



Transient Pulmonary Hypertension in Patients with Graves' Disease



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INTRODUCTION

Hyperthyroidism produce changes in cardiac contractility, blood pressure, and systemic or pulmonary vascular resistance. In almost all cases these cardiovascular changes are reversible when the underlying thyroid disorder is recognized and treated. Pulmonary hypertension (PAH) has been associated with thyroid dysfunction, but primarily with hyperthyroidism. The vast majority of patients with this form of PAH are usually older with toxic multinodular goiter.

The aim of this study was to determine the clinical correlates of PAH in patients with Graves' disease (GD).

METHODS

Our study is retrospective one concerning patients with GD referred for echography before using any treatment. PAH has been diagnosis when pulmonary artery systolic pressure was elevated.

RESULTS

- Among **22** consecutive patients with **GD** referred for echocardiography, **6 patients** (27,32 %) had PAH measured by continuous-wave Doppler echocardiography (**pulmonary artery systolic pressure >35 mmHg**).
- The patients with PAH had significantly higher pulmonaryvascular resistance (PVR), cardiac output and thyroid-stimulating hormone receptor antibody (TRAb) compared to those without (Table 1-2).
- Pulmonary artery systolic pressure may had a good correlation with TRAb, but was not related to free T4.
- All this patients have a reversible pulmonary hypertension after treatment.

Table n= 1 Characteristics of patients with Graves' Disease

| Patients | Without PAH (n=16) | Patients with PAH (n=6) |
|---|--------------------|-------------------------|
| Pulmonary artery systolic pressure mmHg | 12 | 48 |
| TRAb (UI/L) | 4 | 11 |

Table n= 2 Affects of treatment in patients with GD and PAH

| Treatment (n=6) | Before | After treatment (euthyroidisme) |
|---|--------|---------------------------------|
| Pulmonary artery systolic pressure (average) mmHg | 48 | 14 |

DISCUSSION

- The exact reasons for the development of pulmonary hypertension in hyperthyroidism are unclear. Proposed mechanisms include high cardiac output-induced endothelial injury, increased metabolism of intrinsic pulmonary vasodilating substances resulting in elevated pulmonary vascular resistance, and autoimmune phenomenon [1].
- In addition to the effect of thyroid hormone on the cardiovascular system, autoimmune-mediated pulmonaryvascular remodeling may play a role in Graves' disease-linked elevated pulmonary artery systolic pressure.
- An autoimmune process inducing endothelial damage with GD may play a key role. Future studies should focus on discovering the immunogenetic overlap between autoimmune thyroid diseases and PAH.

CONCLUSION

- This study highlights the importance of considering hyperthyroidism as a cause of idiopathic PAH, and demonstrates the potential reversibility of its complications.
- At present, thyroid function tests should be considered in the investigation of all patients. Hyperthyroidism should be included in the causes of secondary pulmonary hypertension and/or otherwise unexplained right heart failure. This is especially important because hyperthyroidism is a treatable entity and its cardiac manifestations may be completely reversible.

References :

- 1- Lozano HF, Sharma CN, Cardiol Rev. **Reversible pulmonary hypertension, tricuspid regurgitation and right-sided heart failure associated with hyperthyroidism: case report and review of the literature** Cardiol Rev 2004 Nov-Dec;12(6):299-305.