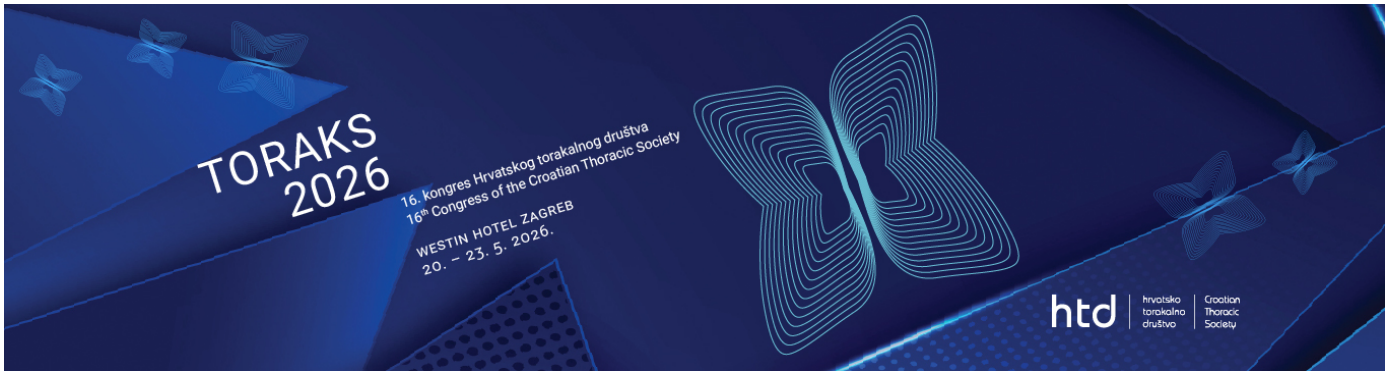


GRAVES' DISEASE MASKED AS PSEUDO-MEIGS' SYNDROME: AN UNUSUAL PRESENTATION OF PULMONARY HYPERTENSION

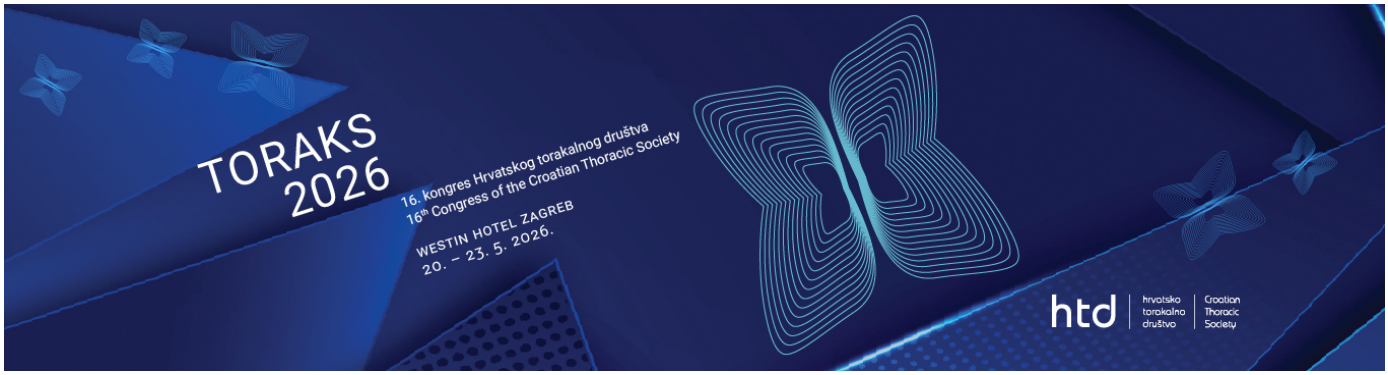
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Background:

Pulmonary hypertension (PH) is a complex pathophysiological condition defined by elevated mean pulmonary arterial pressure confirmed by right heart catheterization. According to the World Health Organization (WHO) classification, PH is most commonly associated with left heart disease (Group 2), chronic lung disease and/or hypoxia (Group 3), or chronic thromboembolic disease (Group 4). It may also appear as idiopathic pulmonary arterial hypertension or as a manifestation of connective tissue diseases and congenital heart defects (Group 1). However, PH may also arise from rare multifactorial conditions classified as WHO Group 5, including thyroid dysfunction. The mechanism involves increased cardiac output and decreased pulmonary vascular compliance.



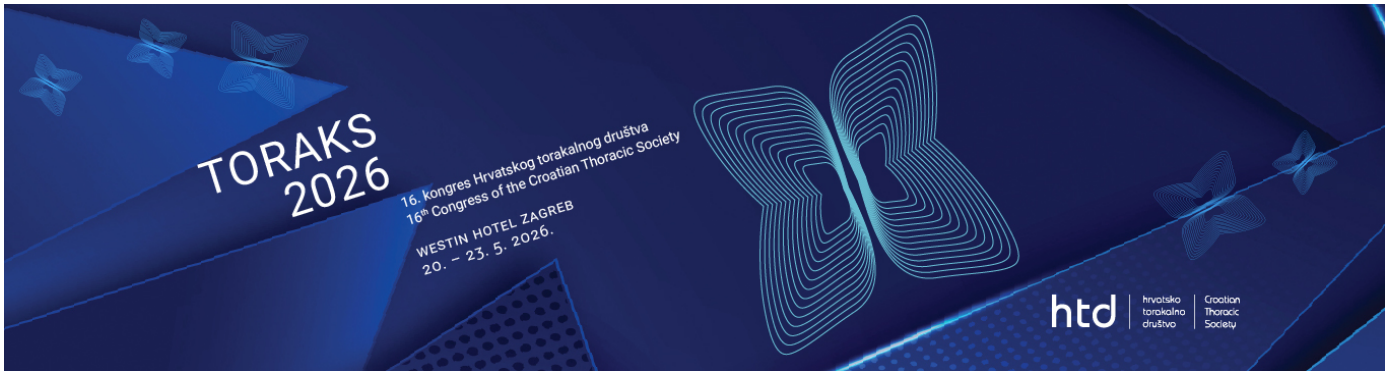
Conclusion:

This case highlights the critical importance of considering Graves' disease in patients with unexplained pulmonary hypertension, hemodynamic instability, and hyperkinetic circulation. Although the initial presentation strongly suggested a gynecological etiology, further evaluation revealed a systemic endocrine cause, emphasizing the complex connection between thyroid function and cardiopulmonary stability.

Case:

A 48-year-old woman presented with a two-month history of significant weight loss, palpitations, and progressive abdominal distension. Clinical imaging revealed massive ascites and bilateral adnexal masses, raising high suspicion of advanced ovarian malignancy. At an outside institution, a diagnostic laparoscopy was converted to a median laparotomy with total hysterectomy, bilateral adnexectomy, and omentectomy. However, definitive histopathological analysis revealed a benign ovarian tumor.

The postoperative course was severely complicated by hemorrhagic shock and consumptive coagulopathy, requiring urgent ligation of the right internal uterine artery. Despite intensive surgical and fluid intervention, the patient remained hemodynamically unstable, with worsening right heart failure and newly diagnosed PH, necessitating high-dose norepinephrine support.



Clinical Note: High doses of norepinephrine may increase pulmonary vascular resistance through alpha-adrenergic stimulation, potentially exacerbating right ventricular strain in the setting of thyrotoxicosis-induced hyperkinetic circulation. This interaction often masks the underlying endocrine trigger by mimicking primary cardiogenic shock.

Following initial stabilization, the patient was transferred to the Department of Pulmonary Diseases Jordanovac for specialized evaluation. Transthoracic echocardiography and right heart catheterization confirmed PH with mild mitral regurgitation in a distinctly hyperkinetic circulatory state. Comprehensive laboratory testing revealed an undetectable TSH (<0.01 mIU/L), significantly elevated free triiodothyronine (FT3) and free thyroxine (FT4), and positive TSH receptor antibodies (TRAb). Thyroid ultrasound demonstrated a diffuse goiter with increased vascularity, confirming previously undiagnosed Graves' disease.

Specific treatment with methimazole and propranolol resulted in rapid clinical improvement and the normalization of hemodynamic status. The patient was discharged in stable condition on beta-blockers, antithyroid therapy, and supportive care.