In conclusion, we fear that by producing more and more positive results in very sensitive assays one does blur the picture of myositis-specific autoantibodies and diminishes their diagnostic value.

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Author’s reply

The conclusion of Drs Mierauc, Dick, and Genth that by producing more and more positive results in very sensitive assays one blurs the picture of myositis-specific autoantibodies and diminishes their diagnostic value is not correct. The autoantibody specificities we measured remain very specific for myositis and as such still are a useful help for diagnosis. However, our data indeed show that anti-Mi-2 antibodies are not completely specific for dermatomyositis (DM) as has been suggested so far, but also occur in 9% of patients with polymyositis (PM). We explained this result by the fact that we used the complete Mi-2 protein (in four overlapping fragments) as antigen, whereas in all previous studies the NM fragment, containing only 26% of the protein, was coated in the enzyme linked immunosorbent assay (ELISA) assay.

Mierauc and colleagues argue that we did not provide information on the diagnostic specificity of the Mi-2 assay we used. The test, in fact, using the four overlapping fragments was evaluated with healthy subjects by testing 84 samples from blood donors. The absorbance values at 405 nm of the mean plus SD were 0.262 for the NT fragment, 0.260 for the M fragment, and 0.115 for the CT fragment. The cut off values for positivity were set distinctly above those values, that is 0.500 for both the NT and M fragments, and 0.450 for the CT fragment.

When this design with rather high cut off values is used, these new tests are specific for the blood donor samples used. To strengthen the argument that anti-Mi-2 antibodies do occur in a significant percentage of PM we successfully confirmed about 50% of the Mi-2 positives by immunoblotting. Because ELISA is much more sensitive than immunoblotting, or one of the other techniques mentioned by Mierauc and colleagues, at this stage of our research we have to accept the fact that 100% confirmation cannot be reached. Our results thus show that anti-Mi-2 is not as specific for DM as was hitherto thought. The take home message for the clinician is that when anti-Mi-2 autoantibodies are found, the underlying disease might not necessarily be DM.

Mierauc and colleagues also correctly state that reported coincidences of myositis-specific antibodies are rare. In our studies, by testing many patients, such coincidences were found more often, as expected, albeit still in a very low percentage of the patients. Again we think that we found these coincidences by virtue of the very sensitive assays we used for the detection of anti-Mi-2 and anti-SRP antibodies. We disagree, however, that such results inevitably mean a loss in specificity.

Apart from the biochemical refinements mentioned above, there could be a second reason for the discrepancy between previous studies and our results, and that is the problem of adequate clinical diagnosis or classification as PM or DM. Moreover, the specificity of myositis related autoantibodies is further blurred by the picture of myositis-specific antibodies one does blur the picture of myositis-related autoantibodies. The consequence of this gain in sensitivity is mostly due to the specificity its HLA association—previously studied in large group of patients with myositis. We wonder if by broadening the anti-Mi-2 specificity its HLA associations—previously described as very strong with HLA-DR7 and a tryptophan on residue 9 of DRB1—might also be altered. It would be especially useful to know the HLA type of the four anti-Mi-2:Jo-1 double positive patients described by Brouwer et al because useful specificities are highly DR3 associated, whereas DR3 is completely absent from all Mi-2 positive patients with DM, whose HLA type has been published so far.

MATTERS ARISING

Less specific myositis autoantibodies?

Brouwer et al studied a very large group of European patients with idiopathic inflammatory myopathies for myositis associated autoantibodies with a variety of methods. This is the largest group of patients with myositis ever studied for autoantibodies. The results substantially increase the existing knowledge about frequencies of autoantibodies in patients with inflammatory myopathies.

Compared with the pre-existing data, clinical associations of certain myositis-specific autoantibodies seem to be widening and are becoming less specific. Whereas the overwhelming majority of patients with anti-SRP are reported to have polymyositis (PM), and anti-Mi-2 was almost completely specific for dermatomyositis (DM), Brouwer et al mention several anti-SRP positive DM and anti-Mi-2 positive PM cases, even in some patients with these autoantibodies and inclusion body myositis. The question is whether this is a matter of test or autoantibody specificity or a problem of adequate clinical diagnosis or classification as PM or DM. Moreover, the specificity of myositis related autoantibodies is further questioned by several violations of the already established rule that coincidences of myositis-specific antibodies are almost non-existent—for example, by patients being positive for anti-Jo-1 and anti-Mi-2 or anti-SRP at the same time.

As the authors correctly state, the detection of more positive results is mostly due to the assay design, especially of those tests with multiple recombinant fragments of the Mi-2 antigen. The consequence of this gain in sensitivity inevitably is a loss in specificity; whether this can be regarded as progress may be a matter for discussion. The authors do not give information about the diagnostic specificity of the new assays, evaluated by testing healthy controls and patients without myositis. Their judgment about the relevance of the results is difficult.

The “new”, non-DM anti-Mi-2 sera differ from the “classical” ones: Brouwer et al mention that their fine specificity is different and that only 8/17 could be confirmed by western blotting. There is no information as to whether they are positive in the traditional Ouchterlony assay and if they show up as antinuclear antibodies (ANA) in HEp-2 cell immunofluorescence (as the “classical” anti-Mi-2 sera do). At least the single anti-MI-2 positive patient with PM previously described by Roux et al was ANA negative.

We wonder if by broadening the anti-Mi-2 specificity it has previously described as very strong with HLA-DR7 and a tryptophan on residue 9 of DRB1—might also be altered. It would be especially useful to know the HLA type of the four anti-Mi-2:Jo-1 double positive patients described by Brouwer et al because useful specificities are highly DR3 associated, whereas DR3 is completely absent from all Mi-2 positive patients with DM, whose HLA type has been published so far.

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Ann Rheum Dis 2001;60:810–816
Usefulness of the HAQ in the clinic

Greenwood et al have questioned the usefulness of the Stanford Health Assessment Questionnaire (HAQ) in the clinic.1 Using Bland and Altman’s limits of agreement procedure,2 they report that an HAQ score change of at least 0.48 units is required to confidently reflect significant change. We have used the HAQ in the care of our patients for many years and think that their conclusions about the extent of change required and the non-usefulness of the HAQ in clinical care is not correct.

We have replicated the authors’ study in paired visits of 443 patients with rheumatoid arthritis (RA) followed up in the clinic by one of us (FW), and in a survey database of 2720 patients. Our data are in general agreement with the authors’ data.

Using the limits of agreement procedure, we found that the 95% confidence intervals (CI) for the HAQ were approximately 0.9 for clinical data and 0.6 for survey data. But we also found the following. To exceed 95% CI, changes in excess of 40 mm/1st h for the erythrocyte sedimentation rate (ESR), 70 mm Hg for grip strength, 10 joints for a joint count, and four units on 0–10 VAS scales for pain, global severity, and fatigue were required.

Although clinicians may have trouble in interpreting HAQ scores, there is no doubt that one does not need a change of 40 mm/1st h for ESR, 70 mm Hg for grip, or 10 joints to detect clinical change. These data, which indicate that we cannot reliably do what we are already reliably doing, suggest two interpretations. Firstly, stable measures of overall health are themselves noisy and do not reflect changes in arthritis clinical status. Secondly, we suggest that the Bland-Altman method for agreement and reliability may not work well and may not have a simple interpretation when extrapolated to settings such as these.

In additional analyses that measured relative accuracy and precision, we examined all of the above variables as well as the SF-36 and WOMAC variables using Lin’s concordance coefficient.3 The HAQ was the most accurate and precise of all of the above variables, with concordance correlations of 0.809 and 0.902 in the two analytic sets. We also believe that it is a mistake to use the HAQ in isolation. One would not treat RA for thyroid dysfunctions.

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Giant cell arteritis associated with demyelinating polyradiculoneuropathy

Peripheral nerve involvement—mononeuritis, mononeuropathies, and polyneuropathies—has been reported in 14% of patients with giant cell arteritis (GCA). GCA associated with acute inflammatory demyelinating polyneuropathy (AIDP) has exceptionally been described. We report an unusual case of this association.

A 67 year old woman presented with a one month history of difficulty in walking, weakness, and proximal myalgia that worsened progressively. Examination showed proximal weakness and abolition of all deep tendon reflexes; there was no sensory deficit and cranial nerve function was normal. Laboratory tests disclosed normal haematological findings with an erythrocyte sedimentation rate (ESR) of 12 mm/1st h; renal and liver function tests, muscle enzymes, and thyroid stimulating hormone were in the normal range. Serum protein electrophoresis, anti-nuclear antibodies and rheumatoid factors, and serology for Lyme disease, Q fever, mycoplasma, Veneréal Disease Research Laboratory test, HIV, and cytomegalovirus were all negative. Chest radiographs and abdominal echography were normal. Electrophysiological investigation disclosed an inflammatory demyelinating polyradiculoneuropathy, with prominence of motor and proximal involvement. The cerebrospinal fluid (CSF) showed four mononuclear cells/ml, protein 0.56 g/l and glucose 3.4 mmol/l.

At this moment, an AIDP or Guillaumin-Barré syndrome was diagnosed, and plasma exchange was performed, using standard procedures. After the second plasma exchange, the weakness had completely disappeared, but the patient started with fever (40°C) and increasing bitemporal and occipital headache. Physical examination showed headache, fever, photophobia, neck stiffness, and decreased level of consciousness. The patient was drowsy and had no visual field defect or papilloedema. Laboratory tests showed elevated white blood count and C-reactive protein. The cerebrospinal fluid showed a marked pleocytosis with neutrophil predominance (109 cells/l) and normal glucose and protein levels. The patient was treated with intravenous methylprednisolone (1000 mg daily) and later switched to prednisone (60 mg daily) due to lack of response. The patient improved gradually over the following weeks. Repeated nerve conduction studies showed an objective improvement.

Ten months later, when she was taking prednisone 10 mg/day, the proximal weakness started again, worsening over the following weeks. Electrophysiological study showed...
progression of the demyelinating polyradiculo-
neuropathy. She had no other symptoms. Haematological and blood chemical findings were all normal, as was the ESR. Plasma exchange sessions were restarted. The diagnosis at this time was an AIDP relapsing form. Prednisone was given at 1 mg/kg weight. She improved rapidly. Because of the serious side effects she had previously had with the steroid treatment (30 kg weight gain, hyperglycaemia, mental changes) and the poor vascular access she had for the plasma exchange, we decided to start treatment with intravenous immunoglobulin pulses, 0.4 g/kg weight every four weeks. Steroids were tapered and the patient remained clinically well during the following year.

To our knowledge, only two cases of GCA associated with AIDP have been previously reported (table 1), and no association of GCA with the AIDP relapsing form has been previously described. The first case refers to a patient who presented a clinical picture of AIDP and in whom temporal arteritis was diagnosed two weeks later. In the second case the patient was diagnosed as temporal arteritis, with a compatible biopsy; he was treated with prednisone and four weeks later he presented a generalised weakness: CSP and electrophysiological study were concordant with AIDP. A sural nerve biopsy was not done in these cases. The neuropathies associated with temporal arteritis and other vasculitides have been attributed to ischaemic lesions of the nerves due to an arteritis of the vasa nervorum. A vasculitic neuropathy was excluded in our case because the clinical course and the electrophysiological study were characteristic of AIDP.

The underlying cause and pathogenic mechanisms of AIDP and GCA are not well understood. Immune cellular mechanisms have an important role: an increasing number of T CD4+ cells and macrophages in demyelinated regions of the nerve are seen in AIDP (1). The histopathology of GCA shows a mixed inflammatory infiltrate with T CD4+ lymphocytes and macrophages secreting proinflammatory cytokines.2

Temporal arteritis associated with inflammatory demyelinating polyradiculoneuropathy may be a coincidence, but a common immunological pathogenesis, probably based on a cellular T cell dependent mechanism and related to a cytokine deregulation, cannot be ruled out. Infectious agents, such as a virus, may be the cause of the onset of both diseases in a subset of patients.3

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Table 1

<table>
<thead>
<tr>
<th></th>
<th>Case 1</th>
<th>Case 2</th>
<th>This study</th>
</tr>
</thead>
<tbody>
<tr>
<td>Age/sex</td>
<td>71/female</td>
<td>73/male</td>
<td>67/female</td>
</tr>
<tr>
<td>Headache</td>
<td>Yes</td>
<td>Yes</td>
<td>Yes</td>
</tr>
<tr>
<td>Fever</td>
<td>No</td>
<td>No</td>
<td>No</td>
</tr>
<tr>
<td>Visual loss</td>
<td>Yes</td>
<td>No</td>
<td>No</td>
</tr>
<tr>
<td>Facial diplopia</td>
<td>Yes</td>
<td>No</td>
<td>No</td>
</tr>
<tr>
<td>PMR* symptoms</td>
<td>No</td>
<td>No</td>
<td>No</td>
</tr>
<tr>
<td>Latency between GCA* and PN*</td>
<td>AIDP occurred 2 weeks before GCA</td>
<td>GCA preceded AIDP by 4 months before AIDP</td>
<td>First episode of AIDP occurred 2 weeks before GCA</td>
</tr>
<tr>
<td>Laboratory tests</td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>ESR* (mm/1st h)</td>
<td>125</td>
<td>125</td>
<td>94</td>
</tr>
<tr>
<td>CSF* Protein 0.1 g/l; acellular</td>
<td>Protein 3 g/l; acellular</td>
<td>GCA</td>
<td>Protein 0.56 g/l; cells 4/ml</td>
</tr>
<tr>
<td>Temporal artery biopsy</td>
<td>Not done</td>
<td>Normal</td>
<td>GCA</td>
</tr>
<tr>
<td>Nerve conduction study</td>
<td>Normal</td>
<td>Normal</td>
<td>AIDP</td>
</tr>
<tr>
<td>Treatment</td>
<td>Prednisone, 80 mg/day</td>
<td>Prednisone 75 mg/day, PEX*</td>
<td>Prednisone 60 mg/day, PEX, IVIg*</td>
</tr>
<tr>
<td>Clinical outcome</td>
<td>Spontaneous improvement of the AIDP symptoms of GCA were controlled with steroids</td>
<td>Cure of the AIDP with PEX</td>
<td>Cure of GCA with prednisone; improvement of the AIDP with PEX and relapse controlled with prednisone and IVIg</td>
</tr>
</tbody>
</table>

*PMR = polymyalgia rheumatica; GCA = giant cell arteritis; PN = polyneuropathy; ESR = erythrocyte sedimentation rate; CSF = cerebrospinal fluid; AIDP = acute inflammatory demyelinating polyradiculoneuropathy; PEX = plasma exchange; TIA = transient ischaemic attack; IVIg = intravenous immunoglobulin.

Oral steroid in the treatment of carpal tunnel syndrome

A range of options are available for the conservative treatment of carpal tunnel syn-
drome (CTS).4 Non-operative methods include immobilisation of the affected hand with wrist splint; local injection of steroids and drugs such as diuretics and non-steroidal anti-inflammatory drugs.5,6 These oral drugs are thought to decrease the volume of swollen tissue within the CTS and are widely used, but there is limited clinical evidence for their role.

This prospective randomised, double blind, placebo controlled study aimed at evaluating the effect of oral steroids in the symptomatic treatment of CTS. We recruited patients with newly diagnosed CTS of more than three months’ duration with confirmatory electrophysiological results (prolonged median nerve distal motor latencies >4 ms or median ulnar palmar sensory latency differ-
ence >0.5 ms) including electromyographic recordings of the abductor pollicis brevis (APB); co-interventions such as drug or injection treatment were withheld during the study. Exclusion criteria included (a) patients with evidence of severe CTS: fibrillation potentials or reinnervation on needle examination of the APB, (b) coexisting disorders or conditions which may mimic CTS, such as cervical radiculopathy or peripheral neuropa-
thy; (c) contraindication to steroid use; and (d) history of underlying disorders associated with CTS, such as diabetes mellitus or rheu-
matoid arthritis.

Patients who fulfilled the criteria were treated conservatively for two months with splinting. If symptomatic after this period, patients were allocated, using a random computer generated code, to a 10 day course of prednisolone 25 mg/day or a 10 day course of placebo. Both were given as single tablets which were identical in appearance. A physician (SMW) unaware of the treatment allocation assessed the mean global symptom score (GSS) at all patients at two and eight weeks. This is a scoring system first devised by Herskovitz which rates symptoms on a scale of 0 (no symptoms) to 10 (severe) in five categories: pain, numbness, paraesthesia, weakness/clumsiness, and nocturnal awakening. The sum of the scores in each category was the GSS.7 Median (interquartile range (IR)) changes in GSS at two and eight weeks from baseline were analysed using the Mann-Whitney test. The null hypothesis was that there was no difference in symptom score between the treatment and placebo groups. Results were considered signif-
ificant at p<0.05 (two sided). The sample population of 36 was planned to enable achievement of 80% power with an α=0.05 for detecting a 50% difference in GSS between the treatment groups, assuming
the response rate in the oral steroid group to be 80% and that of the placebo group 30%.

Thirty-six patients were recruited, of whom half were randomly allocated to receive oral steroids and half to placebo. There was no significant difference in demographics such as age and in the severity of electrophysiologi- cal parameters as shown in table 1. As compared with baseline, patients receiving steroid had a median (IR) change of −12.5 (−15 to −7) at two weeks, whereas the placebo group was −4.5 (−14 to 0), p=0.027 as shown in fig 1A. After eight weeks, the median (IR) reduction of GSS in the steroid group was −9 (−14 to −6) and in the placebo group −2 (−10 to 0), p=0.034 as shown in fig 1B. The median differences between the two groups at two and eight weeks were −6 (−11 to −1) and −6 (−11 to 0) respectively. All patients completed the short course of treatment.

This study shows a small but statistically significant reduction in GSS in the group prescribed a short course of prednisolone as compared with placebo. Steroid may have a role in the treatment of mild to moderate CTS in patients who decline or who are awaiting surgical decompression. Further tri- als with larger sample size and longer follow up, using low dose oral steroid in direct compar- ison with placebo, would further clarify the effect of this treatment.

**Table 1** Patient characteristics

<table>
<thead>
<tr>
<th>Placebo</th>
<th>Steroids</th>
</tr>
</thead>
<tbody>
<tr>
<td>Patient numbers</td>
<td>18</td>
</tr>
<tr>
<td>Age, years (mean (SD))</td>
<td>44.9 (10.0)</td>
</tr>
<tr>
<td>Sex (female/male)</td>
<td>17/1</td>
</tr>
<tr>
<td>Baseline GSS* (median, IR)*</td>
<td>24 (16–26)</td>
</tr>
<tr>
<td>CMAP amplitude, mV (mean (SD))</td>
<td>12.95 (3.40)</td>
</tr>
<tr>
<td>Distal motor latencies, ms (mean (SD))</td>
<td>5.08 (1.20)</td>
</tr>
<tr>
<td>Location of CTS*</td>
<td>10</td>
</tr>
</tbody>
</table>

*GSS = global symptom score; IR = interquartile range; CTS = carpal tunnel syndrome.

Parvovirus B19 infection in Behçet’s disease

We read with great interest the article by Kerr which reviewed present knowledge about the possible association of parvovirus B19 infection with various connective tissue and auto- immune disorders.1 The author concluded that data implicating B19 virus infection in the aetiology/pathogenesis of Behçet’s disease is insufficient and conflicting. Although a signifi- cant number of studies support a possible role for the virus in the pathogenesis of rheuma- toid arthritis, juvenile idiopathic arthritis, systemic lupus erythematosus, and vasculitis, the author believes that B19 infection is only one of a number of triggers.

Behçet’s disease (BD) is a multisystem disorder originally described by the Turkish dermatologist Hulusi Behçet in 1937. The aetiology of BD is unknown, vasculitis is widely accepted as the underlying pathological process.2 As stated by the author, various case reports have been published demonstrating the presence of B19 virus in patients with vasculitic syndromes.3-4 Viral infections have also been pos- tulated as triggering factors in BD. Therefore, in a previous study that was not cited in the report by Kerr, we investigated a possible role of B19 virus in BD.5 We assessed antibodies against parvovirus B19 in serum samples from 41 patients with BD and from 40 age and sex matched controls. Six patients with BD (15%) had anti-B19 IgM antibodies while no IgM antibodies were detected in the control group (p=0.03). However, anti-B19 IgG antibodies were present in 23 patients with BD and 25 controls. There was no correlation between the presence of anti-B19 IgM antibodies and articular and vascular manifes- tations of BD (p=0.9 and p=0.5, respectively).

Therefore, we concluded that our findings did not strongly support the involvement of B19 in the pathogenesis of BD, and we also concluded that serological evidence of acute B19 infection in six patients with BD might have been coincidental. However, the pres- ence of anti-B19 IgM antibodies in patients with BD might provide evidence for the place of B19 infection in the pathobiology of BD. As far as we know our previous report is the only published study investigating the association of B19 virus infection and BD. Therefore, fur- ther studies will be clearly needed to clarify this unresolved issue.

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Idiopathic dilatation of the pulmonary artery in a patient with dermatomyositis complicated by interstitial pneumonitis

Idiopathic dilatation of the pulmonary artery (IDPA) is an uncommon anomaly occurring in 0.6% of patients with congenital heart disease, and may be bilateral or unilateral. We report on a patient with IDPA who concomitantly developed polymyositis (PM).

On 25 August 1997 a 63 year old woman was referred to our hospital because of dyspnoea; the patient was afebrile and did not complain any joint pain, swelling, or limitation of the range of motion. Liver and spleen were not enlarged. Laboratory studies showed increased liver enzymes; aspartate aminotransferase (AST) and lactate dehydrogenase (LDH) were 61 IU/l and 1194 IU/l, respectively. The catheterisation study disclosed dilatation of the PAs without pulmonary hypertension (PH; mean pulmonary pressure was 20 mm Hg). After the diagnosis of IP induced by infection and started treatment with pulse methylprednisolone (169 IU/l, AST 25 IU/l, ESR 122 mm/1st h, and PaCO2 35.3 mm Hg). Laboratory data on discharge (31 October): LDH decreased to 1082 to 738 IU/l, CK from 1885 to 223 IU/l, AST from 60 to 18 IU/l, ESR from 116 to 27 mm/1st h, and white blood cell count from 10 000 to 9600/µl. Data of pulmonary function tests including BGA were unchanged. While taking 30 mg prednisolone a day, she visited crowded stores and thereafter developed pyrexia with severe dyspnoea. BGA showed hypoxaemia (PaO2 45.2 mm Hg, PaCO2 35.3 mm Hg). Laboratory data on readmission showed LDH 1580 IU/l, CK 169 IU/l, AST 25 IU/l, ESR 122 mm/1st h, C reactive protein 212 mg/l, white blood cell count 15 100/µl, and platelets 60 000/µl. We diagnosed her as having exacerbated IP induced by infection and started treatment with pulse methylprednisolone (1000 mg/day for three days) followed by antibiotics, anticyclic pentamidine, and immunoglobulin (high titres for cytomegalovirus).

Despite such treatments, she died of respiratory failure on 10 November. Necropsy disclosed (a) polymyositis showing focal atrophy, fibrosis, and contraction band necrosis; (b) IP with cytoplastic inclusion body; (c) dilatation of the bilateral PAs; and (d) disseminated intravascular coagulation.

Because dilatation of the PA can be caused by rheumatic diseases through secondary PH; diagnosis of IDPA is difficult among such patients. Our patient had PM as evidenced by both IP and PH; we thus initially supposed that she had both IP and PH complicated with PM. Pulmonary catheterisation showed the absence of PH, indicating IDPA. This case indicated that IDPA should be ruled out in patients manifesting PH.

Patients with PM can die of acute respiratory failure induced by infection. To prevent respiratory failure, rheumatologists should be careful about opportunistic infections in patients receiving high doses of corticosteroids or immunosuppressive drugs. Because respiratory failure can be fatal, patients with PM should stay away from crowds while they are taking high doses of immunosuppressive agents.

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antibodies to Epstein-Barr virus, and cytomegalovirus. An x-ray examination of the hands and knees was negative. An ultrasound of the liver and spleen was normal.

His primary physician reported that the abnormal liver function tests had been present for approximately two years. A liver biopsy showed prominent macrovesicular fatty changes, aggregates of parenchymal inflammation, and mild fibrosis with minimal inflammatory infiltrate at some of the portal spaces. Fibrosis and Mallory bodies were not seen at the central vein areas. Iron staining was negative. These findings are consistent with a diagnosis of NASH.

The patient started on a low calorie diet and a few weeks later his liver function tests normalised with complete resolution of his joint symptoms. A few months later he stopped following the low calorie diet and within one week he began to have joint symptoms and the liver enzymes were found to be raised. Reintroduction of the low calorie diet again resulted in complete resolution of his symptoms and abnormal liver function tests.

Polyarthritis or polyarthritis is one of the extrahepatic manifestations of liver diseases, and many chronic liver diseases are associated with rheumatic diseases. Examples include the association between chronic infection with hepatitis B virus and polyarteritis nodosa, the association between chronic infection with HCV and mixed cryoglobulinaemia, the association between chronic infection with HCV and Sjögren’s syndrome, and the association of primary biliary cirrhosis and scleroderma. Immune complexes may be one of the mechanisms of arthralgia associated with liver diseases, and patients with chronic liver disease due to HCV infection have been shown to have an increased prevalence of ANA, rheumatoid factor, and anti-smooth muscle antibodies.

Increasing evidence shows that several cytokines mediate hepatic inflammation and cholestasis in alcoholic and non-alcoholic steatohepatitis. Among these cytokines tumour necrosis factor α (TNFα) is a key factor. TNFα mediates not only the early stages of fatty liver but also the transition to more advanced stages of liver diseases like steatohepatitis and cirrhosis. On the other hand, TNFα is an important inflammatory disease mediator in a wide spectrum of articular diseases, and inhibition of this cytokine led to a significant improvement of symptoms and signs in rheumatoid arthritis (RA). So TNFα may be a common mediator in NASH and arthritis.

We thank Professor Paul Froom for his editorial assistance.

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