that lithium carbonate was implicated. Alcohol excess is known to cause an acute muscular syndrome³ but this is usually painful, asymmetrical, and associated with a recent binge. Secondly, pain is usually the initial symptom of the anterior tibial compartment syndrome and has been thought to be a constant finding.² In this case pain was absent because of the coexistence of a peripheral neuropathy. Again, alcohol abuse was the probable cause of this, as it was reversible with alcohol withdrawal and vitamin supplementation.

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Bladder rupture in multiple sclerosis

We describe two cases of spontaneous bladder rupture in patients with multiple sclerosis.

Case reports

Case 1—A 52-year-old woman presented with a two-day history of haematuria, dysuria, and frequency. She had had difficulty in walking and urgency for five years. On examination she was dehydrated and had loin and abdominal tenderness. She soon became shocked and febrile, and laparotomy showed extensive peritonitis. The bladder was inflamed, hypertrophic, and perforated through the fundus. Neurological assessment during convalescence showed bilateral optic atrophy, spastic tetraparesis, and loss of vibration sense below the iliac crests. Cerebrospinal fluid protein was 0.6 g/l and IgG 0.11 g/l.

Case 2—A 47-year-old woman who had had multiple sclerosis for 11 years presented with vomiting, abdominal pain, and haematuria. She had had recurrent urinary tract infections and urgency for two years. Two weeks before admission she had become incontinent of urine and an exacerbation of her leg weakness had occurred. She was catheterised and treated with distigmine and corticotrophin. On examination she was febrile and dehydrated and had a distended abdomen and loin tenderness. There was noticeable spasticity of all four limbs and optic atrophy. Abdominal radiography showed fluid levels and gas under the diaphragm. At laparotomy peritonitis and a large, trabeculated, hypertrophic bladder with a perforation in the fundus were found.

Discussion

Reflection suggests that rupture of the bladder in multiple sclerosis could result from a combination of several factors. Outflow obstruction from neurogenic sphincter disturbance, chronic bladder infection, direct mechanical trauma from an indwelling catheter, and potentiation of infection by corticotrophin may all play a part. Bladder rupture occurred as a presenting feature in case 1, which further illustrates the need for full neurological assessment of patients, particularly women, who present with retention and chronic infection.

Spontaneous as opposed to traumatic rupture of the bladder is uncommon. Bastable *et al*¹ reviewed 66 cases in 1959. Four patients had neurological disorders, one with paraplegia and three with tabes dorsalis. Most patients had either bladder outflow obstruction or disease of the bladder wall. It appears that these and other factors were present in combination in our two patients.

Urinary symptoms occur in up to 75% of patients with multiple sclerosis, particularly in those with spasticity and sensory loss in the legs.² Symptoms had been present in both of our patients for at least two years. The nature of the bladder dysfunction in multiple sclerosis is complex because of the multiple lesions at different levels in the nervous system. Bladder outflow obstruction may be caused by corticospinal lesions giving rise to spasm of striated muscle in the bladder

neck, external sphincter, and pelvic floor. Infection of residual urine is common, particularly in women. Both of our patients had chronically inflamed bladders. Furthermore, the presence of hypertrophy and trabeculation of the bladder wall in both cases is highly suggestive of chronic outflow obstruction.

Factors other than chronic cystitis and outflow obstruction may have contributed to bladder rupture in case 2. Catheterisation has been reported as causing bladder perforation.³ This patient was also given a parasympathomimetic drug, which, although useful in chronic hypotonic bladder dysfunction, is contraindicated when the bladder neck is obstructed.⁴

Spontaneous bladder rupture is rarely diagnosed before laparotomy, and mortality is high. Our two patients had severe sensory loss, which would favour a late presentation. The use of corticotrophin in case 2 may well have masked the signs of peritonitis and further delayed the diagnosis.

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Mesangiocapillary (membranoproliferative) glomerulonephritis and rheumatoid arthritis

Mesangiocapillary (membranoproliferative) glomerulonephritis (MCGN) is characterised by hypocomplementaemia, proliferation of mesangial cells, and thickening of the glomerular capillary wall. The aetiology is uncertain but it has occurred after upper respiratory tract infection and in systemic lupus erythematosus (SLE), anaphylactoid purpura, chronic bacteraemia, and partial lipodystrophy.^{1,2} We report a case in a patient with rheumatoid arthritis.

Case report

A 64-year-old Chinese woman gave a three-year history of morning stiffness and polyarthritis affecting the ankles, wrists, and proximal interphalangeal joints of the hands, for which she occasionally took prednisolone (5 mg/day), ketoprofen (200 mg/day), and phenylbutazone (200 mg/day). Six months before admission to hospital she developed generalised oedema followed by painless haematuria. She had generalised oedema, pallor, and a blood pressure of 190/100 mm Hg. The fingers showed spindling of all the interphalangeal joints. The wrists and ankles were swollen and movement was limited.

Analysis of urine showed: red blood cells 112×10^3 /l, white blood cells 95×10^3 /l, and granular casts. Urinary protein was 4 g/24 h, serum albumin 19 g/l, serum creatinine 194 μ mol/l (2.2 mg/100 ml), and haemoglobin 9.5 g/dl. Serum C3 was less than 0.05 g/l (normal 0.9–1.2 g/l) and C4 0.24 g/l (normal 0.2–0.4 g/l). Four tests for LE cells gave negative results. Antinuclear antibody was absent. A latex test for rheumatoid factor gave a titre of 1/40 (normal <1/32).

X-ray pictures of the hands and wrists showed reduced joint space, marginal erosions, periarticular osteoporosis, and articular destruction. Renal biopsy showed 15 swollen and lobulated glomeruli with hypercellularity from mesangial cell proliferation. The mesangial matrix was increased. The glomerular lumina were narrowed and many were obliterated. The capillary basement membranes were fragmented and showed a double-contour appearance in places (see figure). The diagnosis of MCGN was confirmed on electron microscopy.

Discussion

The diagnosis of rheumatoid arthritis and MCGN was made after SLE with lupus nephropathy had been excluded by the absence of LE cells, antinuclear factor, and the usual clinical features. The selective depression of C₃ was also more characteristic of MCGN than SLE.

We wondered whether the rheumatoid arthritis was related to the MCGN. The sequence of events in this case suggested that the rheumatoid arthritis preceded the renal disease. It is also uncommon for isolated MCGN to occur at this patient's age. Thus the concurrence of the two diseases in this patient seemed more than coincidental. If there was an association, how might this be explained?

Section of glomerulus showing increased mesangial cell proliferation, thickening of capillary basement membrane, and double-contour appearance (arrowed). (Periodic acid-silver stain $\times 5280$.)

The renal changes usually associated with rheumatoid arthritis are renal amyloidosis and analgesic nephropathy. Experiments have shown, however, that rheumatoid serum may aggravate experimental nephritis.³ Rheumatoid factor may also contribute to the immune-complex formation in the glomerulus.⁴ Furthermore, depression of total haemolytic complement and individual complements—particularly C₄ and sometimes C₃—occurs in some patients with rheumatoid arthritis. Such patients tend to have a high concentration of rheumatoid factor, severe rheumatoid disease, and a high incidence of recurrent infections.⁵

The deposition of immune complexes in the glomeruli with consequent activation of complements has been implicated in the pathogenesis of MCGN.¹ Possibly a similar mechanism may have been operative in this case. Alternatively, the hypocomplementaemia may have preceded rather than followed the formation of the immune complexes. Complement deficiency may cause a defect in immunological defence that results in persistent infection and chronic immune-complex formation and finally leads to MCGN.²

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Non-accidental immersion in bath-water: another aspect of child abuse

New acts are constantly being added to the tragic repertoire that comprises the child abuse syndrome. Deliberate poisoning has recently been added to the well-known forms of acute physical trauma (fractures, dislocations, lacerations, contusions, intracranial haematomata, burns), emotional trauma, deprivation, and pathological neglect.¹ During an extensive total population study of immersion accidents affecting children² a survey of bathtub drownings and near-drownings showed that some of these were deliberately produced by a battering parent.

Incidents

The cases of deliberate immersion that have come to our notice have all occurred in household bathtubs. In each case gross social disorders were pre-existent in the home. Features of these non-accidental immersions, and some differences distinguishing them from bathtub drownings in general, are shown in the table. In one of the cases we encountered the child (who survived the immersion incident) subsequently suffered an unexplained fractured skull at the age of 22 months. In another case we discovered later that a child who was deliberately immersed in the bath at the age of 3½ years was already known to the child protection agencies because of neighbours' reports of child abuse. Another child suspected of being immersed (a handicapped infant) died 12 hours after presentation to hospital from cerebral anoxia. Of the two children described who survived the near-drowning incident one was severely brain damaged as a result.

Common characteristics of children and families in cases of accidental and non-accident immersion in the bath

	Accidental immersions	Non-accidental injury
Age (months)	Usually 9-15*	Usually 15-30
Social class	Usually IV or V	Probably all classes
Pre-accident morbidity:		
Parents	Usually normal	Full sociopathology of parents inflicting non-accidental injury
Child	Usually normal; often youngest in large family; epilepsy or convulsions may be present	May be handicapped; often eldest child of small sibship
Precipitating cause	Household routine upset; child left in bath with other children	Acute parental stress, often domestic altercations
Circumstances	Often many children in bath: older ones get out leaving youngest to drown	Single child in bath; incident occurs at unusual time of day

*Data from Brisbane drowning study.²

The modus operandi seems to be deliberate immersion until unconsciousness ensues. At this point panic or remorse causes the child to be extracted from the water and help summoned. In the cases we have encountered it was the mother (or de facto wife) who was thought to have been responsible. In all cases the child was unconscious and apparently dead when taken from the water.

Comment

Unlike lacerations, fractures, and head injuries,³ but like punitive head shaking and whiplash injuries⁴ and deliberate poisoning⁵ non-fatal immersion accidents leave no pathognomonic stigmata. It is unfortunate that these more subtle forms of child abuse have been emphasised in only the last three years, and further more sophisticated types of battering, at present unrecognised, will probably also come to light.

An older infant or toddler is usually bathed at the end of the day, when a mother is tired and when stress and frustration are usually maximal. It is not surprising that this scenario is the occasion when a disturbed parent finally acts out the temptation and impulses that are usually chronically present, but barely submerged, in this group of families at risk.

Near-drowning in the bath has been ascribed to child neglect in at least one report⁶; but neglect (as opposed to a positive act of wilful child-directed violence) is difficult to define in the context of children's drowning accidents. The parents who get drunk while supervising