Aronow, J Clin Case Rep 2013, 3:11
DOI: 10.4172/2165-7920.1000e120

Journal of Clinical Case Reports
ISSN: 2165-7920

Editorial Open Access

Cardiovascular Manifestations of Hyperthyroidism

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Gupta et al. reported an excellent case report of an acute myocardial infarction caused by thyrotoxicosis in a 23-year old man with a normal coronary angiogram [1]. Acute myocardial infarction may occur in patients with hyperthyroidism and normal coronary arteries due to major coronary artery occlusion by coronary vasospasm induced by a hyperadrenergic state with stimulation of adrenergic receptors on coronary arteries and increased myocardial oxygen demand. This editorial will briefly discuss some other cardiovascular manifestations of hyperthyroidism.

Hyperthyroidism causes an increase in resting heart rate, systolic blood pressure, blood volume, left ventricular mass, stroke volume, left ventricular ejection fraction, and cardiac output and a reduction in systemic vascular resistance with a widened pulse pressure [2]. Hyperthyroidism may cause sinus tachycardia, atrial fibrillation, atrial flutter, paroxysmal atrial tachycardia, premature ventricular beats, premature atrial beats, atrioventricular block, palpitations, left ventricular hypertrophy, myocardial ischemia, angina pectoris, and congestive heart failure [3-5]. These cardiovascular manifestations result from an increased myocardial oxygen demand caused by excessive amounts of the thyroid hormones T4 and T3. The high cardiac output state associated with hyperthyroidism is caused by the peripheral hemodynamic changes as well as by an increase in myocardial contractility caused by excessive amounts of T4 and T3.

A study of 85 patients aged 60 years and older with hyperthyroidism reported that palpitations were present in 42% of patients, angina pectoris in 20% of patients, dyspnea on exertion, orthopnea , or paroxysmal nocturnal dyspnea in 66% of patients, sinus tachycardia in 58% of patients, atrial fibrillation documented by electrocardiography in 45% of patients, cardiomegaly in 11% of patients, a heart murmur heard in 69% of patients, bounding peripheral pulses in 7% of patients, electrocardiographic evidence of St-segment abnormalities in 57% of patients, electrocardiographic evidence of T wave abnormalities in 62% of patients, and electrocardiographic evidence of atrioventricular block in 3% of patients [6]. Hyperthyroidism usually shortens the atrioventricular conduction time and functional refractory period, causing an increased ventricular rate in patients with atrial fibrillation. Hyperthyroidism predisposes to the development of atrial fibrillation in the absence of preexisting structural cardiovascular disease [7]. Shortening of the atrial effective refractory period plus facilitation of the atrial conduction delay increase the propensity for development of atrial fibrillation [7].

Atrial fibrillation in patients caused by hyperthyroidism is associated with thromboembolic stroke, systemic embolism, and congestive heart failure. Patients with atrial fibrillation and a CHADS2 score greater than 1 should be treated with warfarin therapy to maintain an international normalized ratio between 2.0 and 3.0. Because of the high rate of spontaneous cardioversion of atrial fibrillation to sinus rhythm once thyroid hormone levels are normalized, cardioversion to sinus rhythm should not be performed before the euthyroid state is achieved unless the patient is hemodynamically unstable despite intravenous beta blocker therapy. Beta blockers should be used to reduce the rapid ventricular rate associated with atrial fibrillation.

Congestive heart failure is common in patients with hyperthyroidism and heart disease in whom the heart is unable to increase cardiac output appropriately to match the increase in myocardial oxygen demand. Exertional dyspnea and congestive heart failure can result from hyperthyroidism without structural heart disease in all age groups [8]. Hyperthyroidism may also cause congestive heart failure in children without underlying heart disease [9]. Congestive heart failure may be the only clinical manifestation of hyperthyroidism in elderly patients with apathetic hyperthyroidism [6]. The high cardiac output state, increased myocardial oxygen demand, left ventricular hypertrophy, reduced left ventricular contractile reserve, rapid ventricular rate, decreased left ventricular filling time, and atrial fibrillation with a decrease in left ventricular filling because of the loss of atrial contribution and a rapid ventricular rate all contribute to the development of congestive heart failure in patients with hyperthyroidism.

Congestive heart failure may result in patients with hyperthroidism and no underlying cardiovascular disease because of an inadequate cardiac reserve to allow further increase in cardiac function during stress [10]. The increase in left ventricular mass index may lead to systolic dysfunction consistent with hyperthyroid cardiomyopathy reversible with treatment [11]. The acute left ventricular dysfunction which may be caused by hyperthyroidism may mimic ischemic coronary artery disease and is reversible with treatment [12].

Subclinical hyperthyroidism is diagnosed if the serum thyroid-stimulating hormone concentration is below the lower limit of the reference range with normal triiodothyronine and thyroxine values [13]. Cardiovascular findings associated with subclinical hyperthyroidism include increased cardiac contractility, impaired left ventricular diastolic filling, impaired systolic function during exercise, increased left ventricular mass index, increased intraventricular septal thickness, increased left ventricular posterior wall thickness , reduced large and small artery elasticity and a prolonged QTc interval resulting in increased ventricular rate and frequency of premature atrial beats, an increased incidence of atrial fibrillation, decreased exercise capacity, and increased all-cause mortality and mortality from cardiovascular disease [3].

A meta-analysis of 52,674 persons from 10 cohort studies showed that 2,188 persons (4.2%) had subclinical hyperthyroidism [14]. During follow-up, subclinical hyperthyroidism was associated with 24% increased all-cause mortality, with 29% increased coronary heart disease mortality, with 21% increased coronary heart disease events, and with 68% increased atrial fibrillation [14].

Subclinical hyperthyroidism was present in 71 of 5,316 elderly

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Received October 18, 2013; **Accepted** October 23, 2013; **Published** October 25, 2013

Citation: Aronow WS (2013) Cardiovascular Manifestations of Hyperthyroidism. J Clin Case Rep 3: e120. doi:10.4172/2165-7920.1000e120

persons (1.3%) with cardiovascular disease or cardiovascular risk factors [15]. Over 3.4-year follow-up, compared with euthyroid persons, persons with subclinical hyperthyroidism had a 327% increase in the incidence of congestive heart failure [15].

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