indistinct outlines both of the collagen disease subdivisions and of Goodpasture's syndrome itself make this extremely difficult.

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Recurrent Meningitis

T W I Lovel BM (for Arnold Bloom MD FRCP)

A K, male, aged 22. Window cleaner

History: Aged 4, meningitis (organism not known; no other details available). Aged 9, fell off an air-raid shelter; hit head and was probably knocked out for a short time; not admitted to hospital. Aged 18, meningitis; CSF culture – pneumococci; sugar 44 mg/100 ml; cells 3,680/c.mm; responded to penicillin. Aged 19, meningitis; CSF culture – no growth; sugar 20 mg/100 ml; cells 17,280/c.mm; responded to penicillin, streptomycin and chloramphenicol. Aged 20, meningitis; CSF culture – pneumococci; cells 7,680/c.mm; responded to penicillin, sulphadimidine and chloramphenicol. Aged 22 (1963), meningitis; CSF culture – pneumococci; responded to penicillin alone.

Investigations: Serum globulin normal. X-ray skull and sinuses: absent frontal sinuses and no aeration of sphenoid. Fluid from right nostril: sugar content 100 mg/100 ml.

Operation (Mr I McCaul): Right frontal craniotomy. Small adhesions of brain to dura mater were found near right olfactory bulb, with a pinpoint hole in the dura at this point. The olfactory bulb was detached and a piece of temporalis muscle laid over the cribriform plate and the hole. The post-operative course was uneventful and there has been no recurrence of the meningitis.

Discussion

Before the antibiotic era cases like this were extremely rare, although Bronstein (1929) reported one such patient. More recently, numerous case reports have appeared, and the record seems to be held by a patient who suffered 18 attacks of meningitis in five years (Cawthorne 1954).

The cause is either a congenital lesion (usually a defect in a suture line) or defective healing of a fracture. The time elapsing between trauma and meningitis may be short or long. Hoyne & Schultz (1947) described a case where eleven years passed between the trauma and the first of 5 attacks of meningitis. In the case reported by Stenger (1958) there was an interval of thirty-one years between the injury and the first attack; at operation a bony defect was found in continuity with the suture line.

In the present case it is not clear whether the lesion was congenital or acquired. A congenital lesion is suggested by the fact that the first attack of meningitis preceded the head injury, unless it is assumed that this was a spontaneous infection.

The presence of CSF rhinorrhœa is very important. It may be difficult to obtain a history of fluid flowing from the nose, since it is often so infrequent, so small in volume and so dependent upon positioning of the head that the patient dismisses it as of no significance. Our own patient only remembered after a long time that hanging his head upside down out of a window produced a small flow, and a sample was obtained only when he adopted a similar posture. Although CSF rhinorrhœa is highly suggestive of an anterior fossa lesion, the flow may be from the middle ear via the eustachian tube, or from the contralateral anterior fossa (Cairns 1937).

All patients with recurrent meningitis and CSF rhinorrhœa should undoubtedly be offered neurosurgical exploration, and Boe & Huseklepp (1960) have advocated operation in every case, even if no CSF rhinorrhœa can be shown and no bony deficit demonstrated on X-ray.

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Fanconi's Syndrome

M I Lavelle MB (for T Parkinson MD FRCP)

Mr J H, aged 41. Unemployed

The patient was first admitted on 20.12.63 at the request of the public health authorities. He was said to be a known case of disseminated sclerosis who, by virtue of his disease, was incapable of looking after himself.

History: Nine years previously he complained of weak legs and unsteady hands. Seven years ago his hands developed a tremor. These were thought to be hysterical phenomena at the time. Two years ago, when seen again for these complaints, he was found to have dysarthria, ataxic hands and brisk reflexes and the diagnosis was thought to lie between hereditary ataxia and disseminated sclerosis. He remained in this condition, living under appalling social conditions, until his admission.

Past history: Possible rickets as a child.

Family history: An only child whose father and mother died of cancer. He can remember no family history of renal trouble.

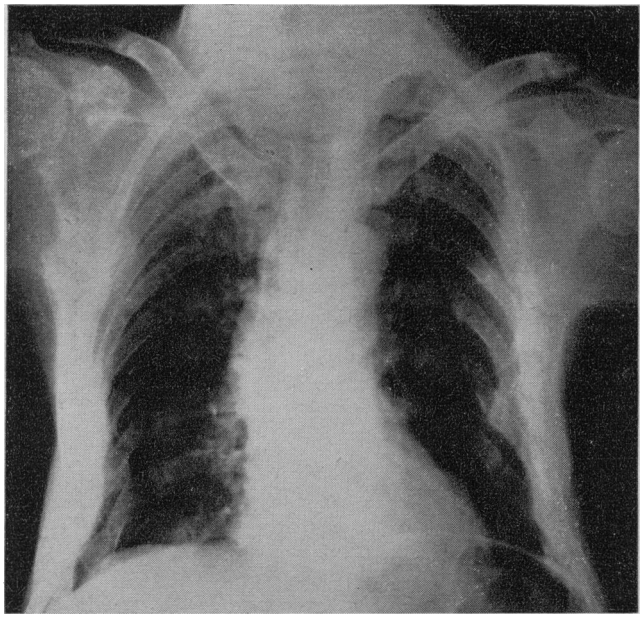

Fig 1 *Chest X-ray showing numerous pseudo fractures*

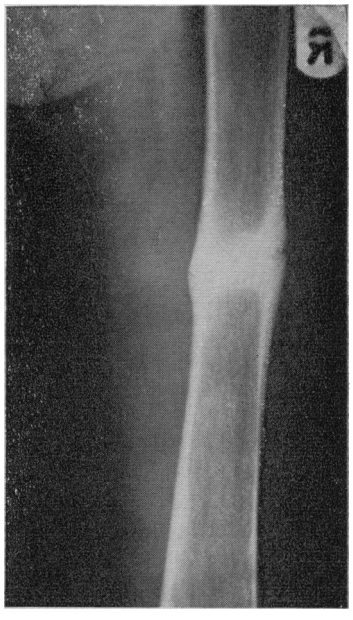

Fig 2 *Pseudo fracture in right femur*

On examination: Frail and emaciated with carious teeth. Reflexes brisk but the plantar reflexes were flexor. Co-ordination was poor in arms and legs and power was generally diminished. Standing was difficult, walking impossible.
Progress: He was regarded as a 'chronic sick' case until two episodes of collapse and flaccidity occurred which led to further investigation.

A chest X-ray showed numerous pseudofractures (Fig 1) and a skeletal survey revealed others (Fig 2) all tending to be symmetrical with characteristics of Looser's zones indicative of osteomalacia. The urine was alkaline and contained a trace of sugar and albumin. The centrifuged deposit showed hyaline and granular casts and culture yielded a growth of *Esch. coli.*
Investigations: Serum calcium 8·0, inorganic phosphate 1·6 mg/100 ml. Alkali reserve 17·8 mEq/1. Serum electrolytes: sodium 143, potassium 3·9, chloride 112 mEq/1. Plasma proteins: albumin 4·5, globulin 1·5 g/100 ml. Urinary calcium 67, phosphate (inorganic) 4·4 mg/24 hours.

Glucose tolerance test showed normal blood levels, but two urine specimens contained sugar. The hæmoglobin, fæcal fat content, serum vitamin B_{12}, and barium pattern of the small intestine were within normal limits. IVP normal renal anatomy and function with an opacity in the right kidney. Liver function tests normal; WR negative.

Investigations so far had revealed many features of the Fanconi syndrome and so the urinary amino-acid excretion was investigated. Chromatography of the patient's urine gave a denser chromatogram than normal and quantitative tests showed a value of 1,580 mg of amino acids per 24 hours, approximately four times the normal level. The serum uric acid was also examined and found to be low at 1·2 mg/100 ml.
Treatment: A normal diet was allowed supplemented by 120 ml daily of a 'polycitrate' solution, containing 1 mEq Na^+, 1 mEq K^+, and 1 mM citrate per ml, and 2·7 g sodium phosphate daily.

Discussion

The adult Fanconi syndrome consists of osteomalacia with hypophosphatæmia, glycosuria and amino-aciduria. Features sometimes present are proteinuria, systemic acidosis, hypo-uricæmia and hypokalæmia. The essential fault lies in the proximal renal tubules, though further degenerative changes due to chronic pyelonephritis with interstitial fibrosis and vacuolar degeneration aggravate the disease.

The case described showed all the listed features. In retrospect the episodes of flaccidity were seen to be due to hypokalæmia. Some unusual features were also present: (1) The absence of bone pain. (2) The low urinary output of calcium and phosphate. Probably his intake of calcium was poor before admission. Prolonged hypophosphatæmia may have led to extensive skeletal decalcification and these two facts may explain the low urinary values. Lastly, it is not possible to substantiate the original diagnosis of disseminated sclerosis. His neurological abnormalities may have a basis in the disordered metabolism and may therefore resolve with treatment.

Treatment is on lines suggested by Wilson &

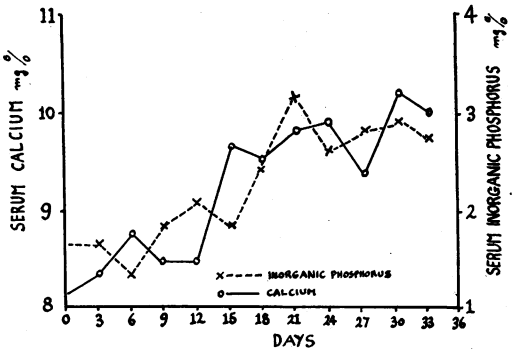

Fig 3 *Chart showing the rise of serum calcium and serum inorganic phosphorus on treatment with 'polycitrate' solution and added phosphate*

Yendt (*Amer. J. Med.*, 1963, **35**, 487). Correction of the metabolic acidosis and low serum phosphate leads to skeletal remineralization, thus avoiding hypercalcæmia with resultant nephrocalcinosis from excessive use of vitamin D. So far the serum calcium, serum inorganic phosphate and alkali reserve have returned to normal (Fig 3) and there has been some improvement in muscle power.

Postscript (20.5.64): Since this case was presented further investigation has shown a greatly lowered copper oxidase activity of 6·0 µl/ml/h. The cæruloplasmin level is also low. These findings suggest that the patient is suffering from Wilson's disease, which would account both for his neurological symptoms and the metabolic disturbance.

Chronic Pneumaturia and Diverticulitis of the Colon

T P Ormerod MB MRCP (for R D Tonkin MD FRCP)

Mr F T, aged 85. Retired head waiter

History: In 1947 he developed an acute urinary infection and passed gas bubbles and fæcal material in the urine. A barium enema showed extensive diverticular disease of the colon. Since then he has had intermittent pneumaturia and coliform urinary infections, both of which respond to treatment with sulphonamides.

In 1960 he was admitted with an unusually severe episode characterized by vomiting and right loin pain associated with pyuria; a diagnosis of pyelonephritis was made. Cystoscopy (Mr Robert Cox) showed gross cystitis with bilateral 'golf-hole' ureters and a fistulous opening halfway up the posterior wall of the bladder on the left side. Sigmoidoscopy was possible to 15 cm only, due to the fixity of the tissues, and no fistula was observed. Intravenous pyelography showed gas in dilated renal calyces and ureter on the right; there was also probably a left duplex kidney and an aneurysm of the abdominal aorta. Chest X-ray showed old pulmonary tuberculosis.

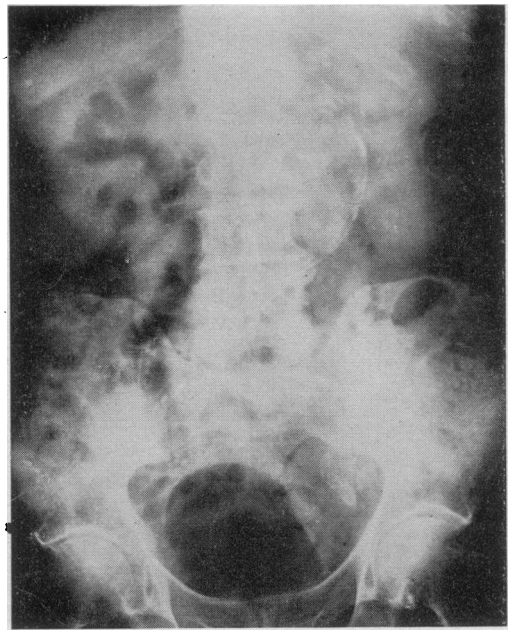

Fig 1 *Plain X-ray showing air in the ureter and renal calyces on the right side, due to the vesicocolic fistula*

His general health is still fairly good. In 1963 the blood urea was 47 mg/100 ml, plain X-ray of the abdomen showed a right pneumo-ureter and pneumonephrosis (Fig 1), and diverticulitis with a fistulous track leading from the sigmoid colon was seen on barium enema.

Discussion

Pneumaturia is commonly secondary to a fistula between the urinary tract and either the gut or vagina. Vesicocolic fistula is a well-recognized complication of diverticulitis, especially in men. The clinical picture is discussed by Bruce (1960). McGregor & Bacon (1960) found 8·3% of fistulæ in 253 patients with diverticulitis. Some other common causes of vesicocolic fistula, such as carcinoma of the sigmoid colon and bladder, are given by Hughes (1960) and Capper (1962).

In most patients the symptoms are severe enough to warrant operation but cases with intermittent symptoms existing for many years have been described by Harrison Cripps (1888) and Lockhart-Mummery (1958). Despite pneumaturia for seventeen years this patient's renal function remains adequate. For this reason and because of his age surgery does not seem indicated.

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