal changes (Fig. 2). A chest radiograph displayed bilateral pleural effusions with possible bibasilar opacities (Fig. 1). Complete blood count results included a white blood cell count, 15.4 K/ μ L; hemoglobin, 13.8 g/dL; hematocrit, 39.2%; and platelets, 257 K/ μ L. Chemistry panel showed sodium, 125 mmol/L; potassium, 4.6 mmol/L; chloride, 93 mmol/L; CO₂, 18 mmol/L; blood urea nitrogen, 30 mg/dL; and creatinine, 1.9 mg/dL. A bedside echocardiogram was conducted, which revealed a small pericardial effusion without tamponade physiology and preserved biventricular function. Blood cultures were drawn, and the patient was subsequently admitted for sepsis secondary to community-acquired pneumonia and acute pericarditis. He was started on broad-spectrum antibiotics. The following day, preliminary blood cultures revealed a gram-negative bacilli bacteremia.

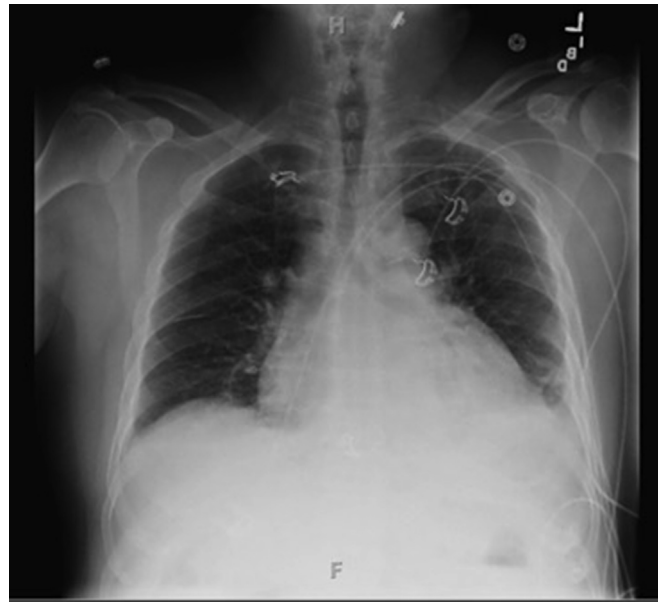


Fig. 1. Chest Radiograph – A chest radiograph exhibiting an enlarged cardiac silhouette and blunting of the costophrenic angles bilaterally.

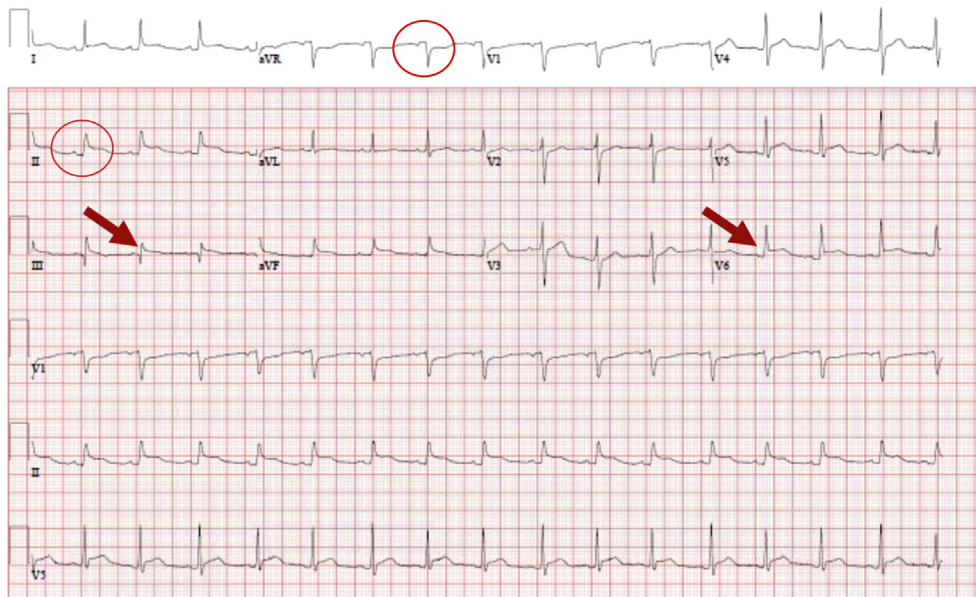


Fig. 2. Electrocardiogram – A 12-lead electrocardiogram revealed ST-segment elevations in leads II, III, aVF, V5 and V6 without reciprocal changes. PR depression is also noted in lead II with PR elevation in lead aVR, which is specific for pericarditis.

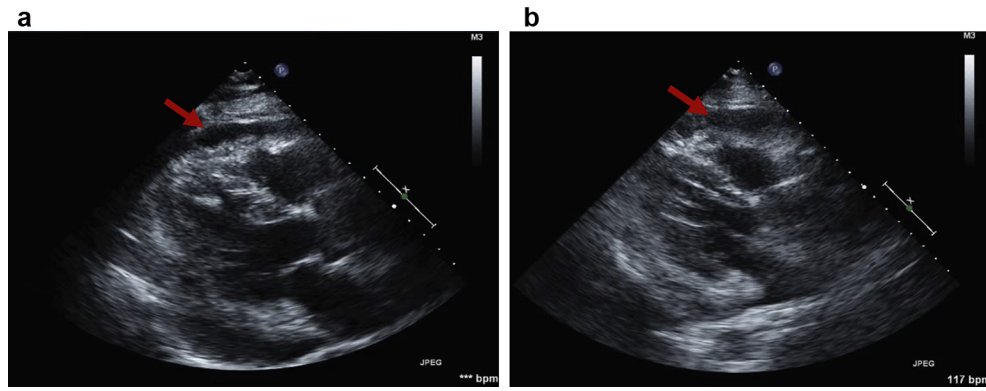


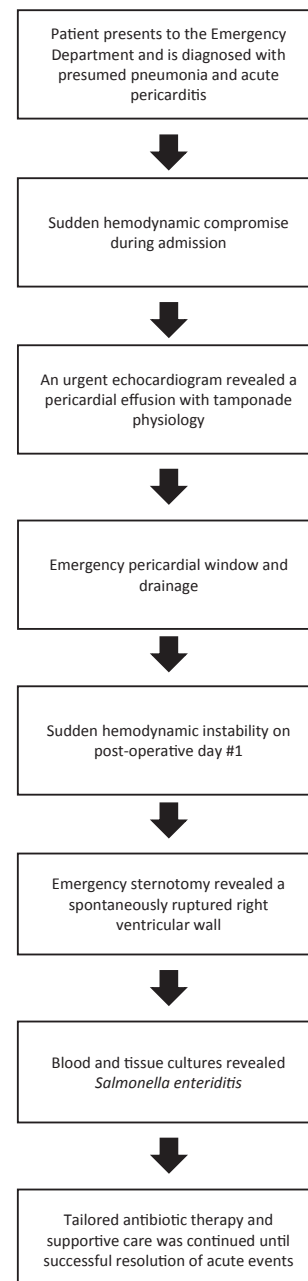
Fig. 3. a: Echocardiogram – The initial echocardiogram shows a pericardial fibrinous pericardial effusion without tamponade physiology. **b: Echocardiogram** – Second echocardiogram reveals an increased pericardial effusion consistent with pericardial tamponade and right ventricular wall collapse.

During the hospitalization, the patient suddenly decompensated becoming hypoxic and hypotensive. An urgent echocardiogram showed no vegetations but displayed a large fibrinous pericardial effusion with constrictive physiology consistent with a pericardial tamponade (Fig. 3). This diagnostic decision was based upon echocardiographic findings of tamponade complicating hemodynamic status. Due to the fibrinous and exudative nature of the effusion, it was felt that the patient would benefit from a surgical pericardial window placement and drainage rather than undergoing a needle pericardiocentesis. Intraoperatively, the pericardium was noted to have a thickened appearance and a complex effusion was encountered. The pericardial tissue was mobilized through the subxiphoid approach with concurrent fluid removal. Two chest tubes were placed, and the patient was sent to the cardiovascular intensive-care unit after hemodynamic stability was reached.

The following day, the patient suddenly developed a large amount of chest tube drainage, hypotension and acidosis, requiring an emergent sternotomy. Upon retraction of the sternum, the right ventricle was noted to be friable and had spontaneously ruptured adjacent to the right atrial appendage and AV groove, where there was a significant amount of active bleeding. After repairing the ventricle, removing the thrombus material and completely excising the pericardium, the visceral pericardium was sent for cultures and pathology. During this time, the initial blood culture drawn in the emergency department was identified as being *Salmonella enteritidis*.

Interestingly, the intraoperative pericardial tissue specimen from the first intervention revealed *Salmonella enteritidis* as well. Antibiotics were tailored and changed to ceftriaxone and ciprofloxacin. On postoperative day four, the patient was successfully extubated and supportive care was continued. Of note, the patient's post-operative period was complicated by transaminemia, acute kidney injury and a partially-occlusive acute deep vein thrombosis involving the right femoral and popliteal veins.

Repeat blood cultures showed clearance of the bacterium and an echocardiogram revealed only a trace pericardial effusion. The patient's stay in the cardiovascular intensive-care unit was lengthy and complicated; however, the patient made a remarkable recovery and was eventually discharged to a sub-acute rehabilitation center with a peripherally-inserted central catheter for ceftriaxone infusions for a total of eight weeks.



3. Discussion

Acute pericarditis is a common but complex condition that may arise secondary to an infectious or non-infectious etiology. Common infectious causes include *Streptococcus pneumoniae*, other *Streptococcus* species and *Staphylococcus aureus* [3]. Though *Salmonella* is often limited to the gastrointestinal tract, infection involving other sites may occur producing characteristic clinical syndromes. Cardiovascular salmonellosis is an extremely rare extra-intestinal manifestation, which can present as endocarditis, myopericarditis or endovascular infection. *Salmonella* pericarditis often presents with cardiac or pulmonic symptomatology, but typical signs of pericardial disease such as pulsus paradoxus or the presence of a friction rub may be absent similar to our patient. Characteristic ECG changes of pericarditis are also uncommon.

In our review of literature, only 19 cases of NTS pericarditis have been reported [1,2,4]. While patients with NTS pericarditis commonly have an identifiable immunosuppressed state, bacteremic patients with endovascular infections were typically older and had underlying conditions similar to our patient [4,5]. Prior to our case, salmonellosis resulting in ventricular rupture has been reported once in 1964 [6]. The patient in the previously reported case similarly complained of progressively worsening dyspnea over preceding weeks and *Salmonella* bacteremia. Unfortunately, the patient deteriorated despite appropriate antibiotic therapy and eventually died. The postmortem autopsy revealed diffuse fibrinous deposits on the pericardial surfaces along with a thin right ventricular wall in the region of the apex. A transmural wall rupture was noted in the area of the affected right ventricular wall similar to our patient.

Patients with NTS bacteremia do not typically exhibit gastrointestinal symptoms; therefore, it often goes unsuspected. Early diagnosis and treatment is crucial in minimizing morbidity and mortality [4,7]. Clinical suspicion based on electrocardiogram and hemodynamic assessment is critical in suspecting pericardial effusion in a patient presenting with nonspecific symptoms and *Salmonella* bacteremia [7]. Furthermore, diagnosis is consolidated based on echocardiogram, which aids to exclude vegetations. The key to clinical recovery involves aggressive treatment, including urgent pericardiectomy and appropriate antibiotic treatment. Surgical drainage is preferred over pericardiocentesis due to fibrinous nature of the exudate and provides assessment of myocardial wall integrity. Unfortunately, overall mortality remains extremely high [1]. Additionally, in any patient presenting with a *Salmonella* bacteremia, it is vital to consider an underlying cardiac etiology.

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Ethical approval

Verbal ethical approval obtained from patient and patient's family.

Sources of funding

There is no source of funding for this manuscript.

Author contribution

Please specify the contribution of each author to the paper, e.g. study design, data collections, data analysis, writing. Others, who have contributed in other ways should be listed as contributors.

Gaurav Chand, DO – primary author and researcher.

Ruby Jhaj, MD – secondary author.

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Prabhat Sinha, DO – secondary author.

Patrick Alexander, MD – secondary author.

Conflicts of interest

We have no conflicts of interest to report for this manuscript.

Guarantor

The Guarantor is the one or more people who accept full responsibility for the work and/or the conduct of the study, had access to the data, and controlled the decision to publish.

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