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Salmonella hadar pericarditis

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Acute purulent pericarditis is an infrequent but potentially life-threatening disease. Early and effective treatment requires a knowledge not only of the clinical course but also of the organisms commonly responsible. However, unusual microbes may be incriminated in immunocompromised hosts. We report a case of *Salmonella hadar* pericarditis in a female with systemic lupus erythematosus (SLE) being treated with oral steroids. Although other non-typhoidal Salmonella species have previously been implicated in the causation of bacterial pericarditis [1–9], infection of the pericardium with this species of Salmonella has not previously been reported.

Case Report

A 28-year-old female diagnosed to be suffering from systemic lupus erythematosus over six months preceding a current illness and maintained on prednisolone 10 mg daily was admitted to the Aga Khan University Hospital, Karachi, Pakistan, with a one-month history of low grade fever and progressive dyspnea on exertion leading to acute exacerbation of dyspnea over three hours prior to presentation. Systemic review was unremarkable. In particular, there was no history of fever, chest pain, cough or expectoration, bowel symptoms or recent procedures of any kind.

On examination, she was tachypneic with respiratory rate of 30 per min, and had a blood pressure of 100/90 mm Hg with a 10 mm drop in systolic pressure during inspiration. Her temperature was 39°C and a heart rate of 88 per minute. Pulse was of low volume. Internal jugular veins were distended to the angle of the jaw in sitting position and she had cushingoid facies. Apex beat was not palpable and heart sounds were reduced in intensity. There was no pericardial rub. Chest was clear on auscultation and liver span was 10 cm in the right mid-clavicular line. The remainder of the examination was unremarkable except for a malar rash and trace pedal edema.

Total leucocyte count was 10.9×10^9/L with 84% polymorphonuclear leukocytes. Erythrocyte sedimentation rate was 63 mm in the first hour by Westergren method. ECG showed low voltage and sinus tachycardia. Chest x-ray showed an enlarged cardiac silhouette. Echocardiography revealed massive pericardial effusion. Pericardiocentesis was performed and 1200 ml of thick purulent fluid was removed. On analysis, this pericardial fluid was exudative with a total leucocyte count of 59,000/ml of which 85% were polymorphonuclear leukocytes. Gram-stain revealed gram negative bacilli. The fluid was cultured on blood agar, chocolate agar, and Mac Conkey agar and incubated aerobically and anaerobically.

Salmonella species was isolated from this pericardial fluid and later sent to the Central Public Health Laboratory in Collindale, London, England, where it was typed as *Salmonella hadar phage type 2* which belongs to the non-typhoidal salmonella group C_2. Blood, collected prior to antibiotic therapy, was culture-negative following incubation of seven days. A stool specimen was also cultured and was found to be negative for any Salmonella species. However, antibiotics had been started prior to the collection of the stool specimen. Another stool specimen, collected after the resolution of the pericarditis also did not grow any organism.

The patient was initially administered ampicillin 1g intravenously every six hours and cefotaxime lg every eight hours. After the identification of the organism and its *in vitro* sensitivity to both ampicillin and cefotaxime, ampicillin was withdrawn. Continuous drainage of the pericardial cavity was performed. Because of continuing hypotension, ampicillin (450 mg/kg/day) was reinstituted and cefotaxime was discontinued on the seventh day of hospitalization. There was good clinical response with only a small amount of pericardial effusion seen echocardiographically 14 days following hospitalization. The patient was discharged on the 18th post-admission day.

Discussion

The etiology of acute bacterial pericarditis is diverse [9]. Cardiac involvement with *Salmonella*, both typhoidal and non-typhoidal infection, has been recognized for many years. Myocarditis and endocarditis due to Salmonella typhi and other Salmonella species have been previously documented [10,11]. The first case of pericarditis in association with typhoid fever was described in 1844 by Volz [12], and reviewed by Levin and Hosier [13]. The first case of pericarditis due to non-typhoidal Salmonella infection was reported by Cohen et al in 1936 [14]. In 1986, Haggman et al summarized 23 cases of non-typhoidal Salmonella pericarditis reported since 1936 [15]. The most common species of Salmonella isolated was *S. typhimurium*. Two patients were found to have systemic lupus erythematosus but none had *Salmonella hadar* isolated from the pericardial cavity. This case report represents the first proven case of *Salmonella hadar* pericarditis.

Non-typhoidal serotypes of Salmonella are found in the intestinal flora from a wide range of animal species which are then transmitted from these animal reservoirs to humans primarily through foods of animal origin [16]. The reservoir of *S. hadar* in the United Kingdom has been identified in turkeys, although the organism has been isolated less frequently from chickens and occasionally from bovines [17]. In Pakistan, turkey is not a commonly eaten food, although chicken and other wild poultry birds may be the carriers of this organism in the country. The prevalence of *S. hadar* in the poultry flocks or bovine herds in Karachi, Pakistan remains unknown.

The pattern of infection characteristic of *S. hadar* in humans has until now been restricted to the abdomen, producing serious illness in elderly and debilitated patients. *S. hadar* has emerged as a common serotype isolated from patients suffering food poisoning in the United Kingdom [18].

It is difficult to determine the source and route of infection in our patient with pericarditis due to *S. hadar*. There was no history of food poisoning or gastrointestinal symptoms. Stool cultures were attempted after the organism was isolated from the pericardial fluid. However, possibly because of the patient's prior antibiotic therapy, the organism was not isolated. It can, nevertheless, be speculated that this patient may have acquired the species from water contaminated with poultry or bovine feces and carried in the gastrointestinal tract without any clinical manifestation. Because of the immunocompromised state, systemic spread occurred which manifested as massive pericardial effusion.

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