stopped. He received bumatadine 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. In these cases the glomerular lesion has been, almost exclusively, the 'minimal change' lesion and there has usually been an associated acute interstitial nephritis. Membranous nephropathy occurring in association with treatment with NSAID is exceedingly rare. Only five cases have been reported. 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 1925 and comprises a pentad of features: consumptive thrombocytopenia, 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. 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 × 10⁹/l to 39 × 10⁹/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 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 aggregation or by a multifactorial release of large molecular weight von Willebrand Factor multimers which augment platelet agglutination. The possible mechanisms underlying TTP have been well summarized recently. The mortality has been much reduced by treatment with plasma infusions or exchange. 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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References
Amiodarone-induced thyrotoxicosis responding to oral steroid therapy

Sir,

Amiodarone-induced thyrotoxicosis is well known for being difficult to treat. Thionamide drugs often fail, and if possible amiodarone should be stopped. Recently there has been much work and discussion on the use of thionamides combined with potassium perchlorate, and the use of corticosteroids in the treatment of this condition.

A 49 year old man presented to clinic having lost 2 stones in weight over a 2 week period. This was associated with watery diarrhoea, heat intolerance and feeling tremulous. Three years earlier he had had an aortic valve replacement, and triple by-pass graft followed by amiodarone therapy 400 mg daily.

He had a smooth goitre, a pulse of 95 sinus rhythm, a proximal myopathy, and laboratory results confirmed thyrotoxicosis. Free thyroxine (T4) > 126 pmol/l (normal range 10-30 pmol/l). Total T4 > 320 nmol/l (normal range 56-154 nmol/l). Serum triiodothyronine (T3) = 5.4 nmol/l (normal range 1.1-2.8 nmol/l). Serum thyroid stimulating hormone (TSH) < 0.1 mU/l (normal range 0.5-5.0 mU/l).

A diagnosis of amiodarone induced-thyrotoxicosis was therefore made. Carbimazole 45 mg/day was recommenced. Amiodarone was withdrawn. After one month T4 remained > 320 nmol/l and prednisolone 30 mg daily was commenced. Within 2 weeks T4 became measurable at 260 nmol/l. Prednisolone was then decreased to 20 mg daily, and carbimazole to 30 mg/day. Within 8 weeks all indices had returned to normal. Prednisolone and carbimazole were slowly reduced over the next 4 months to zero and biochemical euthyroidism persisted.

Recent work has focused on the efficacy of a thionamide drug combined with potassium perchlorate although the rapidity of the response has been questioned. Corticosteroid therapy has been widely advocated often seemingly being effective where other therapies have failed. This case report serves to further underline this message. One month of treatment with carbimazole proved fruitless whilst after 2 weeks of oral prednisolone the patient’s hyperthyroidism had turned the metaphorical corner.

Three months elapsed between diagnosis and cessation of amiodarone, and attaining biochemical euthyroidism. Spontaneous cure for this condition has been quoted as occurring within an average of 6 months. The shorter remission period here and its striking relation to commencement of prednisolone therapy argue against this being a spontaneous cure.

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Idiopathic thrombocytopenic purpura and acute polyneuritis: a coincidence or association?

Sir,

We hereby report the case of a 50 year old woman who presented to us with idiopathic thrombocytopenic purpura (ITP) and developed features of Landry – Guilain–Barré (LGB) syndrome in hospital. The patient presented with an acute onset of generalized purpuric rash. Investigations revealed thrombocytopenia, a normal bone marrow and raised levels of immunoglobulin G. In the absence of any apparent cause of thrombocytopenia, she was diagnosed to be suffering from ITP. She recovered after an 8 week tapering course of oral prednisolone (50 mg/day).

After an asymptomatic intervening period of 6 months, she was re-admitted with generalized purpura, haemoptysis and epistaxis. Platelet count was again found to be low. She was again treated with oral prednisolone (50 mg/day). She was also transfused one unit of fresh whole blood. The following day, the patient complained of weakness of both legs and inability to close her eyes. Physical examination revealed bilateral facial palsy of the lower motor neurone type and an areflexic paraplegia without any sensory involvement. Upper limbs, other cranial nerves, bowel and bladder functions remained unaffected. Further investigations revealed an albuminocytologic dissociation in cerebrospinal fluid and a delay in motor nerve conduction of lower limbs. On immunological investigation, the patient tested negative for human immunodeficiency virus, hepatitis B, cytomegalovirus, herpes simplex and Epstein–Barr viruses. There was no clinical or investigational evidence of malignancy and collagen vascular disease.

ITP is a disorder of uncertain aetiology characterized by reduced platelets and a shortened platelet life span. The presence of anti-platelet antibodies indicates an autoimmune origin. Neurological deficits in ITP usually result from haemorrhage within the nervous system.
Thrombotic thrombocytopenic purpura due to Mycoplasma pneumoniae.
D. Cameron, P. Welsby and M. Turner

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