Transient acute myositis in childhood

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McKinlay, I. A., and Mitchell, I. (1976). Archives of Disease in Childhood, 51, 135. Transient acute myositis in childhood. Eight cases of transient acute polymyalgia with weakness are described. All had abnormal serum creatine phosphokinase levels and most had minor haematological abnormalities at the time of diagnosis. Virological studies were performed with negative findings.

Vague muscular aches and pains are common in viral infections, especially influenza (Lundberg, 1957). Except in coxsackie infection (Thordarson, Sigurdsson, and Grimsson, 1953), however, objective evidence of myositis has seldom been reported (Middleton, Alexander, and Szymanski, 1970; Hoefnagel, 1970; Mejlszenkier et al., 1973). We describe 8 children in whom clinical and biochemical evidence of muscle involvement was found after an upper respiratory tract infection.

Patients

The 8 patients comprised 6 boys and 2 girls whose mean age was 6 years 10 months (range 5 years 11 months to 9 years 8 months). They were admitted to hospital during the period December 1973 to March 1974 from different districts of Edinburgh and the surrounding country. A history of similar illness in 13 contacts was obtained.

Clinical findings. The clinical findings are summarized in Table I. There was a prodromal illness in all cases 1–7 days before the onset of pain and weakness, consisting of an upper respiratory tract infection with fever. 4 patients had skin rashes affecting mainly the head and neck. Severe symmetrical muscle pains and weakness were the presenting symptoms on admission to hospital. Only the calves were affected in 4; calves and thighs in 2; calves, thighs, and pectoral and proximal arm muscles in 1; and mainly neck muscles in 1. Great reluctance to use the affected muscles on account of pain rather than weakness was the striking feature. All affected muscles were tender. The power in apparently affected muscles was initially assessed as Medical Research Council grade 4 in 5 cases. In Cases 1 and 4 the power in calves and thighs was grade 3, while in case 8 it was grade 3 in biceps, pectoralis major, and calf

<table>
<thead>
<tr>
<th>Case no.</th>
<th>Sex</th>
<th>Age (yr) (m)</th>
<th>Prodromal period (d)</th>
<th>Site of weakness</th>
<th>Duration of weakness (h)</th>
<th>Skin</th>
<th>Contacts with similar illness</th>
</tr>
</thead>
<tbody>
<tr>
<td>1</td>
<td>M</td>
<td>6 2</td>
<td>7</td>
<td>Both calves and thighs</td>
<td>24</td>
<td>Normal</td>
<td>Brother and father</td>
</tr>
<tr>
<td>2</td>
<td>M</td>
<td>7 8</td>
<td>2</td>
<td>Both calves and thighs</td>
<td>48</td>
<td>Normal</td>
<td>None</td>
</tr>
<tr>
<td>3</td>
<td>F</td>
<td>8</td>
<td>4</td>
<td>Both calves and thighs</td>
<td>36</td>
<td>Normal</td>
<td>Sister</td>
</tr>
<tr>
<td>4</td>
<td>M</td>
<td>7 9</td>
<td>2</td>
<td>Both calves and thighs</td>
<td>48</td>
<td>Facial flushing</td>
<td>4 neighbouring children</td>
</tr>
<tr>
<td>5</td>
<td>M</td>
<td>5 11</td>
<td>1</td>
<td>Both calves and thighs</td>
<td>24</td>
<td>None</td>
<td>Sister</td>
</tr>
<tr>
<td>6</td>
<td>M</td>
<td>6 6</td>
<td>3</td>
<td>Both calves and thighs</td>
<td>72</td>
<td>Blotchy rash on hands, arms, and trunk</td>
<td>3 children at school</td>
</tr>
<tr>
<td>7</td>
<td>F</td>
<td>9 8</td>
<td>1</td>
<td>Neck</td>
<td>36</td>
<td>Macular rash on face, neck, and trunk</td>
<td>None</td>
</tr>
<tr>
<td>8</td>
<td>M</td>
<td>9 8</td>
<td>7</td>
<td>Upper limbs, thighs, and calves</td>
<td>24</td>
<td>Flushing of face and upper trunk</td>
<td>Sister and father</td>
</tr>
</tbody>
</table>

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muscles bilaterally. The day after admission all patients were improved but had grade 4 power in the affected muscles. Within 72 hours all had normal power and were free from pain. None had hyperalgesia on cutaneous stimulation and there was no sensory impairment. 2 appeared to have extensor planter responses on admission but no other reflex changes. All had normal reflexes on the day after admission and subsequently. There was no recurrence of symptoms on follow-up of 12–16 months' duration.

**Laboratory findings.** The laboratory findings are summarized in Table II. Serum creatine phosphokinase (CPK) levels were estimated by a modification of the Sigma 520 procedure (Sigma Tentative Technical Bulletin No. 520, 1965, Sigma Chemical Co., St. Louis), which is similar to that of Hughes (1962), and results were expressed as IU/l of serum at 37 °C. The upper limit of normal was 55 IU/l. All the patients showed initial levels of between 4 and 64 times this figure. In all cases CPK returned to normal within 2 weeks and remained so during follow-up. 4 patients had transient neutropenia and 6 had thrombocytopenia. All reverted to normal within one week. Culture of nasopharyngeal secretions for virus was positive in only 2 cases (Table II). Paired sera were examined for evidence of infection by viruses and related agents described as being associated with myositis—that is, *myxovirus influenza A* and *B*, parainfluenza type 1, adenovirus, psittacosis, *Rickettsia burnetii*, *Mycoplasma pneumoniae*, respiratory syncytial virus, corona virus, Coxsackie B1–6 and mumps virus. All patients were negative. The following investigations were made and the results were invariably normal: Hb, urea and electrolytes, calcium, phosphate, alkaline phosphatase, ASO titre, antinuclear factor, bacterial culture from throat and urine, x-ray examination of chest and legs. Cerebrospinal fluid was obtained from the patient with neck pain and was normal.

**Treatment.** All patients were put to bed until their pain resolved. 4 patients received paracetamol but not more than three doses in hospital. Two had penicillin and one ampicillin before admission.

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**Discussion**

The patients described here all had a sudden onset of severe myalgia and some weakness after an upper respiratory tract infection. The symptoms were initially alarming but disappeared rapidly and completely. The time scale of the illness, the history of contacts, and the haematological findings suggested a viral cause, but this was not proved. Infections by the most commonly associated agents were excluded. Possibly a virus or viruses might have been obtained from nasopharyngeal secretions during the prodromal illness. Seemingly, some agent or agents other than those previously described may cause an acute myositis. Apart from their close association in time and similarity in clinical features we could not show that all our cases were of the same disease.

Lundberg (1957) described 74 children with similar acute pain and weakness of calf muscles but no viral studies were made or serum CPK values obtained. Middleton *et al.* (1970) reported severe myositis in 26 children, 14 of whom had moderately raised CPK level but normal haematological findings. Most had evidence of infection with influenza A or B virus. Meijlszenker *et al.* (1973) described one child with myositis of both calves five days after an influenza-like illness. Serological evidence of infection by influenza A virus was found and myositis was confirmed histologically. Stevens *et al.* (1974) described a clinically similar illness in 3 cases which they attributed to acute polynarthritis and associated with influenza B virus infection, but they did not report CPK concentrations or nerve conduction data.

There seemed little doubt that our patients showed features of an acute muscular disorder. In the absence of any cutaneous hyperalgesia or joint symptoms the muscle tenderness and the pain and weakness all seemed highly suggestive of

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**TABLE II**

*Creatine phosphokinase levels, haematological findings, and virus infections in 8 cases of transient acute myositis in children*

<table>
<thead>
<tr>
<th>Case no.</th>
<th>Highest CPK (IU/l)</th>
<th>Lowest neutrophil count (mm³)</th>
<th>Lowest platelet count (mm³)</th>
<th>Viral culture</th>
</tr>
</thead>
<tbody>
<tr>
<td>1</td>
<td>3500</td>
<td>1368</td>
<td>150 000</td>
<td>Nil</td>
</tr>
<tr>
<td>2</td>
<td>347</td>
<td>1394</td>
<td>137 000</td>
<td>Adenovirus type 2</td>
</tr>
<tr>
<td>3</td>
<td>587</td>
<td>1326</td>
<td>150 000</td>
<td>Nil</td>
</tr>
<tr>
<td>4</td>
<td>1566</td>
<td>756</td>
<td>52 000</td>
<td>Nil</td>
</tr>
<tr>
<td>5</td>
<td>1262</td>
<td>2340</td>
<td>92 000</td>
<td>Nil</td>
</tr>
<tr>
<td>6</td>
<td>292</td>
<td>2296</td>
<td>88 000</td>
<td>Nil</td>
</tr>
<tr>
<td>7</td>
<td>209</td>
<td>3626</td>
<td>146 000</td>
<td>Nil</td>
</tr>
<tr>
<td>8</td>
<td>2605</td>
<td>5475</td>
<td>122 000</td>
<td>Nil</td>
</tr>
</tbody>
</table>

Normal range Up to 55

|                | 1500–750*         | 150 000–400 000*             |

* * Black and Barkhan (1974).
acute myositis. We considered the raised serum CPK values to be adequate confirmation of this, and we were reassured by their rapid reversion to normal in parallel with clinical recovery. Probably electromyography and, indeed, muscle biopsy might have confirmed the diagnosis but we thought that neither was justified, particularly in view of the transient nature of the patients' illness and their complete recovery.

The highest serum CPK values in some of our cases are unusual in acute myositis (Pearce, Pennington, and Walton, 1964) though not unheard of (Meilszenkier et al., 1973; Pennington, 1974), being usually found only in Duchenne muscular dystrophy (Pearce et al., 1964). The raised serum enzyme reflects abnormal leakage through the muscle cell membrane, CPK being a cytoplasmic enzyme rather than a bound mitochondrial enzyme (Amberson, Roisen, and Bauer, 1965). The details of the mechanism of raised serum CPK levels are unresolved; interference in muscle cell energy-producing mechanisms may well be involved (Zierler, 1961). The subject has been reviewed by Pennington (1974). The haematological findings are of interest and were presumably related to the effects of the causative agent or agents, as all rapidly reverted to normal.

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REFERENCES


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