Salmonella typhimurium Pericarditis

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The salmonellae are primarily intestinal pathogens. The clinical picture after infection may vary from gastro-enteritis to “typhoid-like” illness, to intestinal upset followed by septicaemia. This septicaemic phase may localize to produce meningitis, osteitis, pneumonia, focal abscess. In addition, a septicaemic form occurs without any obvious preceding intestinal involvement; this form eventually shows localized lesions.

Though myocarditis occurring in the course of typhoid fever has been recognized for many years, endocarditis or pericarditis due to typhoid and other salmonella infections is much less common. In 1947 Shulman collected 17 cases of endocarditis, often superimposed on pre-existing valvular disease, and all fatal.

Pericarditis due to Salmonella typhi was apparently well recognized in the 19th century, but has been reported very rarely since. The first case of non-typhoid salmonella pericarditis was described by Cohen, Fink, and Gray (1936). This was a woman of 36 years, who developed pneumonia, pleurisy, and pericarditis, S. choleraesuis being recovered from the blood and faeces but not from the pericardial cavity. She recovered, without specific therapy, after a severe illness lasting 10 weeks.

In 1961 Levin and Hosier reviewed the published reports, collecting six further cases and adding one of their own. Four of these were children under the age of 2 years, and a total of five cases recovered. The organisms identified were S. typhimurium (4), S. paratyphi A, S. Blegdam, and S. Newport.

Case Report

This child, a girl aged 18 months, had been well until she developed a “cough and cold”, with fever, anorexia, and passage of 3 to 5 loose offensive stools daily. Three weeks later she was admitted to hospital with left-sided pneumonia, pleural effusion, and diarrhoea. Cultures from faeces and urine grew S. typhimurium. She was treated for 10 days with chloramphenicol and penicillin, with only slight improvement. At the end of 10 days she had a purulent pericarditis, confirmed by aspiration, which was treated with intrapericardial penicillin, the first cultures being sterile. Penicillin was continued. At the end of 3 weeks, no pus could be aspirated from the pericardium, and she appeared to be improved. During the following week, however, she developed congestive cardiac failure. From this time she deteriorated steadily, with marked venous engorgement, increasing cyanosis and dyspnoea except when in oxygen, oedema in spite of repeated mersalyl, enlarged tender liver, ascites, pleural effusion, oliguria, and albuminuria; she died 7 weeks after admission. The illness lasted 10 weeks.

Bacteriology. S. typhimurium was isolated from faeces and urine throughout the illness, from blood on two occasions during the first week in hospital, and on one occasion from the pericardial exudate.

Necropsy. The main findings, apart from pericarditis, were those of marked general venous congestion, pleural effusion, ascites, left organizing pneumonia, mural thrombosis in the superior vena cava and right auricle, and zonal necrosis of the liver.

The heart was small, fixed in the upper part of the pericardial cavity, and covered by exudate 2.0 to 3.0 cm. thick, both layers of pericardium being greatly thickened (Fig. 1 and 2). The exudate was thick, yellow/white, and almost caseous in appearance, most marked at the base of the heart where the great vessels, especially the superior and inferior venae cavae, were matted together by thick organized exudate. The pericardial sac was firmly adherent to the diaphragm and to the mediastinal pleura.

The heart itself showed no structural abnormality, and all the chambers were small. All valves were normal. Histological examination showed an organizing pericarditis, the “caseous” exudate being composed of eosinophilic debris and degenerate cells. The myocardium and endocardium were normal. No organisms were seen in sections, but at necropsy S. typhimurium was isolated from intestine, gall-bladder, and urine. Culture and guinea-pig inoculation of pericardial exudate for B. tuberculosis were negative. In the liver only a small rim of surviving liver cells was seen around portal tracts, and the findings were interpreted as gross venous congestion, with associated toxemia.
Discussion

Levin and Hosier (1961), in their review, stress the importance of early recognition and treatment. Chloramphenicol has been used in recent cases, with success, but recovery was also obtained in patients treated with penicillin and sulpha drugs, while the case in 1936 had no specific antibiotics. They recommend chloramphenicol, with prednisone during the first 4 to 5 days, as the treatment of choice. It may be that newer drugs such as combinations of trimethoprim and sulphamethoxazole will be of greater value in preventing the serious complications of the salmonella infections (Akinkugbe et al., 1968).

None of the patients described has produced the massive exudate seen in the present case. This may have been due to insufficient treatment, but the infant did receive 13·5 g. chloramphenicol during the first 10 days in hospital, together with a total of 25 mega units of penicillin. The fact that the later cultures and the post-mortem cultures from blood, lung, and pericardium were all negative suggests that the invasive stage had been controlled by the antibiotics. The organisms were still present in gall-bladder, intestine, and urine. The late inflammatory reaction in the pericardial sac, with its constrictive effects, complicated by the mass of necrotic tissue in the sac, produced a lethal degree of cardiac hindrance.

Summary

A case is described of an infant with Salmonella typhimurium septicaemia who developed pericarditis. Organization of the exudate produced a final picture of constrictive pericarditis.

References