Progress in Pediatric Cardiology 58 (2020) 101264

Contents lists available at ScienceDirect

Progress in Pediatric Cardiology

journal homepage: www.elsevier.com/locate/ppedcard

Mitral valve prolapse in adolescent female with hyperthyroidism

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ARTICLE INFO

Keywords:
Mitral valve prolapse
Tricuspid valve prolapse
Hyperthyroidism
Palpitations
Chest pain
Pulmonary hypertension

ABSTRACT

Mitral valve prolapse is prevalent in about 6% of young healthy females yet two to three times as prevalent in patients with hyperthyroidism [1, 8]. Hyperthyroidism is often missed as an underlying diagnosis when mitral valve prolapse is visualized by echocardiogram. There are many cardiac findings reported in the literature associated with hyperthyroidism. However, hyperthyroidism may be difficult to diagnose in the early stages of the disease process without other classic signs and symptoms. We report a case of an adolescent female who presented to our cardiology clinic with palpitations and chest pain diagnosed with hyperthyroidism after subtle findings of mitral valve prolapse and trivial mitral regurgitation. This case report will review the classic cardiac changes which may be observed on echocardiography in patients with hyperthyroidism.

1. Introduction

The effects of hyperthyroidism on the heart have been well studied, reported, and documented [1]. Hyperthyroidism may produce changes in cardiac contractility, myocardial oxygen consumption, cardiac output, blood pressure, systemic vascular resistance, and atrial fibrillation [1]. In this case report, we discuss an adolescent female newly diagnosed with hyperthyroidism and review her associated echocardiographic findings.

2. Case

A 16-year-old girl was referred to our institution for complaint of palpitations, chest pain, and a new onset heart murmur after prior evaluation at an outside emergency department. Since then, she experienced daily symptoms, both at rest and with exercise, of “rapid heart beating out of her chest” and mid-sternal pressure-like chest pain radiating to her anterior ribs and back. In addition, she complained of headaches, shortness of breath, and fatigue. Prior to this emergency department visit, she was in good health without any past medical history or significant family history of cardiac disease.

Due to concurrent COVID-19 pandemic, her initial clinic visit with cardiology was conducted via telemedicine. During this appointment, she was discovered to have reproducible chest pain, in addition to her palpitations, and recommended to start non-steroidal anti-inflammatory medications and provided with a 48-h Holter monitor. However, she complained of persistent cardiac symptoms 2 weeks later at her follow-up in-person cardiology clinic visit. Her Holter monitor revealed an average heart rate of 116 beats per minute with 4 isolated premature ventricular contractions. Her blood pressure was elevated at 147/71 mmHg, and her heart rate was elevated at 127 beats per minute. Her height was 168 cm and weight was 63 kg. Physical exam revealed bilateral exophthalmos and thyromegaly with a nontender goiter. She was tachycardic and hypertensive on exam without appreciable murmur, rub, click or gallop. Thyroid stimulating hormone was low at 0.01 IU/mL. Total T4 was elevated at 23.1 μg/dL. Free T4 was elevated at 2.9 ng/dL. Total T3 was elevated at 499 ng/dL, Thyroglobulin antibody was elevated at 295 IU/mL, and Anti-thyroid peroxidase antibody was higher than 900 IU/mL, all consistent with Grave's thyrotoxicosis.

ECG revealed sinus tachycardia and was otherwise normal. Transthoracic echocardiogram showed mitral valve prolapse with trivial mitral regurgitation, normal tricuspid valve with trivial regurgitation, normal estimated right ventricular systolic pressure, normal biventricular chamber size and systolic function with left ventricular ejection fraction of 64% (Fig. 1). Due to concurrent COVID-19 pandemic, endocrinology was consulted via telemedicine prior to leaving the cardiology clinic. Our patient was started on atenolol and methimazole for Graves thyrotoxicosis.

3. Conclusion

Hyperthyroidism is associated with pulmonary hypertension, mitral and tricuspid valve regurgitation, myocardial remodeling, and even

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https://doi.org/10.1016/j.ppedcard.2020.101264
Received 14 May 2020; Received in revised form 15 June 2020; Accepted 22 June 2020
Available online 25 June 2020

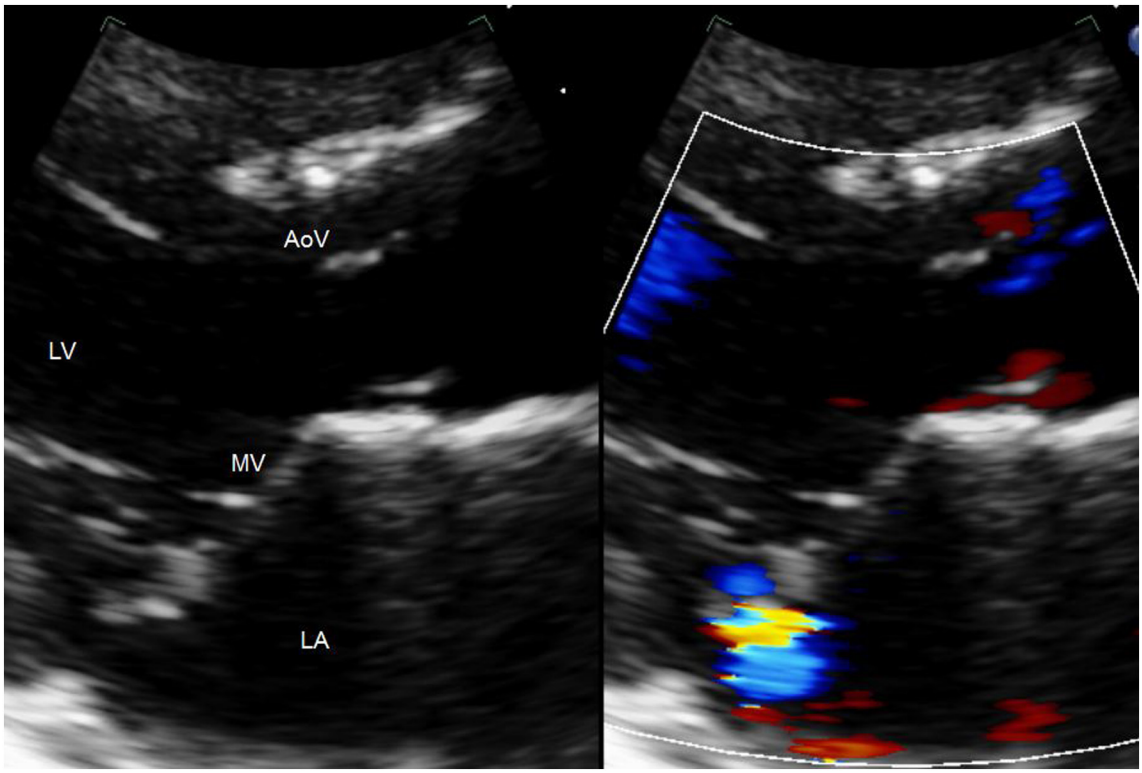

Fig. 1. Parasternal long axis view showing a thickened and redundant mitral valve prolapsing into the left atrium during systole with posteriorly directed trivial mitral regurgitation.
AoV aortic valve, *LA* left atrium, *LV* left ventricle, *MV* mitral valve.

cardiac failure. The pathophysiology of pulmonary hypertension in the setting of hyperthyroidism has not been fully elucidated and felt to be multifactorial with similar clinical presentation to other autoimmune induced pulmonary hypertension [2,3]. One theory which may explain the association of pulmonary hypertension with thyrotoxicosis includes the stimulation of a baroreceptor reflex in the pulmonary veins by a higher pressure within the cardiovascular system leading to constriction of the pulmonary vasculature, increased right ventricular pressure and contractility, higher pulmonary vascular resistance, and pulmonary hypertension [4]. Pulmonary pressure typically normalizes once a euthyroid state is reestablished [2].

Tricuspid valve prolapse, mitral valve prolapse, and myocardial remodeling have direct and indirect associations with hyperthyroidism. It has been postulated that tricuspid regurgitation and prolapse are the direct result of right ventricular dilation secondary to increased systemic venous return in the hyperthyroid state [5,6]. Channick et al. (1981) suspected mitral valve prolapse in the setting of hyperthyroidism was secondary to increased adrenergic tone [7]. Patients with autoimmune thyroid disease may have excess glycosaminoglycan deposition in the periorbital and pretibial dermal and subcutaneous skin layers and in the cardiac valves, especially the mitral valve [8]. This, in conjunction with enhanced cardiac contractility, in the setting of elevated thyroid hormone, may lead to chordae tendinae rupture in a vulnerable mitral valve [9].

Skelton (1982) postulated several mechanisms which may lead to cardiac remodeling, specifically cardiac hypertrophy, in hyperthyroidism [10]. Cardiac volume overload, increased contractility, and tachycardia all place a higher demand on the heart for increased energy generation [10]. Accelerated energy turnover then results in activation of protein-synthesizing processes directly by thyroid hormone [10]. Thyroid hormone may exert a direct effect on myocyte-specific gene transcription by regulating proteins involved in cardiac contractile function [11].

Our patient's echocardiographic findings do not completely encompass the cardiac changes which may potentially be seen in hyperthyroidism. However, hyperthyroidism should be included in the differential diagnosis for patients with the subtle finding of mitral valve prolapse who present with palpitations, tachycardia, chest pain, and hypertension. Early recognition of hyperthyroidism in our patient prevented further morbidity and progression of her cardiac findings. We plan on following our patient's mitral valve prolapse for any progression or improvement after she returns to a euthyroid state.

Funding

This research did not receive any specific grant from funding agencies in the public, commercial, or not-for-profit sectors.

Declaration of Interest

Authors have no conflicts to disclose.

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