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Acute dyspnea following knee joint total endoprosthesis – a diagnostic surprise

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Abstract: Acute dyspnea, sometimes dramatic, is most often caused by cardiovascular or respiratory disease. However, although rare, thyroid dysfunction may present a similar clinical picture with equally serious and life-threatening consequences. Therefore, every occurrence of acute dyspnea represents a special medical challenge in diagnosis and treatment. We present a case of 81-year-old male admitted to the rehabilitation department five days after a knee joint total endoprosthesis and developed acute dyspnea two days later. An emergency diagnostic was performed and confirmed an airway stenosis through a previously undiagnosed enlarged thyroid gland. Although in most cases an acute life-threatening postoperative dyspnea indicates a cardiac or pulmonary problem, other diseases must be taken into consideration.

Keywords: Airway Obstruction; Diagnosis; Dyspnea; Goiter

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1. Introduction

Acute dyspnea, sometimes dramatic, is most often caused by cardiovascular or respiratory disease. However, although very rare, thyroid dysfunction may present a similar clinical picture with equally serious and life-threatening consequences. Therefore, every occurrence of acute dyspnea represents a special medical challenge in diagnosis and treatment.

2. Case presentation

An 81-year-old man was admitted to rehabilitation five days after a knee joint total endoprosthesis implantation. He was an obese patient and was being treated for hypertension, diabetes mellitus and hyperlipidemia, without any other diseases in his medical history.

Additionally, the patient had been prescribed rivaroxaban before surgery for thrombosis prophylaxis. Two days after admission to the ward, the patient complained of a sudden onset of dyspnea on minimal exertion. Physical examination revealed a blood pressure of 132/87 mm Hg with a regular pulse rate (88/min). His respiratory rate was 24 breaths/min with normal oxygen saturation (97%) in room air and a temperature of 36.7 °C. The remaining physical examination was unremarkable. Routine blood tests showed a hemoglobin level of 10.8 mg/dL (normal range 12.7-16.3 mg/dL), leukocyte count of 8.5 giga/L (normal range: 2.6-7.8 giga/L), platelet count of 554 giga/L (normal range: 130-330 giga/L), sodium level of 140 mmol/L (normal range: 136-145 mmol/L), potassium level of 4.4 mmol/L (normal range: 3.4-4.5 mmol/L), creatinine level of 96 μmol/L (normal range: 59-104 μmol/L), glomerular filtration rate of 61 ml/min/1.73 m^2, aspartate aminotransferase level of 27 U/L (normal range: <35 U/L), alanine aminotransferase level of 22 U/L (normal range: 10-35 U/L), and C-reactive protein level of 17 mg/L (normal value <5 mg/L). Arterial blood gas analysis was unremarkable with a pO2 of 11.3 kPa (normal range: 11.07-14.4 kPa), pCO2 of 4.2 kPa (normal range: 4.27-6.00 kPa), and O2 saturation of 97%. Troponin I levels were within the normal range, as well as N-terminal pro-brain natriuretic peptide (NT-pro BNP). The D-dimer was 570 ng/ml (normal range <500 ng/ml).

An electrocardiogram showed first-degree AV block, as well as one ventricular extrasystole. The initial radiological findings of the conventional chest radiograph indicated no major abnormalities, only mild signs of pulmonary emphysema. Transthoracic echocardiography revealed an enlarged left ventricle with increased pulmonary pressure to 41 mmHg. Ultrasound of the thyroid gland showed an extremely enlarged thyroid gland with inhomogeneous glandular parenchyma and multiple nodules. A computed tomography angiography scan was performed to rule out pulmonary embolism. While it was negative for pulmonary embolism, it revealed a massive goiter with gross enlargement of the entire, especially the right lobe of the thyroid gland, causing severe narrowing of the trachea (Figure 1).

To assess thyroid function, additional laboratory tests were performed. They showed a thyroid stimulating hormone (TSH) level of <0.002 mU/L (normal range: 0.27-4.20 mU/L),

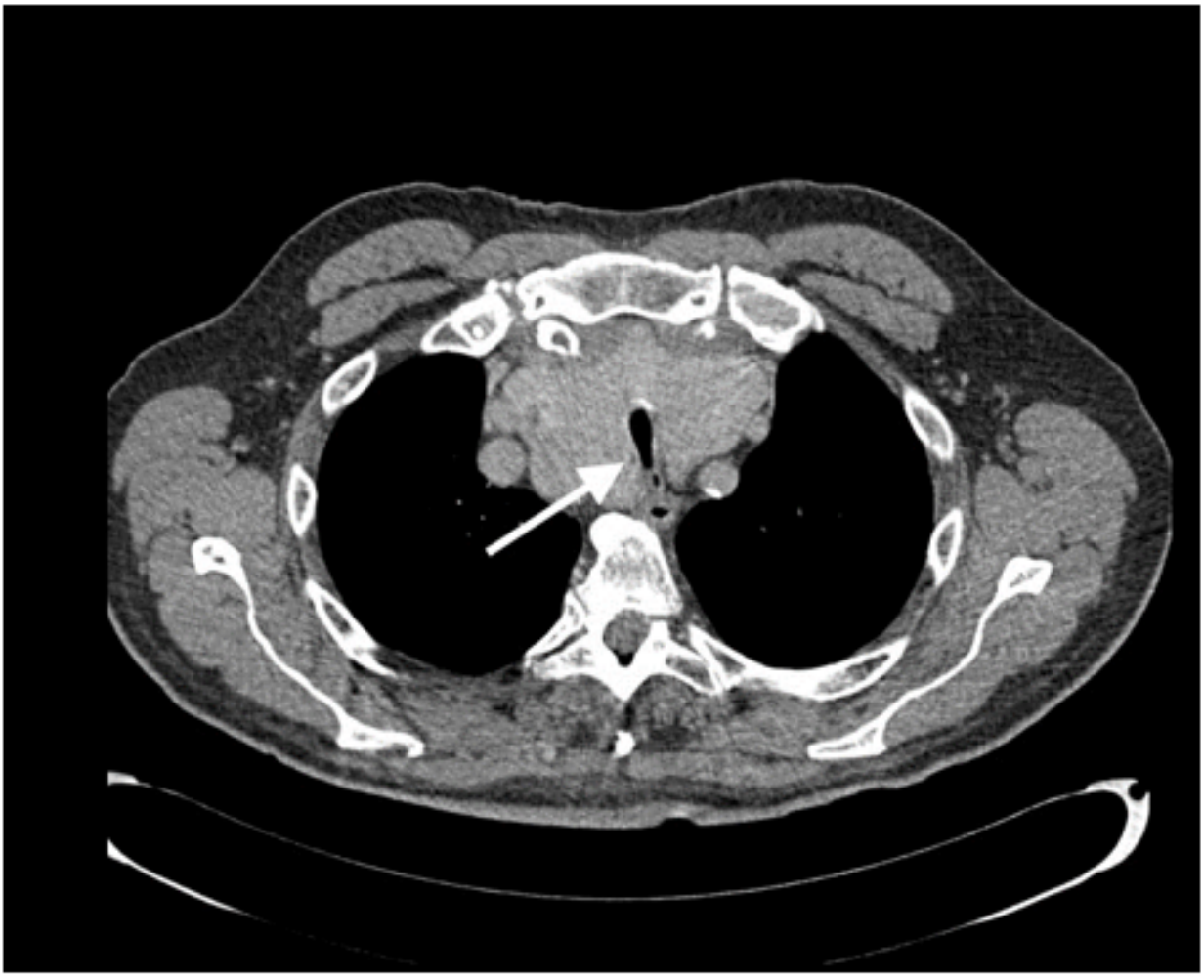

Figure 1 Arrow: goiter with severe narrowing of the trachea

a free triiodothyronine (fT3) level of 4.6 pmol/L (normal range: 3.1-6.8 pmol/L), and a free thyroxine (fT4) level of 22.5 pmol/L (normal range: 12-22 pmol/L). Antibodies to thyroperoxidase (anti-TPO) were 10 U/ml (normal range <80 U/ml) and there was an elevated thyroglobulin level of 141 μg/ml (normal range <75 μg/ml). The diagnosis of high-grade tracheal stenosis due to a newly diagnosed goiter and hyperthyroidism was established. The patient was transferred to the surgical department for further treatment. A total thyroidectomy was performed with an uncomplicated course. The patient was able to continue the interrupted rehabilitation program after ten days.

Under hormonal substitution therapy, the patient was asymptomatic thereafter and pulmonary hypertension decreased.

3. Discussion

Postoperative dyspnea, sometimes presenting dramatically, is usually attributed to cardiovascular or respiratory disease. However, thyroid dysfunction may occasionally manifest a similar clinical picture. The thyroid gland exerts various effects on the heart, vasculature, and respiratory system. Although the precise pathophysiological link between hyperthyroidism and pulmonary hypertension remains unclear, increased pulmonary vascular resistance and endothelial damage may lead to pulmonary hypertension and dyspnea (1,2).

Mild progressive dyspnea during daily activities is a common manifestation in patients with thyroid dysfunction, especially hyperthyroidism (3). Upper airway obstruction due to an enlarged thyroid gland has been reported in up to one-third of patients, presenting as a gradual airway obstruction over years. Typically, it manifests with dyspnea, stridor, or respiratory infections and is often mis-diagnosed as asthma (4). Unfortunately, the slow growth of goiter often goes unnoticed. Local findings during inspection and palpation can sometimes raise suspicion of thyroid enlargement. However, the growth of the thyroid gland in the retrosternal part usually remains unnoticed until goiter complications arise. Obesity in patients, often accompanied by fat deposition in the neck area, significantly complicates clinical ex-

amination and suspicion of thyroid enlargement, as was evident in this case. While thyroid ultrasound provides valuable information about the thyroid gland's size, and laboratory hormone analysis indicates hypo- or hyperfunction of the thyroid gland, these tests are not routinely performed in every individual but only when a thyroid disorder is suspected. CT scans are recommended for a quick, easily accessible, and valuable examination, offering insights into the thyroid gland's size and its relationship with other structures. CT scans enable the desired assessment and planning of further procedures. Kadhim et al. reported that a goiter that has grown slowly over years can also lead to acute deterioration with life-threatening airway obstruction (5). Regular monitoring and medication adjustments, or planned surgical inter-vention, can prevent such situations. Considering that benign goiter grows slowly, regular moni-toring of the goiter's size should prevent the complication of acute tracheal obstruction. An increase in the goiter's size and its retrosternal growth direction simultaneously increases the risk of acute airway obstruction. However, certain conditions, such as bleeding or acute respiratory infections of the airways leading to tracheal mucosa edema and secretion retention, can also increase the risk of acute obstruction. This implies that individuals with a known goiter require more intensive moni-toring, even during milder respiratory infections. The airway's mucous membrane reacts similarly in smokers, exposing them to a higher risk of developing acute airway obstruction if diagnosed with thyroid goiter, especially in combination with an acute respiratory infection. Asymptomatic thyroid goiters leading to a gradual tracheal narrowing, especially if accompanied by normal hor-monal levels, indicate elective surgery.

Conservative drug treatment in slow-growing goiters with hyperthyroidism, as in our case, can contribute to reducing goiter mass. However, acute obstruc-tion causing significant symptoms and potentially endangering the patient's life remains a primary indication for urgent surgery. Acute dyspnea due to airway obstruction without local signs of thyroid enlargement in previously undiagnosed goiters remains a rarity (6). Such a clinical presenta-tion is rare, occurring in only up to 0.6% of cases (7). Therefore, this initial manifestation of thyroid dysfunction in the postoperative course may lead to a false diagnosis and potentially a lethal event (8). In such situations, rapid diagnosis and early surgery are crucial to prevent respiratory failure and a potentially lethal outcome. Surgical intervention is associated with increased complications, including heightened hemorrhage, tracheal injury, laryngeal recurrent nerve injury, pneumothorax, and tracheomalacia, but it remains the preferred choice of treatment (9,10).

4. Conclusion

Although in most cases, postoperative acute dyspnea indicates a cardiac or pulmonary problem, other causes such as thyroid dysfunction with a large goiter (as an initial manifestation or pre-existing condition) have to be taken into consideration, and a differential diagnosis is essential.

5. Declarations

5.1. Acknowledgement

None.

5.2. Authors' contribution

The authors meet all criteria for authorship based on the recommendations of the International Committee of Medical Journal Editors (ICMJE).

5.3. Conflict of interest

None.

5.4. Funding

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References

1. Sugiura T, Yamanaka S, Takeuchi H, Morimoto N, Kamioka M, Matsumura Y. Autoimmunity and pulmonary hypertension in patients with Graves' disease. Heart Vessels. 2015;30(5):642-6.
2. Mathot V, Oosterwerff E, van Pampus MG, Riezebos R. Pulmonary hypertension in a pregnant patient with thyrotoxicosis due to Graves' disease: considerations with respect to treatment. BMJ Case Rep. 2014, bcr2013201916.
3. Zuhur SS, Baykiz D, Kara SP, Sahin E, Kuzu I, Elbuken G. Relationship among pulmonary hypertension, autoimmunity, thyroid hormones and dyspnea in patients with hyperthyroidism. Am J Med Sci. 2017;353(4):374-80.
4. Chen DC, Williams DM, Aronowitz PB. Long-standing goiter causing acute airway compromise. J Gen Intern Med. 2016;31(12):1539-40.
5. Kadhim AL, Sheahan P, Timon C. Management of life-threatening airway obstruction caused by benign thyroid disease. J Laryngol Otol. 2006;120(12):1038-41.
6. Aghajanzadeh M, Asgary MR, Mohammadi F, Darvishi H, Safarpour Y. An investigation into symptoms, diagnosis, treatment, and treatment complications in patients with retrosternal goiter. J Family Med Prim Care. 2018;7(1):224-9.
7. Dias T, Santos A, Mesquita C, Santos RM. Acute airway obstruction due to benign multinodular goitre. BMJ Case Rep. 2019;12(4):e228095.
8. Dell'Aquila M, De Matteis A, Bolino G, Urciuoli P, Fineschi V, Maiese A. Death due to external compression of the trachea in a patient with multinodular hemorrhagic goiter. Forensic Sci Med Pathol. 2019;15(3):509-12.
9. Ito T, Shingu K, Maeda C, Kitazawa M, Mizukami Y, Hiraguri M, et al. Acute airway obstruction due to benign

asymptomatic nodular goiter in the cervical region: a case report. Oncol Lett. 2015;10(3):1453-5.
10. Bayhan Z, Zeren S, Ucar BI, Ozbay I, Sonmez Y, Mestan M, et al. Emergency thyroidectomy: due to acute respiratory failure. Int J Surg Case Rep. 2014;5(12):1251-3.