In view of his deteriorating renal function, resistant hypertension, and widespread atheromatous disease, renal artery stenosis was suspected, and he underwent intravenous digital subtraction angiography. This showed a non-functioning right kidney and postostial stenosis of the left renal artery, with poststenotic dilatation. Renal vein renin studies showed no lateralisation, and peripheral concentrations were within the normal range. Because of his poor medical condition he was admitted for percutaneous transluminal renal angioplasty. Arterial angiography confirmed a tight stenosis with a pressure gradient of 100 mm Hg, falling to zero after dilatation. His blood pressure remained unchanged that evening at 200/120 mm Hg. There were no immediate postangioplasty complications, and his antihypertensive treatment was continued.

Over the next 24 hours he had a considerable diuresis and lost 2 kg in weight. On the morning after angioplasty, while sitting in a chair, he lost consciousness and was incontinent of urine; his pulse could barely be felt. On return to bed he regained consciousness; his blood pressure at this time was 130/80 mm Hg and his pulse rate 80 beats/min and regular. There were no new neurological deficits, and his supine blood pressure settled at 180/100 mm Hg. His antihypertensive treatment was reduced, and he suffered no further syncopal attacks. On the morning of his syncopal attack his packed cell volume had increased from 0·34% to 0·39% and total protein concentration from 70 g/l to 80 g/l. His serum creatinine concentration settled at 161 μmol/l, and his blood pressure was easily controlled with slow release nifedipine 20 mg twice daily at around 150/80 mm Hg; antiplatelet treatment was also started.

Comment

This patient had a solitary functioning kidney with a stenosed arterial supply and normal plasma renin activity. This is comparable to the one kidney-one clip model of experimental renovascular hypertension, which is not renin dependent.[2] After a technically successful dilatation his blood pressure did not fall until 12 hours later, after a large diuresis. This diuresis may have been induced by pressure or volume or related to poor tubular function.

We conclude that a large diuresis after percutaneous transluminal renal angioplasty may precipitate orthostatic hypotension. It is therefore important to monitor urine output and, if this is excessive, to ensure adequate volume replacement.

Hypovolaemia and hypotension should be avoided as such patients may also have cerebrovascular disease, and strokes may be precipitated.[3] It is important to observe these precautions after angioplasty in patients with single functioning kidneys or bilateral renal artery stenosis.

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Cerebrospinal fluid fistula after lumbar puncture

Cerebrospinal fluid leaking into the surrounding soft tissues is well known after lumbar puncture and is thought to be the most important factor in the genesis of postpuncture headache. We describe a unique case of hitherto unknown chronic external leak of cerebrospinal fluid through a fistula which developed after lumbar puncture.

Case report

A 16 year old boy with a clinical diagnosis of cauda equina syndrome was subjected to lumbar puncture. An 18 gauge needle was introduced in the L3-4 intervertebral space in the left lateral position and about 6 ml fluid collected in a single prick with no evidence of traumatic tap. Simultaneously 6 ml lipid soluble contrast medium was introduced. Subsequent myelography showed nothing abnormal. Following the custom in this hospital, we did not remove the lipid contrast after the myelography. The patient lay prone for the next three hours with his feet raised.

Twenty four hours after the lumbar puncture cerebrospinal fluid started oozing through the puncture wound. Pressure dressings with adhesive tape did not help. Epidural blood patching[1] was also tried but without success. Two months later the leak persisted and the fistula was sutured subcutaneously. The leak stopped for three days but then started again. After three months of persistent leak the patient was treated by absolute bed rest in the prone position with the head down and cisternal punctures were done daily for seven days, 10-20 ml cerebrospinal fluid being withdrawn each time. This completely stopped the leak within a week, and after a further two weeks' bed rest in the same position the patient was discharged. No leak was detected over the next eight months.

Comment

Internal cerebrospinal fluid leak of clinical importance is manifested by postpuncture headache. The average incidence of headache after lumbar puncture is 41%,[2] but this may be appreciably reduced by preventive measures.[3] These measures fail in about a third of patients, however, who therefore have clinically significant cerebrospinal fluid leak into the surrounding soft tissues. External cerebrospinal fluid fistulas occur in patients with head injury, infections, neoplasms, and hydrocephalus[4] and present as rhinorrhoea or otorrhoea, but there is no definite report of such a fistula developing after a lumbar puncture.[5]

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Equality in death: disappearance of differences in postneonatal mortality between northern and southern regions of England and Wales

Evidence is increasing that the state of health in adulthood is closely linked to that in childhood.[1-3] For many years postneonatal mortality was higher in the industrial north of England than the more affluent south because of its greater proportion of working class families. After a plateau in the figures for England and Wales in the 1960s, however, postperinatal mortality fell during the 1970s.[4] We therefore looked at whether this fall also applied to postneonatal mortality and examined the present trend in northern and southern regions of England and Wales.

Methods and results

All figures were obtained from the Registrar General's annual statistical reviews and surveys (DH3 series) of the Office of Population Censuses and Surveys. Northern regions were taken as the north east, Yorkshire, the east Midlands, the west Midlands, the north west, and Wales. Southern regions were taken as East Anglia, London and the south east, the south, and the south west.

The figure shows the trends in postneonatal mortality in the northern and southern regions. Rates remained higher in the northern regions until the fall in the early 1970s, which brought equality with the south of England, and this persisted. Rates in the south of England remained the same after 1960. The fall in postneonatal mortality in the 1970s in northern regions fully explained the fall in the figures for England and Wales, which happened at the same time.

Comment

Postneonatal mortality shows the effects of environmental conditions during the first year of life free from the effects of fetomaternal interactions