A Rare Case of Amiodarone-Induced Hypothyroidism Presenting with Cardiac Tamponade

Ali Motabar, M.D.,¹ Ramtin Anousheh, M.D., M.P.H.,¹ Rimon Shaker, M.D.,¹ and Ramdas G. Pai, M.D.¹

ABSTRACT

Subclinical pericardial effusions are common in patients with untreated hypothyroidism and usually resolve with thyroid replacement therapy, but cardiac tamponade is a rare presentation of prolonged untreated hypothyroidism. We report the first case of cardiac tamponade due to hypothyroidism produced by administration of amiodarone.

KEYWORDS: Cardiac tamponade, hypothyroidism, amiodarone therapy

Accumulation of proteinaceous fluid in vital body cavities of patients with hypothyroidism is widely documented. The most common sites for fluid accumulation are the pericardial, pleural, and peritoneal cavities.¹ The reported incidence of pericardial effusion in hypothyroid patients is ~3% in an early mild stages to 80% when myxedema is present,^{2,3} but cardiac tamponade is very rare and happens in prolonged untreated hypothyroidism.⁴ Hypothyroidism is common in patients receiving amiodarone, but cardiac tamponade secondary to this has not been reported to our knowledge.⁵⁻⁷ We report the first case of cardiac tamponade caused by hypothyroidism produced by administration of amiodarone.

CASE REPORT

A 69-year-old Caucasian man with medical history of hypertensive cardiomyopathy, colon cancer, and atrial fibrillation presented with progressive dyspnea on exertion and fatigue over a period of weeks. He was on long-term amiodarone therapy; 200 mg twice a day for several

years. Physical examination revealed blood pressure of 95/54 mm Hg and heart rate of 67 beats per minute with distended jugular veins and distant heart sounds.

The chest X-ray revealed massive cardiomegaly (Fig. 1A). Chest computed tomography scan performed to evaluate the metastatic cancer revealed a large pericardial effusion (Fig. 1B). Echocardiography confirmed the diagnosis of massive pericardial effusion with tamponade physiology consisting of markedly dilated inferior vena cava and respirophasic variations in transvalvular flows (Fig. 1C). The clinical suspicion of hypothyroidism was supported by an elevated thyroid stimulating hormone level of 77.7 μ IU/mL (normal 0.4 to 4.0 μ IU/mL) and decreased plasma-free thyroxine level of 0.62 ng/dL (normal 0.8 to 2.0 ng/dL).

The patient underwent emergent echocardiographically guided pericardiocentesis with drainage of 1.4 L of straw-colored fluid. The patient had marked improvement in symptoms and blood pressure with pericardiocentesis. The protein content of the fluid was 2.5 g/dL and cytology was negative for malignant cells. Follow-up chest X-ray showed a dramatic reduction in

¹Department of Cardiology, Loma Linda University Medical Center, Loma Linda, CA.

Address for correspondence and reprint requests: Ramdas G. Pai, M.D., Professor of Medicine, Division of Cardiology, Loma Linda University Medical Center, Loma Linda, CA 92354 (e-mail: Rpai@llu.edu).

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Figure 1 (A) Chest X-ray on admission showing prominent cardiomegaly. (B) Chest CT scan revealed massive pericardial effusion (PE) with no evidence of neoplastic pericardial involvement. (C) Echocardiography confirmed presence of large pericardial effusion. LV, left ventricle; PE, pericardial effusion; RV, right ventricle.

cardiac size and echocardiographic images showed absence of pericardial fluid as presented in Fig. 2. The patient was treated with thyroxine replacement with no recurrence of pericardial effusion in 12-month follow-up.

DISCUSSION

Cardiac tamponade is a rare presentation of hypothyroidism.⁴ The extravasations of mucopolysaccharides into the pericardial space along with increased capillary

Figure 2 (A) Chest X-ray after pericardiocentesis consistent with resolution of pericardial effusion. (B) Echocardiography confirmed resolution of pericardial effusion. LA, left atrium, LV, left ventricle; RA, right atrium; RV, right ventricle.

permeability, decreased lymphatic drainage, and increased retention of salt and water are postulated mechanisms of pericardial effusions in hypothyroid patients.³ Hypothyroidism should be in differential diagnosis of patients with asymptomatic moderate- to massive pericardial effusions and usually does not require pericardiocentesis and responds to hormone replacement therapy.

Patients with cardiac tamponade usually present with tachycardia; absence of tachycardia despite hypotension was a clinical clue to hypothyroid etiology in our patient.⁵ The pericardial fluid in our patient was a transudate which is consistent with hypothyroidism and was negative for any malignancy or infection.

Amiodarone-induced thyroid dysfunction is a common complication of amiodarone therapy and its prevalence varies according to dose and duration of therapy.⁵⁻⁷ The reported incidence of hypothyroidism in patients taking amiodarone ranges from 5 to 25%.^{7,8} However, presence of secondary pericardial effusion in amiodarone treatment is unusual and tamponade has not been reported to our knowledge. We have reported this case because of its rarity, classic tamponade with bradycardia, and to alert the clini-

cians that cardiac tamponade is possible with amiodarone.

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