A purulent pericarditis caused by *Salmonella typhimurium*

Fusun Can,1 Muge Demirbilek,1 Birsel Erdem,2 Ugur Ciftci,3 Mine Tunaoglu3 and Yahya Laleli3

1Department of Microbiology and Clinical Microbiology, Bas¸kent University School of Medicine, Ankara, Turkey
2Department of Microbiology and Clinical Microbiology, Ankara University School of Medicine, Ankara, Turkey
3Duzen Laboratories, Ankara, Turkey

A case of *Salmonella typhimurium* pericarditis is reported. The diagnosis was based on blood and pericardial effusion cultures.

**Introduction**

Salmonellosis is an important health problem worldwide (Haggman et al., 1986). Poor sanitation conditions and lack of careful monitoring of food processing cause large outbreaks of salmonellosis, especially in developing countries. *Salmonella* infections may occur in different clinical forms in humans. Extraintestinal *Salmonella* infections are seen in less than 30 % of cases. Localized infections are an important form of *Salmonella* infection and are frequently seen during salmonella bacteraemia, but may also occur with enteric fever or gastroenteritis. Urinary tract, central nervous system, soft tissue, bone and joint infections due to different *Salmonella* serotypes have been reported previously (Aksoycan et al., 1958; Erler et al., 1994). Although the pericardium is rarely involved, accounting for less than 2 % of cases, the mortality rate is as high as 50 % or more (Cohen et al., 1987).

We describe a case of purulent pericarditis caused by *Salmonella typhimurium*.

**Case report**

A 42-year-old man was admitted to hospital with a history of fever and dyspnoea. He gave a 2-week history of chest pain, palpitations, nausea and diarrhoea. On examination he was tachycardic and tachypnoeic with a temperature of 38.2°C, and heart sounds were diminished. Examination of the abdomen showed no organomegaly or ascites. Jugular venous pressure was raised and peripheral oedema was not observed. The white cell count was normal (8.6 × 10⁹ l⁻¹), the erythrocyte sedimentation rate was 75 mm h⁻¹ and the level of C-reactive protein was 58 mg l⁻¹. He was presented to us with a diagnosis of pericardial effusion demonstrated by transthoracic echocardiography. Pericardiocentesis was carried out and haemorrhagic fluid was drained. Three blood cultures were drawn. The patient died 24 h after he was admitted to the hospital. Gram-negative rods were seen by Gram staining and lactose-negative colonies grew on EMB agar (Oxoid) after overnight incubation of pericardial fluid culture. *Salmonella* was identified by sugar fermentation and gas and H₂S production on triple-sugar–iron agar and by urea hydrolysis tests. Identification was confirmed by the Vitek identification system (bioMérieux). Slide agglutination with polyvalent antisera for *Salmonella* serogroup O antigen showed that it was included in group B of the Kauffmann–White scheme. Based on the agglutination reactions with specific antisera to O and H antigens the organism was identified as *Salmonella typhimurium*. It was also isolated from blood cultures.

Both of the isolates (from pericardial fluid and blood) were found to be sensitive to ampicillin and ciprofloxacin.

**Discussion**

With the use of antibiotics in medicine, bacterial infection of the pericardium has become uncommon; however, is still a life-threatening condition (Badawi et al., 2002). Although Gram-positive bacteria such as *Streptococcus pneumoniae*, *Staphylococcus aureus* and *Streptococcus pyogenes* and Gram-negative bacilli are the most important causative agents of bacterial pericarditis, Gram-negative rods are isolated more frequently than Gram-positive bacteria from patients with pericardial infection (Klacsmann et al., 1977). Extraintestinal salmonellosis more frequently affects patients with some predisposing factor and/or underlying disease than patients without these features (Rodriguez et al., 1998). Pre-existing pericardial effusion due to uraemia, thoracic surgery and other factors and the immuno-status of patients are important predisposing factors for the development of purulent pericarditis. Transient salmonella bacteraemia from the gastrointestinal tract has also been previously noted (Sabeel et al., 1997). Our patient was known to have pericardial
effusion symptoms at least 2 weeks before his presentation. Gastroenteritis might have preceded the illness and *Salmonella typhimurium* could have entered the pericardium from the blood.

Purulent pericarditis is a rare complication of *Salmonella* infections (Doig et al., 1991). Cohen et al. (1936) described the first case of non-typhoidal salmonella pericarditis in a 36-year-old woman in 1936. The majority of cases of salmonella pericarditis were in children in the preantibiotic era; in the antibiotic era the majority of cases are in adults (Cohen et al., 1987).

Salmonella pericarditis cases are diagnosed by either pericardial fluid culture or Gram staining of pericardial fluid. Using these criteria, fewer than 30 non-typhoidal salmonella pericarditis cases have been reported in the literature (Kiughi et al., 1998). In a review of the published literature up until 1987 by Cohen et al. (1987), there were 10 cases of non-typhoidal salmonella pericarditis: seven of the patients had positive pericardial fluid cultures during life, and two had positive cultures and one had Gram-negative rods at autopsy. In our case we isolated *Salmonella typhimurium* from both the blood and pericardial fluid cultures. *Salmonella typhimurium* is the most common organism isolated in non-typhoidal suppurative pericarditis, representing about 50% of the reported cases (Sanchez-Guerrero & Alarcon-Segovia, 1990). Other *Salmonella* serotypes isolated from pericardial fluid in the literature are *Salmonella enteritidis*, *Salmonella paratyphi* A, *Salmonella choleraesuis*, *Salmonella newport*, *Salmonella panama* and *Salmonella paratyphi* D (Victor et al., 1997).

In conclusion, we reported a case of non-typhoidal salmonella pericarditis secondary to bacteraemia in a 42-year-old man. It is a rare complication of *Salmonella* infection and atypical clinical findings in particular cause high mortality rates.

**References**


