An intravenous pyelogram was performed using 20 ml Conray 280 (a total dose of 5.6 g iodine). The child vomited during the injection of contrast but showed no immediate anaphylactic reactions. There was prompt excretion of contrast from both kidneys which showed normal pyelograms. 24 hours after the investigation the child complained of right-sided facial pain and developed a unilateral, localized swelling of the right parotid gland but was afebrile. There was local tenderness with erythema, unilateral conjunctivitis, photophobia, and lacrimation. Iodide allergy with sialadenitis was diagnosed and diphenhydramine hydrochloride 50 mg, 3 times daily, was started and the swelling subsided within 3 days. Mumps antibody titres showed negative results (mumps S+V antibody, titres 1/8 in both acute and convalescent sera).

Comment

In addition to renal clearance, inorganic iodide is removed from plasma by the breasts, the thyroid, the stomach, and the salivary glands (Mason, Harden, and Alexander, 1966). Though the salivary glands trap substantial amounts of iodide, this accumulation is independent of thyroid function (Fellinger *et al.* 1956; Gabrielsen and Kretschmar, 1956).

Iodide sialadenitis, a painful swelling of the salivary glands, has been reported after infusion pyelography in a uraemic adult (Nakadar and Harris-Jones, 1971). This was associated with a conspicuous rise in plasma inorganic iodide, but these workers also showed the presence of similarly raised levels in reversible renal failure with no clinical evidence of salivary gland enlargement. Furthermore, the plasma inorganic iodide remained raised until the ureteric obstruction responsible for the renal failure was relieved. Though the pathogenesis of iodide sialadenitis is uncertain, it seems, therefore, to depend upon individual sensitivity and not upon the absolute level of inorganic iodide. This was not measured in our patient, but with normal renal function and after a standard amount of contrast medium, a prolonged rise in plasma inorganic iodide seems unlikely.

A review of published reports revealed only 6 cases of iodide sialadenitis, 5 were adults with apparently normal renal function and one case was in a uraemic adult. The condition followed either oral or intravenous iodide. The glandular swelling occurred typically within 48-96 hours of iodide administration and clinical resolution was complete by the 7th day, as in our patient. In the 6 reported cases, the adenitis was either unilateral or bilateral and involved the submandibular or parotid glands, though 1 patient experienced abdominal pain with vomiting and coexistent pancreatitis was diagnosed. However, in none of these cases was

mumps excluded by appropriate serological investigation.

Tucker and Di Bagno (1956) reviewed 2000 cases given intravenous iodide for pyelography but found no case of parotitis within this group.

Despite the relative infrequency of this form of salivary gland enlargement, iodide sialadenitis should be considered in the differential diagnosis of acute submandibular or parotid swelling in childhood. A history of recent intravenous pyelography suggests the diagnosis, but in this context it should be remembered that iodide-containing proprietary cough mixtures can be freely purchased by parents, unknown to their family doctor. A careful drug history should be obtained, therefore, in all cases of acute salivary gland enlargement where the clinical features or history seem atypical.

Summary

An 8-year-old child developed an acute swelling of the right parotid gland after intravenous pyelography. The possible relation of this sialadenitis to iodide sensitization is discussed.

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D. C. DAVIDSON,* J. A. FORD, and E. G. FOX
Division of Medical Paediatrics, Stobhill General Hospital, Glasgow G21 3UW.

*Correspondence to Dr. D. C. Davidson.

Bilateral renal venous thrombosis Recovery after peritoneal dialysis

Renal venous thrombosis is a well-recognized entity of early infancy, 60% of cases occurring in babies under the age of 2 months. The thrombosis originates in the small intrarenal veins (Johnston,

1968), though less commonly it may start in the inferior vena cava (Verhagen, Hamilton, and Genel, 1965). The consequences of the thrombosis are acute tubular necrosis with or without renal infarction, there being a wide variation in severity. Though cases may occur in previously healthy babies, renal venous thrombosis is frequently secondary to dehydration or sepsis. The diagnosis is usually evident clinically, with loin masses, haematuria, and azotaemia, and while its confirmation may be desirable radiologically, the risk of renal medullary necrosis with high doses of contrast media (Gilbert *et al.*, 1970) does not in general justify angiography.

Case report

A male infant was delivered at term after an uneventful pregnancy, weighing 3.15 kg. At the age of 26 days he was transferred to this hospital from another where he had been admitted earlier the same day with a history of intermittent vomiting from birth, diarrhoea for 1 week, and haematuria for 24 hours. The family history was noncontributory.

On examination he was markedly dehydrated, though not collapsed, and weighed 2.65 kg. Blood pressure was 110 mmHg systolic (1" cuff). Both kidneys were considerably enlarged, approximately 6 cm in length, hard, and fixed. The bladder was not palpable and small amounts of heavily bloodstained urine were present in a urine bag applied some hours previously.

Laboratory investigations. Hb 10.4 g/100 ml; WBC 19,000/mm³ with 82% neutrophils, 15% lymphocytes, and 3% monocytes; platelet count 67,000/mm³; plasma sodium 133 mEq/l.; potassium 7.3 mEq/l.; urea 405 mg/100 ml; calcium 8.3 mg/100 ml; magnesium 2.14 mg/100 ml; total protein 5.6 g/100 ml; albumin 2.5 g/100 ml; arterial blood pH 7.31; Pco₂ 18 mmHg; standard bicarbonate 16 mEq/l.; blood culture sterile; urine culture (suprapubic aspiration) *Esch. coli*; chest x-ray normal; straight x-ray abdomen—outline of an enlarged left kidney visible.

Management. Peritoneal dialysis was instituted once assessment was completed. It was complicated by short-lived seizures some hours after onset, easily controlled with diazepam. No cause was found for the seizures and they did not recur. Gentamicin 1 mg/kg was given intramuscularly, subsequent dosage being regulated by serum levels. Oral feeding was started within hours of admission, and after 96 hours, when the plasma urea was 111 mg/100 ml, the dialysis was discontinued and did not have to be restarted. The kidneys remained enlarged until the 4th day and then progressively became smaller until at the time of discharge the left one was palpable, but normal in size and consistency. Platelet count remained low for 5 days and then quickly recovered, while Hb stabilized at 9 g/100 ml. Creatinine clearance at day 5–6 was 9 ml/min per 1.73 m² with plasma creatinine 2.16 mg/100 ml and

glomerular filtration rate on the 15th day, calculated from the plasma clearance of ⁵¹Cr-labelled EDTA, 30 ml/min per 1.73 m². The baby was thriving at this time and has continued to do so. When reassessed at the age of 4 months, physical examination was normal and the blood pressure was 100/70 mmHg. Urine was free of protein. Creatinine clearance was 43 ml/min per 1.73 m² and a ⁵¹Cr EDTA clearance was 58 ml/min per 1.73 m². Intravenous pyelogram was normal.

Discussion

The diagnosis of renal venous thrombosis is usually evident on clinical grounds, though it cannot be formally shown without pathological examination. The evidence presented here, namely firm enlargement of both kidneys, oliguria, heavy proteinuria, azotaemia, and thrombocytopenia, is very strongly suggestive of bilateral renal venous thrombosis. In the past the management has been essentially expectant, giving intravenous fluids, antibiotics, and sometimes anticoagulants with generally a poor salvage rate. Survival with conservative measures has been reported occasionally since Fallon's case in 1949 and obviously depends on the severity of the thrombosis and the extent of kidney destruction. In 1965, Verhagen *et al.* performed the first successful thrombectomy for a case of inferior caval and bilateral renal venous thrombosis. Only twice since then has thrombectomy been reported as being successful (Lowry *et al.*, 1970; Mauer *et al.*, 1971).

Before operation can be undertaken, the extent of the thrombosis must be accurately shown, remembering the attendant risks of the investigation and that the ultimate outcome of operation depends on the extent of the infarction and on adequate clearance of the main renal veins relieving the obstruction in the smaller venous radicles. The claim of surgical success must obviously be set against the equally satisfactory result described in this case report. Peritoneal dialysis has only been described once previously as being successful in a case of bilateral renal venous thrombosis, the baby dying at the age of 5 months of chronic renal failure (Lugo *et al.*, 1969).

The purpose of this case report is to show that with the use of peritoneal dialysis the prognosis for bilateral renal venous thrombosis may not be so poor now as previously believed, not only because of its low risk compared to operation, but also because of its facility and availability.

Summary

Complete recovery after peritoneal dialysis in a neonate with acute renal failure and clinical evidence of bilateral renal venous thrombosis is described. The use of dialysis in similar cases is advocated.

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RALPH COUNAHAN*

The Hospital for Sick Children, Great Ormond Street, London.

*Correspondence to Dr. R. Counahan, Queen Elizabeth Hospital for Children, Hackney Road, London E2 8PS.

Spinal cord damage in a newborn infant

The obstetrician may sometimes have to decide between rapid delivery, with the risk to the baby of traumatic injury, and delay with its risk of severe birth asphyxia. Strain imposed on the neck of the baby during delivery may in certain cases damage the brain stem and spinal cord (Yates, 1959; Towbin, 1969). The mechanical effects on the spine of manipulation of the head and trunk are important especially during breech delivery, but so is the state of the baby during delivery in that if asphyxiated it will usually be hypotonic and therefore be unable to resist stretch. It is the purpose of this paper to draw attention to the likelihood of spinal injury to the asphyxiated baby during delivery.

Case report

After a 41-week, normal pregnancy, a primigravid mother was admitted to hospital with breech presentation. Labour was induced by anterior rupture of the membranes. Fetal heart rate during labour was 120-150/min. About 3 hours after the onset of labour the cord prolapsed. As the cord was nonpulsatile and the liquor was meconium stained, the mother was delivered by breech extraction, forceps being applied to the after-coming head. A male infant was born, limp, cyanosed,

and apnoeic weighing 2.7 kg. He was resuscitated by intubation and intermittent positive pressure ventilation. Later the baby was found to be neurologically abnormal and to have a chronically distended bladder. The neurological abnormalities were hypotonia, absence of Moro reflex, sluggish limb movements (especially lower limb movements), and a patulous anus. X-ray of the spine revealed no bony defect.

Aged one week the baby weighed 2.7 kg and his head circumference was 35.0 cm. He had a high-pitched cry but sucked normally and turned towards diffuse light. Spontaneous movements were present in the upper but not in the lower limbs, and the trunk and lower limbs were conspicuously hypotonic. The traction, grasp, crossed extensor, asymmetric tonic neck, and Moro reflexes were absent. His tendon and abdominal reflexes were sluggish, his cremasteric reflexes normal, and his plantar responses extensor. Urine was passed in dribbles and the bladder was distended. Clonic fits occurred in the following week and were controlled with phenobarbitone.

At 3 weeks the infant had some flexor tone in his upper limbs, fed well, and often gazed steadily at the person feeding him. A cystogram showed a slightly trabeculated bladder and a urinary infection with *Esch. coli* was treated with trimethoprim and sulphamethoxazole (Septrin).

When about 4 weeks old he was discharged from hospital. 4 weeks later he was readmitted with severe hypothermia and died the same day.

Necropsy findings were spinal cord atrophy involving about 2.5 cm in the midcervical region with thickened adherent dura mater, a small subdural haematoma in the right temporo-parietal area, and moderate haemorrhagic cystitis.

Discussion

The spinal cord, blood vessels, and dura mater are protected by the vertebral column, ligaments, and muscles, and can normally withstand the stresses imposed during labour and delivery. When muscle tone is inadequate, the ligaments may permit the vertebral column to be unduly stretched and flexed with elongation of the spinal cord, blood vessels, and dura mater. These structures could therefore be compressed and torn without associated bony injury. In several early reports of spinal injury in the newborn infant (Burr, 1920; Crothers, 1923; Ford, 1925; Crothers and Putnam, 1927) emphasis was given to the mechanical effects of breech delivery but not to the state of the baby during delivery. It is possible that a baby asphyxiated *in utero* runs a higher risk of sustaining a spinal injury because it is hypotonic.

Yates (1959) reported spinal injuries in 27 of 60 unselected perinatal deaths. The affected infants were born by breech delivery, normal delivery, or caesarean section. There were extradural and subdural haemorrhages and haemorrhages into