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Letter to the Editor

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Letter to the Editor from Martin-Grace and Crowley: "Myxedema Heart and Pseudotamponade"

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Abbreviations: AI, adrenal insufficiency; fT4, free thyroxine; TSH, thyrotropin.

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We read with interest the recently published case series of pericardial effusions caused by severe hypothyroidism [1], which highlighted the relative hemodynamic stability of these patients despite echocardiographic features of cardiac tamponade. We have previously reported a case of cardiac tamponade in the setting of central hypothyroidism [2], which we believe highlights an additional aspect of this rare presentation.

A 53-year-old woman with a 20-year history of secondary amenorrhea was diagnosed with a moderate pericardial effusion (maximum depth, 20 mm) with no evidence of tamponade on echocardiography. Serum thyrotropin (TSH) was within the reference range, but free thyroxine (fT4) was not measured on initial presentation. She was treated conservatively, but was readmitted 2 weeks later, hypotensive and tachycardic. An echocardiogram showed minimal change in effusion volume but demonstrated new right atrial diastolic collapse. In view both of the clinical and echocardiographic signs of tamponade, an emergency pericardial drain was inserted and 1100 mL of straw-colored pericardial fluid was removed. Repeat thyroid function tests and cortisol measurements prompted by the presence of new hyponatremia demonstrated central hypothyroidism (TSH 0.73 mIU/L [range, 0.27-4.4 mIU/L], fT4 6.3 pmol/L [range, 12-22 pmol/L], 3,5,3′-triiodothyronine 0.44 nmol/L [range, 1.3-3.1 nmol/L]), and an inappropriately low random cortisol (43 nmol/L).

The patient was subsequently diagnosed with panhypopituitarism with an empty sella and commenced first on hydrocortisone and then levothyroxine replacement therapy. She had no history suggestive of previous pituitary apoplexy. Previous estradiol, luteinizing hormone and follicle-stimulating hormone results were consistent with hypogonadotrophic hypogonadism, suggesting a prolonged period of pituitary dysfunction prior to this presentation. Serial echocardiograms demonstrated complete resolution without recurrence once appropriate hormone replacement therapy was commenced.

Our case highlights the importance of considering concurrent adrenal insufficiency (AI) in patients with hypothyroidism-related pericardial effusions. AI can coexist in the setting both of primary and central hypothyroidism. It is imperative that, when this occurs, glucocorticoid replacement be commenced before thyroxine replacement is given. If AI cannot be excluded in the acute setting, empiric glucocorticoid replacement should be commenced without delay [3]. Failure to do so risks precipitating an adrenal crisis [4]. This is of particular importance in the setting of a large pericardial effusion whereby the hypotensive effect of an adrenal crisis could provoke a "low-pressure tamponade." This situation can arise when a reduction in cardiac filling pressure results in tamponade at a relatively low or previously tolerated pericardial

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pressure [5]. It is possible that an adrenal crisis precipitated the rapid progression to tamponade in our case, which may also explain the patient's tachycardia, a feature not typical of hypothyroidism-related pericardial effusions [1].

In conclusion, whether a large pericardial effusion is caused by primary or secondary hypothyroidism, the management is the same. Concurrent AI is rare but has important implications for the safe initiation of levothyroxine replacement. Irrespective of whether AI occurs in the setting of primary or central hypothyroidism, the principles of glucocorticoid replacement remain the same. We echo the authors' and European Thyroid Association's guidance that empiric stress-dose glucocorticoid replacement be considered if it is not possible to exclude AI prior to commencing levothyroxine replacement [3].

Additional Information

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References

1. Baldwin C, Newman JD, Vallejo F, Peck V, Greene LW, Goldberg IJ. Myxedema heart and pseudotamponade. *J Endocr Soc*. 2021;**5**(1):bvaa125.
2. Martin-Grace J, Ahmed M, Mulvihill N, Feeney ER, Crowley RK. Getting to the heart of hypopituitarism. *Clin Med (Lond)*. 2017;**17**(2):140-142.
3. Persani L, Brabant G, Dattani M, et al. 2018 European Thyroid Association (ETA) guidelines on the diagnosis and management of central hypothyroidism. *Eur Thyroid J*. 2018;**7**(5):225-237.
4. Fleseriu M, Hashim IA, Karavitaki N, et al. Hormonal replacement in hypopituitarism in adults: an Endocrine Society clinical practice guideline. *J Clin Endocrinol Metab*. 2016;**101**(11):3888-3921.
5. Sagristà-Sauleda J, Angel J, Sambola A, Alguersuari J, Permanyer-Miralda G, Soler-Soler J. Low-pressure cardiac tamponade: clinical and hemodynamic profile. *Circulation*. 2006;**114**(9):945-952.