Pericardial effusion complicating psittacosis infection

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SUMMARY A 13-year-old schoolboy developed a moderately large pericardial effusion during the course of infection with psittacosis. The value of echocardiography in differentiating between pericardial effusion and myocarditis is shown.

Pericarditis complicating psittacosis infection is rare and to our knowledge no well-documented case of pericardial effusion caused by this infection has been described.

Case report

A 13-year-old schoolboy was admitted to hospital with a 2-day history of headache, left-sided chest pain, cough, and thick clear sputum. There had been no similar illness among his 5 brothers or 7 sisters. The poultry on the farm where he lived had not been diseased recently.

On examination he looked flushed, ill, and toxic, with a temperature of 39.2°C, and a coated tongue. His respiratory rate was 20/minute and the pulse was 130/minute and regular. Blood pressure was 117/50 mmHg. There were expiratory rhonchi in both lower lobes. There were no abnormal heart sounds or murmurs and no friction rub was audible. There was no rash or lymphadenopathy.

The clinical diagnosis was bilateral bronchopneumonia. The chest x-ray film showed patchy inflammatory changes in both lower zones with a normal cardiac outline (Fig. 1a). The erythrocyte sedimentation rate was 54 mm in the first hour, Hb was 13.3 g/dl, and the white cell count 9800 x 10^9/l, with no neutrophilia. Sputum culture failed to grow bacterial pathogens. Acute viral studies showed normal antibody titres.

The patient was treated with ampicillin and cloxacillin to which oxytetracycline was added after 48 hours. The pyrexia subsided slowly and after 7 days these antibiotics were stopped. The pyrexia returned promptly and cotrimoxazole was started. At this time the erythrocyte sedimentation rate was 77 mm in the first hour and the Hb was 10.8 g/dl. Coombs' test was negative. A pericardial friction rub was audible and the electrocardiogram showed widespread ST and T wave changes consistent with pericarditis. A second chest x-ray film showed increase in the heart size, the contour suggesting pericardial effusion (Fig. 1b).

Fig. 1 (a) Chest x-ray film on admission. (b) Chest x-ray film showing pericardial effusion.
The patient was transferred to the Cardiac Unit of the Belfast City Hospital where an echocardiogram confirmed the presence of a moderate pericardial effusion (Fig. 2a). The patient showed no signs of pericardial tamponade so pericardiocentesis was not performed. With bed rest the pericardial effusion resolved spontaneously. Four days later the echocardiogram showed only a small amount of pericardial fluid. Two weeks after the onset of the illness the psittacosis/lymphogranuloma venereum complement-fixing antibody titre had risen from <10 on admission to 320.

The patient was transferred back to St. Columb’s Hospital where a further chest x-ray film showed restoration of a normal cardiac contour, and complete resolution of the inflammatory changes in the lung fields. The erythrocyte sedimentation rate fell to 21 mm in the first hour. The Hb was 12.2 g/dl and the white cell count was $8 \times 10^9/1$, with mild lymphocytosis.

The psittacosis antibody titre level remained 320 at the time of the patient’s discharge from hospital 4 weeks after the onset of his illness, but had fallen to 40 one month later. Fourteen weeks after the onset of infection, the patient was clinically well and the electrocardiogram had returned to normal. The diameter of the cardiac shadow was at the upper limit of normal. The echocardiogram showed no pericardial fluid (Fig. 2b). The psittacosis antibody titre was 20.

**Discussion**

Psittacosis in humans usually affects the respiratory system, the mode of infection being by inhalation. Isolated cases of cardiac involvement have been reported, usually taking the form of myocarditis (Coll and Horner, 1967; Dymock et al., 1971). Clinical evidence of pericarditis is rare. Definite clinical and serological evidence of psittacosis was found in only 3 out of 599 unselected cases of acute perimyocarditis reported by Sutton et al. (1967).

In the present case the diagnosis of acute psittacosis infection was established by the clinical features and the typical acute rise and fall in complement fixing antibody titre to psittacosis-LGV agent. There were no clinical signs of lymphogranuloma venereum. The source of infection remains unclear. The patient was responsible for cleaning out the chicken house weekly on the farm where he lived and would sometimes find a dead chicken there. However examination of the flock subsequently by a veterinary surgeon was negative for psittacosis. Grist and McLean (1964) reported 16 cases in whom there was no obvious source of infection.

The development of a substantial pericardial effusion, which was the most noteworthy aspect of this case, was established conclusively by the typical radiological and echocardiographic findings. In another case in which a pericardial effusion was suspected during life, necropsy showed only a little pericardial fluid and that most of the increase in the diameter of the cardiac shadow was the result of severe myocarditis (Dymock et al., 1971). The value of echocardiography in differentiating between myocarditis and pericardial effusion was seen well in the present case. It is surprising that pericardial
Pericardial effusion has not been described more frequently in human psittacosis. In birds it is one of the commonest findings (Yow et al., 1959). The increasing availability of echocardiography may show that small effusions are commoner than has been believed.

The relatively slow fall in the patient's temperature despite vigorous treatment with oxytetracycline, and the secondary rise in temperature after discontinuing this drug may have been because of difficulty in eradicating the infection from the pericardial effusion. The demonstration of such effusions and their differentiation from myocarditis may be useful in estimating the prognosis of patients with cardiac involvement in psittacosis. This patient's full recovery from a pericardial effusion stands in distinct contrast to the fatal outcome from myocarditis in the cases described by Dymock et al. (1971) and Vosti and Roffwarg (1961).

References

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