CASE REPORT

Atrioventricular block: an unusual complication of Graves' disease

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SUMMARY

We report the case of a 10-year-old girl treated with atenolol and carbimazole for tachycardia and hypertension associated with Graves' disease who developed symptomatic 2:1 heart block. 2:1 heart block resolved following cessation of atenolol, reduction in carbimazole dose and treatment of suspected tonsillitis. First-degree atrioventricular block persisted, but gradually normalised following improvement in thyroid status.

BACKGROUND

Atrioventricular (AV) conduction defects are rare but significant complications of hyperthyroidism. Beta-blockers and coexistent infection further increase the risk of such conduction abnormalities. We discuss the likely pathophysiology of 2:1 heart block in this case and highlight the importance of ECG assessment prior to using a beta-adrenoceptor antagonist in patients with hyperthyroidism.

CASE PRESENTATION

A 10-year-old girl presented to her general practitioner with a 2-year history of hyperthyroid symptoms. Blood tests confirmed thyrotoxicosis and she was referred to the general paediatric team for management. At presentation, she appeared thyrotoxic, had associated cardiac symptoms with heart rate 142 beats per minute, blood pressure 133/87 and also complained of sore throat and abdominal pain. She was commenced on daily atenolol 32 mg (1 mg/kg/day) and carbimazole 40 mg.

Two days later, she re-presented with nausea, sore throat, pyrexia and chest pain. A baseline bradycardia of 60 beats per minute with sudden episodes of self-limiting bradycardia to 28–45 beats per minute and hypotension (lowest blood pressure 57/43) was noted. This haemodynamic instability was associated with chest pain, dizziness and nausea. There was a positive maternal family history of thyroid disorders.

On examination, exophthalmos, diplopia on lateral gaze, a smooth non-tender goitre, fine tremor, erythematous tonsils with exudate and mild abdominal discomfort were evident.

INVESTIGATIONS

The 12-lead ECG identified 2:1 AV block with prolongation of the P-R interval (317 ms) during the conducted beats (figure 1). Biochemistry confirmed hyperthyroidism (free T4: 42.4 pmol/L, free T3:

8.9 pmol/L, TSH <0.01 mIU/L; normal ranges: 10–23 pmol/L, 3.1–6.8 pmol/L, 0.27–4.2, respectively) with mildly elevated inflammatory markers (white cell count 13.7, neutrophils 8.2, C reactive protein 43). Echocardiogram showed no evidence of structural heart disease.

Treatment

The patient was admitted for cardiac monitoring; atenolol was discontinued and neither chronotropes nor pacing was required. Carbimazole was reduced to 10 mg twice daily and a course of oral phenoxymethylpenicillin was commenced for suspected tonsillitis.

OUTCOME AND FOLLOW-UP

Repeat ECG within 48 hours of admission revealed resolution of second-degree heart block with persistence of P-R prolongation (240 ms) (figure 2). She was subsequently discharged on carbimazole 10 mg twice daily and a course of phenoxymethylpenicillin. No bacterial growth was identified on throat swab or respiratory secretions. Three months after presentation, and following an increase in carbimazole dose to 40 mg daily, there is improvement in thyroid function: free T4: 14.3 pmol/L, free T3: 9 pmol/L, TSH <0.01. TSH receptor antibodies were positive. Repeat ECG showed a P-R interval of 185 ms with a normal heart rate (figure 3). Hypertension is still present and the patient remains under endocrine follow-up.

DISCUSSION

A number of factors may have contributed to AV block in this patient. Although reported rates of P-R prolongation in adult patients vary between 3% and 30%, this phenomenon is felt to be a rare complication of hyperthyroidism and is more commonly seen in hypothyroid patients. Case reports also describe higher degrees of AV block in association with hyperthyroidism.¹ Osman *et al*,² for example, describe two cases of second-degree and third-degree AV block associated with hyperthyroidism. This effect is, however, not well reported in the paediatric population.

Proposed mechanisms for this association include hyperthyroidism-induced hypervagotonia as well as a direct effect of thyroid hormones on cardiac conduction at the AV node.^{1,3} Coexistent infection, hypercalcaemia and concurrent drug use have also been implicated as contributory factors to AV block in these patients.³ Beta-adrenoceptor antagonists,

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Figure 1 ECG at presentation.

which form standard treatment for symptomatic tachycardia in paediatric hyperthyroidism,⁴ block sympathetic stimulation to both the sinoatrial and AV nodes. It is therefore unsurprising that higher degrees of AV block can occur in hyperthyroid patients receiving such medication and that this effect may be exacerbated by infection.

In the absence of a 12-lead ECG prior to initiating beta-adrenoceptor antagonist therapy, it is unknown if a pre-existing conduction delay was present in our patient. Although cardiac monitoring was instituted at initial presentation, conduction delays may have easily been missed. The staged resolution of

Figure 3 ECG 3 months after initial presentation.

AV block, however, implicates high circulating thyroid hormone levels as the cause for first-degree heart block. It is most likely that atenolol, together with the concurrent effects of an upper respiratory tract infection, propagated this and caused 2:1 AV block. As described in other reports, resolution of conduction defects occurred following a reduction in thyroid hormone levels.^{1 5}

Contributors AA and TV were responsible for reviewing the case and drafting the manuscript. MA and SM reviewed and revised the manuscript for important intellectual content. All authors have read and approved the final manuscript.

Figure 2 ECG 48 hours after admission.

Learning points

- ▶ Although rare, atrioventricular (AV) conduction abnormalities are significant complications of hyperthyroidism.
- ▶ Beta-adrenoceptor antagonists—drugs commonly used in thyrotoxicosis—act on the AV node and also impair AV conduction.
- ▶ In view of these well-described associations, we would recommend that a baseline 12-lead ECG is performed to look for evidence of AV conduction abnormalities prior to initiating beta-blocker therapy in children with hyperthyroidism.

Competing interests None declared.

Patient consent Obtained.

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