Previous reports of peripheral nerve lesions in haemophilia have only rarely recorded or recommended surgery. Ehrman found that 37·5% of peripheral nerve lesions in his study showed only partial or no recovery.[1] The rapid and complete clinical recovery shown in this case suggests that when intraneural haemorrhage is possible early surgical decompression with adequate factor VIII replacement is the treatment of choice.

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Diabetogenic effects of nifedipine

Nifedipine is widely used to treat angina pectoris and hypertension. Some studies of its effects on carbohydrate metabolism and insulin secretion have shown it to be potentially diabetogenic,[1 2] although another study found that it had no appreciable effect on glucose tolerance.[3] We report on one patient in whom diabetes deteriorated and another in whom it developed during the use of nifedipine; in both cases these effects disappeared after withdrawal of the drug. We have not been able to find any other such reports, and neither the adverse drug reaction monitoring team in Kuwait nor the manufacturers of nifedipine have been notified of such side effects.

Case reports

CASE 1

A 56 year old man was diagnosed as having diabetes mellitus in 1982. He had had hypertension and type IV hyperlipidaemia since 1969 and a myocardial infarction in 1978. His diabetes was controlled by diet and he was prescribed propranolol 320 mg/day, chlorthalidone 50 mg thrice weekly, isosorbide dinitrate 30 mg/day, and clofibrate 2 g/day. In August 1983 his fasting plasma glucose concentration was 7·9 mmol/l (142·4 mg/100 ml), and oral nifedipine 30 mg/day was started to control his hypertension and mild angina. Ten days later he complained of fatigue and polyuria, and his fasting plasma glucose concentration was found to be 30·8 mmol/l (555 mg/100 ml). He was admitted to hospital, and insulin injections were started.

Examination did not show any abnormality. His blood pressure was 120/80 mm Hg and his temperature normal. Blood tests showed: white cell count $7{\cdot}5 \times 10^9$/l, erythrocyte sedimentation rate 61 mm in the first hour, blood urea concentration 19 mmol/l (114 mg/100 ml), creatinine concentration 336 μmol/l (3·8 mg/100 ml), and normal serum electrolyte concentration. Urine and throat cultures were sterile.

Despite twice daily injections of insulin zinc suspension (Monotard) and neutral insulin (Actrapid), his plasma glucose concentrations during the day were 19·6-22·4 mmol/l (353-404 mg/100 ml) with no ketonuria. Three days later nifedipine was stopped. His plasma glucose concentrations fell gradually and the doses of insulin were tapered down. He was discharged, taking a small dose of insulin, when his fasting plasma glucose concentration was 8·2 mmol/l (148 mg/100 ml). During follow up insulin was withdrawn, and two months later his fasting plasma glucose concentration was 5·4 mmol/l (97 mg/100 ml) without any medication.

CASE 2

A 67 year old man, hypertensive since 1973 and taking propranolol 180 mg/day, had suffered a myocardial infarction in the same year. His fasting plasma glucose concentration was 5·5 mmol/l (99 mg/100 ml) in December 1982, and oral nifedipine 20 mg/day was started for angina. A month later he started to complain of polyuria, and his fasting plasma glucose concentration was 10·4 mmol/l (187 mg/100 ml). No cause could be found for this sudden development of diabetes mellitus. During the next few months his fasting plasma glucose concentration remained between 8·8 and 9·8 mmol/l (159 and 177 mg/100 ml). Nifedipine was stopped in November 1983 when his symptoms became worse. Three days later his fasting plasma glucose concentration was 5·2 mmol/l (94 mg/100 ml) and he stopped complaining of polyuria, although his angina worsened.

Comment

The administration of nifedipine to both patients was associated with raised plasma glucose concentrations, which returned to normal after this drug was withdrawn. We could not find any other factor in either patient to explain these changes. In view of reports that nifedipine may be diabetogenic,[1 2] we conclude that it was responsible for the deterioration in glucose homeostasis in these two patients. Verapamil, another calcium antagonist, has been experimentally shown to inhibit the release of insulin.[4] Although more studies are needed to confirm the effect of nifedipine on glucose tolerance, we suggest that plasma glucose concentrations should be monitored in diabetic and non-diabetic patients receiving this drug.

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Traumatic dislocation of the hip in mini rugby

Traumatic dislocation of the hip in children is rare and seldom presents to an individual surgeon. Most reports have combined the experience of several surgeons. We report two cases of traumatic dislocation of the hip incurred in mini rugby, both of which presented to the same orthopaedic surgeon within six months.

Case reports

Case 1—A 10 year old boy was kneeling, having been tackled, when he was knocked forwards on to his hands and knees by another player who then sat on his right buttock. The posterior disclocation thus caused was reduced with ease under general anaesthesia within a few hours. He then spent four weeks in skin traction, and the joint remained non-weightbearing for a further two months, when he used crutches. His recovery was without complication: within eight months he had regained a full and painless range of movement of the hip and was back in the rugby team.

Case 2—A 10 year old boy was injured after scoring a try. He was on his hands and knees when another player jumped on his back, causing a posterior dislocation of the hip. Reduction was achieved with ease within three hours of injury. He was immobilised in fixed traction with a Thomas splint for four weeks, and the joint remained non-weightbearing for a further two months, when he used crutches. Six months after the injury his hip was normal, both clinically and radiologically.

Comment

During games of mini rugby both these boys sustained a posterior dislocation of the hip without any associated fracture; neither showed any complication during the subsequent nine months. Posterior dislocation of the hip usually occurs when a force is directed proximally up the shaft of the femur from the knee to the flexed hip. This can happen in younger children who fall on to their knees or in older children who suffer greater trauma—for example, by falling from a height or in road traffic accidents. In our cases the knee was pressed to the ground and the dislocating force was applied to the pelvis via another player's body weight. This mechanism of injury has been described previously in a child aged under 2 who was on his knees and elbows when his father fell on his back.[1]

The major complications of traumatic dislocation of the hip include avascular necrosis of the femoral head, traumatic arthritis, coxa

magna, nerve injuries, premature epiphyseal fusion, recurrent dislocation, and persistent limp. After such dislocation the overall incidence of abnormality of the hip at skeletal maturity is around 30%.[2-5] The incidence of avascular necrosis of the femoral head, which occurs up to two years after injury, is about 10%.[4] Factors that predispose to complications are age above 6 years, delay in reduction, severe trauma (usually found in older children), associated fractures, and possibly open reduction. The method of immobilisation after reduction and the length of time beyond four weeks for which the joint is non-weight bearing are not important.

The incidence of complications of traumatic dislocation of the hip in children is thus appreciable though lower than that in adults; however, dislocation occurs with less force. This has an important bearing on mini rugby, in which children as young as 7 or 8 may play. Children may have a tendency to assume the "knee elbow" position on the ground, so leaving them vulnerable to traumatic dislocation of the hip. Our two cases, both sustained in mini rugby, presented to the same orthopaedic surgeon within a short time; this suggests that this injury may be more common than previously thought.

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Reversible acute on chronic renal failure during captopril treatment

Treatment with captopril controls the blood pressure well in most patients with severe hypertension. Nevertheless, some patients may develop reversible renal insufficiency, especially when there is bilateral renal artery stenosis.[1] We describe three patients with reversible acute on chronic renal failure in the absence of renal artery stenosis.

Case reports

Case 1—A 51 year old man with a long history of analgesic abuse had been treated for hypertension with clonidine. He had stage 1 hypertensive retinopathy. An ultrasonic tomogram and intravenous urogram with rapid sequence films showed bilateral small kidneys with delayed excretion. There was no evidence of renal artery stenosis. Ten days after starting captopril serum creatinine concentration rose from 380 to 681 μmol/l (43 to 77 mg/l) and renal biopsy was performed. Histological examination showed thickening of Bowman's capsule, hyperplastic atherosclerosis of the interlobular arteries, and spontaneous fluorescence of tubular epithelium related to analgesic abuse.

Case 2—A 54 year old man with known hypertension had grade 3 hypertensive retinopathy. There was no suggestion of secondary hypertension. After 11 days of combined treatment with captopril (75 mg/day) and frusemide (80 mg/day) serum creatinine concentration rose from 491 μmol/l (55·5 mg/l) to 673 μmol/l (76 mg/l). Renal biopsy was performed and examination by light microscopy showed an increase in mesangial matrix and capillary thickening.

Case 3—A 45 year old man had been treated with metoprolol, prasosin, and thiazide. Renal failure was noted before he started taking captopril and an intravenous urogram with rapid sequence films had shown atheromatosis of the aorta and atrophic kidneys, but no evidence of renal artery stenosis. He had stage 3 hypertensive retinopathy. Combined treatment consisted of captopril 25 mg, frusemide 80 mg, and atenolol 50 mg daily. Serum creatinine concentration rose to 885 μmol/l (100 mg/l) and haemodialysis was performed on day 11. A renal biopsy specimen obtained 10 days after starting captopril showed only an increase in mesangial matrix and capillary thickening.

Renal function improved in all three patients with no change in their treatment. Proteinuria was always less than 0·5 g/l and no allergic phenomena appeared. Immunofluorescent examination of biopsy specimens did not show significant immune deposits. There was no correlation between changes in blood pressure and those in serum creatinine concentration.

Comment

In all three patients there was a substantial decline in renal function during the first 10 days of captopril treatment. Hypotension and dehydration did not occur in these patients and there was no evidence of immunological or toxic renal damage induced by captopril.

Severe functional and reversible renal failure has been observed during captopril treatment in patients with bilateral renal artery stenosis or with a solitary kidney with renal artery stenosis.[2] In such patients renal perfusion pressure is low and angiotensin II is important for maintaining renal blood flow and glomerular filtration. Blocking the converting enzyme in this condition may lead to renal failure.[3] A similar mechanism could have been responsible for the renal failure in our patients since renal perfusion may be considerably impaired in patients with severe arterial hypertension even without renal artery stenosis.[4] It is unlikely that diuretics or β blockers contribute to this form of reversible renal failure since acute renal failure can occur on captopril alone, and renal function may improve rapidly once captopril is withdrawn even though β blockers and diuretics are continued.[2] In addition, giving diuretics or β blockers, or both, to patients on captopril does not adversely affect renal function.[1]

In our patients renal function improved despite continuing captopril treatment. Reversability of captopril induced renal failure has also been noted by Brunner and others in patients who had renal failure before starting captopril.[5] These cases suggest that in some patients the decline in renal function may be temporary and that stopping the captopril is not always necessary. The improvement in renal

Clinical details of the three cases

	Captopril daily dose (mg)	Other drugs taken during treatment with captopril (mg)	Blood pressure (mm Hg)	Body weight (kg)	Serum creatinine (μmol/l)	Serum urea (mmol/l)	Plasma renin activity (ng/ml/h)
Case 1							
Before captopril			240/120	56·9	380	17·5	2·47
At highest creatinine concentration	75	Atenolol 100 Frusemide 40	135/98	55·2	681	45·3	
After 3 months' treatment	75	Atenolol 100 Frusemide 40	170/100	59·0	354	20·7	
After 9 months' treatment	75	Atenolol 100 Frusemide 40	170/95	66·0	283	15·3	
Case 2							
Before captopril			210/120	70·7	491	18·8	3·25
At highest creatinine concentration	75	Frusemide 80	150/95	69·7	673	42·3	
After 3 months' treatment	150	Frusemide 80 Atenolol 50	160/100	69·0	398	23·3	
After 9 months' treatment	150	Frusemide 80 Atenolol 50	160/100	71·7	266	16·8	
Case 3							
Before captopril			170/130	47·5	557	47·8	6·15
At highest creatinine concentration	25		170/95	47·7	885	73·0	
After 3 months' treatment	12·5	Frusemide 80 Atenolol 50	170/100	49·5	504	39·5	
After 9 months' treatment	12·5	Frusemide 80 Atenolol 50	120/80	47·5	389	24·2	