Amiodarone-induced thyrotoxicosis responding to oral steroid therapy

Sir,

Amiodarone-induced thyrotoxicosis is well known for being difficult to treat. Thionamide drugs often fail,¹ 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 commenced. Amiodarone was withdrawn.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 perchlorate² although the rapidity of the response has been questioned.³ Corticosteroid therapy has been widely advocated⁴,⁵ 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.⁶ 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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References


Idiopathic thrombocytopenic purpura and acute polynuropathy: 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–Güllain–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.⁷
LGB syndrome has also been related independently to a disordered immune system with evidence of both delayed hypersensitivity as well as antibodies to peripheral nerve myelin. Suggested initiating causes include viruses, Hodgkin's lymphoma, the autoimmune deficiency syndrome, rarely specific autoimmune disorders and blood transfusion. The above mentioned causes with the exception of blood transfusion have also been implicated independently in the aetiology of thrombocytopenia. All these factors are only initiating mechanisms, both disorders in effect being immune mediated. It is therefore surprising that the description of association of ITP and LGB syndrome in literature is limited to a single report. The therapeutic use of corticosteroids and more recently human gammaglobulin in the two disorders may further indicate a link between them. Until such time that a definite relation is established, this association may be considered coincidental. However we suggest that serial platelet counts in patients with LGB syndrome and nerve conduction studies in patients with ITP may be useful in detecting subclinical abnormalities substantiating any such association.

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References