FVC% in all our subjects.[4] Fortunately, the ventilatory impairment is of little clinical importance but it offers the clinician an objective assessment of progress: the absence of any improvement in the first week makes it unlikely that treatment is effective, thereby saving time and allowing exploration of alternative therapeutic approaches.

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Acute renal failure due to polymyositis

Crush injury is a recognised cause of acute renal failure, histologically giving characteristic myoglobin casts in biopsy specimens. A similar syndrome can be precipitated by muscle damage associated with barbiturate overdose[1] and carbon monoxide poisoning.[2] We report a case of acute renal failure in which the renal biopsy specimen showed a typical "crush injury" appearance due to severe acute polymyositis.

Case report

A 56-year-old theatre technician was admitted with a ten-day history of increasing proximal muscle pain, tenderness, and weakness. He was afebrile but had dark urine and increasing oliguria. Investigations showed blood concentration of urea 34 mmol/l (205 mg/100 ml), creatinine 569 μmol/l (6·4 mg/100 ml), potassium 6·9 mmol(mEq)/l, creatinine phosphokinase 681 IU/l (normal 26-200 IU/l), and alanine transferase 1004 IU/l (normal range 0-40 IU/l). The blood concentration of urea rose to 77 mmol/l (464 mg/100 ml) with a creatinine of 1164 μmol/l (13·2 mg/100 ml) over the subsequent three days. An arteriovenous shunt was inserted and haemodialysis started. A specimen obtained by renal biopsy to determine the cause of his acute renal failure showed casts in tubules reminiscent of those seen in "crush injury." Electromyography showed irritability to needle provocation at rest with repetitive potentials and abundant fibrillation. During contraction there were many polyphasic and brief-duration potentials compatible with severe polymyositis. A muscle biopsy specimen of the vastus internus showed necrotic fibres undergoing phagocytosis and a perivascular infiltration of mononuclear cells and lymphocytes.

He was given prednisolone, 100 mg/day. He remained anuric for 10 days requiring repeated haemodialysis. Subsequently his renal function returned to normal. He continued to lose weight with weakness, wasting, and tenderness of the proximal muscles, and he lost 20 kg in weight in three weeks before improving. An electromyogram six weeks later while he was taking prednisolone 10 mg/day showed all muscles electrically silent at rest with no fibrillation activity. During contraction there were full interference patterns of motor unit potentials. A muscle biopsy specimen showed an obvious improvement with a few scattered atrophic fibres and no interstitial inflammation. In view of his occupation as a theatre technician, specimens of muscle were examined in vitro for sensitivity to halothane and other anaesthetic agents. No abnormality was detected.

When last seen his muscle bulk and power had returned to normal, as had his serum biochemical values. He was well and no longer receiving steroids. The results of investigations for autoimmune disorders, viral infection, and malignancy had been negative.

Discussion

Polymyositis is a disease of unknown aetiology. It presents as proximal, symmetrical muscle weakness with pain and tenderness. The urine is frequently dark red, due to myoglobin. Muscle biopsy specimens show muscle fibre degeneration with a variable, often perivascular, inflammatory cell infiltrate. Electromyography shows spontaneous activity and myopathic changes[3-5] and the serum creatinine phosphokinase concentration is raised. These features were all present in this patient, supporting a diagnosis of polymyositis. The disease may occur in association with connective tissue disorders, such as rheumatoid arthritis or dermatomyositis, carcinoma, and viral infections. There was no evidence of such conditions in this patient.

Treatment is with corticosteroids, initially prednisolone 30-100 mg/day, the dose depending on the severity of the disease, reducing to a maintenance dose of 5-15 mg/day, depending on the clinical response. This patient made a satisfactory and rapid response to treatment. With reduction and cessation of this there has been no evidence of any relapse and he remains asymptomatic.

This case is unusual in as much as the muscle lysis was produced by a polymyositis of unknown cause, severe enough to produce a "crush injury" type kidney with reversible acute renal failure.

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Long-term self-administered subcutaneous heparin in pregnancy

Antenatal thromboembolism is an important complication of pregnancy and a major cause of maternal death. Patients previously advised against pregnancy now present to the obstetrician with prosthetic heart valves or a history of major thromboembolism[2] and require anticoagulants throughout pregnancy. Oral anticoagulants may increase the perinatal mortality and are probably teratogenetic.[1 3] If their administration during the first trimester and the last three weeks is avoided the risks to the fetus can be reduced.[2] Heparin, which does not cross the placenta, is safer but the need for long-term parenteral treatment is a disadvantage. Self-administration of subcutaneous heparin is safe and effective,[4] and we describe our experience.

Patients, methods, and results

Nineteen patients received subcutaneous heparin during 22 pregnancies. Thromboembolism occurred in 14 pregnancies and subcutaneous heparin was preceded by an intravenous heparin infusion (40 000 units daily). Eight patients with a history of thromboembolism started receiving prophylactic subcutaneous heparin after their first antenatal visit, around 12 weeks. One patient taking long-term warfarin was changed to heparin at six weeks' gestation. The patients were instructed to give their own injections in a manner similar to diabetics. The site of injection was left to the patient, the thigh being preferred to the abdomen. When they were considered competent and their dosage had been established, the patients were allowed home. The dose was monitored in most cases by the activated partial thromboplastin time (APTT). A result lower than the control was taken as an indication for increasing the dose. If the APTT result was comparable with or higher than the control the same dose was maintained. In a few patients antifactor Xa assay was performed and the dose altered to keep the level in the prophylactic range. Those patients breast-feeding were maintained on heparin for about six weeks post partum and those electing to bottle feed were changed on to warfarin.

The duration of treatment in the antenatal period ranged from two to 34 weeks. The total antenatal experience was 374 weeks (169 weeks in the