stem can result from static or dynamic subluxation of the cervical spine or from direct pressure by synovial pannus [2]. We present a case of periodontoid rheumatoid pannus formation in which symptomatic relief and regression of pannus (imaged by MRI scanning) was achieved following a short course of infliximab therapy.

A 65-yr-old female with a 33-yr history of rheumatoid arthritis presented with a 2 month history of severe intractable cervical spine (neck) pain and occipital headaches. Her peripheral arthritis was controlled on 10 mg of methotrexate. ESR was 11mm/hr and CRP was <7 mg/l.

Cervical spine X-rays showed marked osteoarthritis in the lower cervical spine. There was no instability on flexion and extension views, and in particular there was no atlanto-axial subluxation.

An MRI scan of her cervical spine showed that there was abnormal low-signal intensity material, which was seen anterior and superior to the odontoid peg. This was in keeping with pannus formation associated with synovial hypertrophy, which extended into the spinal canal on the left side indenting the thecal sac (Fig. 1A).

The patient subsequently received three infusions of 500 mg of methylprednisolone intravenously in an attempt to relieve her symptoms. However, there was no resolution of the symptoms and a subsequent MRI scan showed no reduction in the size of the pannus. There was extensive pannus formation around the odontoid peg, which extended into the spinal canal (Fig. 1B). The patient’s occipital headaches persisted despite methylprednisolone therapy and a left occipital nerve block. Pregabalin was ineffective in relieving her symptoms. Neurological examination remained unremarkable at all stages of her treatment. The patient was subsequently given three infusions of infliximab therapy at 5mg/kg over a 6-week period. After the third infusion her symptoms of neck and occipital pain improved. A repeat MRI scan 4 months following the infliximab therapy revealed that the tissue planes around the odontoid peg were better defined when compared with the previous MRI scans with a significant reduction in the tissue bulk at this level (Fig. 1C).

Infliximab therapy should be considered early in the treatment of symptomatic cervical spine disease where rheumatoid pannus is shown to be causing neurological symptoms or compromise. Early treatment with anti-TNF therapy may have a significant impact on the development of rheumatoid myelopathy and prevent the need for surgery.

Rheumatology key message

- Infliximab reduces periodontoid rheumatoid pannus which therefore prevents progressive myelopathy and the need for surgery.

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A. J. Robinson1, D. H. Taylor2, G. D. Wright1

1Department of Rheumatology and 2Department of Radiology, Musgrave Park Hospital, Belfast, Northern Ireland

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Correspondence to: A. J. Robinson, Department of Rheumatology, Musgrave Park Hospital, Belfast, Northern Ireland.

E-mail: arobinson13@hotmail.co.uk


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An unusual cause of abnormal liver function in a patient with rheumatoid arthritis

Sir, A 70-yr-old male with long-standing seropositive rheumatoid arthritis (RA) was seen as part of his routine follow-up. His disease had been difficult to control, requiring numerous joint replacements, and over the last several years he had been managed with a combination of i.m. gold and methotrexate. Three months prior to review these were stopped and he had been commenced on adalimumab.

On review, his joints were quiescent, but he complained of vague abdominal discomfort. Routine bloods revealed CRP 87,
Alk P 359, ALT 253, AST 175, GGT 362 and bilirubin 32. He was reviewed again a few days later and described himself as being well, denying any recent infection, alcohol use or weight loss. Examination revealed 2 cm of tender hepatomegaly.

Further investigation revealed an elevated Epstein–Barr virus (EBV) immunoglobulin (Ig)M suggesting recent EBV infection. His ultrasound abdomen revealed numerous echo-poor lesions throughout the liver, which were suspicious for metastatic deposits. CT imaging revealed no evidence of a primary neoplasm and no lymphadenopathy. The lesions were poorly visualized, and CT appearances were not typical for metastases. Therefore, other causes such as hepatic abscesses were considered.

While an inpatient, he was noted to have a swinging pyrexia. A blood culture grew coagulase-negative Staphylococcus that was considered to be a contaminant. However, on the advice of Infectious Diseases Department, he was commenced on i.v. flucloxacillin and co-amoxiclav. His pyrexia remained despite antibiotic therapy. Further cultures were negative.

After initial reluctance by the Radiology Department, he underwent an ultrasound-guided liver biopsy. Histology revealed infiltration by tumour cells. Immunohistochemical staining was positive for CD45, CD20 and BCL-6. Proliferation rate was >95%. This was consistent with high-grade non-Hodgkin’s lymphoma (NHL) of diffuse large B-cell subtype. A bone marrow examination did not reveal any evidence of infiltration. The antibiotics were stopped and he was treated with CHOP-R (cyclophosphamide, doxorubicin, vincristine, prednisolone and rituximab) therapy. His liver function tests normalized within 2 weeks of induction. At present, he is currently maintained in remission, with his arthritis also much improved as a result of his chemotherapy.

RA is thought to develop as a result of immuno-incompetence, with evidence of contraction of T-cell diversity and premature ageing of the immune system. Immune failure, especially impairment of T-cell function is thought to be the main risk factor for the development of NHL [1]. EBV replication may be associated with impairment of T-cell function and a high EBV load is frequently implicated in the pathogenesis of lymphoma [2]. This may explain a mechanism for the increased incidence of lymphoma in RA. While essentially a disease related to T-cell dysfunction, lymphomas occurring in RA are predominantly NHL, and more specifically of diffuse large B-cell subtypes. Chronic B-cell stimulation, at the germinal centre stage of development, results from T-cell-dependent antigen exposure. Mutations in the variable region Ig genes occur, causing B-cell proliferation and lymphomagenesis. Furthermore, increased resistance to apoptosis that occurs in many autoimmune conditions may enhance the carcinogenic effects of chronic B-cell proliferation [3].

In the past, a number of disease-modifying drugs have been implicated in the development of lymphoma. Although epidemiological studies have not found any association between methotrexate use and the development of lymphoma, there are reports of individuals developing lymphoma while taking methotrexate. A significant proportion of these are EBV positive and NHL often regresses following cessation of methotrexate [4, 5]. Anti-tumour necrosis factor (TNF) therapies have revolutionized the management of RA. Recent studies suggest that the risk of lymphoma may not be increased beyond that of the general population of RA patients [6].

Primary hepatic lymphoma (PHL) is rare; accounting for <1% of all extra-nodal lymphomas. Patients classically present complaining of abdominal pain and nausea [7], with B symptoms (fevers, night sweats and weight loss) present in 37–86% of the cases. Hepatomegaly and abnormal liver function tests are found in the majority. PHL may present as a solitary mass or multiple lesions, the pattern being of little or no prognostic value [8]. Diagnosis of PHL can often be complex. Ultrasonographic appearance may mimic metastases, abscess formation and liver cirrhosis, and CT scanning does not provide better results for similar reasons [9, 10]. Tissue diagnosis is therefore of paramount importance.

Although a rare cause of abnormal liver function, this case highlights the need for adequate investigation. PHL often presents with non-specific features and mimics a number of other conditions. Abnormal liver function in RA patients may not always be related to drugs. Tissue sampling is essential in the investigation and management of patients whose differential diagnosis lies between infection, malignancy and adverse drug reaction.

### Rheumatology key message

- Abnormal liver function in RA may not always be attributable to drugs and should be comprehensively investigated.

### Disclosure statement

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J. G. Boulton, D. E. Bax

Royal Hallamshire Hospital, Glossop Road, Sheffield, UK.

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Correspondence to: J. G. Boulton.

E-mail: jgboulton@btinternet.com


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**Complete heart block after infliximab therapy**

Sir, we report the case of a 78-yr-old woman who developed complete heart block (CHB) following her third dose of infliximab.

She was diagnosed with RA RF-positive at age 50 and was treated sequentially with sulphasalazine, azathioprine, methotrexate and leflunomide. Each DMARD was discontinued either due to side-effects or lack of efficacy. She was then treated with etanercept from summer 2003 to October 2004. This had to be discontinued due to hypertension and headaches (which improved with stopping etanercept). Subsequently, she was treated for 12 months with adalimumab, which was ineffective (discontinued in December 2005).

Her RA continued to be active on prednisolone 10 mg once daily and in August 2006 she started infliximab (3 mg/kg) and methotrexate (7.5 mg once weekly). Her other medical problems were hypertension, osteoporosis, indigestion and nausea for which she was taking lisinopril, amlodipine, residronate, omeprazole and