First and only manifestation of Hashimoto's disease: pericardial tamponade

Hashimoto hastalığının ilk ve tek bulgusu: Perikart tamponadı

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Summary– Pericardial effusion in hypothyroidism is a common finding, but an effusion which causes cardiac tamponade is a rarity. A 39-year-old man without previous medical history presented with progressive shortness of breath. Transthoracic echocardiography revealed massive pericardial effusion with tamponade findings. Thyroid function analysis showed raised thyroid-stimulating hormone associated with a severe decrease of free thyroxine and triiodothyronine. Antithyroglobulin and antithyroperoxidase antibodies were significantly high. The echocardiography-guided pericardiocentesis was followed by thyroid replacement therapy with the diagnosis of Hashimoto's disease. We report a case of pericardial tamponade as the first and only manifestation of Hashimoto's disease.

Özet– Perikart efüzyonu hipotiroidizmde sık görülmesine rağmen nadiren kalp tamponadına neden olmaktadır. Daha önceden sağlıklı olan 39 yaşındaki erkek hasta ilerleyici nefes darlığı yakınması ile başvurdu. Transtorasik ekokardiyografide tamponada neden olan masif perikart efüzyonu saptandı. Tiroit fonksiyon testlerinden tiroit stimüle edici hormonun düşük, serbest tiroksin ve triiyodotironin yüksek olduğu saptandı. Antitiroglobulin ve antiperoksidaz antikorları anlamlı derecede yüksekti. Ekokardiyografi kılavuzluğunda apikal yaklaşımla perikardiyosentez işlemi uygulanan hastaya Haşimato hastalığı tanısı konularak tiroit replasman tedavisine başlandı. Bu yazıda, Hashimoto hastalığında ilk ve tek bulgu veren perikart tamponatlı olgu sunuldu.

Pericardial effusion (PE), occurring in about one-third of patients with hypothyroidism, usually improves slowly and does not cause tamponade.[1]

In this report, we present a case of pericardial tamponade as the initial and only manifestation of Hashimoto's disease.

CASE REPORT

A 39-year-old man without previous medical history presented with progressive shortness of breath for a month. The initial physical examination showed jugular venous distention, and distant and muffled heart sounds. Blood pressure and heart rate were 100/50 mmHg and 47 bpm respectively. Respiratory and other system examinations were normal. Electrocardiography (ECG) revealed sinus bradycardia with low voltage QRS complexes in all leads (Fig. 1a). The chest X-ray showed radiographic evidence of significant cardiomegaly. Transthoracic echocardiography (TTE) revealed a large PE with a diastolic collapse of the right heart chambers and a significant respiratory variation of mitral inflow velocity (Fig. 2). The ejection fraction (EF) was 60%. Blood tests were normal except creatine kinase (CK) and thyroid hormones. Serum CK level was 3026 U/L (normal range 33-211 U/L). Thyroid hormone profile revealed

Abbreviations:

CK	*Creatine kinase*
ECG	*Electrocardiography*
T_3	*Triiodothyronine*
T_4	*Thyroxine*
PE	*Pericardial effusion*
TG	*Thyroglobulin*
TPO	*Serum thyroperoxidase*
TSH	*Thyroid-stimulating hormone*
TTE	*Transthoracic echocardiography*

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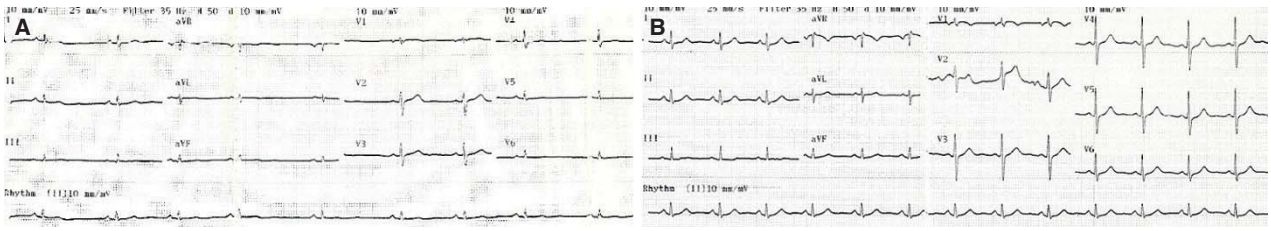

Figure 1. **(A)** Initial electrocardiogram showed sinus bradycardia with low voltage QRS complexes. **(B)** Control electrocardiography showing sinus rhythm with a normal axis.

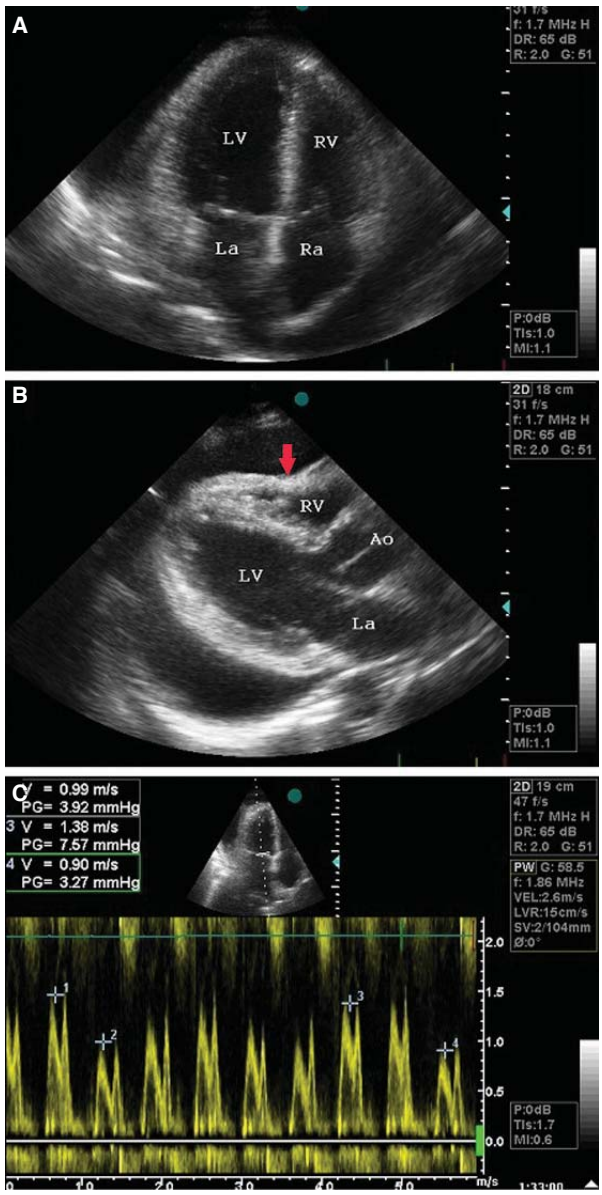

Figure 2. Two-dimensional transthoracic echocardiographic demonstration: **(A)** Massive pericardial effusion from apical 4-chambers view. **(B)** Right ventricular diastolic collapse from parasternal long-axis view (arrow). **(C)** Continuous Wave Doppler revealed significant (37%) respiratory variation of mitral inflow.

reduced free T_3 and T_4 and raised thyroid-stimulating hormone (TSH). Serum thyroperoxidase (TPO) and thyroglobulin (TG) antibody levels were significantly high (Table 1). On the course, we performed thoracoabdominal computed tomography to identify any possible etiology of PE and it did not reveal any abnormal findings which could explain the etiology of effusion. Eight hundred milliliters of straw colored PE was withdrawn by echocardiography-guided pericardiocentesis with an apical approach and the removed fluid was examined for bacterial, cytological and chemical analysis. Cytological analysis revealed that there were mesothelial cells with neither inflammatory nor malignant cells. Bacterial culture was negative. Total protein was 6.9 g/dl. Under the diagnosis of hypothyroidism due to Hashimoto's thyroiditis, replacement therapy with levothyroxin was started. After two months of treatment, CK, free thyroxine (FT4), TSH and anti-TG antibody levels returned to normal values (Table 1). A control ECG showed sinus rhythm with normal QRS voltage (Fig. 1b) and TTE revealed no residual PE (Fig. 3).

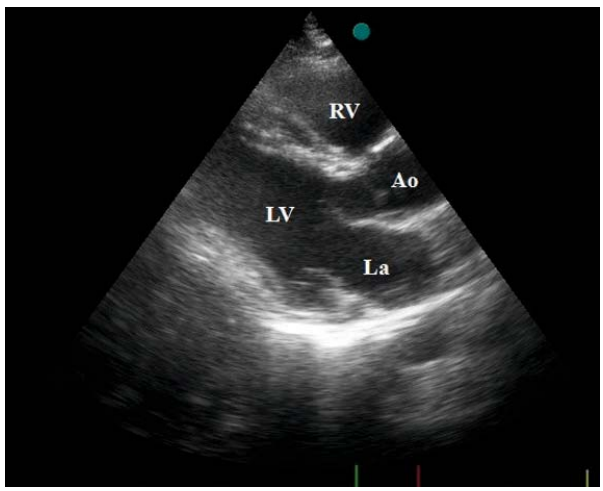

Figure 3. Control transthoracic echocardiography showing no residual pericardial effusion.

Table 1. Labaratory findings of the patient

	Initial	Control*	References
Creatine kinase (U/L)	3026	150	33-211
Free triiodothyronine (pg/mL)	0.87	5.41	2.3-4.5
Free thyroxine (ng/dL)	0.25	1.26	0.74-2
Thyroid-stimulating hormone (μIU/mL)	>150	0.66	0.55-4.78
Anti-TG antibodies (IU/mL)	30.96	2.1	0.9-4
Anti-TPO antibodies (IU/mL)	>969	>969	0-9

*After two months. TG: Thyroglobulin; TPO: Serum thyroperoxidase.

DISCUSSION

Hashimoto's disease is an autoimmune thyroid gland disorder, characterized by the production of antibodies in response to thyroid antigens (antibody producing) and the replacement of normal thyroid structures with lymphocytes and lymphoid germinal centers.[2] It is most commonly diagnosed after the age of 40, when patients usually become hypothyroidic.

PE in hypothyroidism is common and the mechanisms of myxedematous PE are increased permeability of capillaries with subsequent leakage of protein rich fluid into the interstitial space, impaired lymphatic drainage and salt and water retention.[3-5] But an effusion which causes cardiac tamponade is rarely seen. The rarity of cardiac tamponade in hypothyroid patients with PE is attributed to the slow accumulation of fluid and the remarkable distensibility of the pericardium. Therefore pericardiocentesis is usually unnecessary in these patients.[6-10] Also, myxedematous PEs tend to regress slowly and ultimately disappear over a period of several months after patients have returned to the euthyroid state.[11] Although emergent pericardiocentesis should be performed in clinical cardiac tamponade, patients with echocardiographic tamponade signs without a paradoxical pulse should be treated with thyroxine initially. For patients diagnosed with cardiac tamponade without sinus tachycardia, hypothyroidism should be highly suspected.[12] In our case, initial TTE revealed pericardial tamponade, which was the first and only manifestation of hypothyroidism due to Hashimato's disease. The accompanying bradycardia interestingly obscured the clinical findings of tamponade such as tachycardia.

Our objective of publishing this case is to point out the possibility of encountering patients who have pericardial tamponade as the first manifestation of hypothyroidism. However, unlike the usual benign course of PE in these patients, an effusion causing tamponade and resisting to medical therapy is a clear indication for pericardial drainage as in our case.

Conflict-of-interest issues regarding the authorship or article: None declared

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Key words: Cardiac tamponade/etiology; electrocardiography; Hashimoto disease/complications/diagnosis; pericardial effusion.

Anahtar sözcükler: Kalp tamponadı/etyoloji; elektrokardiyografi; Hashimoto hastalığı/komplikasyonlar/tanı; perikart efüzyonu.