wound healed well, her symptoms rapidly resolved, and she went home 2 weeks after her operation.

Iatrogenic meningocoels have been reported following spinal surgery, usually for disc disease. The majority are due to failure of recognition of a surgical injury, or failure of a repair attempt. It is possible that the puncture wound for myelography may be responsible for a few cases. They have been reported after intradural operations but at higher levels when the problem has been cord compression.

The recurrent symptoms may be caused by adhesion of a nerve root to the fistulous track or prolapse through it with resultant traction on the root. This did not apply in the case reported here. It is possible that the distention of the sac excited nerve endings in the muscles and periosteum of the laminae upon which it pressed, the radiation being due to common innervation. These meningocoels are found when patients are investigated for recurrence of symptoms: I have been unable to find reports of any not thus discovered, although this does not mean they do not occur.

One factor known to be associated with failure of a dural repair is the presence of a clip upon the dural edge. None were used in this case. However temporary sutures were used to retract the dural edges. It is possible that the small hole left by one of these gradually enlarged and led to the defect found at operation, for it did not appear to be related to the suture line. Care must therefore be used in the employment of retraction sutures, and perhaps all dural repairs ought to be covered with gelfoam or a similar preparation to encourage fibrinous reaction. Such materials alone are not recommended for dural repair.

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References

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Recurrent localised myositis

Sirs: Polymyositis is typically a diffuse disease throughout its course, but rarely may begin as a localised process. We describe a case with recurrent localised myositis with fluctuating elevation of serum creatine kinase (CK) levels, but who clinically merely had relapsing focal muscle lesions, without diffuse progression. All other cases of localised myositis previously published have progressed rapidly to the typical generalised pattern.

A 66 year old shepherd presented in August 1985 with a 5 week history of tender swellings and progressive disability in walking. Normally, he walked 20 miles a day on his farm, but at the time of presentation could only walk with the aid of a stick. He had no relevant past medical history. General examination revealed no evidence of systemic disease, in particular there were no heart murmurs, his heart size was normal and his peripheral pulses were normal, and he was normotensive. Abnormal physical findings were confined to the calf muscles. Both were tender, and very indurated with nodular swellings. Movement of the affected muscles was limited by pain. There was no obvious loss of power, wasting or fasciculation, tendon reflexes were retained, and there was no sensory deficit.

Creatine kinase was elevated at 1190 (normal 40-150 IU/l). Sedimentation rate, haemoglobin and white cell count, radiographs of legs and chest, complement levels, toxoplasma serology and autoantibody screen were all normal. There was no eosinophilia. No antibodies to coxsackie virus were detected. Stools contained no cysts or ova. Sputum cytology was negative. Electromyograph (EMG) of gastrocnemius showed no spontaneous activity, and polyphasia with good recruitment and abundant small motor units. Needle muscle biopsy of uninvolved muscle (left quadriceps) was normal. Open muscle biopsy of left gastrocnemius revealed well marked changes of polymyositis. There were foci of active muscle necrosis, with collections of lymphocytes and an increase in interstitial connective tissue. There were also lymphocytic infiltrates around blood vessels. Direct immunofluorescence showed C3 and IgM within blood vessels.

While being investigated, his clinical condition spontaneously improved. The induration receded, he was able to walk unaided and the CK level fell to 370 IU/l. In view of this improvement, it was decided to withhold steroid therapy.

Following discharge, with regular review, he has been able to continue his work, walking 8-10 miles per day. His only complaint normally is of stiff calves in the morning. He has had two further clinical episodes of focal myopathy. In December 1985, he redeveloped tender induration in both calves, but again spontaneously settled.

Table Change in CK value (normal 40-150 IU/l) during relapses and remissions of recurrent localised myositis

<table>
<thead>
<tr>
<th>Months from onset</th>
<th>CK</th>
<th>Site of nodules</th>
<th>Number</th>
</tr>
</thead>
<tbody>
<tr>
<td>1-125</td>
<td>1404</td>
<td>gastrocnemius/soleus</td>
<td>2</td>
</tr>
<tr>
<td>1-50</td>
<td>1190</td>
<td>&quot; &quot;</td>
<td>2</td>
</tr>
<tr>
<td>1-75</td>
<td>370</td>
<td>&quot; &quot;</td>
<td>1</td>
</tr>
<tr>
<td>4-50</td>
<td>1374</td>
<td></td>
<td></td>
</tr>
<tr>
<td>5-00</td>
<td>1521</td>
<td></td>
<td>2</td>
</tr>
<tr>
<td>5-50</td>
<td>1100</td>
<td></td>
<td>1</td>
</tr>
<tr>
<td>6-00</td>
<td>1106</td>
<td></td>
<td></td>
</tr>
<tr>
<td>7-50</td>
<td>1350</td>
<td></td>
<td></td>
</tr>
<tr>
<td>10-00</td>
<td>2281</td>
<td></td>
<td></td>
</tr>
<tr>
<td>12-00</td>
<td>953</td>
<td></td>
<td></td>
</tr>
<tr>
<td>13-00</td>
<td>4084</td>
<td>both hamstring &amp; right calf</td>
<td>3</td>
</tr>
<tr>
<td>13-125</td>
<td>891</td>
<td></td>
<td>2</td>
</tr>
<tr>
<td>16-00</td>
<td>106</td>
<td></td>
<td></td>
</tr>
<tr>
<td>17-00</td>
<td>722</td>
<td></td>
<td></td>
</tr>
<tr>
<td>20-00</td>
<td>2454</td>
<td></td>
<td></td>
</tr>
<tr>
<td>23-00</td>
<td>6985</td>
<td></td>
<td></td>
</tr>
<tr>
<td>36-00</td>
<td>62</td>
<td></td>
<td></td>
</tr>
</tbody>
</table>

Letters
within a week. In July 1986, he developed tender induration in both calves and the right thigh. Again, spontaneous remission was swift. At no stage has there been any evidence of a diffuse myopathy or systemic illness. Throughout the course, the CK has been markedly elevated (see table), only once falling into the normal range.

Polymyositis typically presents as a symmetrical proximal myopathy. The clinical diagnosis is confirmed by muscle biopsy showing lymphocytic inflammatory cell infiltrate and fibre necrosis. This is supported by elevated CK and other sarcoplasmic enzymes and characteristic EMG findings of myopathy—spontaneous fibrillation, positive sharp waves and polyphasic motor unit potentials.

Cumming et al first described the focal presentation of this disease, and for it coined the term “localised nodular myositis”. Since then there have been rare cases, the majority of which rapidly progressed to the full clinical syndrome, despite steroid therapy. We describe a patient with three definite episodes of localised nodular myositis, including biochemical, EMG, and muscle biopsy support of the diagnosis. At the time of his first presentation his quadriceps biopsy was histologically normal, when his gastrocnemius was very inflamed. Despite a continuous marked elevation in CK levels, the patient has remained well, fulfilling criteria of CK and other sarcoplasmic enzymes.

Immunofluorescence testing revealed IgG and IgM within blood vessels, supporting the evidence of a vasculitic element in the pathogenesis of polymyositis. We have chosen not to treat an elderly man with steroids since he has no disability, despite the fluctuating CK levels.

In the past, there has been confusion between localised nodular myositis, chronic focal polymyositis, and focal myositis. The first two are unusual manifestations of polymyositis, whereas focal myositis is a benign inflammatory pseudotumour. In chronic focal polymyositis, highly selective muscle wasting and weakness remains confined to individual groups of muscles. This has been described for the forearm flexors, brachio-radialis and quadriiceps, and, more recently, the bulbar muscles. Focal myositis presents as a lymphocytic infiltration, scattered muscle fibre necrosis, and interstitial fibrosis. There is never sign of diffuse muscle involvement or systemic disease, and both blood levels of CK and ESR remain normal. The aetiology is unknown.

Whether the condition of localised nodular myositis is a distinct entity is not clear. In the literature, previously reported cases have been a mode of presentation of polymyositis. It is still possible that a generalised myopathy may develop in this man. It is remarkable that it has not happened in the 3 years that he has been under review.

Localised nodular myositis must be remembered in the differential diagnosis of the focal tender muscle mass, which includes myositis ossificans, proliferative myositis, pseudosarcomatous fascitis, granulomatosis and pyomyositis.

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References


Rhabdomyolysis associated with simultaneous Epstein-Barr virus infection and isolation of echovirus 6 from muscles: a dual infection

Sir: Acute rhabdomyolysis has been associated with a number of viral infections, which has been confirmed by a significant rise in serum antibodies or by isolation of virus from throatwashing, CSF, urine or stool. Previous studies reveal that only influenza A and B, adenovirus type 2 and Coxackie virus type A9 have been isolated from muscle tissue. We report a case of rhabdomyolysis associated with a dual viral infection. Epstein-Barr virus (EBV) was identified serologically and echovirus 6 isolated from muscle.

A 17 year old boy was suffering from a flu-like disease with headache, fever (40°C), and progressive weakness of legs and arms. When after 3 days he started voiding dark-coloured urine and when amoxicillin medication failed to lower his body temperature, he was admitted to hospital. His previous medical history was non-contributory. Previously he had been a healthy young man attending secondary school without exercise-related muscle complaints and his family history gave no evidence of muscle disorders.

On admission the rectal temperature was 39°C and the patient appeared mildly dehydrated. Blood pressure was 120/60 mm Hg and pulse rate 92 beats per minute. Some small axillary and inguinal lymph nodes were palpated. Liver and spleen were not enlarged. Thigh muscles were tender to palpation and weak against resistance. The patient was unable to stand alone without support. The result of the remainder of the physical examination was within normal limits. On admission a ESR of 3 mm/h was noted. WBC was 9.2×10⁹/l with 17% segmented forms, 14% bands, 61% lymphocytes, of which many were atypically shaped, and 8% monocytes. Serum enzymes were strongly elevated: CK 54 050 U/l (normal 15–91 U/l), ASAT 864 U/l (normal 7–25 U/l), ALAT 198 U/l (normal 5–25 U/l) and LDH 5100 U/l (normal 155–275 U/l). Gamma-GT was normal. Urine analysis showed proteinuria (2+) and many pigmented casts were detected. Immunoassay for myoglobin revealed a very high level in serum of 11 100 ng/ml (normal 0–85 ng/ml) and presence in urine of 824 500 ng/ml. AST and ANF were normal and circulating immune complexes absent. The qualitative monoclonal slide agglutination test was positive. By indirect immunofluorescence IgM and IgG antibodies to EBV were detected at titres of 1:512 and 1:2048 respectively. Complement fixation antibody titres to adenovirus, influenza virus, mycoplasma pneumoniae, parainfluenza virus, measles and herpes simplex virus were negative.

On the 9th day after admission a needle biopsy of the quadriceps muscle was carried out. The structure of muscle fascicles was well preserved. Necrosis was absent; some mononuclear cell infiltration could be still
Recurrent localised myositis.

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