cells and granulation tissue [5]. When the acute inflammatory process improves, FDG uptake should be diminished, at least in the affected vessels. This was confirmed in the two patients for whom we have a follow-up F18-FDG-PET. Nevertheless, this pathophysiological basis can cause some false-positive results, as FDG uptake can occur in atherosclerotic lesions with an accumulation of inflammatory cells. Usually these images are less clearly positive and the distribution is different.

F18-FDG-PET not only seems to be a useful tool in the diagnosis of giant cell arteritis itself, but it also seems to be helpful in the evaluation of the extent and activity of the disease.

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Normalization of methotrexate-induced high levels of serum transaminases after ursodeoxycholic acid administration in a rheumatoid arthritis patient

Str, Administration of ursodeoxycholic acid (UDCA) can improve liver function in the course of several hepatic disorders; in particular, this drug can decrease serum transaminase levels in patients suffering from chronic hepatitis C virus (HCV)-related hepatitis [1], autoimmune hepatitis [2] and primary biliary cirrhosis [3].

Methotrexate (MTX) is effective in rheumatoid arthritis (RA) but it can induce elevation of serum transaminases. Serum aminotransferase (AST) levels seem closely correlated with histological damage of the liver caused by this drug [4]. Sometimes, folate supplementation can reduce the incidence of elevated liver enzymes during MTX administration, with a little concurrent loss of efficacy in the treatment of RA [5, 6].

Here we describe the case of an RA patient in whom UDCA was able to normalize high levels of serum transaminases induced by MTX. As a consequence, our patient continued the use of this drug as effective treatment of her arthritis.

A 64-yr-old Caucasian woman presented in November 2000 with a 4-yr history of seronegative RA. In the previous course of the disease, MTX (10 mg/week) had been effective, inducing complete remission of the symptoms. Unfortunately, MTX also caused a conspicuous increase in transaminases (more than three times the normal value of ALT) and consequently it was stopped. At that time, folate was not being administered. Subsequent therapeutic attempts with other disease-modifying anti-rheumatic drugs (gold salts and hydroxychloroquine) were unsuccessful.

At admission to our division, her treatment consisted of non-steroidal anti-inflammatory drugs and low doses of steroids (8 mg/day of methylprednisolone). Physical examination revealed symmetrical painful swelling of the metacarpophalangeal and proximal interphalangeal (PIP) joints, wrists and elbows. Considerable functional limitation (with ulnar deviation) of the fingers and subankylosis of the wrist were also present. Radiographs showed several erosions (mainly in the PIP joints) and a bilateral erosive/fusive carpitis. Pathologic laboratory findings were: erythrocyte sedimentation rate, 54 mm/h; C-reactive protein, 7.9 mg/dl (normally <0.5 mg/dl); and serum haemoglobin, 10.3 mg/dl. Searches for HBsAg (hepatitis B surface antigen) and HCV antibodies were negative. Risk factors for liver disease (such as alcohol consumption) were excluded.

We decided to administer MTX at 7.5 mg/week parenterally. Folinic acid (7.5 mg three times a week) was also administered.

A progressive increase in transaminases was recorded. Thirty-two days after the beginning of treatment, ALT reached 112 IU/l (normally <30 IU/l) and AST 43 IU/l (normally <30). Other liver tests (bilirubin, alkaline phosphatase, γ-glutamyl transpeptidase, albumin) were normal. At that time, folate was replaced by UDCA (450 mg/day). Transaminases returned gradually to the normal range over 2 weeks. MTX became progressively more effective and led to the remission of the arthritis in 2 months.

The patient has continued treatment with MTX and UDCA for 21 months of follow-up without alterations in liver enzymes.

In some patients treated with MTX, high levels of transaminases are not an important problem, because temporary drug discontinuation [6] or folate supplementation [4, 5] can normalize liver enzymes. However, in some subjects these expedients are not effective. Also in our case, MTX discontinuation, dose reduction
and folate administration were unable to normalize serum transaminases. The use of UDCA (450 mg/day) progressively reduced liver enzymes to normal levels. The protective role of UDCA on the liver has been described in many hepatic diseases, such as HCV-related hepatitis [1], autoimmune hepatitis [2] and primary biliary cirrhosis [3]. In the course of these disorders, UDCA can decrease the serum levels of transaminases. The beneficial effect of this drug is related to cytoprotective, anti-apoptotic, membrane-stabilizing, anti-oxidative and immunomodulatory effects [7]. Furthermore, UDCA can oppose the toxic action of exogenous substances such as ethanol [8] and flutamide (a non-steroidal anti-androgen) [9]. Our case report indicates a possible role of UDCA in treating toxic liver damage due to MTX.

In some liver disorders, UDCA-induced improvement in laboratory tests does not reflect a reduction in histological activity [2]. Consequently, if the role of UDCA in normalizing MTX-related high levels of transaminases is to be confirmed, studies involving liver biopsy or the detection of serum aminoterminal propeptide of type III procollagen [10] may be required in order to establish the absence of drug-induced damage.

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Cutaneous vasculitis associated with rofecoxib

SIR, We read with interest the recent letter concerning a celecoxib-induced cutaneous vasculitis [1]. There are no such case reports concerning rofecoxib. There have now been several case reports recently documenting cutaneous vasculitis in association with the cyclo-oxygenase 2 (COX-2)-specific non-steroidal anti-inflammatory drug (NSAID) celecoxib [1–3]. One case of fatal allergic vasculitis, described in Lancet [2], raised the possibility that the sulphonamide moiety of celecoxib could be contributory. Rofecoxib differs from celecoxib in having a methylsulphone group rather than a sulphonamide. Cutaneous vasculitis has not been reported previously in association with rofecoxib.

We report a case of cutaneous vasculitis that was apparently associated with rofecoxib.

In July 2002, a 62-year-old Caucasian female was commenced on rofecoxib 12.5 mg/day for exacerbation of her osteoarthritic symptoms. She had presented with increasing pain and stiffness in her wrists, shoulders, knees and lumbar spine. She made a good response to the treatment but was troubled by pedal oedema. This resolved on discontinuation of the drug but due to exacerbation of her pain the treatment was recommenced. She subsequently developed an extensive palapable petechial rash on her lower legs and feet that extended to just above the knees. The rofecoxib was discontinued and she was admitted to hospital for further investigation.

Her past medical history included chronic suppurrative lung disease secondary to alpha 1 antitrypsin deficiency and a left nephrectomy for reflux disease. Examination revealed no evidence of systemic vasculitis, but the extensive petechial rash was noted. Investigation revealed +++ blood and ++ protein on the urine dipstick, with mild renal impairment (creatinine 143 µmol/l). However, serum creatinine was not significantly changed from her previous measurements. Twenty-four-hour urinary protein excretion was increased to 1.06 g/24 h, although serum albumin was normal. Her erythrocyte sedimentation rate was elevated, at 22 mm/h, and the C-reactive protein concentration was 26 mg/l. The following tests were all normal or negative: complete blood count, complement levels, immunoglobulins, cryoglobulins, antinuclear antibodies, extractable nuclear antigens, antineutrophil cytoplasmic antibodies, rheumatoid factor, anticardiolipin antibodies and hepatitis serology.

A biopsy of involved skin showed a moderate perivascular lymphocytic infiltrate associated with widespread red-cell extravasation. No haemosiderin deposits were seen and there were only small