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.

I thank Dr S Varnoos and Dr A Farshid for permission to refer to their cases, and Mr S E Haratunian and Mr D Ehsani for the laboratory studies.

[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,

the severe night sweats stopped after two courses of treatment, and his temperature returned to normal over one month. No other active treatment was given and his symptoms remained well controlled for two months before the disease eventually progressed and he died.

Discussion

We attribute the relief of symptoms to levamisole. No other active form of treatment for Hodgkin's disease was used in the second patient during the asymptomatic period, and in the first patient three months of the same combination chemotherapy had completely failed to alleviate her symptoms. Control of severe night sweats occurred after two courses of levamisole in both cases.

The pathogenesis of the systemic manifestations of Hodgkin's disease is obscure and the mechanism of action of levamisole in abolishing the night sweats and fever in our patients is not clear. There is indirect evidence of circulating immune complexes in the plasma of patients with Hodgkin's disease[2 3] and their association with systemic symptomatology has been suggested.[3] Nephrotic syndrome in association with immune complexes and active Hodgkin's disease has been described.[1]

We speculate that levamisole potentiated the activity of phagocytes and thymus-dependent lymphocytes in these patients, with subsequent ingestion of immune complexes[3] or temporary elimination of disease-associated antigen.[5] We propose that levamisole may have a place in the management of Hodgkin's disease as an adjunct to radiotherapy and chemotherapy, especially in symptomatic cases.

[1] Ramot, B, *et al*, *New England Journal of Medicine*, 1976, **294**, 809.
[2] Sutherland, J C, *et al*, *Cancer Research*, 1974, **34**, 1178.
[3] Amlot, P L, Slaney, J M, and Williams, B D, *Lancet*, 1976, **28**, 449.
[4] Lokich, J J, Galvanek, E G, and Moloney, W C, *Archives of Internal Medicine*, 1973, **132**, 597.
[5] Order, S E, and Hellmann, S, *Journal of the American Medical Association*, 1973, **223**, 174.

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Use of hyperbaric oxygen in paralytic ileus

Paralytic ileus may complicate major abdominal surgery or present as a sequel to peritoneal infection. Rarely it may be purely neurogenic, usually after abdominal injury. Conventional treatment occasionally fails and abdominal distension and general toxicity increase. Inhalation of 100% oxygen, which has been used to treat abdominal distension for many years,[1] is thought to act by reducing the partial pressure of nitrogen in the blood and therefore increasing the speed of absorption of nitrogen from the bowel. There is every reason to believe that hyperbaric oxygen would have a similar action. While it is generally known that hyperbaric oxygen has a profound effect on anaerobic bacteria,[2] it is not so well known that it also kills or slows the growth rate of aerobic pathogenic bacteria.[3 4] It is thus reasonable to expect hyperbaric oxygen to be of value in paralytic ileus, whatever the cause, as part of treatment.

Patients, methods, and results

During the past five years 12 cases of paralytic ileus with varying and increasing degrees of distension and toxicity have been treated with hyperbaric oxygen at this hospital (see table). Ten cases occurred after acute infection—five from acute appendicitis, two from perforations of the large intestine (stab wound and caecal carcinoma), two with no obvious cause found at laparotomy, and one from empyema of the gall bladder—one after vagotomy and pyloroplasty, and one after laparotomy for abdominal trauma, at which a retroperitoneal haematoma was the only finding. All the patients had shown either no improvement or a worsening of their condition despite continuous gastric suction, intravenous fluids, and correction of biochemistry. When indicated laparotomy had been performed, the cause treated, sepsis drained, and antibiotics given.

Hyperbaric oxygen was begun on the second day of illness in four cases, the third day in five cases, and the fourth, seventh, and eight days in the remaining cases. It was given over one hour twice daily in a Vickers single-person chamber at two-and-a-half atmospheres, gastric suction and intravenous fluids being continued.

The patients received four to 10 hours of hyperbaric oxygen, and all were improved, 11 recovering completely (table). The remaining patient (case 7), with a perforated carcinoma of the caecum, died on the third day of treatment, although his bowel function was recovering. There were no complications referable to the hyperbaric oxygen.

Comment

It was surprising how quickly recovery occurred and how well the patients felt after even the first treatment. Measurement of abdominal girth gave no real indication of success, as an abdomen as tight as a drum at the beginning might be of the same size but quite soft after one hour's treatment. Only a post-traumatic neurogenic distension in a young girl (case 12) diminished (by 8·5 cm in 48 hours).

I do not suggest that any steps in the conventional treatment of paralytic ileus should be omitted—which should possibly include giving sympathetic nervous blocking agents[5]—but that when improvement in the patient's condition is not obvious after a reasonable interval hyperbaric oxygen should be tried.

I thank Mr D W Bracey, consultant surgeon, for enthusiasm and help, and Sister J Burbage and her nursing staff in the intensive care unit, who administered the hyperbaric oxygen.

[1] Bailey, H, and Love, R J M, *Short Practice of Surgery*, 15th edn, p 75. London, Lewis, 1971.

Clinical details and outcome of treatment

Case No	Age (years)	Sex	Cause of paralytic ileus	Symptoms: Distension	Symptoms: Toxicity	Day of illness hyperbaric oxygen begun	Hours of treatment	Outcome
1	11	F	Appendicitis	+ + + +	+ + +	3	8	Temperature normal in 24 hours; distension resolved in three days
2	58	M	Appendicitis	+ + +	+ + +	2	7	Temperature 38 C and pulse 120 min falling to normal in 48 hours; bowels open in 48 hours
3	58	M	Appendicitis	+ + + +	+ +	8	3	Pulse 100 min falling to 80 min and distension subsiding in 48 hours
4	14	M	Appendicitis (abscess)	+ +	+ + + +	3	8	Temperature 38·4 C and pulse 120 min falling to normal in 48 hours
5	12	M	Appendicitis	+ +	+ + + +	7	4	Temperature 38·5 C and pulse 130 min falling to normal; distension reduced in 24 hours
6	26	M	Perforated colon (stab wound)	+ +	+ + + +	2	8	Pulse 120 min falling to 80 min in 24 hours
7	84	M	Perforated caecum (carcinoma)	+ +	+ +	2	6	Bowel sounds started; patient died on third day
8	26	M	Peritonitis (no cause found)	+ +	+ + + +	4	4	Pulse 140 min falling to 80 min in 24 hours
9	1 12	M	Peritonitis (no cause found)	+ + +	+	2	6	Bowels open and patient hungry in 24 hours
10	64	F	Empyema of gall bladder	+ +	+ + + +	3	10	Temperature 39·5 C and pulse 140 min falling to 36 C and 90 min respectively in 48 hours
11	58	M	Vagotomy and pyloroplasty	+ + +	+ +	3	4	Pulse 110 min falling to 78 min, no distension, and bowels open in 48 hours
12	8	F	Retroperitoneal haematoma	+ + + +	+ +	3	4	Temperature 38 C and pulse 130 min falling to 36·5 C and 100 min respectively in 48 hours; girth 68·5 cm decreasing to 60·0 cm in 48 hours