A progressive pericardial effusion caused by psittacosis

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SUMMARY A patient developed signs of a progressive pericardial effusion over a period of eight days. Diagnostic and therapeutic pericardiocentesis was performed. Viral titres to psittacosis rose from <1:8 to >1:256 indicating recent infection.

Chlamydial infections produce a wide variety of clinical problems but rarely affect the heart.¹ We describe a case of acute pericarditis caused by psittacosis in which a large pericardial effusion required pericardiocentesis.

Case report

In a 55 year old woman a central constricting chest pain developed and became worse over two days. It was associated with dry cough and breathlessness. There was no past history of heart disease. She was taking thyroxine (200 µg daily) for longstanding primary hypothyroidism and prednisolone (5 mg daily) for late onset asthma. She smoked 20 cigarettes per day.

On examination she looked flushed but was otherwise well. Her temperature was 38°C, her pulse was 86 per minute and regular, and her blood pressure was 160/90 mm Hg. The jugular venous pressure was not raised and the heart sounds were normal. No friction rub was heard. Chest examination revealed an expiratory wheeze and the peak expiratory flow rate was 270 l/min.

The white cell count was $13.7 \times 10^9/l$ (79% neutrophils) and the erythrocyte sedimentation rate was 68 mm/h (Westergren). Cardiac enzymes, the electrocardiograph, and chest radiograph were normal. Eighteen hours after admission left anterior pleuropericardial pain and atrial fibrillation developed. Examination of the arterial blood gases showed pH 7.438 (normal range 7.35-7.45), partial pressure of oxygen (P_{O_2}) 11.1 kPa (normal range 12-15 kPa), partial pressure of carbon dioxide (P_{CO_2}) 4.63 kPa (normal range 4.5-6.0 kPa) on air. An electrocardiogram confirmed atrial fibrillation but

was otherwise unchanged, and the chest radiograph remained normal. Pulmonary embolism was suspected and treatment with heparin (15 000 units twice daily) was started. Twelve hours later a further electrocardiograph showed sinus rhythm with atrial supraventricular extrasystoles and concave upward ST elevation in both the anterior and inferior leads. Anticoagulation was stopped because of suspected pericarditis. A repeat chest radiograph showed no change of heart size.

Three days after admission she developed further pain, rapid atrial fibrillation and the blood pressure fell to 90/60 mm Hg. A pericardial friction rub was heard at the left sternal edge. A chest radiograph now showed an increase in the size of the cardiac silhouette and a left basal pleural effusion. Cross sectional echocardiography confirmed a pericardial effusion and showed a cystic lesion in the region of the posterior tricuspid valve leaflet (fig 1). Antinuclear antibodies and anticardiac antibodies were negative; serum viral titres for Coxsackie B1-B6, influenza A+B, mumps, and psittacosis/lymphogranuloma venereum were all <1:8. A Mantoux reaction was positive at a 1:1000 dilution.

She improved after treatment with digoxin, indomethacin (50 mg three times daily), and erythromycin (500 mg four times a day). Sinus rhythm was restored, blood pressure rose, and she had less pain. An intermittent fever (up to 38.5°C) persisted.

During the next week the size of the pericardial effusion increased considerably on the chest radiograph (fig 2) and cross sectional echocardiograph. The jugular venous pressure rose to 9 cm and the blood pressure averaged 100/70 mm Hg. Diagnostic and therapeutic pericardiocentesis was performed because of the signs of progressive subacute tamponade. We withdrew 500 ml of blood stained pericardial fluid. Microbiological studies of the fluid including examination for acid and alcohol

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Fig 1 Cross sectional echocardiograph performed three days after admission. EFF, effusion; X, cystic lesion; RA, right atrium; RV, right ventricle; LA, left atrium; LV, left ventricle.

fast bacilli as well as cytological examination were negative. After the procedure the cystic lesion was no longer visible on cross sectional echocardiographs.

Fifteen days after admission the titre of antibody to psittacosis/lymphogranuloma venereum measured by complement fixation and immunofluorescence techniques rose to $> 1:256$ from the initial value of $< 1:8$. Oxytetracycline (500 mg four times daily) was started. The temperature became normal over five days and the left basal pleural effusion disappeared over two weeks.

A one month course of treatment was planned, but after three weeks the pericardial and pleural effusions and the chest pain recurred. The effusions resolved within one week with bed rest and indomethacin (500 mg four times daily). Oxytetracycline was continued for two months in total. No further relapses occurred during four months of follow up and the

chest radiograph and electrocardiograph became normal. The psittacosis/lymphogranuloma venereum titre fell to 1:8.

On enquiry the patient reported brief exposure to a parrot two weeks before admission. The owner refused to let us investigate the bird.

Discussion

Acute *Chlamydia psittaci* infection is usually transmitted by inhalation of the dried excreta of psittacine birds and in most cases produces a respiratory illness. The diagnosis is by serology, and an antibody titre of 1:16 to the group specific antigen of the psittacosis/lymphogranuloma venereum group is regarded as positive.² Cardiac involvement is rare and often complicates psittacosis related respiratory disease.³ Both endocarditis^{4,5} and myocarditis^{6,7} have been reported and the small number of necropsy reports show endocardial involvement as the major pathological feature.¹ We found only three reported cases of documented pericardial effusion caused by psittacosis.^{3,8} To our knowledge this is the first reported case of the development of a large pericardial effusion requiring pericardiocentesis after psittacosis. The rise and fall of the antibody titre to the *Chlamydiaceae* group specific antigen was considerable and there were no features of ocular or genital disease or cat scratch fever to suggest *Chlamydia trachomatis* infection, which is a cause of false positive results.⁹ The lungs were only slightly affected; she had a dry cough at presentation and a small left sided pleural effusion developed. The response to standard antibiotics was slow but this was a feature of the case reported by Kundu and Scott⁸ and may reflect the difficulty in

Fig 2 Chest radiographs taken on admission (a) and nine days after admission (b).

eradicating the organism from the pericardial effusion.

Sutton *et al*, in a review of 599 unselected cases of acute pericarditis, found nine cases with serological evidence of psittacosis, with pericardial effusions in two.³ In four cases heart disease was the only clinical manifestation. In six patients the diagnosis was by serology, with an antibody titre > 1:32. No titres > 1:16 were recorded in a control population of 1000. Five patients recovered completely but their treatment regimens were not reported.

The nature of the cystic lesion seen on initial echocardiography remains uncertain. It most probably represents encysted pericardial fluid and rupture during pericardiocentesis may account for its disappearance.

It is often difficult to obtain an accurate history of bird exposure in psittacosis.¹⁰ In the present case the history of exposure was obtained only after serological diagnosis. We therefore recommend the measurement of antibody titres of psittacosis/lymphogranuloma venereum titres in all cases of acute pericarditis regardless of pulmonary involvement. If the diagnosis is confirmed treatment with oxytetracycline should be continued for at least two months.

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