

Grave's Disease (Hyperthyroidism) in a Child Presenting with Tachycardia

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Abstract: We report here, a six year old Saudi girl who presented with history of tachycardia, she was, then, diagnosed to have hyperthyroidism due to Grave's disease. The importance of including thyroid function in the management of tachycardia is highlighted.

Keywords: Grave's Disease, hyperthyroidism , tachycardia.

INTRODUCTION

It has long been recognized that some of the most characteristic and common signs and symptoms of thyroid disease are those that result from the effects of thyroid hormone in the heart and cardiovascular system [1-3]. Hyperthyroidism is associated with palpitations, tachycardia, exercise intolerance, dyspnea on exertion, widened pulse pressure and sometimes atrial fibrillation.

In the present report, we describe a 6-year-old Saudi girl with hyperthyroidism who presented mainly with tachycardia. This case has reports the importance of recognizing this fact, and the need to order appropriate thyroid function test to establish the diagnosis.

CASE REPORT

A 6-yearsold Saudi girl was referred from the primary care clinic with tachycardia and palpitation. She was assessed initially in the local hospital, where sinus tachycardia was documented, and heart rate of 150/minutes (Fig. 1). Echocardiogram was normal. Her past medical history was unremarkable; in particular there was no history of hypermetabolism. Medical examination revealed a pleasant young girl, with a height of 115 cm, at the 50th percentile and weight of 17 kilogram, at the 25th percentile, and a heart rate of 160/minute, with hyperdynamic circulation.

Thyroid was not palpable and the rest of examination was unremarkable.

Laboratory investigations revealed normal complete blood count, renal and liver functions. Thyroid function showed high free thyroxine (FT 4) of more than 80 Pmol/L (normal FT 10-25) with suppressed thyroid stimulating hormone (TSH) of less than 0.01 IU/L (N;0.5-5). Thyroid stimulating immunoglobulin (TSI) was positive, Bioscentia Laboratory, Germany. Chest X-ray was normal.

Tc^{99m} thyroid scan showed a homogeneous gland with high uptake of 27% (normal 0.5-4%) (Fig. 2). The diagnosis of hyperthyroidism (Grave's disease) was entertained and she was started on Neomercazole 5mg three times daily and Propranolol 10mg twice daily. She was clinically and biochemically euthyroid on Neomercazole within 6 months of therapy. However, she experienced four relapse while tapering Neomercazole with the first 10 years of treatment. Finally, a radioactive iodine therapy was given. She received clinically and biochemically euthyroid for the first 2 years, then developed hypothyroidism which repeated L-thyroxine therapy.

Currently, at the time of writing this report, she is 20 years-old and clinically and biochemically euthyroid in 75 µg of L-thyroxine therapy.

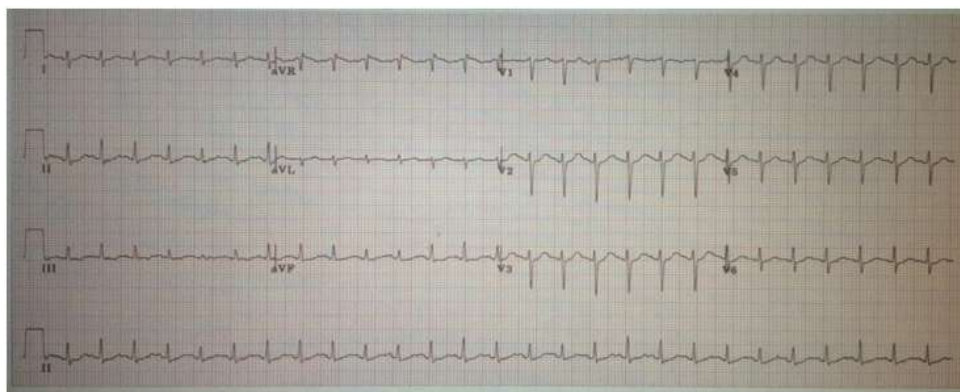


Fig. 1: An electrocardiogram (ECG) showing sinus tachycardia



Fig. 2: Technetium (Tc) 99m thyroid scan showing, a homogeneous gland with high uptake of 27% (normal; 0.5-4%)

DISCUSSION

Hyperthyroidism was diagnosed based on elevated levels of free thyroxine (FT₄), with suppressed level of thyroid-stimulating hormone, (TSH). Grave's disease was suggested by the positive thyroid stimulating immunoglobulin (TSI) antibodies. The thyroid gland was not enlarged, as the case, in 5%. No goiter, or eye signs (exophthalmus) [4, 5].

Patients with hyperthyroidism present with characteristic signs and symptoms, many of which related to the heart such papillitis and tachycardia. Cardiac contractility is enhanced, and resting heart rate and cardiac output are increased. Cardiac output may be increased by 50 to 30% over that of normal subject. As a result of combined effect of in resting heart rate, contractility, ejection fraction, and blood volume. Rapid diagnosis of hyperthyroidism and successful treatment is associated with a reversion to sinus rhythm in a majority of patients within two to three months.

It has been suggested that hyperthyroidism resembles a hyperadrenergic state, however, no edema suggest that thyroid hormone excess enhance the sensitivity of the heart to adrenergic stimulation [6]. In hyperthyroidism, serum level of catecholamines remain low or normal [7]. Treatment of hyperthyroidism with β -adrenergic blockade improves may, if not all, of the cardiovascular signs and symptoms associated with

hyperthyroidism [8]. Heart rate is showed but the enhanced diastolic performance is not altered after treatment which indicates that Thyroxine (T₃) acts directly on the heart to increase calcium cycling [9, 10].

Treatment involves alleviation of symptoms and correction of the thyroxic state. Adrenergic hyperfunction is treated with beta-adrenergic blockade correcting the high thyroid hormone levels can be achieved with anti-thyroid medication e.g. Nomercazole that block the synthesis of thyroid hormone or by the treatment with radioactive iodine [11-13].

Finally, pediatricians should be aware of this possibility. Thyroid function should be included in the work-up of patients with tachycardia.

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REFERENCES

1. Klein I, Ojamaa K; Thyroid hormone and the cardiovascular system. *N Engl J Med.*, 2001; 344(7): 501-509.

2. Danzi S, Klein I; Thyroid hormone and the cardiovascular system. *Minerva Endocrinologica*, 2004; 29(3): 139-150.
3. Kahaly GT, Dillmann WH; Thyroid hormone action is the heart. *Endocrine Rev.*, 2005; 26(5): 704-728.
4. Volpe R; Immunoregulation in autoimmune thyroid disease. *Thyroid*, 1994; 4(3): 373-377.
5. Weetman AP; Grave's disease. *N Engl J Med.*, 2000; 343 (17): 11235-48.
6. Nakazawa H, Lythall DA, Harada SMC, Noh J, Ishikawa N, Sugino K *et al.*; Is there a place for the late cardioversion of atrial fibrillation? A long-term follow-up study of patients with post-thyrotoxic atrial fibrillation. *Eur Heart J.*, 2000; 21(4): 327-333.
7. Hoit BD, Khoury SF, Shao Y, Gabel M, Liggett SP, Walsh RA; Effects of thyroid hormone in cardiac β -adrenergic responsiveness in conscious baboons. *Circulation*, 1997; 96: 592-598.
8. Ventrella S, Klein I; Beta-adrenergic receptor blocking drugs in the management of hyperthyroidism. *The Endocrinologist*, 1994; 4: 391-399.
9. Hartong R, Wang N, Kurokawa R, Lazar MA, Glass CK, Aprilletti I *et al.*; Delineation of three different thyroid hormone-response elements in promotes of rat sarcoplasmic reticulum Ca²⁺ ATPase gene. *J Biol Chem.*, 1994; 269(17): 13021-13029.
10. Mintz G, Pizzarello R, Klein I; Enhanced left ventricular diastetic function in hyperthyroidism: non-invasive assessment and response to treatment. *J Clin Endocrinol Metab.*, 1991; 73(1): 146-150.
11. Cooper DS; Antithyroid drugs. *N Eng J Med.*, 2005; 352(9): 905-917.
12. Kalinyak JE, Mc Dougall IR; How should the dose of iodine-131 be determined in the treatment of Grave's hyperthyroidism. *J Clin Endocrinol Met.*, 2003; 88(3): 975-977.
13. Mumtaz M, Lin LS, Sharifuddiz A; Radioiodine I-131 for the therapy of Grave's disease. *Malays J Med Sci.*, 2009; 16(1): 25-33.