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 aminotransferease (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 IU/l). 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...
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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 action of this drug is related to cytprotective, 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.

The authors have declared no conflicts of interest.

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