then it is probably not too late to stop immunosuppression and remove the kidney without the risk of tumour transfer.

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SHORT REPORTS

Pulmonary fibrosis associated with hypersensitivity to gold salts

The pulmonary fibrosis associated with rheumatoid arthritis (RA) is usually thought to be a part of the systemic disease. In some cases an adverse reaction to a drug may be an equally valid explanation. We report a patient with rheumatoid disease in whom gold seems to have been responsible for the development of pulmonary fibrosis.

Case report

A 54-year-old nurse presented with pulmonary fibrosis in 1973. Rheumatoid arthritis had been diagnosed in 1962 and treated with analgesics until gold was started in December 1972. At that time she had no respiratory symptoms and her chest *x*-ray picture was normal.

In April 1973 after she had received 655 mg of sodium aurothiomalate she had a dry cough and was breathless on minimal exertion. The chest *x*-ray film showed diffuse bilateral shadowing. The gold was stopped. The only other drugs taken over this period were indomethacin and ferrous sulphate (Feospan), both of which she had taken for some years.

A lung biopsy in July 1973 showed fibrosing alveolitis. She refused steroid treatment. Her breathlessness steadily improved and in July 1974 she could climb three flights of stairs. At this time gold was restarted for an exacerbation of her arthritis. In October 1974 she again had a dry cough and was breathless and by March 1975 she could walk only 9 metres (10 yards) on the flat. Gold was stopped and prednisone 15 mg a day started. Over the next nine months her symptoms resolved, lung function improved, and the chest radiograph cleared slightly.

On examination in April 1975 there was no clubbing, and crackles were heard over both lung fields. The pulse was regular (80/min), blood pressure was 130/80 mm Hg, and there was no evidence of cardiac failure. There were the changes of RA affecting the knees, wrists, and hands. The remainder of the physical examination was negative.

Investigations while taking gold showed haemoglobin 11·0 g/dl, white cells 6·4 × 10^9/l (6400/mm^3), differential count normal, and no eosinophilia. Serum urea, electrolyte, and enzyme levels were repeatedly normal. The creatinine clearance was 106 ml/min, and there was no significant proteinuria. The sheep cell agglutination test gave a positive result (1/5120), and there were no LE cells. Lung function test results are shown in the table. Lymphocyte transformation to gold was tested by the method of Denman and Denman[1] using tritiated thymidine incorporation and was strongly positive. Lymphocyte transformation was negative in four normal subjects and also in four patients with rheumatoid disease taking gold without adverse reactions.

Lung function test results

	Predicted	June 1973	April 1975	Aug 1975	Nov 1975
Forced expiratory volume in 1 s (l)	2·25	1·30	1·15	1·60	1·85
Vital capacity (l)	2·70	1·50	1·25	1·70	1·95
Carbon monoxide transfer factor (T_LCO: mmol/min/kPa) ..	7·7	2·67	*	*	3·04

*Volumes too low to permit measurement of T_LCO.
Conversion: SI to traditional units—T_LCO: 1 mmol/min/kPa ≈ 3·0 ml/min/mm Hg.

Discussion

The appearance of pulmonary fibrosis during four months' gold treatment, the complete resolution of symptoms when gold was withdrawn, the recurrence of symptoms when gold was restarted, and resolution again on withdrawal strongly suggests that the gold was responsible for the fibrosis. A positive lymphocyte transformation response to gold has been shown to correlate well with adverse reactions to gold,[1] although not with pulmonary fibrosis. Gold treatment has been reported to cause cough and dyspnoea[2] and also pulmonary eosinophilia[3] but not pulmonary fibrosis.

Reports on patients with rheumatoid lung often omit details of treatment. In one survey of 126 patients with seropositive rheumatoid disease nine had extensive pulmonary parenchymal disease.[4] Six of these had received gold, and in five the gold was stopped because of a serious adverse reaction. Similarly in a study of lung function tests in patients with RA[5] 47% of those with a reduced carbon monoxide transfer factor (T_LCO) had received gold compared with 25% of those with a normal T_LCO.

Gold may therefore be the cause of the pulmonary fibrosis in some patients with rheumatoid lung. Differences in prescribing habits may partly explain the reported differences in the incidence of rheumatoid lung.

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Requests for reprints should be addressed to Dr D M Geddes, senior medical registrar, Westminster Hospital, London SW1P 2AP.

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Middlesex Hospital and Medical School, London W1

D M GEDDES, MB, MRCP, medical registrar
JONATHAN BROSTOFF, MRCP, MRCPATH, senior lecturer, department of immunology

Disintegration of cellulose dressings in open granulating wounds

Doctors are responsible for the steady healing of their patients' granulating wounds but the wounds are dressed by nurses. The most common type of dressing is a gauze roll soaked in half-strength eusol and packed into the open wound once or twice daily. Gauze rolls were made of spun cotton until 1974, when the warp was changed to rayon or spun cellulose. When cellulose absorbs wound exudate it expands to a greater volume than cotton and causes discomfort in a shorter time.

Patients, methods, and results

Patients with a variety of granulating wounds are seen at a weekly clinic in the University Hospital of Wales. During the past three years about 100 patients a year have been treated. The dressing is removed. The wound is inspected carefully and washed with a cotton gauze square soaked in saline. The skin is then shaved with a scalpel blade and the wound scrupulously cleaned once more to remove all hair.

Many patients have arrived with cellulose gauze dressings in their open granulating wounds. Six among the last 60 patients were found to have one