LETTERS TO THE EDITOR

SIR — Retroperitoneal fibrosis (RPF) is an uncommon condition of uncertain aetiology. It has been
monitored for over 2 decades with symptoms uncon-
trolled by diacerein slow release 100 mg daily led to
the introduction of intramuscular gold sodium
athiomalate (GST), beginning with a 10 mg test dose followed by 50 mg once per week. After a total of
210 mg GST the patient began passing 3–4 loose
pale motions daily. She did not report these changes and a further 150 mg GST was given over the
following 3 weeks. Weight loss was noted amounting to 5 kg in 3 weeks. Her other medication was lorazepam 1.5 mg daily for several years. She had never suffered lower GI symptoms previously, although a hiatus hernia had been diagnosed many years earlier and she suffered intermittent heart-
burn. On examination she was thin and not clinically dehydrated. She had mil bilateral ankle oedema. Signs of active RA were noted. Investigation results were Hb 10.5 g/dl (12.0 before the onset of diarrhoea), WCC 7.8×10^9/l, no eosinophils, platelets 58×10^9/l, ESR 60 mm/h, electrolytes and creatinine normal, urea 9.0 mmol/l, alkaline phosphatase 330 U/l (normal 100–285). Bilirubin, albumin, AST, ALT, γGT were normal.

Stool microscopy revealed white cells ++ + , no RBC, organisms, parasites or ova. No pathogenic organisms grew on stool culture.

Sigmoidoscopy to 15 cm revealed changes consist-
tent with mild colitis.

GST was stopped and the diarrhoea controlled initially with codeine phosphate. The diarrhoea set-
tled over 2 days and did not recur when codeine phosphate was withdrawn. Rectal biopsy revealed mucosal oedema with a mild increase in eosinophils and marked congestion of the blood vessels. No evidence of amyloidosis, vasculitis, or chronic inflammatory bowel disease was seen.

The patient’s active arthritis was treated with prednisolone 5 mg daily. At follow-up 5 months later, no further diarrhoea had occurred and repeat sigmoidoscopy and rectal biopsy were normal. The diagnosis of gold-induced enterocolitis is supported by the close temporal relationship of the diarrhoea with the initiation and cessation of gold treatment together with characteristic rectal biopsy findings [2–4].

Diarrhoea was originally noted in dogs treated with intravenous gold for tuberculosis [5]. There have been approximately 20 reports of gold-
induced colitis in RA since 1935, with a variable range of presentation ranging from fulminating colitis associated with perforation and high mortality to cases which responded to conservative management [6–12]. The report by McCormick and col-
leagues [13] demonstrates that a fatal outcome may still occur.

The toxic effects are thought to be due to elemental gold because several different gold salts have caused this complication, including auranofin [11]. All the reported cases have occurred in patients at the start of gold treatment (total dose, 74–438 mg), with the exception of one patient beginning a second course of treatment. Five reports comment on the presence of eosinophils in rectal biopsy specimens [2–4,10,11] and in two cases an associated peripheral blood eosinophilia was present [2,4]. The suggestion that this eosinophilic infiltrate may mark a group with a better prognosis [3] is not supported by reports of severe disease with this histological picture [2,10].

Our case demonstrates that gold-induced enterocolitis can have a less dramatic presentation than previously reported and suggests that the onset of diarrhoea in any patient beginning gold treatment should be taken seriously.

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2. Fam AG, Paton TW, Shammes CJ, Lewis AJ. Fulmi-
nant colitis complicating gold therapy. J Rheumatol
1980;7:479–85.
1567–70.
21–6.
12. Eaves R, Hanksy J, Wallis P. Gold induced entero-

Retroperitoneal Fibrosis in Radiotherapy-treated Ankylosing Spondylitis

SIR — Retroperitoneal fibrosis (RPF) is an uncommon condition of uncertain aetiology. It has been
reported following the use of various drugs, including methysergide, and in association with a variety of fibrotic conditions, including mediastinal fibrosis, Reidel's thyroiditis, Peyronie's disease, sclerosing cholangitis and pseudotumour of the orbit. Recent evidence suggests an aetiological relationship with atheromatous aneurysms of the abdominal aorta [1]; antigens in atheroma may promote an autoimmune immunological response, the resulting chronic periaortic inflammation leading to progressive fibrosis. This may be sufficiently extensive to obstruct the ureters and cause renal failure.

One report describes three cases of RPF in patients whose treatment for Hodgkin's lymphoma had included radiotherapy to the para-aortic and pelvic lymph nodes [2]. We describe a case of biopsy proven RPF in a man with ankylosing spondylitis (AS) who had received spinal radiotherapy 15 years previously.

The patient had AS diagnosed in 1956 at the age of 34 years on the basis of typical symptoms, signs and radiological features in the pelvis and spine. Symptoms were of moderate severity and usually controlled by anti-inflammatory agents. In 1971 his back pain was sufficiently severe to merit radiotherapy. The upper and lower thoracic spine and lumbar spine each received 896 rads at 250 kV, given in eight divided fractions over 24 days, followed 1 month later by 900 rads in six fractions over 17 days to the sacroiliac joints. This treatment was successful and he did well until lost to follow-up in 1978.

In 1986 he presented with lower abdominal pain radiating to the groins and back, which interfered with sleep, and was associated with weight loss of 3 kg. He was a smoker of 15 cigarettes daily, and was moderately hypertensive on atenolol 100 mg daily. Abdominal examination revealed a pulsatile mass, thought to be an aortic aneurysm. Ultrasound showed an echogenic mass 1 cm thick overlying the abdominal aorta and inferior vena cava. Both RPF and a leaking aneurysm were considered possible. The kidneys looked normal, and a subsequent IVP confirmed normal ureteric anatomy and function. Haematological and biochemical tests were normal, and the sedimentation rate (ESR, Westergren) was 14 mm/h.

A computerized tomography scan also suggested leakage from an aneurysm, with possible dissection, and laparotomy was performed in mid-January 1987. A marked inflammatory process covered the aorta and extended to involve the left renal vein. The aorta, although difficult to visualize, appeared normal.

Histological examination showed the tissue to be highly cellular, with lymphoplasmacytic aggregates, numerous eosinophils, foreign-body-type giant cells, fibrin, and a modest degree of fibrosis, indicative of RPF.

No resection was done, but the patient improved on oral prednisone 15 mg/d. Attempts to decrease the dose have resulted in return of abdominal pain. The ESR remains normal at 10 mm/h and a recent IVP was normal.

RPF has previously been recognized in association with the presence of sacroiliitis and HLA-B27 [3,4], although the nature of this relationship is highly speculative. There are no previous reports linking RPF to radiotherapy treated AS. However, five cases of RPF with ureteric obstruction were found among 1416 patients given radiotherapy for cervical carcinoma [5], while in another report the only three cases of RPF among 423 patients with Hodgkin's disease surviving 10 years or more, were from the group which had received abdominal radiotherapy in addition to standard chemotherapy [2].

Our patient developed RPF 15 years after his radiotherapy, an interval broadly comparable to the 9–13 years in the patients described by Chao et al. [2]. As with all case reports, our patient may represent merely a chance finding, but the possible link to HLA-B27 is certainly worthy of further investigation.

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Fatal Hypersensitivity Reaction to Sulphasalazine

Sir—Sulphasalazine has found increasing favour with rheumatologists during recent years as a second-line agent for the treatment of rheumatoid arthritis (RA). Studies have demonstrated the effectiveness of the drug and it is an alternative agent to gold and penicillamine [1–3]. We report a case which may have been a fatal hypersensitivity reaction to sulphasalazine.

A woman aged 59 years with psoriatic arthritis was given sulphasalazine (Salazopyrin EN), at a starting dose of 500 mg once daily. She had a 7-year history of minor psoriasis affecting her arms and legs and for 12 months, progressive, active arthritis in her right hand, wrist and foot. Her joint symptoms were not adequately controlled by non-steroidal anti-inflammatory drugs which together with bendrofluazide for mild hypertension, were her...