usage of domperidone. It must now be added to the long list of causes of drug-induced gynaecomastia.

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Recurrence of a reactive arthritis following streptokinase therapy

Sir,

We write to report a case of a delayed reaction to streptokinase therapy, probably an immune-complex vasculitis now increasingly associated with the drug, in contrast to the immediate allergic reaction commonly seen.^{1–3} Similar vasculitic reactions have been reported with anisoylated plasminogen streptokinase activator complex.⁴

A nulliparous woman of 46 was admitted as an emergency with a diagnosis of myocardial infarction. She gave a history of hypertension for which she had been treated for 8 years with diltiazem, captopril and frusemide. She was treated with streptokinase 1.5 million units intravenously over 1 h. On the 5th in-patient day, she developed a widespread macular rash, predominantly over the limbs; no purpuric element appeared. Simultaneously, she felt considerable pain in the knees, shoulders and elbows, which were hot and stiff symmetrically but with no effusions. She was not febrile, there was no pericardial rub, and dipstick testing of the urine was normal. The arthritis and rash settled spontaneously over two days on ibuprofen. She recovered and was discharged on the 10th day. At the time of the arthritis, blood film showed a leucocyte count of $14 \times 10^9/l$, ESR 54 mm/h. Antinuclear factor was negative and rheumatoid factor (RAPA) borderline at 1/80. C3 and C4 186 and 14 mg/100 ml (elevated and normal, respectively). IgG 19.9, IgA 3.9 (both elevated) and IgM 1.1 g/l. Urine protein excretion 0.26 g/24 h.

She subsequently revealed that she had experienced the same reaction, with arthritis affecting the elbows and knees, and a rash, in 1980 when she had had an episode of pneumonia for which she had received parenteral antibiotics. At that time, no pathogen had been identified, but she had taken penicillin subsequently with no ill effect.

This patient's problem is interesting because of the previous reaction to a pneumonia, and we may surmise that this was again a reaction to a streptococcal compon-

ent antigen, although we have no proof that this was so. The reaction would then appear to be a form of reactive arthritis, and this suggests that investigation of future patients should include studies of synovial fluid (if accessible), anti-streptokinase antibodies and lymphocyte and neutrophil responses to streptococcal antigens.

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Spontaneous pneumomediastinum following myocardial infarction

Sir,

Spontaneous pneumomediastinum is a rare condition that may simulate the features of myocardial infarction in the absence of actual ischaemic heart disease. We report a case of asymptomatic spontaneous pneumomediastinum that followed acute myocardial infarction.

A 52 year old woman presented with a 2-h history of severe retrosternal chest pain associated with dyspnoea and nausea but no vomiting. Clinical examination was unremarkable but the electrocardiogram showed acute anteroseptal myocardial infarction. She was given intravenous streptokinase infusion. A chest radiograph at admission revealed free air within the mediastinum but no evidence of pneumothorax. The patient had no further chest pain or other complications. Acute myocardial infarction was confirmed by elevated serial enzymes. The radiographic appearance resolved over the following week.

Spontaneous pneumomediastinum is caused by non-traumatic rupture of marginal pulmonary alveoli allowing air to travel along interstitial and vascular routes.¹ It occurs in situations where there is a sudden increase in intra-alveolar pressure such as severe coughing, straining or Valsalva manoeuvres, and has been associated with acute asthma,² violent exercise³ and childbirth.⁴ This is the first reported case of pneumomediastinum following myocardial infarction and the pathogenesis is uncertain. We do not believe that the concurrence of these condi-