http://www.banglajol.info/index.php/JACEDB

Case report

DOI: https://doi.org/10.3329/jacedb.v2i1.78433

Journal of Association of Clinical Endocrinologist and Diabetologist of Bangladesh
January 2023, Vol. 2, No. 1, pp. 24-27

ISSN (Online): 2959-8176
ISSN (Print): 2958-0307

Reversible thyrotoxic valvulopathy: A case report

*Rana MS[1], Fariduddin M[2], Mahmood SA[3], Hossain MS[4], Khan MA[5], Ahmed CM[6]

[1]Md Sohel Rana, Resident, Department of Endocrinology, Bangabandhu Sheikh Mujib Medical University, Dhaka, Bangladesh; [2]Md Fariduddin, Professor, Department of Endocrinology, Bangabandhu Sheikh Mujib Medical University, Dhaka, Bangladesh; [3]Syed Azmal Mahmood, Resident, Department of Endocrinology, Bangabandhu Sheikh Mujib Medical University, Dhaka, Bangladesh; [4]Md Solaiman Hossain, Resident, Department of Endocrinology, Bangabandhu Sheikh Mujib Medical University, Dhaka, Bangladesh; [5]Murshed Ahamed Khan, Assistant Professor, Department of Endocrinology, Bangabandhu Sheikh Mujib Medical University, Dhaka, Bangladesh; [6]Chowdhury Meshkat Ahmed, Professor, Department of Cardiology, Bangabandhu Sheikh Mujib Medical University, Dhaka, Bangladesh.

Abstract

A 52-year-old female was admitted to the hospital with chronic diarrhoea, vomiting, and weight loss. She also reported dyspnea on exertion, palpitation, and hypertension. She had recently been diagnosed with Grave's thyrotoxicosis with carbimazole-induced agranulocytosis with lithium toxicity. Physical examination revealed signs of thyrotoxicosis with right heart failure with atrial fibrillation. Thyroid gland examination revealed a diffuse, firm, nontender goiter. On cardiovascular examination, a grade III pansystolic murmur in the mitral area was found. ECG showed atrial fibrillation. Transthoracic echocardiography showed a flail posterior mitral leaflet (PML) with severe mitral regurgitation (MR). After initial conservative treatment, radioactive iodine ablation was done and her dyspnea, leg edema and diarrhoea gradually improved and she achieved stable thyroid and cardiac function with β blocker only. A follow-up visit after 6 months documented the absence of cardiac symptoms. There were no murmurs clinically and only mild MR on echocardiography. This case demonstrates the importance of cardiac evaluation in hyperthyroidism as treatment of thyroid abnormalities can reverse these cardiac manifestations. *[J Assoc Clin Endocrinol Diabetol Bangladesh, January 2023; 2 (1): 24-27]*

Keywords: Thyrotoxic valvulopathy; Hyperthyroidism; Mitral valve prolapse

***Correspondence:** Md. Sohel Rana, Resident, Department of Endocrinology, Bangabandhu Sheikh Mujib Medical University, Dhaka, Bangladesh. Email: m.s.rana34@gmail.com; Cell no: 01724455696

Introduction

Hyperthyroidism has many systemic effects and exerts many changes on the cardiovascular system. Thyrotoxic valvulopathy is one of the rare presentations of hyperthyroidism. Hyperthyroidism can affect cardiac valves in many ways. Thyrotoxicosis may damage cardiac valves through a combined effect of direct myxomatous valve degeneration causing primary valve insufficiency and hemodynamic volume overload causing cardiac chamber dilatation and further valve incompetency.[1] We present a case of an elderly female with severe mitral regurgitation (MR) due to mitral valve prolapse associated with Graves' disease with carbimazole-induced agranulocytosis and lithium toxicity. This case highlights the importance of maintaining a high index of suspicion for hyperthyroidism when faced with valvular heart disease as treatment of the thyroid abnormalities can reverse these cardiac findings.

Case presentation

A 52-year-old hypertensive female was admitted to the hospital with chronic diarrhoea, vomiting, and weight loss for 3 months. She also reported dyspnea on exertion and palpitation. Initially, she was admitted to a tertiary care hospital one month back and after thorough evaluation diagnosed with Graves' disease on the basis of thyroid function test (TFT), thyroid scan, radioactive iodine uptake (RAIU) test and was put on carbimazole 45 mg daily. After 2 weeks she developed carbimazole-induced agranulocytosis and switched to lithium 800 mg/day. A few days later she developed lithium toxicity (serum lithium was 1.7 mmol/L) with profuse diarrhoea, vomiting, psychotic behaviour and

electrolyte imbalance. She was then managed in the intensive care unit after stopping lithium and was referred to our hospital. On admission, she was found mildly anemic, respiratory rate was 28 breaths/min; pulse was 109 beats/min, irregularly irregular, high volume; blood pressure 110/70 mm Hg; jugular venous pressure (JVP) raised 4 cm from sternal angle; mild ankle edema. The hands were warm and sweaty with fine tremors. Thyroid gland examination revealed mild diffuse goiter which was firm, mobile and non-tender without any signs of thyroid eye disease.

On examination of the cardiovascular system, the apex beat was shifted to the left, thrusting in character. 1st heart sound was variable, with pan systolic murmur present in the whole precordium most prominent in the mitral area. Her Burch-Wartofsky score on admission was 40 (10 points for diarrhoea, 10 points for tachycardia, 10 points for moderate heart failure, and 10 points for atrial fibrillation) being suggestive of an impending thyroid storm. ECG revealed atrial fibrillation with a fast-ventricular rate (Figure-1). Echocardiography showed flail posterior mitral leaflet (PML) (due to ruptured chordae) with severe MR, dilated left atrium (LA), mild tricuspid regurgitation (TR), pulmonary artery systolic pressure (PASP) 70 mm Hg, no regional wall motion abnormalities (RWMA) (Figure-2). Chest radiograph showed no acute pathology.

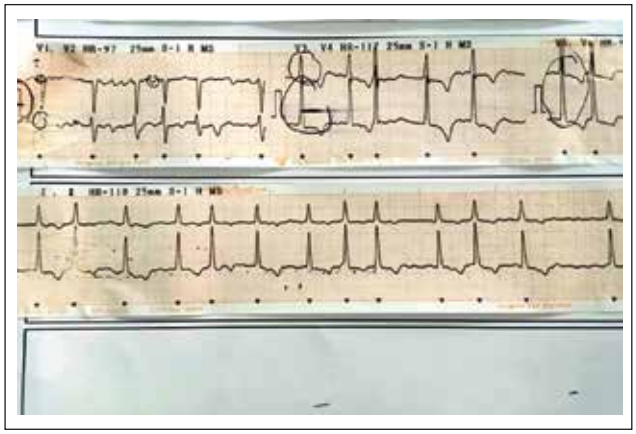

Figure-1: ECG showing atrial fibrillation.

Laboratory investigations after admission revealed a hemoglobin level of 9.8 gm/dl and a total count of WBC 9000/mm^3. Serum creatinine was 0.49 mg/dL, albumin 2.77 gm/dL, serum glutamate pyruvate transaminase (SGPT) 38 U/L, serum alkaline phosphatase 225 U/L, random blood glucose 5.5 mmol/L. TFT showed TSH 0.02 µIU/ml, FT4 was 4.7 ng/ml, FT3 13.9 pg/ml, thyrotropin receptor antibody (TRAb) 9.54 U/L, serum

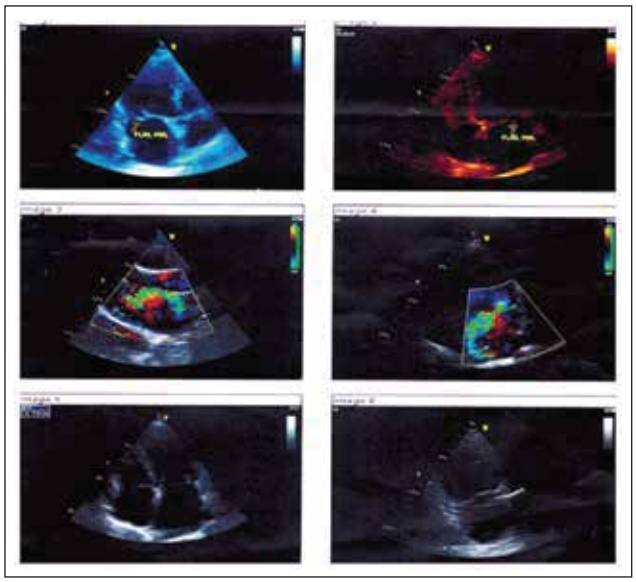

Figure-2: Echocardiographic finding before treatment showing flail posterior mitral leaflet (?due to ruptured chordae) with severe mitral regurgitation (MR), mild dilated left atrium (LA), pulmonary artery systolic pressure (PASP)=70 mm Hg, and left ventricular ejection fraction (LVEF) 50%.

Lithium level 0.20 mmol/L.

The patient was initially managed with Lugol's iodine, propranolol, phenobarbitone, rivaroxaban, lithium SR, furosemide + spironolactone (20/50), and phenoxymethylpenicillin. Radioactive iodine ablation was done later on. Her dyspnea, leg edema and diarrhoea gradually improved and she achieved stable

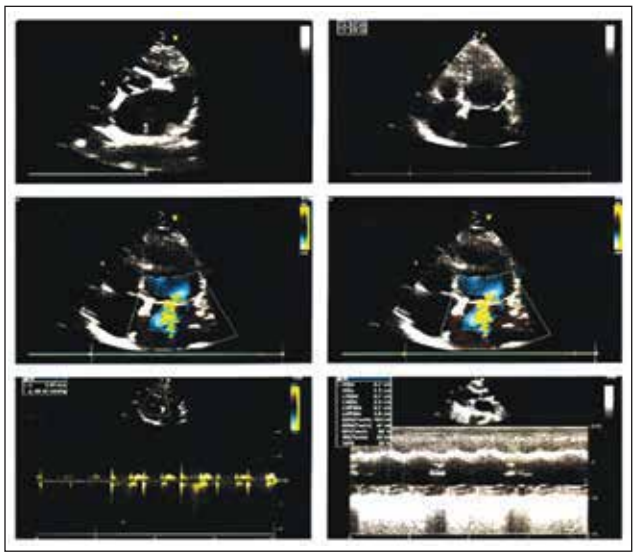

Figure-3: Echocardiographic findings after restoring near normal thyroid function showing posterior mitral leaflet (PML) prolapse, moderate mitral regurgitation (MR), pulmonary artery systolic pressure (PASP) 48 mmHg, and left ventricular ejection fraction (LVEF) 60%.

thyroid and cardiac function with β blocker and levothyroxine 50 µg daily. Follow-up visits after 6 months documented the absence of cardiac symptoms and no murmur was noted clinically. Repeated transthoracic echocardiography revealed the intensity of MR decreased from grade III to grade II (Figure-3) and then grade I MR (Figure 4) within 6 months of radioactive iodine ablation.

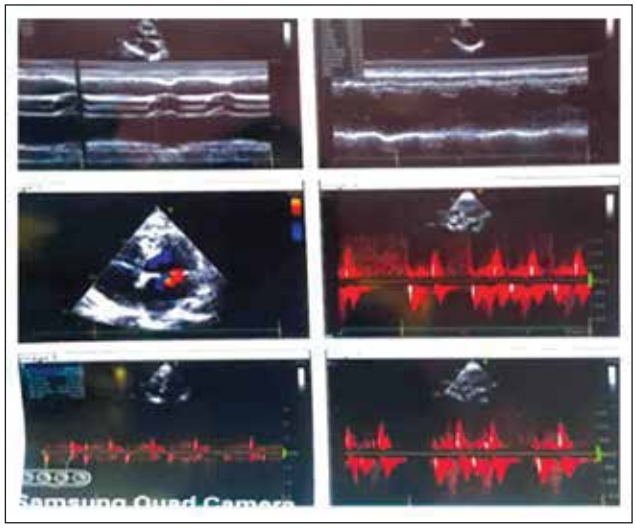

Figure-4: Echocardiographic findings 6 months after radioactive iodine (RAI) ablation showing mild (grade I) mitral regurgitation (MR), pulmonary artery systolic pressure (PASP) 33 mmHg, and left ventricular ejection fraction (LVEF) 61%

Discussion

More than 200 years ago, Dr. Caleb Parry, an English physician, was the first to identify the cardiovascular consequences of hyperthyroidism.[2] Our patient presented with right-sided heart failure due to mitral valve prolapse with severe mitral regurgitation. A three-fold higher incidence of mitral valve prolapse (16.3%) in 126 patients with Grave's disease compared to 111 healthy controls (5.4%) was reported around 40 years back.[3] Reversible mitral valve prolapse and mitral regurgitation can also occur in children with hyperthyroidism, especially those with Graves' disease.[4] It is reported that thyrotoxic mitral regurgitation usually disappears after successful treatment of hyperthyroidism as in the index case.[5] In a study by Syriou et al., a 48-year-old woman with hyperthyroidism and refractory heart failure was treated for several months, leading to the resolution of her symptoms and the echo findings. She also had significant tricuspid regurgitation and pulmonary hypertension.[6]

The inotropic and chronotropic effects of thyroid hormones, which change cardiac rhythmicity (for example, atrial fibrillation) or heart muscle function, are thought to contribute to making changes in thyrocardiac function. (e.g., hypertrophy and biventricular enlargement).[7] There are various known effects of thyrotoxicosis, particularly Grave's disease, on connective tissues. For instance, Grave's ophthalmopathy has been linked to the development of fibroblasts and inflammatory cell infiltrates in the soft tissue of the extraocular muscles while Grave's dermopathy is brought on by the deposition of glycosaminoglycan in the skin dermis.[8,9] Similar myxomatous changes can also affect the mitral valve causing significant primary MR in association with rheumatic valve disease and secondary to heart failure.[10,11] As a result, myxomatous degeneration caused by thyrotoxicosis may potentially directly affect the connective tissue of heart valves. Excess thyroid hormone directly affects the heart and circulatory system, causing intrinsic papillary muscle disease.[5] At the same time, the prolapsed mitral valve, containing defective collagen fibrils and excess spongiosa, appears to withstand elevated left ventricular systolic pressures poorly compared with the normal mitral valve, resulting in an increased tendency to chordae tendineae rupture.[12] Therefore, thyrotoxic valvulopathy should be viewed as a combination of both primary changes brought on by a direct damaging effect on the valve structures and secondary changes brought on by cardiac chamber dilatation causing valve leaflet malcoaptation. Furthermore, Autoimmunity is a well-known causative factor in Graves' disease and recently it has been mentioned also in the etiology of mitral valve prolapse.[13] The demonstration of lymphocytic infiltration of the mitral valve leaflets in patients with chronic lymphocytic thyroiditis and mitral valve prolapse would support a possible autoimmune cause for mitral valve prolapse in patients with autoimmune thyroid disorders.[14]

Conclusion

Hyperthyroidism should be considered in the differential diagnosis of heart failure in general and also in coexisting valvular heart disease. Definite treatment of hyperthyroidism may escape patients from unnecessary cardiac intervention.

Acknowledgements

We are grateful to our patient for giving the consent to report the case.

Disclosure

The authors have no multiplicity of interest to disclose.

Financial Disclosure

The author(s) received no specific funding for this work.

Data Availability

Any inquiries regarding supporting data availability of this study should be directed to the corresponding author and are available from the corresponding author on reasonable request.

Ethics Approval and Consent to Participate

Written informed assent was taken from the patient.

How to cite this article: Rana MS, Fariduddin M, Mahmood SA, Hossain MS, Khan MA, Ahmed CM. Reversible thyrotoxic valvulopathy: A case report. J Assoc Clin Endocrinol Diabetol Bangladesh, 2023; 2(1):24-27

Publication History

Received on: 14 October 2022

Accepted on: 23 November 2022

Published on: 1 January 2023

References

1. Pierre K, Gadde S, Omar B, Awan GM, Malozzi C. Thyrotoxic valvulopathy: case report and review of the literature. Cardiol Res 2017;8(3):134-38. DOI: 10.14740/cr564w.
2. Skelton CL. The heart and hyperthyroidism. N Engl J Med 1982;307(19):1206-08. DOI: 10.1056/NEJM198211043071909.
3. Brauman A, Algom M, Gilboa Y, Ramot Y, Golik A, Stryjer D. Mitral valve prolapse in hyperthyroidism of two different origins. Br Heart J 1985;53(4):374-77. DOI: 10.1136/ hrt.53.4.374.
4. Liu YP, Tsai WY, Wang JK, Wu MH. Reversible mitral valve prolapse and mitral regurgitation in children with Graves' disease. J Pediatr Endocrinol Metab 2004;17(9):1211-13. DOI: 10.1515/jpem.2004.17.9.1211.
5. Fekri K, Michel CM, Tamilia M. Reversible, severe mitral regurgitation in thyrotoxic Graves' disease. BMJ Case Rep 2021;14(2):e239626. DOI: 10.1136/bcr-2020-239626.
6. Syriou V, Plastiras SC, Paterakis T, Moyssakis I, Vlachoyiannopoulos P. Severe reversible right heart failure in a patient with hyperthyroidism. Int J Clin Pract 2008;62(2): 334-36. DOI: 10.1111/j.1742-1241.2006.00979.x.
7. Chiha M, Samarasinghe S, Kabaker A. Thyroid storm: An updated review. J Intensive Care Med 2015; 30:131-40. DOI: 10.1177/0885066613498053.
8. Bahn RS. Graves' ophthalmopathy. N Engl J Med 2010;362(8):726-738. DOI: 10.1056/NEJMra0905750.
9. Peacey SR, Flemming L, Messenger A, Weetman AP. Is Graves' dermopathy a generalized disorder? Thyroid 1996;6(1):41-45. DOI: 10.1089/thy.1996.6.41.
10. Harirforoosh S, Cohen G, Glovaci D, Patel PM. Right heart failure in the setting of thyrotoxic valvulopathy: The pathophysiology of an often neglected diagnosis: A case report. Eur Heart J Case Rep 2022;6(9):ytac305. DOI: 10.1093/ ehjcr/ytac305.
11. Cavros NG, Old WD, Castro FD, Estep HL. Case report: Reversible mitral regurgitation and congestive heart failure complicating thyrotoxicosis. Am J Med Sci 1996;311(3):142-144. DOI: 10.1097/00000441-199603000-00007.
12. Chen AW, Wee HC, Sonawane V. Flail mitral valve: a rare complication of a thyroid storm. AACE Clin Case Rep 2019;5(1):e4-e6. DOI: 10.4158/ACCR-2018-0137.
13. Guy TS, Hill AC. Mitral valve prolapse. Annu Rev Med 2012;63:277-92. DOI: 10.1146/annurev-med-022811- 091602
14. Baştuğ S, Sarı C, Aslan AN, Bayram NA, Ayhan H, Kasapkara HA, et al. Evaluation of autoimmune thyroid disease in patients with mitral valve prolapse. J Clin Pract Res 2015; 37(3): 98-101. DOI: 10.5152/etd.2015.0017