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When the thyroid threatens the heart; Cardiac tamponade as the initial presentation of severe hypothyroidism: A case report

Ivana Čičak[1]*, Tomislava Bodrožić-Džakić Poljak[2], Ivana Petrović Juren[3], Marin Viđak[2], Šime Manola[2], Fran Šaler[2]

[1]General Hospital "Dr. Josip Benčević", Slavonski Brod, Croatia
[2]Dubrava University Hospital, Zagreb, Croatia
[3]General Hospital "Dr Anđelko Višić" Bjelovar, Bjelovar, Croatia

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***ADDRESS FOR CORRESPONDENCE:** Ivana Čičak, Opća bolnica "Dr. Josip Benčević", A. Štampara 42, HR-35000 Slavonski Brod, Croatia. / Phone: +385992826177 / E-mail: ivanacicak07@gmail.com

ORCID: Ivana Čičak, https://orcid.org/0009-0006-2380-1355 • Tomislava Bodrožić-Džakić Poljak, https://orcid.org/0000-0002-7293-3972 Ivana Petrović Juren, https://orcid.org/0000-0002-2793-3455 • Marin Viđak, https://orcid.org/0000-0003-0341-9598 Šime Manola, https://orcid.org/0000-0001-6444-2674 • Fran Šaler, https://orcid.org/0000-0002-1428-3940

Introduction: Pericardial effusion is a well-recognized complication of profound hypothyroidism (with an incidence of 3%-37%).[1] Effusion occurs due to increased permeability of the epicardial blood vessels and reduced lymphatic drainage of albumin, leading to the accumulation of protein-rich fluid in the pericardial space.[2] However, rapidly progressive cardiac tamponade with hemodynamic collapse is highly uncommon, particularly in young patients without pre-existing chronic conditions, trauma, or underlying malignancies. This case report highlights a 19-year-old female whose critical, undiagnosed, endocrine emergency was initially presented with a life-threatening tamponade, masked by a concurrent severe respiratory infection.

Case report: 19-year-old female with no significant medical history was initially hospitalized at a regional hospital due to pleuropneumonia and a pleural empyema requiring urgent drainage. During her hospital stay, she experienced cardiovascular deterioration. Transthoracic echocardiography (TTE) revealed a circumferential pericardial effusion with signs of impending cardiac tamponade, including partial right atrial collapse, a distinct 'swinging heart' phenomenon, and significant respiratory variations in transmitral and transtricuspid flows. An emergency apical pericardiocentesis was performed, evacuating 600 mL of serous fluid that transitioned from bright yellow to a reddish hue. Comprehensive cytological, biochemical, rheumatologic, and tumor marker analyses classified the fluid as a transudate, effectively ruling out primary infectious, malignant, or systemic autoimmune etiologies. Subsequent laboratory evaluation to determine the etiology of the massive transudative effusion revealed severe, advanced primary hypothyroidism. The thyroid stimulating hormone (TSH) levels were markedly elevated at 168 μIU/mL (ref.values between 0.4 and 4.0 mIU/L (or μU/mL), accompanied by undetectable free T3 and T4 levels, alongside positive anti-TPO antibodies. These findings established a definitive diagnosis of Hashimoto's thyroiditis. Following fluid evacuation, immediate hemodynamic stability was achieved. In coordination with an endocrinologist, tailored hormone replacement therapy with levothyroxine was initiated, combined with a targeted anti-inflammatory therapy consisting of ibuprofen and colchicine. A predischarge echocardiogram demonstrated near-complete resolution of the effusion, revealing only a minor residual collection of 8 mm along the posterior wall with minimal trace fibrin. The patient was discharged after two days, and follow-up visit after three weeks displayed further regression of pericardial effusion with improvements in symptoms. Six weeks follow up revealed no pericardial effusion.

Conclusion: This case underscores the necessity of a broad diagnostic mindset when managing massive pericardial effusions. Confounding intrathoracic inflammatory processes can easily mask the primary etiology, making routine thyroid screening essential for all unexplained transudative effusions. Ultimately, combining timely pericardiocentesis with early thyroid hormone replacement is critical to achieve rapid hemodynamic stabilization and prevent clinical recurrence.

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