SHORT REPORTS

Pleuropericardial lesion in Q fever

In most reported series about half of all patients with Q fever have pneumonic lesions, yet pleurisy with effusion is uncommon. During an epidemic of Q fever affecting 100 New Zealand and British troops in Italy,[1 2] I encountered 50 with pneumonic lesions and five with pleural effusions—two with generalised unilateral effusions, two with encysted paravertebral effusions, and one with an interlobar effusion. Out of 300 cases reported from Southern California,[3] three had pleural effusions.

I can find no reported case of pericarditis with or without effusion, and it is not mentioned in current texts on Q fever. I therefore report our experience of Q fever in Southern Iran during the past five years. Out of 80 patients diagnosed clinically and serologically as having the disease, two had pericarditis with effusion and one pleural effusion.

Case 1

A 52-year-old Iranian man became ill on 13 April 1976 with fever, sweating, and headache and was treated at home. Twelve days later he developed chest pain, which was localised to the precordium and was aggravated by deep breathing. On admission to hospital his temperature was normal, pulse 72/min, and blood pressure 110/80 mm Hg. His heart was not enlarged. There was a loud pericardial friction rub over the left sternal border.

On 28 April chest radiography showed minor pneumonitis and plate atelectasis of the right lower zone and a costoparietal band in the right costophrenic sinus. The cardiac silhouette was somewhat enlarged, the right border passing directly to the diaphragm. On 4 May the lesion at the right lower zone had disappeared and the costophrenic sinus was clear. The cardiac silhouette was smaller and the angle between the right border and the diaphragm was less, suggesting a minimal pericardial effusion. On 25 April an electrocardiogram (ECG) had shown minimal S-T elevation and depression of T waves in leads I, aVL, aVF, and V 1-4. These changes persisted for 10 days. On 30 June the ECG was normal. The white cell count was 7×10^9/l (7000/mm^3) (polymorphs 64%, lymphocytes 33%) and the erythrocyte sedimentation rate 51 mm in the first hour. On 9 May the complement-fixing antibody titre to Q fever was 1/256.

The patient was given 90 mg prednisone daily for seven days and analgesics and the symptoms quickly subsided, the pleural friction subsequently disappearing. He was discharged on 9 May. A month later in London Sir Ronald Gibson reported that he had made a good recovery and that the cardiac function was normal.

Case 2

This patient, a 22-year-old Iranian man, had developed precordial pain on deep respiration in August 1976 while in Isfahan. He had a low-grade fever and dyspnoea. Pericarditis was diagnosed. After 25 days he was transferred to this hospital complaining of chest pain and loss of weight.

Temperature was 36·7 C, pulse 90/min, and blood pressure 110/70 mm Hg. Pericardial friction was heard over the precordial area during systole and diastole. The lung fields were resonant and air entry was normal. A week later the pericardial rub was barely audible. On 4 September chest radiography showed enlargement of the cardiac silhouette of the left ventricular type. The lung fields were clear. On 11 September the cardiac silhouette was smaller. On 2 September the ECG showed minimal S-T elevation in leads I, V5, and V6, and on 13 September flat and inverted T waves in leads II, III, aVF, V5, and V6. Complement-fixing antibody titre to Q fever was 1/8 on 6 September and 1/32 seven days later.

The patient was given ampicillin and co-trimoxazole and made a good recovery. He was discharged from hospital after 23 days and remained well. On 12 December the ECG was normal.

Case 3

A 40-year-old Englishman developed a febrile illness early in June 1976 that responded to ampicillin, and 10 days later noticed a sharp pain in the right chest on deep respiration, coughing, and when he lay on his right side. He sweated profusely while feverish and lost a lot of weight. Temperature was 36·7 C, pulse 80/min, and blood pressure 130/80 mm Hg. The trachea was not displaced and there was no clubbing. The heart was not displaced. There was dullness to percussion over the right lower chest posteriorly, with absent breath sounds and no vocal fremitus up to the fifth intercostal space. The tentative diagnosis was pleurisy with effusion due to tuberculosis or Q fever. He elected to return to the United Kingdom immediately without further investigation.

Ten weeks later Dr S A C Hunter, of the Epsom Chest Clinic, reported as follows: "Pleural biopsy showed a non-specific chronic inflammatory change. Pleural fluid showed 80% eosinophils. Q fever CFT titres were: on 30 July 640, on 5 August 320, on 10 August 160, and on 20 September 80. With a short course of oral steroids combined with tetracyclines the pleural effusion, which had persisted for several weeks, quickly resolved. Tetracyclines were continued for 8 weeks."

Comment

In areas where Q fever is endemic, and among people who may be at risk of infection, such as farming personnel, workers in abattoirs, and those who keep household pets, Q fever should be considered in the diagnosis of pericardial or pleural lesions.

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[1] Adams, A B, *et al*, *British Medical Journal*, 1946, **1**, 227.
[2] Caughey, J E, and Dudgeon, A, *British Medical Journal*, 1947, **2**, 684.
[3] Beck, M D, *et al*, *Public Health Reports*, 1949, **64**, 41.

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Symptom relief with levamisole in stage IV Hodgkin's disease

The anthelmintic drug levamisole was found to have important immunopotentiating effects on thymus-dependent lymphocytes. Ramot[1] showed in-vitro and in-vivo potentiation of T-cell function in patients with Hodgkin's disease. We used the drug in two patients with advancing Hodgkin's disease who had failed to respond to intensive chemotherapy, hoping to control progression of their disease by improving cell-mediated immunity. Both patients suffered troublesome fevers and night sweats and we report the dramatic response of these symptoms to levamisole.

Case reports

Case 1—A woman aged 27 presented in January 1967 with night sweats and cervical lymphadenopathy of nodular, sclerosing Hodgkin's disease. She received five courses of radiotherapy and chemotherapy, remaining in remission until May 1974. The mustine, vincristine (Oncovin), procarbazine, and prednisone (MOPP) regimen with bleomycin was then started for spleen and pancreatic node involvement and intractable night sweats. She was disease-free until January 1976 when symptoms recurred and night sweats became particularly troublesome. These were unremitting until September despite combinations of doxorubicin, bleomycin, vinblastine, and dacarbazine and oral cyclophosphamide, razoxane, methotrexate, and prednisone. Levamisole was then added to the latter combination in a dose of 200 mg/day at weekends only and the night sweats stopped after two courses. Three months later she was well and there were no further night sweats, though her disease otherwise remained static.

Case 2—A man aged 45 presented in September 1971 with Stage IA lymphocyte-depleted Hodgkin's disease. He remained well on radiotherapy until March 1973 when fevers, severe night sweats, and weight loss necessitated eight courses of MOPP chemotherapy. From September 1974 he had radiotherapy and intensive chemotherapy until October 1975, when symptomatic recurrence in liver and bone marrow necessitated change to doxorubicin, bleomycin, vinblastine, and dacarbazine. These regimens failed to control the persistent fevers and night sweats, and toxicity became unacceptable. He was started on levamisole 200 mg/day for three days each week in June 1976. There was a remarkable improvement in his well-being,