revealed pancytopenia [white blood cell count (WBC) $2.3 \times 10^3/mm^3$, haemoglobin (Hb) 6.8 g/dl, haematocrit (Hct) 17.5, platelets $37\,000/mm^3$]. Prior to infliximab therapy her WBC was $8.0 \times 10^3/mm^3$, Hb 11 g/dl, Hct 35 and platelet count $200\,000/mm^3$. She developed fever up to 102°F (38.9°C) and abdominal pain over the next 24 h, and ascitis was present. Peritoneal fluid was cloudy, with 322 red blood cells/mm^3, 4950 white blood cells/mm^3 and 96% neutrophils. Fluid cultures later grew *Candida albicans*. According to the patient's wishes, only blood transfusions, antibiotics and supportive care were given, and she expired the next day. No autopsy was done.

Tumour necrosis factor (TNF) inhibitors are being investigated in the treatment of a variety of rheumatic disorders, including scleroderma [2, 3]. Early results with etanercept indicate marginal clinical improvement, especially of skin involvement. Due to the severity of the patient's clinical picture, infliximab therapy was given. This was followed by pancytopenia and fungal infection. She was not receiving any other therapy that may have induced her haematological complication, which led us to implicate infliximab as an important contributor. This case should be added to the cases of existent pancytopenia already reported in association with anti-TNF-α therapy [4, 5].

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Reply

We read with interest this letter from Menon *et al.* [1] reporting another case of pancytopenia following infliximab infusion, but this case was a bit different from ours. It concerned a patient with scleroderma (our patient was affected by rheumatoid arthritis), and the pancytopenia was strictly linked to infliximab treatment, whereas the bone marrow toxicity occurring after the infliximab infusion in our patient may have been partially due to the fact that he was being treated with methotrexate and allopurinol and had been receiving leflunomide until a few weeks before the infliximab treatment. As it was not clear which drug(s) or combination of drugs was reponsible for the severe adverse reaction, we stressed the importance of careful patient monitoring when switching from one anti-rheumatic drug to another, especially in the case of the new and powerful immunosuppressive agents. It is interesting to note that both patients had impaired renal function and, although we cannot know whether this condition may be relevant in such cases, in our opinion it must clearly be kept in mind when starting infliximab therapy. At least one lesson that can be learned from these two cases of pancytopenia following infliximab infusion is that such powerful biological agents should be used with caution in rheumatic patients debilitated by other conditions and years of drug therapy.

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Fate of inflammatory neutrophils within the joint

Sir, We agree with Ottonello *et al.* [1] that the fate of neutrophils at inflammatory sites, especially within the rheumatoid joint, is an important issue which needs to be clarified. Their study, recently reported in this journal, is one of a very few which examines the influence of inflammatory synovial fluid on apoptosis in neutrophils [1, 2]. In their studies, joint fluid from eight of 11 RA patients produced inhibition of spontaneous and stimulated apoptosis in cultured neutrophils. Evidence is presented that this effect relates to adenosine levels within the joint fluids. The authors suggest that their findings support the view that, within the inflamed rheumatoid joint, synovial fluid factors (especially adenosine) tend to inhibit apoptosis and prolong neutrophil lifespan, thus maintaining the inflammatory response.

Their results appear to conflict with some of our own findings [2] in which we reported that synovial fluids from a variety of arthritic patients generally promoted neutrophil apoptosis, a finding at odds with