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

Details of treatment and outcome of pregnancy

Case No	Parity	Reason for treatment	Time started (weeks)	Dosage of heparin	Duration of treatment (weeks)	Complication of treatment	Fetus
1	1+0	DVT at 30 weeks	30	IV 10 000 units 6-hourly, SC 5000 units bd, increasing to 12 500 units bd	10 + puerperium	None	Well, 3400 g, breast-fed
2	0	PE at 22 weeks	22	IV 10 000 units 6-hourly, SC 8000 units 8-hourly	17	None	Well, 3350 g, bottle-fed
3	1+1	DVT at 20 weeks (previous postpartum DVT)	20	IV 10 000 units 6-hourly, SC 10 000 units bd, increasing to 10 000 units tds	20	None	Well, 3230 g, bottle-fed
4	1+3	DVT at 31 weeks (chemical diabetic)	31	IV 10 000 units 6-hourly, SC 8000 units bd, increasing to 10 000 units bd	7 + puerperium	None	Well, 3400 g, breast-fed
5	0	DVT + PE at 34 weeks	34	IV 10 000 units 6-hourly, SC 12 000 units bd	6	None	Well, 2940 g, bottle-fed
6	1	DVT at 30 weeks (previous pregnancy DVT)	30	IV 10 000 units 6-hourly, SC 8000 units bd, increasing to 10 000 units bd	10	None	Well, 3260 g, bottle-fed
7	0	Prophylaxis. DVT on pill 1973, recurrence 1974	12	SC 5000 units bd, increasing to 8000 ml tds	28 + puerperium	None	Lower segment caesarean section under epidural, well, 3260 g, breast-fed
8	1	Prophylaxis see case No 7	12	SC 8000 units bd	28	None	Lower segment caesarean section, well, 4060 g, breast-fed
9	0	Cortical vein thrombosis and right femoral vein thrombosis (previous DVT on pill with multiple PE, inferior vena cava complication)	12	IV 10 000 units 6-hourly, SC 5000 units bd, increasing to 15 000 units bd	28 + puerperium	Recurrence of DVT at 20 weeks while on SC heparin 5000 units bd	Lower segment caesarean section, well, 4300 g, breast-fed
10	0	Prophylaxis on long-term warfarin after DVT × 4 after laminectomy	6	SC 2000 units bd, increasing to 7500 units bd	34 + puerperium	? DVT on 2000 units bd at 6 weeks, degenerating fibroid at 19 weeks	Well, 3780 g, breast-fed
11	0+2	Prophylaxis. DVT with missed abortion	13	SC 5000 units bd	27, stopped one week after delivery	None	Well, 3520 g, breast-fed
12	1+2	Prophylaxis as in case No 11	10	SC 5000 units bd	30, stopped on discharge	None	Well, 3380 g, breast-fed
13	2	DVT at 28 weeks	28	IV 10 000 units 6-hourly, SC 7500 units tds	12	None	Well, 3600 g, bottle-fed
14	3	Prophylaxis see case No 13	12	SC 7500 units bd	28	None	Well, 3450 g, bottle-fed
15	2	DVT at 32 weeks	32	IV 10 000 units 6-hourly, SC 7500 units bd	6	None	Well, 3500 g, breast-fed
16	2	DVT after appendicectomy	24	IV 10 000 units 6-hourly, SC 5000 units bd	16	None	Well, 3750 g, bottle-fed
17	2	DVT at 36 weeks	36	IV 10 000 units 6-hourly, SC 7500 units bd	2 + puerperium	None	Well, 3560 g, breast-fed
18	1	DVT at 34 weeks	34	IV 10 000 units 6-hourly, SC 5000 units bd	3	None	Well, 3960 g, bottle-fed
19	2	DVT at 16 weeks (previous DVT)	16	IV 10 000 units 6-hourly, SC 5000 units bd, increasing to 7500 units bd	22	None	Well, 4050 g, breast-fed
20	1	Prophylaxis. Previous PE on warfarin	36	SC 5000 units tds	2 + puerperium (4 weeks)	None	Well, 3300 g, breast-fed
21	2	PE at 26 weeks	26	IV 10 000 units 6-hourly, SC 10 000 units bd	10 + puerperium	None	Well, 3240 g, breast-fed
22	0	Prophylaxis. Previous PE	8	SC 7000 units bd, increasing to 15 000 units bd	28	None	Well, 3910 g, breast-fed

DVT = Deep vein thrombosis. PE = Pulmonary embolus. IV = Intravenous. SC = Subcutaneous. bd = Twice daily. tds = Thrice daily.

treatment group and 205 weeks in the prophylaxis group). The daily dose of subcutaneous heparin at the start of treatment ranged from 4000 to 24 000 units; by the end of pregnancy the range was 10 000-30 000 units daily. Most patients required an increased dose as pregnancy progressed.

Two patients receiving a low dose (4000 units and 10 000 units daily) showed evidence of deep venous thrombosis, which disappeared with intravenous heparin. There were no haemorrhagic complications. Three patients had caesarean section with no notable increase in operative blood loss. Bruising at the injection site was common, sometimes producing appreciable tenderness, but alternating the site from thighs to abdomen largely overcame this problem. No other problems arose.

Discussion

Oral anticoagulants are hazardous in early pregnancy and labour, and to advocate their withdrawal at 37 weeks makes no allowance for possible premature labour. Subcutaneous heparin appears safe and effective and it seems particularly important that all women exposed to the risk of pregnancy and who require long-term anticoagulant treatment should be given subcutaneous heparin. The effective dose range is between 5000 and 15 000 units twice daily. Because of increasing blood coagulability a dosage increase around the 28th week may be required. A dose of 10 000 units twice daily should be safe and effective throughout pregnancy, even without knowing the APTT result or the antifactor Xa level. Renal disease may cause higher serum heparin concentrations,[4] making some control of treatment, preferably estimating antifactor Xa, necessary.

Recent work[5] suggests that warfarin is no longer contraindicated in breast-feeding women.

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Correction

Severe envenomation from "harmless" pet snake

We regret that an error occurred in the figure of this article by Dr Mather and others (20 May, p 1324). The scale for fibrinogen concentration should have been calibrated from 0 to 1·0 g/l in 0·2 g/l steps.