Pentazocine-induced fibromyositis and contracture

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Summary

We report a case of myopathy, accompanied by widespread contractures predominantly involving the elbow and knee joints, following long-standing pentazocine abuse.

Keywords: pentazocine; myopathy; contractures

Complications of parenteral narcotic abuse such as focal tissue damage (localised sclerosis of skin and subcutaneous tissue, indurations and nonseptic ulcerations) are well documented.^{1,2} There are a few reports of myopathy following chronic pentazocine administration,^{3,4} but the association with contractures around the shoulder and hip joints is still rare.^{5,6} We report a patient with myopathy, accompanied by widespread contractures predominantly involving the elbow and knee joints, following long-standing pentazocine abuse. A detailed electrophysiological study of different muscle groups and biopsies from involved and healthy sites was also carried out.

Case report

A 38-year-old doctor (figure 1) presented with a painless and progressive restriction of flexion around both the knee joints for 4 years. This had been followed by a similar problem involving both the elbow joints a year later. His main complaints were inability to squat, difficulty in dressing/undressing and generalised stiffness. Over the last 6 months he had mild limitation at the extremes of abduction of the right shoulder, and bilateral hip extension for which he had developed a compensatory lumbar lordosis. He had no complaints of any muscle weakness within the limited range of possible movements. He had been addicted to injection

of Fortwin (pentazocine) 1–2 ml intramuscular bid for the past 18–20 years. The site of injection was the right deltoid for the first 8 years, followed by sequential use of both glutei, quadriceps and the left deltoid. He had a history of frozen shoulder on the right side which subsequently improved. He was non-diabetic and non-hypertensive. There was no family history of any neuromuscular disorder.

Physical examination revealed a well-built man with marked contractures involving both the knee and elbow joints and mild limitations of abduction of right shoulder and extension of both hip joints. He walked with a lordotic gait and had marked woody indurations of the deltoids, biceps, glutei and quadriceps. Both elbows were semi-flexed with a 20° range of movement on either side. The knees could not be flexed beyond 80° and the right hand could not be abducted beyond 90°. Distal joints were normal. Muscle power was normal within the limited range of movements, and there was no sensory deficit. Laboratory studies disclosed the following values: haemoglobin 15.0 g/dl; white cell count $6.0 \times 10^9/l$ (neutrophils 67%, lymphocytes 20%, monocytes 3%, eosinophils 10%); platelets $275 \times 10^9/l$; erythrocyte sedimentation rate 2 mm 1st hour; serum urea 8.9 mmol/l; serum creatinine 0.09 mmol/l; aspartate transaminase 27 U/l; alanine transaminase 9 U/l; alkaline phosphatase 56.8 U/l; creatine kinase 324 U/l. Radiography revealed normal joint architecture. Electromyography (EMG) of the deltoids, triceps and quadriceps showed a reduced number of potentials, low in amplitude and of short duration. Polyphasias were increased (>40%) with a full recruitment pattern. EMG of the supraspinati was normal. Muscle biopsy showed a normal right soleus, whereas there was extensive fibrosis involving the left quadriceps (figure 2).

It was explained to the patient that his drug addiction was responsible for the condition, and he received drug rehabilitation treatment as well as vigorous physiotherapy with passive and active stretching exercises. At 6 months follow-up he had successfully overcome his addiction, but there was no change in his deformity apart from some increase in range of mobility around both the elbow joints to 40–45°, from the previous range of 20°.

Discussion

Cutaneous complications of pentazocine injections were first described by Schlicher *et al.*¹ Swanson *et al.*, in their study of psychiatric aspects of pentazocine abuse,⁷ noted a 33%

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Figure 1 The patient (reproduced with his permission)

Figure 2 (A) Muscle biopsy from right soleus showing normal morphology; (B) muscle biopsy from left quadriceps showing scar tissue consisting of mature fibrosis tissue (Haematoxylin & eosin stain; bar = 50 μ m)

Learning points

- pentazocine-induced myopathy is a rare condition and should be looked for in all patients using intramuscular pentazocine
- it should be considered in the differential diagnosis of any atypical form of myopathy with contractures
- multiple muscle groups should be evaluated by EMG/biopsy as the disease may be restricted to specific muscle groups
- differentiating this iatrogenic myopathy from polymyositis/dystrophy is of immense prognostic value

incidence of brawny induration of skin and underlying tissue. The list of complications was later extended by Steiner *et al* and Joong *et al* who described a fibrous myopathy with intramuscular pentazocine injections.^{3,4} Their patients had woody induration of muscles with secondary contractures. Deltoid contracture causes a fixed abducted posture, aptly termed 'arm levitation sign', a signal of chronic injection myopathy.⁸ The main histopathologic change is extensive fibrosis in skin and muscle. Endarteritis, vascular thrombosis, granulomatous inflammation and fat necrosis may also be seen but are non-specific.⁹

The history of progressive decline in mobility with generalised stiffness and a lordotic gait in our patient might suggest joint disease. However, the extensive induration of soft tissue with resultant contractures and normal radiographic studies point to the nonarticular nature of his deformity. Although he did not have the arm levitation sign,⁸ he did have a history of frozen shoulder. Differential diagnosis of this clinical picture would include infiltrative myopathies such as amyloidosis, eosinophilic fasciitis and the stiff man syndrome. Rarely, an adult form with a forme-fruste manifestation of

Emery Dreifuss muscular dystrophy may bear a close resemblance. The history of chronic pentazocine injection in conjunction with extensive fibrosis on biopsy is sufficient to exclude these conditions. The normal EMG of the supraspinati and normal muscle biopsy of the soleus point against a generalised muscle involvement, despite the raised creatine kinase.

The mechanism of this condition remains unclear. Repeated injections of other medications are not commonly reported to result in diffuse muscle fibrosis. Pentazocine is most soluble under acidic conditions, and precipitation in the alkaline pH of extracellular tissue with secondary inflammation has been postulated.¹ Birefringent crystals have been noted in the areas of induration. The role of repeated muscle trauma, microhaemorrhages, and infections is unknown. Elevations of muscle enzymes, as seen in our patient, have been recorded and suggest ongoing muscle destruction. Animal studies utilising repeated injections of pentazocine show induction of localised sclerosis in guinea pigs but not in rats.⁴

Pentazocine-induced fibrous myopathy should be included in diagnostic considerations when a parenteral narcotic agent is required for management of chronic pain.

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Spontaneous rupture of a saphenous vein graft

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Summary

We present a case of spontaneous rupture of a right coronary bypass vein graft in a 57-year-old woman 10 years after coronary by-pass surgery. Although rare, this diagnosis should be considered in such patients presenting with appropriate symptoms.

Keywords: coronary artery bypass graft; false aneurysm; saphenous vein graft

Coronary artery rupture as a consequence of interventional procedures is well described both for native coronary vessels and for bypass grafts.¹ Spontaneous rupture in the absence of intervention is rare, though described for native vessels.^{2,3} Spontaneous rupture of a bypass graft is an even rarer occurrence.

Case report

A 57-year-old woman presented with sudden onset of chest pain, described by her as 'explosive', and so severe as to 'knock her to the ground'. The pain was in the anterior chest, radiated through to the back, and was exacerbated by deep respiration and by movement. She felt nauseated and was breathless. Moderately intense pain continued until admission to hospital 4 hours later, by which time she had experienced some minor haemoptysis. She had undergone coronary surgery 15 years earlier, with vein grafts to the left anterior descending artery and diagonal branch; 10 years ago the vein grafts were occluded, and she underwent further coronary surgery, with grafts to the left anterior descending artery (left internal mammary artery) and the right coronary artery (saphenous vein graft). Two years prior to the current admission, angiography demonstrated impaired left ventricular function, with an ejection fraction of 45%, occluded left anterior descending artery, with a good distal internal mammary artery graft, severe native circumflex disease, and an occluded native right coronary with the distal vessel supplied by an ectatic bypass vein graft, measuring up to 2 cm in diameter (figure 1). Medical management was pursued.

Examination showed her to be in mild distress, warm, heart rate 90 beats/min, with no signs of heart failure and normal cardiac auscultation. Saturation on finger plethysmography was 76%. Chest X-ray showed cardiomegaly with clear lung fields. Electrocardiography (ECG) showed no new changes.

The most likely diagnosis was felt to be a pulmonary embolus, though aortic dissection was also considered. Computed tomography (CT) scan revealed a unremarkable aorta, with an

Figure 1 Angiography of the venous bypass graft to the right coronary artery. This figure shows a 6 French diagnostic coronary catheter intubating the ectatic right coronary graft.

abnormal structure lying behind the heart. Transthoracic echocardiography (TTE) revealed a small 1 × 2 cm fluid-filled structure lying behind the left atrium. Twelve hours later repeat TTE showed that this structure had enlarged dramatically, resulting in severe compression of the left atrium. Transoesophageal echocardiography (TOE, figure 2) showed a large mass behind and to the right side of the heart. This structure had a thick rim, with spontaneous contract echo. Laterally there was a thick rimmed structure divided by a thick septum. Repeat CT scan of the chest (figure 2) showed that contrast leaked into the posterior structure. At urgent thoracotomy, a large false aneurysm of the right coronary saphenous by-pass graft was found, with extensive extravasation of blood into the surrounding tissues. Haemostasis was achieved, but it was not possible to wean her from bypass, and she died.

Discussion

Late rupture of a coronary bypass graft seems very rare. In this case, rupture led to extrinsic compression of the left atrium, and subsequent left heart failure, hypoxaemia, haemoptysis and death. Of particular interest was the difficulty in establishing the correct diagnosis, which was in part due to its rarity. At presentation, the sudden onset of pleuritic chest pain, with hypoxaemia and haemoptysis led to an initial diagnosis of pulmonary embolism, though common forms of vascular catastrophe, including aortic dissection, were considered. Initial

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