Reversible cause of complete heart block: an unusual presentation of thyrotoxicosis

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Received 12 July 2015
Accepted 17 August 2015

The Egyptian Society of Internal Medicine
2016, 28:28–29

Thyrotoxicosis is a clinical entity that is often very difficult to diagnose without biochemical confirmation, as its clinical features can be highly varied. It is more common in women, with a female to male ratio of up to 5 : 1 between the ages of 20 and 40. Thyroid hormone is important at a cellular level, affecting nearly every type of tissue in the body. Very rarely it can cause heart blocks. We report a case of complete heart block associated with thyrotoxicosis that reverted to sinus rhythm with medical management alone.

Keywords:
complete heart block, thyrotoxicosis, complete heart block, thyrotoxicosis, sinus rhythm

Egypt J Intern Med 28:28–29

1110-7782

Introduction

Thyrotoxicosis is characterized by weight loss despite increased appetite, as well as sweating, palpitations, tremor, heat intolerance, weakness, visual disturbances, diarrhea, loss of libido, and oligomenorrhea. The most common cardiac manifestation is resting sinus tachycardia, which can cause supraventricular tachycardia, including atrial fibrillation. Atrioventricular blocks are rarely reported in thyrotoxicosis. We report the case of a 38-year-old woman who presented with thyrotoxicosis with complete heart block.

Case report

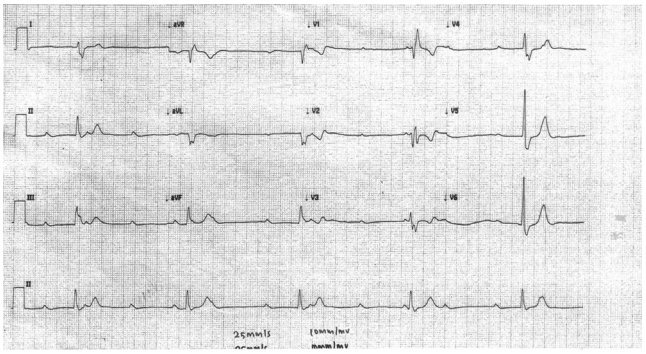
A 38-year-old woman was admitted at our institute, PSG Institute of Medical Sciences and Research, Coimbatore, Tamilnadu, India, with breathlessness, palpitation, weight loss of around 8 kg despite increased appetite, diarrhea since 4 months, and oligomenorrhea. She had no other cardiac or toxic symptoms, nor a history of fever or syncope. Her menstrual cycles before 6 months were normal. There was no significant past medical or drug history. She was emaciated with BMI of 16.44 with diffuse nontender goiter and fine tremor in the upper extremities. Her pulse rate was regular at 30/min, with blood pressure of 130/80 mmHg. She showed no signs of heart failure. Her abdomen was soft with no organomegaly or ascites. Her hemoglobin level was 10 gm%, erythrocyte sedimentation rate was 40 mm/h, total leukocyte count was 8000/cm^3, and platelet count was normal. Peripheral smear showed microcytic hypochromic anemia. Chest radiograph was normal. She was HIV-negative. Urine analysis, liver function tests, blood sugar, renal function tests, and stool examination were normal. Serum sodium was 138 Meq/l, serum potassium was 3.56 Meq/l, and serum calcium and magnesium were normal.

Her ECG showed complete heart block at 30/min with narrow QRS escape rhythm (Fig. 1). ECG performed 2 years earlier during a routine health checkup was normal. Thyroid function tests revealed toxic state with thyroid stimulating hormone 0.013 μIU/ml (normal 0.34–4.25 μIU/ml), free tri-iodothyronine 5.65 pg/ml (normal 2.4–4.2 pg/ml), and free thyroxine 7.77 ng/dl (normal 0.8–1.7 ng/dl). She was positive for thyroid peroxidase antibodies. Transthoracic echocardiogram was normal. She was managed with carbimazole 20 mg twice daily along with iron supplementation for anemia. ECG at 6 weeks showed sinus rhythm (Fig. 2). She was on medical management for 18 months and was subsequently lost to follow-up. She did not require pacing. During the follow-up period, her ECG reverted to sinus rhythm and all her symptoms improved.

Discussion

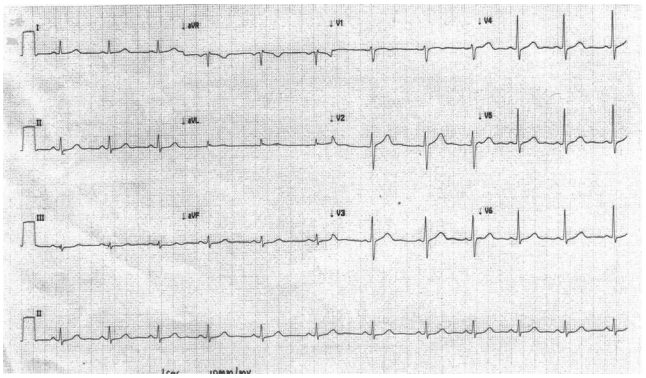
Thyrotoxicosis can cause a variety of cardiac arrhythmias including resting sinus tachycardia, atrial fibrillation, and atrial flutter with or without heart failure because of the intrinsic effects of thyroxine on sinoatrial node.[1,2] In thyrotoxic patients the intrinsic activity of the sinus node is increased. This

Figure 1



Complete heart block with narrow QRS complexes.

Figure 2



ECG on follow-up shows normal sinus rhythm.

could possibly be a direct consequence of thyroid hormone excess, rather than an effect of extrinsic influences exerted by the autonomic nervous system on sinus node activity.[2] Atrioventricular conduction defects and sinoatrial blocks have been reported in patients with hyperthyroidism.[3] Second-degree and third-degree heart blocks are very rare. In view of the potential aggravation of atrioventricular conduction disturbance by β-adrenergic blocking agents, thyrotoxic patients should be carefully screened for ECG evidence of conduction disturbance before the administration of rate control drugs.[3] Cases of complete heart block complicating thyrotoxicosis have been reported.[4,5] Lack of awareness of the above association and atypical presentations may delay diagnosis and treatment and unwarranted pacemaker implantation. The exact mechanism of atrioventricular conduction blocks is unknown. However, focal myocarditis around the atrioventricular node and interstitial inflammation of the atrioventricular node and bundle have also been reported in autopsies and also dilated ventricles, myocyte hypertrophy, myocyte necrosis, myocardial edema, interstitial, and perivascular fibrosis.[6,7]

Our patient presented with thyrotoxicosis, which was confirmed by biochemical tests, and her heart rate was 30/min instead of tachycardia. The ECG showed complete heart block. She was treated with carbimazole, and the repeat ECG at 6 weeks showed sinus rhythm. Her ECG from 2 years ago was normal. We have not used rate control drugs as she had complete heart block. She could have developed heart block because of thyrotoxicosis. Only a few reported cases show complete to partial reversal of conduction abnormalities and heart failure.[8,9] Therefore, cardiac and electrophysiological decompensation due to thyrotoxicosis may be reversible. Our patient's symptoms and complete heart block improved with antithyroid drugs alone and she did not require pacing.

Conclusion

Atrioventricular blocks especially complete heart block can occur in thyrotoxicosis. There should be a high index of suspicion for thyrotoxicosis in the differential diagnosis of patients who present with heart blocks associated with abnormal features like weight loss, diarrhea, and palpitation. The clinical status and conduction abnormalities are reversible with medical management. The administration of rate-controlling drugs can worsen the heart block associated with thyrotoxicosis. We present this case for the rarity of association of complete heart block with thyrotoxicosis and its reversal to sinus rhythm with medical management.

Financial support and sponsorship

Nil.

Conflicts of interest

There are no conflicts of interest.

References

1 Rives JD, Shepard RM. Thyroid crisis Am Surg 1951; 17:406–418.

2 Valcavi R, Menozzi C, Roti E, Zini M, Lolli G, Roti S, *et al.* Sinus node function in hyperthyroid patients. J Clin Endocrinol Metab 1992; 75:239–242.

3 Kramer MR, Shilo S, Hershko C. Atrioventricular and sinoatrial block in thyrotoxic crisis. Br Heart J 1985; 54:600–602.

4 Miller RH, Corcoran FH, Baker WP. Second and third degree atrioventricular block with Graves' disease: a case report and review of the literature. Pacing Clin Electrophysiol 1980; 3:702–711.

5 Topaloglu S, Topaloglu OY, Ozdemir O, Soylu M, Demir AD, Korkmaz S. Hyperthyroidism and complete atrioventricular block – a report of 2 cases with electrophysiologic assessment. Angiology 2005; 56:217–220.

6 Ortmann C, Pfeiffer H, Du Chesne A, Brinkmann B. Inflammation of the cardiac conduction system in a case of hyperthyroidism. Int J Legal Med 1999; 112:271–274.

7 Shirani J, Barron MM, Pierre-Louis ML, Roberts WC. Congestive heart failure, dilated cardiac ventricles, and sudden death in hyperthyroidism. Am J Cardiol 1993; 72:365–368.

8 Osman F, Ayuk J, Dale J, Franklyn JA, Gammage MD. Thyrotoxicosis with heart block. J R Soc Med 2001; 94:346–348.

9 Martí V, Ballester M, Obrador D, Moya C, Carrio I, Pons-Lladó G. Active myocardial damage in hyperthyroidism. A concurrent mechanism of heart failure reversed by treatment. Eur Heart J 1995; 16:1014–1016.