

Case Report

Pulmonary Embolism in Case of Hyperthyroidism: A Rare Case Report

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ABSTRACT

Patients of hyperthyroidism widely reported to have several coagulation and fibrinolytic disorders. Hyperthyroidism and Pulmonary embolism (PE) are one of the rare associations. We have reported a case of Thyroiditis with Multinodular Goitre (known case of Hyperthyroidism) complicated by pulmonary embolism. In this patient, PE was suspected on basis of clinical presentation with 2D Echo suggestive of severe pulmonary hypertension (PAH), with ECG suggestive of S₁Q₃T₃ pattern. PE was confirmed on CT pulmonary angiography. This patient treated with heparin followed by oral anticoagulants. In conclusion, hyperthyroidism is associated with increased risk venous thromboembolism including PE and necessitates the need of Thyroid evaluation if other conventional factors causing PE are not present.

Key words: Hyperthyroidism, Pulmonary embolism, CT pulmonary angiography.

INTRODUCTION

Hyperthyroidism is a common disorder, with an incidence of 0.5-2.5% worldwide, and women are much more likely to develop this disorder than men. [1-3] Hyperthyroidism is associated with a hypercoagulable state. [4,5] Several previous studies suggest that hyperthyroidism represent a potential hypercoagulable and hypofibrinolytic state, which may contribute to the increased risk of thromboembolism. [4,6-8] Most reports have focused on only the venous thromboembolism risk, and few of them have studied specifically the association between hyperthyroidism and pulmonary embolism (PE). [9-12] In another much larger study from Taiwan, the researchers used the country's National Health Insurance Database and calculated that the incidence of pulmonary embolism in the patients with acute hyperthyroidism was 0.16%, a 2.3 fold increase from a

baseline of 0.06% in patients without thyroid disorder. [9] Despite a low overall occurrence, pulmonary emboli accounted for up to 18% of all deaths from thyrotoxicosis. [13]

CASE REPORT

A 55-year-old female patient, whose mother was affected by Multinodular Goitre and operated for the same, had been treated for Toxic Multinodular Goitre since 1992. This diagnosis was based on hyperthyroid symptoms with elevated serum total T₃ level (200 ng/dl, normal range 90 to 190 ng/dl) with normal serum total T₄ and serum TSH. USG chest was suggestive of multiple Thyroid nodules with Thyroid extending retrosternally into superior mediastinum. Possibility of T₃ toxicosis was considered.

Patient was put under antithyroid drugs: Carbimazole at a dose of 5mg once a

day. Patient shown poor compliance with the drugs and again presented with hyperthyroid symptoms in June 2011. At that time patient was having elevated serum total T₄ level (19.49ug/dl, normal range 4.5 to 12.5 ug/dl) with suppressed serum TSH (<0.005 mIU/ml, normal range 0.3 to 4.5 mIU/ml) and Thyroid scan suggestive of thyroid uptake (10.5%, normal range 0.24 to 3.34%). At this time patient received Low dose Radioiodine Therapy and continued with tablet carbimazole once a day. Since last 4 years patient was on regular medications and regular follow up.

Now again in August 2015, patient suddenly had shortness of breath, dyspnea, tachypnea. Patient's thyroid status immediately performed which was euthyroid i.e. all the thyroid hormone levels within normal range. At the same time 2D echo performed which was suggestive of severe pulmonary hypertension (PAH) with Right Atrium, Right Ventricle, Pulmonary Artery dilated and Peak Pulmonary Gradient 70 mm of Hg. ECG had S₁Q₃T₃ pattern. USG thyroid had Thyroiditis with multinodular goiter. All this scenario lead us to suspect pulmonary embolism and CT pulmonary angiography, confirmed the diagnosis of PE which shown grey matter white area on Right and Left pulmonary artery (Figure 1). At this time patient was thrombolysed followed by inj. heparin and discharged on oral anticoagulants and Tablet carbimazole.

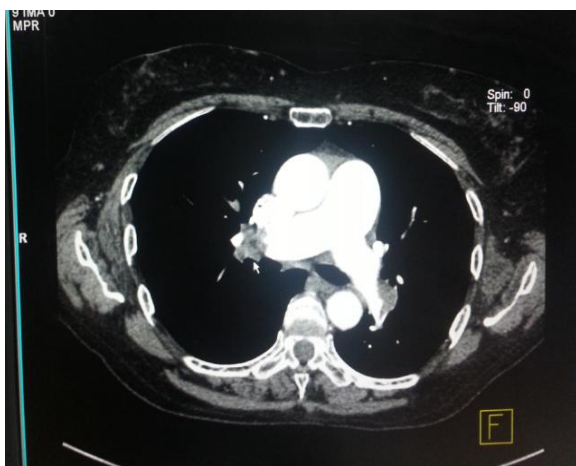


Figure 1: CT pulmonary angiography suggestive of bilateral pulmonary embolism. (Arrow showing thrombus in Right pulmonary artery)

DISCUSSION

Hyperthyroidism has widespread effects on the heart and cardiovascular system; it can increase the heart rate and blood pressure, [15] resulting in increased risk of atrial fibrillation and ventricular dysrhythmias. [16] In addition, a previous study reported that thyroid dysfunction may modify the physiologic processes of primary and secondary hemostasis, and lead to bleeding or thrombosis. [4,17,18] A systematic analysis supports an increased risk of venous thrombotic complications, including cerebral venous thrombosis (CVT), deep vein thrombosis (DVT) and PE in patients with hyperthyroidism. [14] Possible predisposing factors for the development of venous thrombosis and PE in patients with thyrotoxicosis are also in line with this triad. Indeed, patients with hyperthyroidism may often have accompanying endothelial dysfunction, decreased fibrinolytic activity, and hypercoagulable states, which contribute to the development of venous thrombosis and increased risk of PE. [4,9,18] There are few reports of PE associated with hyperthyroidism. A recent study, using a nationwide population-based dataset, aimed to estimate the risk of PE among hyperthyroidism patients during a five year period. [9] The study included 8903 patients with hyperthyroidism as a study cohort and 44515 randomly selected patients without hyperthyroidism as a comparison cohort. After adjustment for potential confounders, the risk of having PE during the five year follow-up period was 2.31 times greater (95% confidence interval 1.20-4.45, $P = 0.012$) for patients with hyperthyroidism than for patients in the comparison cohort. [9] However, several other mild to moderate abnormalities of the coagulation-fibrinolytic systems have been reported in patients with overt hyperthyroidism, predisposing these patients to a hypercoagulable state rather than to a bleeding tendency. [19]

Our case presented with pulmonary embolism in known case of hyperthyroidism and Diagnostic investigations are previous

thyroid profile reports, ECG, 2D Echo and the diagnosis of pulmonary embolism was confirmed on CT pulmonary angiography report. Prognosis in this case depends on severity of pulmonary hypertension. Similarly in our case prognosis is poor due to severe pulmonary hypertension. The treatment of pulmonary embolism was directed towards thrombolysis and shift her to on oral anticoagulants.

CONCLUSION

We present this case of pulmonary embolism in case of hyperthyroidism who did not receive any prevention for hypercoagulability in the form of oral anticoagulants.

This case highlights the importance of preventive strategy for pulmonary embolism in case hyperthyroid patients as soon as it is diagnosed. Conversely it is important to consider hyperthyroidism as one of the cause of pulmonary embolism when all the conventional causes have been ruled out.

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