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.

We would like to thank Dr F H Scadding for permission to report this case and Mrs Sue Pack for excellent technical help.

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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

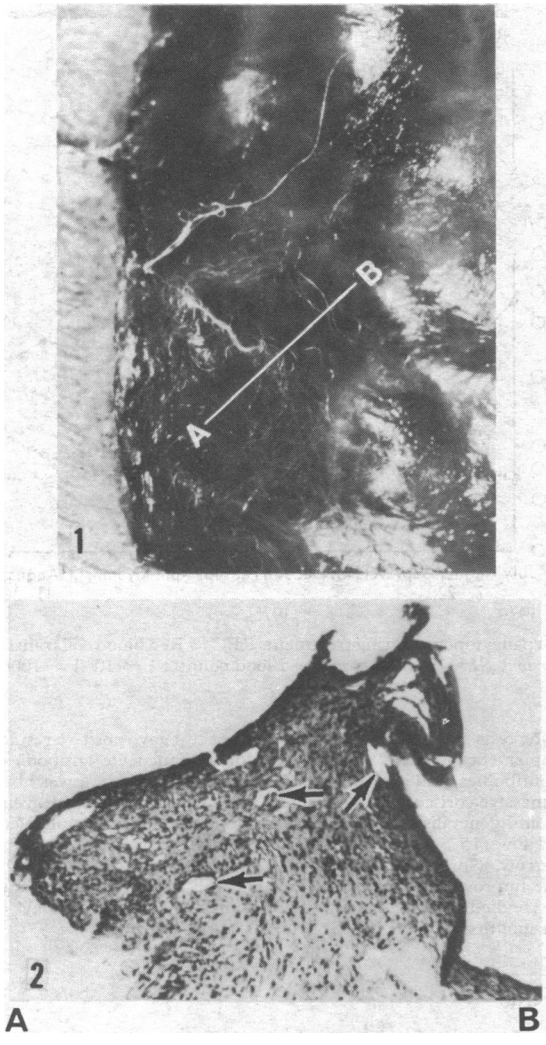

FIG 1—Edge of open granulating wound showing cellulose dressing on and embedded in granulation tissue. FIG 2—Section of tissue from area A-B (fig 1), seen by polarised light microscopy, showing birefractile cellulose particles.

or more strands of cellulose dressing on and embedded in the granulation tissue (fig 1). The strands were removed with forceps. At the point where embedding had occurred tissue for biopsy was taken with a scalpel blade and stained with haematoxylin and eosin.

When the slides were viewed under polarised light cellulose fibres were seen embedded in the granulation (fig 2). In all cases there was positive histological evidence of cellulose deep in the granulation tissue, including two cases in which the patients presented at the clinic without a dressing in their unhealed granulating wounds but had previously had cellulose dressings. Strands of cellulose dressings viewed under polarised light by an experienced pathologist were identical in appearance with those in the granulation tissue from wounds.

Discussion

The *British Pharmaceutical Codex*[1] states that cellulose wadding quickly disintegrates when wet. Certain factors have led to the widespread use of cellulose dressings. A shortage of cotton made dressings more expensive, and cheaper materials such as cellulose were substituted. Cellulose dressings are much used in the National Health Service. There is no warning on the packets that cellulose is unstable when wet and the Department of Health has not warned hospital regions or district nurses. In our local health authority area the standard sterile "dressing pack" from central supply contained cotton wool balls to swab the wound and cellulose gauze squares for the dressing. Nurses were not told that the cellulose squares were unstable when wet and therefore unsuitable. This has now been corrected.

These cases are a small percentage of those in which granulating wounds are being dressed with cellulose. Many must contain cellulose particles which act as foreign bodies and may delay healing. Their removal has been associated with accelerated healing.

I thank the Departments of Pathology and of Medical Photography, Welsh National School of Medicine, for their help.

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Circulating immune complexes in a patient with meningococcal disease

Allergic complications occur in meningococcal disease, and it has been suggested[1 2] that these are due to the deposition of immune complexes containing meningococcal antigens. We report here a case of severe meningococcal disease with some hitherto unreported immunological findings.

Case report

An 18-year-old woman was admitted to Enfield District Hospital, Chace Wing, with a history of malaise, sore throat, and neck pain. She was shocked and semi-comatose, with signs of severe meningitis and neurological deficit. After several grand mal fits she improved on penicillin and by the fifth day was answering simple questions, but extensive cranial nerve and cauda equina lesions persisted. Bilateral knee effusions developed on day 15. Aspiration yielded turbid fluid containing many polymorphs but no organisms; repeated cultures were negative. Arthroscopy showed a thickened synovium and synovial biopsy showed non-specific subacute inflammation.

Turbid cerebrospinal fluid (CSF), which contained 295 · 10^6 white blood cells/l (295/mm^3) and 400 · 10^6 red blood cells/l (400/mm^3), was obtained on lumbar puncture. The protein concentration was 9 g/l and the sugar level 1·6 mmol/l (28 mg/100 ml). Gram-negative diplococci were found and culture of both CSF and blood grew type C *Neisseria meningitidis*.

Haematological investigations showed evidence of disseminated intravascular coagulation: the platelet count was 22 · 10^9/l (22 000/mm^3), fibrin degradation products were 0·04 g/l, and both prothrombin and partial thromboplastin times were prolonged.

The most striking finding was the demonstration in both sera and synovial fluid of circulating immune complexes. These were detected simultaneously by radiobioassay,[3 4] a modified Clq-binding test,[5] and a modified polyethylene glycol (PEG) precipitation test.[4] The results summarised in the table show the early appearance of complexes and their rise with the onset of joint effusions and subsequent disappearance on recovery.

The PEG-precipitated material was dialysed against glycine saline buffer, pH 3·0, and the solution tested for meningococcal antigen by counter-current immunoelectrophoresis, using a polyvalent antiserum (Burroughs Wellcome meningococcal group A, B, C, D), but no antigen was shown. Snap-frozen synovial biopsy specimens and air-dried unfixed preparations of synovial fluid white cells were examined by direct immunofluorescence with fluorescein-isothiocyanate-conjugated anti-human IgG, IgM, and IgA, and antihuman C3 and by indirect immunofluorescence with polyvalent anti-meningococcal antiserum, but there was no evidence of complex deposition.

Immune complexes and immunoglobulins in serum and synovial fluid

	Immune complexes			IgG (g/l)	IgM (g/l)	C3 (g/l)
	Radiobioassay* (% inhibition)	Clq-binding† (%)	Total protein in 4% PEG precipitation test‡ (g/l)			
9 August:						
Serum	24	50	0·18	22	2·1	0·76
22 August:						
Serum	40	73	0·295	25·5	2·52	0·63
SF	41	75	0·290	25·0	2·0	0·24
December:						
Serum	13	9	0	15·2	1·95	0·76

*Normal ≤15% inhibition. †Normal 8-15%. ‡Normal 0·02 g/l.
SF = Synovial fluid.