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Case report

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Graves´ disease presenting with complete heart block in a South African lady from Mthatha: a case report

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Abstract

Graves´ disease (GD) is an autoimmune thyroid disease first described by Robert Graves in 1835. Patients with GD usually present with symptoms such as heat intolerance, palpitations, tremors, and weight loss. Tachyarrhythmias are the typical cardiac rhythm abnormalities in thyrotoxicosis due GD. We report on a patient with thyrotoxicosis due to Graves´ disease who presented to our institution with symptomatic bradycardia characterized by dizziness and syncope. Electrocardiogram confirmed complete heart block. Therefore, Graves´ disease can present unusually with complete heart block instead of a typical tachyarrhythmia.

Introduction

Graves´ disease (GD) is an autoimmune thyroid disease first described by Robert Graves in 1835 [1]. It accounts for up to 80% of hyperthyroidism cases and is estimated to affect 0.5% of the population [2]. Patients with GD usually present with manifestations such as heat intolerance, palpitations, tremors, and weight loss. Among the unusual manifestations reported in association with GD are pulmonary arterial hypertension (PH), right heart failure, myocardial infarction, and heart block [3]. We herein report a case of a 52-year old woman who presented in complete heart block and was found to be GD.

Patient and observation

A 52-year old lady was referred to Nelson Mandela Academic Hospital (NMAH) from a district level hospital for permanent pacemaker implantation due to persistent complete atrioventricular (AV) block. The patient began to have palpitations and progressive weakness few weeks before her admission. She had also reported pre-syncopal as well as syncopal episodes. Her medical history included systemic arterial hypertension treated with hydrochlorothiazide and enalapril. On admission, she was bradycardic with a pulse rate of 38 beats per minute. Blood pressure was normal at 125/70 mmHg. She was pyrexic with a temperature of 37.9 degree Celsius. She had fine tremors of the outstretched hands as well as clubbing of the fingers. Mild diffuse, non-tender goiter was also noted, and the remainder of her clinical examination was unremarkable. The electrocardiogram (ECG) revealed complete atrioventricular block with a heart rate of 38 beats per minute (Figure 1). Echocardiography revealed a structurally normal heart with left ventricular ejection fraction of 61%. Coronary angiogram revealed no abnormalities in the coronary vessels. Laboratory examination results at presentation as depicted in Table 1 showed serum free thyroxin (FT4) concentration of 33 pmol/l (normal 12-22 pmol/l), free triiodothyronine (FT3) of 8.8 pmol/l (normal 3.1-6.8 pmol/l) and Serum thyroxin stimulating hormone (TSH) concentration of <0.01 mIU/l (normal 0.27-4.2 mIU/l). TSH receptor antibodies (TRAb) level was 7.46 U/L (normal, <1.8 U/L), the haemoglobin, white cell count, platelets and Erythrocyte sedimentation rates were all normal; 12.2 g/dl, 9.400/mm^3, 278 x109/L and 12 mm/hr respectively. Anti-nuclear and anti-double stranded DNA antibodies were both negative. Blood and urine cultures were also negative. She was HIV negative. Chest radiograph was unremarkable. The final assessment was that of Graves´ disease with complete heart block (CHB). Antithyroid treatment was commenced. Body temperature returned to normal and three weeks after admission, her Thyroid function test normalised. Complete heart block, however, persisted and this necessitated the insertion of a dual chamber pacemaker with no recurrence of dizzy spells and syncope. She was discharged home one week after pacemaker insertion to be followed up at our Cardiac and Endocrine Outpatient Clinics.

Discussion

Our patient presented with CHB and the unexpected finding of thyrotoxic Graves´ disease. Indeed, hyperthyroidism due to Graves´ disease is extremely rarely associated with CHB [4]. The mechanism of AV block in hyperthyroidism is still controversial. Suggested mechanisms for CHB in thyrotoxic Graves´ include direct effect of thyroid hormones on conduction pathways, autoimmune mediated injury of the cardiac conduction pathway, and hyperthyroidism triggered hypervagotonia [4,5]. Typical causes of CHB such as ischemia; infiltrative diseases like sarcoidosis and amyloidosis, malignancy, infections such as Lyme disease and iatrogenic causes (typically medications, but also surgical complications) will need to be excluded. In support of a direct effect of thyroid hormones in the causation of CHB include a report of CHB in association with thyrotoxic Graves´ with restoration of sinus rhythm on normalization of thyroid function [6]. There is also a report of

3rddegree AV block following excessive thyroid hormone replacement for hypothyroidism [5]. Our case extends the reports of the rare association of CHB and thyrotoxic Graves´ disease. However, unlike the report by Dave *et al*. our patient remained in CHB despite restoration of euthyroid status. This could be because of irreversible thyrotoxic Graves´ related conduction pathway damage or existence of another, yet to be established cause for CHB in our patient. Hyperthyroidism may be associated with interstitial and perivascular fibrosis, cellular infiltration, and myocyte necrosis [7]. Indeed, focal myocarditis affecting the region around the AV node has also been postulated to result in heart block [5]. We can only speculate on the cause of CHB in our patient.

Conclusion

We report a rare finding of Graves´ disease presenting with complete heart block. This case report serves to remind our colleagues about this association and to continue to have a high index of suspicion and a low threshold to investigate any young patient presenting with an unexplained bradycardia.

Competing interests

The authors declare no competing interests.

Authors' contributions

All the authors contributed to conception, design, drafting and revising of the manuscript. All authors read and approved the final manuscript.

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Table and figure

Table 1: laboratory results at presentation

Figure 1: ECG at presentation showing complete heart block

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Table 1: laboratory results at presentation		
	At presentation	**Normal range**
TSH [mIU/L]	< 0.01	0.27-4.2
FT4 [pmol/L]	33	12-22
FT3 [pmol/L]	8.8	3.1-6.8
TRAb [u/L]	7.46	< 1.8
Hb [g/L]	12.2	12-15
WBC [x10^9/L]	9.4	3.9-12.6
Platelets [x10^9 /L]	278	186-454
Haematocrit [l/L]	0.36	0.36-0.46
MCV [fL]	80.6	78.9-98.5
MCH [pg]	26.1	26.1-33.5
MCHC [g/dL]	32.8	32.7-34.9
Na+ [mmol/L]	136	136-145
K+ [mmol/L]	4.1	3.5-5.1
Cl− [mmol/L]	101	98-107
Urea [mmol/L]	4.3	1.4-5.4
Creatinine [umol/L]	68	49-90
Total protein [g/L]	78	57-80
Albumin [g/L]	41	29- 42
Bilirubin [umol/L]	15	5-21
ALT [U/L]	21	7-35
AST [U/L]	23	13-35
GGT[U/L]	16	< 40
ALP [U/L]	98	42-98
TRAb, TSH receptor antibodies; ALT, alanine aminotransferase; AST, aspartate aminotransferase; GGT, gamma-glutamyl transferase, ALP, alkaline phosphatase		

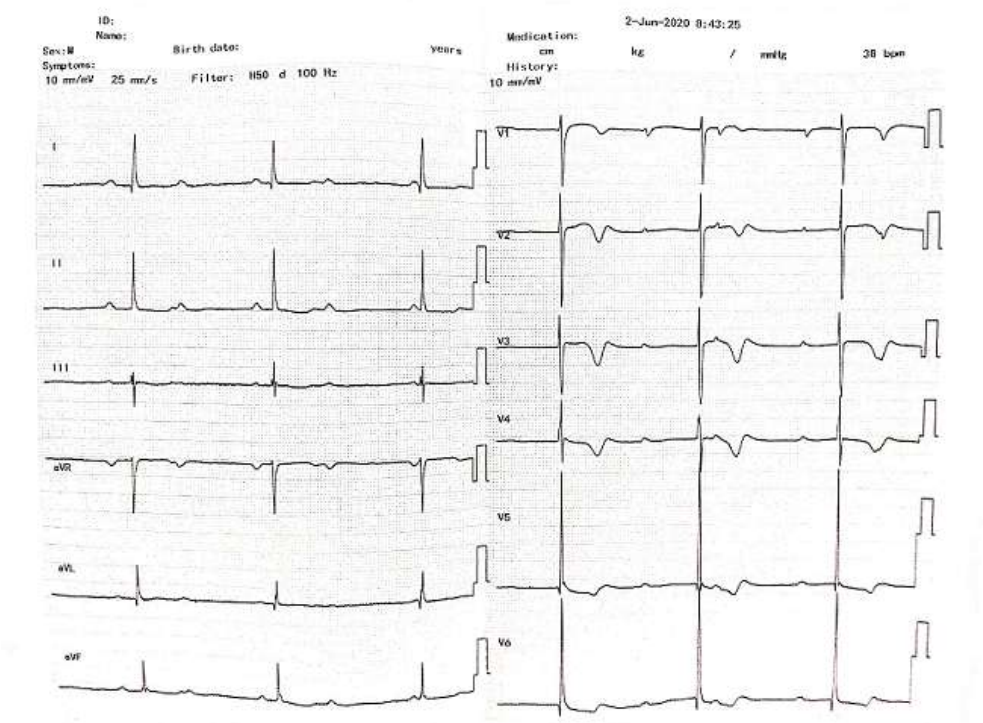

Figure 1: ECG at presentation showing complete heart block