Benign postinfection polymyositis

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Summary and conclusions

Six patients developed persistent muscular cramps, aching pain, and fatigability after an influenza-like illness. Electromyography showed myopathic changes, although results of routine laboratory investigations were normal in all but one patient, whose serum creatine kinase concentration was slightly increased. All but one of the patients improved: three were asymptomatic within one to two years. The syndrome was probably a benign form of polymyositis.

Introduction

A symptom complex of muscular aches, pains, and cramps related to exercise is common in clinical practice. Investigation rarely discloses a definite cause, and the complaints are often thought to be psychogenic. Several metabolic myopathies may present in this way, but these disorders are rare.

We describe six patients who presented with persistent but ill-defined muscular complaints after an influenza-like illness. Although routine laboratory investigations were unhelpful, electromyography (EMG) showed changes of a mild myopathic disorder. We suggest that the illness was a benign form of polymyositis, probably induced by viral infection.

Patients and methods

The six patients were aged 35-58 years. Five were women. In each case routine haematological and biochemical investigations were carried out and the serum creatine kinase (CK) concentration measured. Routine EMG was performed on biceps brachii and deltoid in every patient. Other muscles, including quadriceps, were sampled in most cases. In each patient single-fibre EMG was used to determine fibre density in biceps brachii (average number of single muscle fibre action potentials recorded from 20 different motor units[1]) and measure neuromuscular jitter (variability of neuromuscular transmission[2]). One patient had a muscle biopsy.

CLINICAL FEATURES

The features in each case were similar (table I). The illness had begun with fever, headache, cough, and catarrh, often associated with diarrhoea and vomiting. One or two days after onset aching muscular pain occurred, especially in the lower back and thighs. The acute illness resolved in a few days but the muscular complaints—fatigue, cramps, and aching muscular pain—continued, often in relation to exercise. All the patients complained of weakness, but no muscle tenderness or weakness was found and none had neurological signs. All had been treated for several months with salicylates and analgesics by their general practitioners before referral, and one (case 5) had been taking corticosteroids for a few weeks. Three patients recovered; cases 1 and 5 after one year, and case 4 after two years. Cases 2 and 3 were improved 12 and 15 months later respectively. One patient (case 6) had similar symptoms after two-and-a-half years. The following is a typical case.

Case 4—While on holiday in Wales a 41-year-old woman became ill with fever, headache, vomiting, and diarrhoea. The illness lasted three days. On the second day she noticed severe muscular aching pains in her back, thighs, and calves. These were aggravated by walking. The pains were severe for about four days and were treated with codeine phosphate. She gradually improved but three weeks later, while gardening, noticed aching pains and twitching in her arms and legs. For a year these pains recurred after exercise, although gradually lessening in severity. During this time she also complained of undue fatigability. The symptoms interfered with her daily life. Two years after onset all her symptoms had resolved.

Four other patients, three of them women, presented with similar symptoms but were excluded because they had no antecedent illness, no improvement occurred over several months, and their EMGs were normal. Two were thought to be depressed, and one had cervical and lumbar spondylosis.

INVESTIGATIONS

In all cases routine biochemical and haematological values, including erythrocyte sedimentation rate (ESR), were normal. The serum CK concentration (normal < 60 IU/l) was raised only in case 5 (62 IU/l). All the patients, however, showed EMG abnormalities (table II). Increased insertional activity was seen in cases 4 and 6 but no fibrillation potentials or positive sharp waves were recorded. One patient (case 4) showed pseudomyotonic discharges. In each case several short-duration polyphasic motor unit action potentials of low to normal amplitude were observed on volition. In case 6 double discharges were present. The interference pattern was full in all patients. Single-fibre EMG showed increased fibre density in biceps brachii in cases 2, 3, and 4. There was increased jitter in one potential in cases 4 and 6, and in case 6 this potential also showed impulse blocking. Three patients (cases 1, 2, and 6) were followed up by EMG two to three months after their initial investigation. In cases 1 and 2 the findings were normal. In case 6 mild myopathic potentials remained.

Biopsy of the right deltoid was performed in case 6, the only patient who did not show clinical improvement. Centrally placed nuclei were found in 9% of the fibres in the specimen (normal $< 4\%$). The distribution of type 1 and type 2 fibres was normal, and there were no structural changes in individual fibres. There was a sparse interstitial infiltration of mononuclear cells. Blood vessels were normal.

Discussion

These six patients had a feverish illness associated with muscular pain in the acute phase followed by a persistent symptom complex of muscular cramps, aching pain, and fatigability, often related to exercise. These symptoms, which were particularly noticeable in the legs, were most severe during the first few months. There was no objective weakness but all the patients were restricted in their daily activities by pain and fatigue.

Myalgia is a common feature of acute infectious illnesses, particularly influenza,[3] in which pain in the muscles of the legs and lower back is characteristic. It usually improves rapidly but may be intense for several days. The serum CK concentration may be slightly raised during this time.[4 5] Weakness and fatigability are common in influenza and other feverish illnesses, but weakness cannot usually be measured objectively. Friman,[6] however, using an isometric measuring system, reported slight weakness soon after resolution of the acute illness in patients

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TABLE I—*Details of patients studied*

Case No	Age and sex	Antecedent illness	Duration of illness before referral	Clinical features	Outcome
1	35 F	Fever, sore throat, muscular aches	4 months	Muscular cramp and pain on exercise; fatigability	Recovered in 1 year
2	39 F	Fever, muscular aches, diarrhoea, drowsiness	9 months	Fatigability, muscular aches	Improved in 1 year
3	40 M	Flu-like illness	1 year	Fatigability, muscular aches	Improved in 15 months
4	41 F	Fever, vomiting, diarrhoea, and muscular pain for four days	1 year	Fatigability, muscular pain after exercise	Recovered in 2 years
5	55 F	Headache, weakness, and muscular aches for four days	9 months	Fatigue and muscle pain after exercise	Recovered in 1 year
6	58 F	Flu-like illness	2 years	Fatigability, muscular pain, and tenderness after exercise; depression	Unchanged in 2½ years

TABLE II—*ESR, serum CK concentrations, and EMG findings*

Case No	ESR (mm in 1st h)	CK (normal <60 IU/l)	Initial EMG findings	Single-fibre EMG (biceps brachii)	
				Fibre density* (normal <1·8)	Neuromuscular jitter†
1	12	19	Occasional myopathic potentials	1·3	Normal
2	4	15	Myopathic potentials	2·4	Normal
3	2	35	Occasional myopathic potentials	2·6	Normal
4	3	25	Increased insertional activity, some myopathic potentials, pseudomyotonic discharges	2·1	One potential with increased jitter. No blocking
5	12	62	Myopathic potentials	1·4	Normal
6	6	40	Increased insertional activity, many myopathic potentials, double discharges	1·8	One potential with increased jitter and blocking

*Average number of single muscle fibre action potentials recorded from 20 different motor units.
†Variability of neuromuscular transmission.

with influenza. Transient changes in neuromuscular transmission may also occur in the acute myalgic phase of influenza and echovirus infections.[7] These findings suggest that muscles are affected in some acute viral infections. There have been several reports of polymyositis complicating influenza[8] and other viral infections.[9 10] Furthermore, influenza virus may replicate in tissue cultures of human skeletal muscle cells,[11] and influenza[8] and other viral infections[9 10] have been associated with subacute polymyositis, often principally affecting the calves.

Our patients showed myopathic EMG abnormalities, but these were not prominent. Single-fibre EMG disclosed abnormalities in four patients. Two had an increased neuromuscular jitter in single potential pairs, and one of them showed impulse blocking. Fibre density was increased in three of our patients. Increased fibre density occurs in various neurogenic and myopathic disorders, including polymyositis.[12] In classical polymyositis, however, the EMG is usually grossly abnormal. Prominent myopathic changes are found, sometimes associated with fibrillation potentials, and the fibre density is usually greater than in our patients.[12] Although the EMG abnormalities were not severe in our patients, the combination of clinical features, EMG findings, and clinical course suggests that they had a myopathy: probably a mild polymyositis. In case 6 the histological features were consistent with this diagnosis.

In several of our patients a psychogenic disorder was initially considered as the most likely diagnosis, although overt depressive symptoms were present only in case 6. The clinical features in our patients were not well defined, and similar complaints may be a presenting feature of depression or vertebral spondylosis. Nevertheless, the history of antecedent illness and the results of investigations, particularly EMG, were positive features lacking in these other disorders, as, for example, in the four patients we excluded after investigation. A further possibility is epidemic myalgic encephalomyelitis. In this condition[13] there may be signs of central nervous system disorder, and exhaustion with emotional lability and lack of concentration are characteristic. None of our patients had symptoms other than those of muscular disease. In our cases only symptomatic treatment was needed, and five of the six patients recovered or were improved two years later.

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ONE HUNDRED YEARS AGO Marks of serious violence, which may be mistaken for acts of murder, are often seen on the bodies of persons found dead in rivers. The throat may be cut, and the wound involve the carotid arteries; or there may be a gunshot wound, involving the heart or the brain. Such injuries, being likely to destroy life speedily, may be pronounced to be inconsistent with suicidal drowning subsequent to their infliction; and, unless the circumstances are known, a jury may be disposed to return a verdict of wilful murder. An inquest was held during the last week by Mr St Clair Bedford on the body of a Mr O Smith, recovered from the Serpentine. Dr Blackett stated that he found a bullet-wound through the chest on the left side. It involved the heart and must have caused instantaneous death. The evidence showed that the deceased was heard to discharge a revolver, and his body then fell over the bridge into the Serpentine. The facts were thus easily explained. (*British Medical Journal*, 1878.)