infants who showed evidence of permanent brain damage are the first and last cases described in the text.

We are grateful to Dr Declan Meagher, master of the National Maternity Hospital, for his invaluable support; to Dr Niall O'Brien and Dr Helen Mulcahy, who assessed the newborn infants; to Dr Francis Geoghegan and Dr Joan Gearty, who performed the post-mortem examinations; and to the nursing sisters for their co-operation.

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SHORT REPORTS

Retrobulbar neuritis and infectious mononucleosis

In a recent leader in the *British Medical Journal*[1] on the aetiology of optic neuritis it was stated that, "By no means all cases of optic neuritis are due to multiple sclerosis and other causes, particularly in the older patients, should be remembered." We would like to support this statement by reporting a case of retrobulbar neuritis complicating infectious mononucleosis in an 18-year-old male student.

Case Report

An 18-year-old student was admitted to hospital with a 24-hour history of loss of vision. Two weeks previously he had had an upper respiratory tract infection and for one week bitemporal headache, pain when moving both eyes, and tiredness. On examination he was found to have bilateral retrobulbar neuritis, in addition to palatal petechiae and generalised lymphadenopathy and splenomegaly. Apart from the ocular signs no other neurological abnormality was shown then or subsequently.

Investigations showed haemoglobin 14·4 g/dl, erythrocyte sedimentation rate 28 mm in 1 h, white blood count $6{\cdot}2 \times 10^9$/l (6200/mm^3) (neutrophils 19%, lymphocytes 76%, monocytes 5%). Blood film showed many atypical lymphocytes. A Monospot test gave a strongly positive result; the titre of the Paul-Bunnell test was 1/1286; cerebrospinal fluid and Lange's test result were normal; and the Epstein-Barr virus titre (fluorescent antibody test) was 80. Vision in the right and left eyes was 6/36-1 and 6/60 respectively.

He was treated with 1 mg ACTH (Synacthen Depot) twice weekly. The patient reported some minimal improvement in his vision, but there was no objective evidence of this. After nine weeks the ACTH was increased to three times weekly but the optic atrophy progressed, particularly on the left. Because of the severity of the visual defect a prolonged course of treatment seemed justified. Treatment was finally stopped after a further nine weeks, his visual acuity remaining unchanged.

Discussion

Neurological complications in infectious mononucleosis are well known but seldom lead to significant permanent damage. The ocular manifestations of infectious mononucleosis were reviewed by Tanner[2] in 1954. He divided the clinical features into two groups: those possibly due to direct involvement of the eye, such as conjunctivitis, eyelid and periorbital oedema, episcleritis, uveitis, optic neuritis, papilloedema, retinal oedema, and haemorrhage; and those due to a more remote lesion in the central nervous system such as extraocular muscle palsies, ptosis, and disturbances of conjugate deviation.

Probably the earliest report of optic neuritis in glandular fever was by Clemens[3] in 1907, but corroborative details are lacking. The first authenticated report of retrobulbar neuritis without other central nervous system involvement was by Ashworth and Motto[4] in 1947; subsequent reports, together with a case report, were reviewed by Frey[5] in 1973—a total of 8 cases.

All the cases reported to date did well, with or without steroid therapy, and complete recovery was the rule. Unfortunately our patient did not follow this pattern. Possibly he has some other condition, but as yet none has been shown.

We suggest, therefore, that the possibility of infectious mononucleosis as a cause of retrobulbar neuritis should not be overlooked.

We thank Dr C R S Jackson and his staff, at Princess Alexandra Eye Pavilion, Royal Infirmary, Edinburgh, for their help in management of this case, and Mrs G B McKenzie for her secretarial help.

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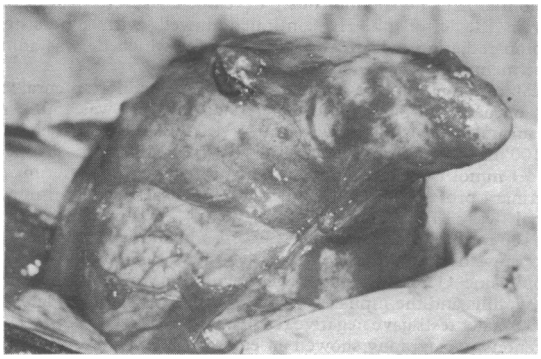
Hydatid cysts in pancreas

In only two out of 780 cases of hydatid disease affecting various tissues and organs operated on in one surgical unit in Baghdad during the period 1963-75 was the cyst located in the pancreas—an incidence of 0·25%.

Case reports

The first case was that of a 27-year-old woman complaining of a gradually growing abdominal lump and heaviness in the left upper abdominal quadrant for the past year. There was a palpable mass ($7{\cdot}6 \times 10{\cdot}2$ cm) notched in its lower pole, in the left hypochondrium. It moved vertically with respiration, was smooth, mobile, and not tender and was thought to be a large spleen. The results of *x*-ray examination of the chest and abdomen, routine haematological investigation, liver function tests, and Casoni's test were all normal.

At laparatomy a large hydatid cyst was found protruding below the stomach and adherent to the transverse colon, mesocolon, and omentum. It arose from the body of the pancreas at its junction with the tail and had a bud on its lower pole, producing the notch mentioned above (see fig). The liver and other abdominal viscera were normal. The cyst cavity was full of milk-coloured hydatid fluid, with many daughter cysts and shredded membranes.



Hydatid cyst of body of pancreas.

The contents were evacuated and all but about one-eighth of the ectocyst, which projected freely into the peritoneal cavity was excised, leaving a disc of ectocyst attached to the pancreas. The main pancreatic duct was eroded and communicated with the cyst cavity, pancreatic juice flowing freely from its orifice. The communication was closed and a corrugated drain left in the area. The patient made an uneventful recovery.

The second case was that of a 45-year-old man complaining of right upper abdominal pain radiating to the back and right shoulder and accompanied by nausea and occasional vomiting with intolerance to fatty meals. These disturbances continued for a year. Icterus then appeared and became progressively more intense with clay-coloured stools and dark urine. The patient was very cachectic and had lost 20 kg in weight over a few months. He was feverish (38°C); the liver was enlarged, smooth, and not tender; and the gall bladder was distended. The results of investigations suggested an obstructive jaundice, and carcinoma of the head of the pancreas was diagnosed.

At laparotomy the liver was found to be enlarged and darkly stained, the gall bladder distended, and the common bile duct dilated. A cystic mass projected just lateral to the second part of the duodenum. The mass was located in the head of the pancreas and on aspiration a crystal clear fluid was obtained. Five minutes after injecting 5% formalin the ectocyst was incised and a single endocyst the size of a small orange was completely extracted. A corrugated drain was left in the area. The patient improved after the operation and his jaundice subsided.

Comment

These cases exemplify two different modes of presentation of pancreatic hydatid cysts, according to the part of the pancreas affected. One mimicked an enlarged spleen and the other a carcinoma of the head of the pancreas. The pancreatic juice flowing into the cyst cavity explained the unusual milky colour of the hydatid fluid and daughter cysts in the first case.

I thank Mr H Al-Hashimi for allowing me to report the second case, in which the patient was under his care.

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Unilateral renal vein thrombosis and nephrotic syndrome

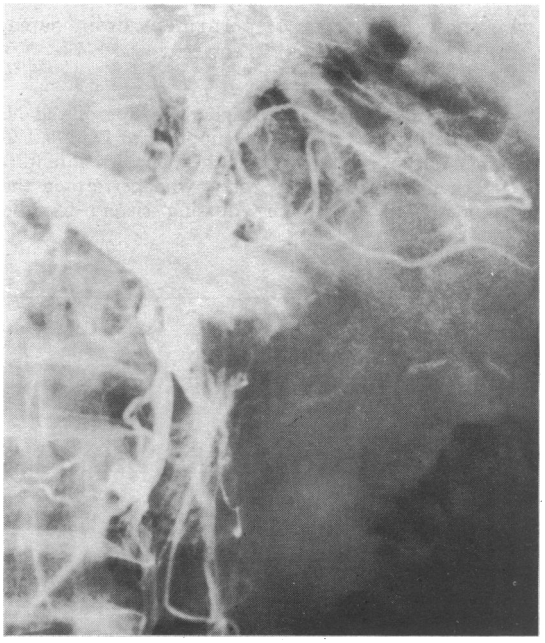
The pathogenesis of nephrotic syndrome in patients with thrombosis of one or both renal veins remains obscure despite an increasing number of case reports. The following case strongly supports the theory that the nephrotic syndrome is the cause and not the consequence of renal vein thrombosis (RVT).

Case Report

In July 1973 a previously healthy 48-year-old man developed insidious bilateral ankle swelling. There was no history of sore throat, skin infection, drug consumption, or trauma. and no familial history of diabetes mellitus. Three months later he spontaneously developed a severe pain of one day's duration in the left flank. He continued unwell, with a persistent, dull aching, non-radiating pain in the left flank, for which he was admitted to our department.

Physical examination showed peripheral oedema, left pleural effusion, and pain on palpation of the left flank. Blood pressure was 150/90 mm Hg. Routine urine analysis disclosed protein 4+. Blood urea was 4·3 mmol/l (26 mg/100 ml), creatinine 84 μmol/l (0·95 mg/100 ml), fasting blood glucose 4·1 mmol/l (73 mg/100 ml), total proteins (47 g/l), serum albumin 18 g/l, urinary proteins 9 g/24h, and creatinine clearance 92 ml/min. Serum cholesterol was 13·0 mmol/l (501 mg/100 ml), triglycerides 2·80 mmol/l (248 mg/100 ml), total serum lipids 13·4 g/l. Qualitative analysis of urinary proteins showed a typical non-selective glomerular-type pattern. The antistreptolysine O and C3 titres were within normal limits. Serological tests for syphilis and the lupus erythematosus cell preparation, latex fixation, and Rose-Waaler tests gave negative results.

Intravenous pyelography showed an enlarged left kidney, with stretching of the pyelocalyceal cavities, which suggested a swelling of the renal parenchyma; the right kidney was normal. Aortography only confirmed the enlarged left kidney. Inferior venacavography with selective right and left renovenograms showed complete left renal vein thrombosis (see fig) without involvement of the other vessels.



Left renal phlebography: complete thrombosis of proximal segment of renal vein with increased collateral circulation.

Immediately after the selective phlebography the patient developed an episode strongly suggestive of right renal vein occlusion—a possible complication of phlebography. Thrombolytic treatment was started, and despite a pulmonary infarction, complete recovery occurred rapidly. Management was continued with intravenous heparin for four weeks and dicoumarol for eight months. The patient improved gradually and was readmitted in September 1974 for needle renal biopsy. Typical features of membranous glomerulonephritis were found: mild basement membrane thickening on periodic acid-Schiff stains, which was confirmed by the presence of irregular spikes on the external face of the basal membrane on Gomori stain. There were no amyloid deposits. Fourteen months later the patient was participating in normal activities and his renal function remained normal despite continued proteinuria.

Discussion

This observation led us to consider the relation of RVT to the nephrotic syndrome. There is evidence to suggest that RVT is a complication of nephrotic syndrome rather than a cause of it. In experimental animals, except when the contralateral kidney has been removed, occlusion of one renal vein does not produce a nephrotic syndrome.[2 3] De Laurentis and Iyengar reported the same observation in man.[1] In several cases of unilateral RVT and nephrotic syndrome in which separate urine specimens were collected the urinary protein excretion was symmetrical.[5] On the other hand, experimental venous ligature does not produce morphological changes of membranous glomerulonephritis.[2 3] Moreover, in patients with unilateral RVT changes of membranous glomerulonephritis have been found in both kidneys.[3 5] All these experimental and clinical data suggest strongly that RVT occurs with rather than causes the nephrotic syndrome which is in fact due to glomerulonephritis. The clinical history of our patient agrees with this view: indeed, oedema preceded signs of RVT by several months.

On the other hand, there is a significantly higher incidence of thromboembolism of any kind in patients with the nephrotic syndrome; recent studies to elucidate the causes of this phenomenon have shown existence of an hypercoagulable state in these patients.[4] We thought that since RVT is only an event illustrating the hypercoagulable state of the nephrotic syndrome the primary treatment of this condition should be medical. This treatment proved successful in our patient.

We are indebted to Dr A Gossuin for referring this patient to us.

[1] De Laurentis, D A, and Iyengar, S R K, *American Journal of Surgery*, 1970, 120, 41.