Thyrotoxicosis presenting with complete heart block

Sheikh Jalal, *DM,* ***Khurshid A. Khan,*** *DM,*
Malik A. Rauoof, *MD,* ***Vicar M. Jan,*** *DM,*
Nazir A. Lone, *MD,* ***Hilal A. Rather,*** *DM,*
Kaiser Habib, *MD.*

Disorders of cardiac rhythm and repolarization are frequently observed in thyrotoxicosis. Of these, sinus tachycardia and atrial fibrillation are the most common. Less commonly, conduction abnormalities including prolongation of PR interval and high-grade atrioventricular (AV) block may be encountered, usually in the presence of precipitating factors like infection and drugs.[1-3] We report 3 cases of untreated thyrotoxicosis presenting with symptomatic complete heart block (CHB) in the absence of any known precipitating factors.

A 20-year-old female presented with one day history of recurrent syncopal attacks. She reported history of palpitations, heat intolerance and weight loss of 3 months duration. Examination revealed a thin and lean female with prominent stare, lid lag, and warm and moist skin and fine finger tremor. Her pulse rate was 34 beats per minute and blood pressure 160/70 mm Hg. Thyroid was diffusely enlarged with an audible bruit over it. Cardiac examination was unremarkable. Electrocardiogram (ECG) showed AV dissociation (atrial rate 100/minute, ventricular rate 34/minute). Chest x-ray was normal. Thyroid function tests showed triiodothyronine (T_3) 7.8 ng/ml (normal range 0.7-2.5 ng/ml), thyroxine (T_4) 16.9 µg/dl (normal range 5.5-13.5 µg/dl) and thyroid stimulating hormone (TSH) 0.2 µIU/ml (normal range 0.4-5.0 µIU/ml). A transvenous right ventricular pacing lead had to be inserted in addition to carbimazole 10 mg 3 times daily. The patient reverted to sinus rhythm on the fifth day.

Another patient, a 33-year-old female reported with a single episode of syncope. She had a preceding history of excessive sweating, palpitations and exertional dyspnea of 6 months duration. On examination, diffuse thyroid enlargement with a bruit was present. Pulse was 41 beats per minute and blood pressure 170/80 mm Hg. Cardiac examination revealed a grade II/VI systolic murmur at left parasternal area. Electrocardiogram showed AV dissociation (atrial rate 110/minute, ventricular rate 41/minute). Chest x-ray was normal. Thyroid function tests revealed T_3 6.7 ng/ml, T_4 15.8 µg/dl and TSH 0.3 µIU/ml. Patient reverted to sinus rhythm the following day with intravenous atropine and isoprenaline. Carbimazole 10 mg 3 times a day was also given.

The third patient, a 38-year-old female was hospitalized for evaluation of cardiac syncope noticed 7 days prior to admission. She also had history of palpitations, weight loss, heat intolerance and excessive sweating for the past 8 months. Examination revealed an anxious look with warm and moist skin and diffuse thyromegaly. Pulse rate was 124 per minute, blood pressure was 130/60 mm Hg. Cardiac auscultation was normal. Electrocardiogram on admission showed sinus tachycardia. Thyroid function tests were T_3 7.9 ng/ml, T_4 16.1 µg/dl and TSH undetectable. The patient was given carbimazole 15 mg 3 times a day. She developed symptomatic CHB on third day of admission (atrial rate 105, ventricular rate 38 per minute) for which temporary pacing was performed. Patient reverted to sinus rhythm after 6 days.

Complete blood count, sedimentation rate, urine examination, serum electrolytes, serum immunoglobulins, antistreptolysin O, serum creatine kinase and liver function tests were normal in all 3 patients. Echocardiographic examination was also normal. Intracardiac electrophysiologic study, performed in sinus rhythm, was within normal limits and the block could not be reproduced. On 6 months follow up, all the 3 patients were euthyroid. There was no recurrence of CHB.

Hyperthyroidism is commonly associated with a spectrum of cardiovascular abnormalities that range from asymptomatic elevation of cardiac function indices to overt heart failure. Direct stimulant action of thyroid hormones on heart and their indirect effects via sympathovagal imbalance underlie most of these cardiovascular mainfestations.[3,4] Rhythm disturbances, particularly sinus tachycardia and other atrial arrhythmias including atrial fibrillation are also encountered frequently in thyrotoxic patients. Intra-atrial and intra-ventricular conduction disturbances each occur in approximately 15% of patients. Abbreviated refractory period of the AV node and shortened AV conduction time indicate a generally facilitated AV conduction in hyperthyroidism. However, AV conduction disturbances usually in the form of prolonged PR interval, can occur in up to 5% of thyrotoxic patients.[3] Reports of CHB complicating thyrotoxicosis are only anecdotal. The present report of 3 cases of symptomatic CHB underscores the importance of serious conduction disturbance as an important clinical problem in thyrotoxicosis. Complete heart block was the dominant albeit transient cardiovascular manifestation of the thyroid disorder in all of the 3 patients. High grade AV block occurring in the context of thyrotoxicosis usually occurs in the presence of other conditions

such as infection, myocarditis, electrolyte disturbances, or the use of drugs such as digoxin, propanolol, reserpine, or ajmaline.[3,5,6] However, the etiology of CHB in our patients of thyrotoxicosis is unclear as none of the above-mentioned conditions was present nor was there any evidence of other known precipitators of AV conduction disturbance. Further, the quick resolution of conduction abnormality within days bore no relation to the achievement of euthyroid state, which does not usually occur until at least 6-8 weeks of continuous antithyroid drug therapy.[7] Whether AV conduction disturbances can be included as primary disturbances in the spectrum of thyrocardiac disease remains elusive at present.

Received 16th May 2004. Accepted for publication in final form 9th August 2004.

From the Department of Cardiology, Sher-I-Kashmir Institute of Medical Sciences, Soura, Srinagar, Kashmir, India. Address correspondence and reprint requests to Prof. Sheikh Jalal, Head, Department of Cardiology, Sher-I-Kashmir Institute of Medical Sciences, Post Bag No.27, Soura, Srinagar, 190 011, Kashmir, India. Tel. +91 (194) 2400064 Ext. 2355. Fax +91 (194) 2403470. E-mail: marauf2903@yahoo.co.in

References

1. Blizzard JJ, Rupp JJ. Prolongation of the PR interval as a manifestation of thyrotoxicosis. ***JAMA*** 1960; 173: 1845-1846.
2. Colzani RM, Emdin M, Conforti F, Passino C, Scarlattini M, Tervasi G. Hyperthyroidism is associated with lengthening of ventricular repolarization. ***Clin Endocrinol (Oxf)*** 2001; 55: 27-32.
3. Williams GH, Lilly LS, Seely EW. The heart in endocrine and nutritional disorders. In: Braunwald E, editor. Heart Disease. A Textbook of Cardiovascular Medicine. 5th ed. Philadelphia (PA): WB Saunders Company; 1997. p. 1887-1913.
4. Burggraaf J, Julen JH, Lalezari S, Schoemaker RC, DeMeyer PH, Meinders AE, et al. Sympathovagal imbalance in hyperthyroidism. ***Am J Physiol Endocrinol Metab*** 2001; 281: 190-195.
5. Davis AC, Smith HC. Complete heart block in hyperthyroidism following acute infections. A report of six cases with necropsy findings in one case. ***Am Heart J*** 1933; 9: 81-89.
6. Stern P, Jacobs RL, Duncan W. Complete heart block complicating hyperthyroidism. ***JAMA*** 1970; 212: 2117-2118.
7. Jameson JL, Weetman AP. Disorders of thyroid gland. In: Braunwald E, Fauci AS, Kasper DL, Hauser SL, Longo DL, Jameson JL, editors. Harrison's Principles of Internal Medicine. 15th ed. New York (NY): McGraw-Hill; 2001. p. 2060-2083.

Simultaneous bilateral tubal pregnancy after ovulation induction with clomiphene citrate

Mariam Mathew, *MD, MRCOG,*
Shabnam Saquib, *MBBS, MD,*
Andrzej Krolikowski, *MD, PhD.*

Simultaneous bilateral ectopic pregnancy is a rare and difficult to diagnose preoperative condition. The frequency of bilateral ectopic pregnancy has been estimated at 1 in 200,000 uterine pregnancies and from 1/725 to 1/1,580 ectopic pregnancies.[1] We report a case of simultaneous bilateral tubal pregnancy; one ruptured and the other unruptured, following ovulation induction in a 25-year-old Indian woman.

The patient was a gravida-1 para-0, at 9 weeks gestation admitted with acute abdominal pain of 2 hours duration. This pregnancy resulted from ovulation induction with 100 mg of clomiphene citrate followed by intrauterine insemination (IUI). A transvaginal ultrasonogram 3 weeks after IUI showed a small intrauterine gestational sac without fetal pole. Her first pregnancy was a right-sided tubal pregnancy, which was also a result of ovulation induction with clomiphene citrate. She was treated with laparoscopic methotrexate (MTX) injection. This was 3 years ago. Clinical examination on admission revealed pallor and lower abdominal tenderness with stable vital signs. Vaginal examination showed tenderness on both fornices and cervical excitation. Transvaginal sonography revealed a small intrauterine gestational sac without fetal pole, a complex mass in the right adnexa and free fluid in the pouch of Douglas. Diagnostic laparoscopy followed by laparotomy and dilatation and curettage (D&C) was performed immediately. We have found 100 cc of fresh blood and some clots in the peritoneal cavity. Right tube showed a large hematosalpinx, friable and adherent to the ovary. Left tube showed a 2 cm unruptured ampullary ectopic. Right salpingectomy and left linear salpingostomy were carried out. Moderate amount of curetting was obtained on D&C. Histopathological examination identified chorionic villi in each tube, thus, confirming the presence of a bilateral tubal pregnancy and secretory endometrium with decidual changes in the endometrial curetting. The postoperative course was unremarkable and the patient was discharged on day 5 after the operation.

Bilateral tubal pregnancies and other unusual forms of ectopic gestations are seen more often today, as part of the rising incidence of ectopic