the left with complete right facial palsy but no neck stiffness.

Investigations showed a white cell count of 20.6 x 10⁹/l, with 90% neutrophils. A computed tomographic (CT) scan was entirely normal. However, the patient's clinical condition rapidly deteriorated. She developed marked neck stiffness, became drowsy and her temperature increased to 40°C. A lumbar puncture showed 100 white cells/mm³ predominately lymphocytes with normal protein concentration. No organism was found on Gram staining. A tentative diagnosis of meningoencephalitis was made and the patient was commenced on acyclovir, ampicillin and gentamicin. C-reactive protein on admission was 195 mg/l (normal <6 mg/l).

Four days after admission Listeria monocytogenes was isolated from the cerebrospinal fluid (CSF) culture. The patient remained drowsy and continued to deteriorate. Chloramphenicol was added to the drug regime. A repeat CT scan was again normal.

Four weeks after admission the patient developed acute renal failure complicated by disseminated intravascular coagulation and died. A post-mortem examination revealed necrotic areas in thepons and medulla on the right side of the brain but no other pathology was found. L. monocytogenes is a Gram-negative organism which enters the body through the consumption of contaminated food. It mainly affects pregnant women, immunocompromised patients or the elderly. The most common central nervous system manifestation of L. monocytogenes is menin gitis. Eck reported the first case of brain stem encephalitis in 1957. Since then a few cases have been reported and notably most of these patients were previously in good health.

The clinical presentation of rhombencephalitis shows a unique bimodal pattern. The prodromal phase of fever, malaise, headache, vomiting and leucocytosis usually precedes the neurological event by 4–10 days. This is followed by sudden appearance of pontomedullary dysfunction with cranial nerve palsy, however, nuchal rigidity is a rare occurrence. The CSF usually shows a normal glucose concentration with marginally elevated proteins but the white blood cell count is elevated predominantly with lymphocytes.

The diagnosis is based on isolation of L. monocytogenes from the blood cultures or occasionally from CSF cultures. The organism has rarely been identified on initial Gram staining. The issue of optimal antibiotics treatment is unclear and awaits clinical trials. The bacterium is sensitive to a wide variety of antibiotics but ampicillin and gentamicin are the recommended combination.

Listeria rhombencephalitis is a treatable cause of brain stem encephalitis which presents in a characteristic bimodal pattern. Although the differential diagnosis of brain stem encephalitis is vast, this possibility should be reconsidered whenever progressive brain stem encephalitis is accompanied by sterile CSF culture. Our case exhibited signs of brain stem encephalitis which is a very rare manifestation of listeriosis along with signs of meningitis. This case also highlights the fact that brain stem encephalitis mainly affects otherwise healthy subjects.

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References

Membranous nephropathy associated with diclofenac

Sir,

Non-steroidal anti-inflammatory drugs (NSAID) have numerous effects on the kidneys¹ including haemodynamic alterations, hyperkalaemia, acute interstitial nephritis, papillary necrosis, and the nephrotic syndrome associated with the 'minimal change' lesion. Membranous nephropathy has only rarely been reported in this context.² We report a case of nephrotic syndrome due to membranous nephropathy which occurred in a patient taking diclofenac and which resolved completely after withdrawal of the drug.

A 64 year old college lecturer presented with a 3 month history of leg swelling. He gave a history of subtotal thyroidectomy for hyperthyroidism 34 years previously and an episode of pancreatitis 4 years previously. He had taken diclofenac for non-specific arthritis for 18 months. On examination he had marked oedema. His blood pressure was 160/95 mmHg. Urinalysis showed heavy proteinuria but no haematuria. Twenty-four hour urinary protein excretion was elevated at 3.7 g. Creatinine clearance was normal at 87 ml/min (serum creatinine 106 μmol/l). Serum albumin was reduced at 25 g/l whilst serum bilirubin, aspartate transaminase, and alkaline phosphatase were all normal. Chest X-ray, abdominal ultrasound examination, and serum thyroxine were normal. Antinuclear factor, C3 and C4 components of complement, and serum protein electrophoresis were normal.

On renal biopsy 20 glomeruli were examined by light microscopy and showed mild mesangial increase but no capillary wall or other abnormality. Two glomeruli were examined by immunofluorescent microscopy and both showed diffuse granular capillary wall positivity for IgG. One glomerulus was examined by electron microscopy and showed numerous discrete subepithelial deposits in many of the capillary loops with no evidence of subendothelial or mesangial deposits. A diagnosis of early membranous glomerulonephritis (Stage 1 WHO classification) was made. Treatment with diclofenac was
stopped. He received bumetanide to control his oedema. Three months later his proteinuria had completely resolved, his serum albumin has risen to 43 g/l and his serum creatinine remained normal at 102 µmol/l.

The nephrotic syndrome is one of the many recognized renal complications of treatment with NSAID.1 In these cases the glomerular lesion has been, almost exclusively, the 'minimal change' lesion and there has usually been an associated acute interstitial nephritis.2 Membranous nephropathy occurring in association with treatment with NSAID is exceedingly rare. Only five cases have been reported.2 In our case the nephrotic syndrome due to membranous nephropathy occurred in a patient on long-term diclofenac therapy and resolved rapidly with no specific treatment apart from stopping the drug. There was no evidence of any other condition known to be associated with membranous nephropathy. Membranous nephropathy is a further manifestation of NSAID-related nephrotoxicity.

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References

Thrombotic thrombocytopenic purpura due to Mycoplasma pneumoniae

Sir,
The syndrome of thrombotic thrombocytopenic purpura (TTP) was first described by Moschowitz in 19253 and comprises a pentad of features; consumptive thrombocytopения, microangiopathic haemolytic anaemia, fluctuant neurological abnormalities, renal impairment and fever. We report a patient with TTP precipitated by Mycoplasma pneumoniae infection: there has been only one previous such report.4

A previously well 27 year old man presented with a 4 day history of myalgia, arthralgia and upper respiratory tract symptoms. On the day of admission he had become drowsy, incoherent and faecally incontinent, and had received a single dose of oral penicillin. He had a fever of 40°C, and some petechiae in his right axilla.

He failed to respond to commands, and could only utter incomprehensible noises. He was hypotonic with absent reflexes and normal plantar responses. His eyes opened spontaneously and he responded symmetrically to painful stimuli. Fundoscopy was normal and there were no signs of meningeal irritation.

A chest X-ray showed patchy right lower zone consolidation. The haemoglobin was initially 15.8 g/dl, falling to 11.1 g/dl within 24 h. The peripheral blood film showed spherocytes and red cell fragments with a normal white cell count. The platelet count fell from 105 × 10^9/l to 39 × 10^9/l within 24 h. The prothrombin time was 20 s (control 15 s), PTTK 38 s (control 35 s), fibrinogen 4.0 mg/dl (normal range 1.5–4.0 mg/dl), D Dimer 0.5–1.0 (normal range <0.25 g/l), haptoglobin 0.2 g/l (0.3–2.0 g/dl) and lactic dehydrogenase 580 U/l (100–300 U/l). The urea rose to 12.9 mmol/l, with a creatinine of 364 µmol/l. Urinalysis showed proteinuria, with red and white cell casts. A computed tomographic brain scan was normal and the cerebrospinal fluid was sterile, containing eight white cells and a protein of 438 mg/l. Acute Mycoplasma pneumoniae infection was confirmed with a positive IgM with a subsequent rise in antibody titre from 1:32 to 1:256.

On admission a diagnosis of atypical pneumonia, possibly Legionnaire's disease, was made, and treatment commenced with intravenous erythromycin, penicillin and rifampicin. Once TTP was confirmed additional treatment with fresh frozen plasma was undertaken, and improvement was rapid and so plasmapheresis was not required. Although our patient had received a single dose of oral penicillin before admission, it is unlikely that this was pathogenically significant because the TTP resolved despite the continued use of high dose intravenous penicillin.

TTP is associated with microvascular platelet thrombosis leading to multiple organ ischaemia and is perhaps caused by a deficiency in an immunoglobulin which normally inhibits platelet aggregation5 or by a multifactorial release of large molecular weight von Willebrand Factor multimers which augment platelet aggregation.4 The possible mechanisms underlying TTP have been well summarized recently.6 The mortality has been much reduced by treatment with plasma infusions or exchange.6

Acute Mycoplasma pneumoniae infection is an unusual initiating cause of TTP but it deserves mention as early treatment may benefit the patient.

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