PERICARDIAL EFFUSION IN A PATIENT WITH HYPERTHYROIDISM: A CASE REPORT

M.O. Fabowale[1], O.S. Ogah[1,2,3], A.M. Kehinde[1], F.I. Olusola[1], I.J. Okafor[1], T.A. Bakare[1], V. Obasuyi[1], O.O. Orimolade[1], A. Aje[1], and A. Adebiyi[1,2]

1. Cardiology Unit, Department of Medicine, University College Hospital, Ibadan, Nigeria.
2. Cardiology Unit, Department of Medicine, University of Ibadan, Ibadan, Nigeria.
3. Institute of Advanced Medical Research and Training, College of Medicine, University of Ibadan, Nigeria.

Correspondence:
Dr. O.S. Ogah
Cardiology Unit,
Department of Medicine,
University College Hospital,
Ibadan,
E-mail: osogah56156@gmail.com

ABSTRACT

Pericarditis and pericardial effusion are commonly associated with hypothyroidism. It is an uncommon association with hyperthyroidism. We present a case of pericarditis/pericardial effusion in a 28-year-old Nigerian lady with hyperthyroidism. There was resolution of the pericardial effusion with antithyroid medications and steroid therapy. We recommend a high index of suspicion of this association in patients with hyperthyroidism and/or Graves' disease.

Keyword: Thyroid disease, Hyperthyroidism, Pericarditis, Pericardial effusion

INTRODUCTION

Hyperthyroidism is a syndrome associated with excess thyroid hormone production. Patients typically present with irritability, unintentional weight loss, heat intolerance, malaise, diaphoresis, gastrointestinal hypermotility and diarrhoea. Cardiovascular complications of hyperthyroidism include high or normal output heart failure, arrhythmias and tachycardia-associated cardiomyopathy.[1, 2]

Pericardial effusion is rare in hyperthyroidism when compared to hypothyroidism. Autoimmune pericarditis has been reported in Graves' disease.[3]

Herein, we present the case of a 28-year-old woman who was admitted with features of hyperthyroidism and pericardial effusion, the effusion resolved following treatment of hyperthyroidism.

CASE REPORT

A 28-year-old lady presented to the Medical Outpatient Clinic of our hospital on account of a three-months history of recurrent palpitations, easy fatigability and exertional dyspnea. Palpitations occur most times of the day but worse at rest. There was history diaphoresis but no chest pain or pressure like sensations on the

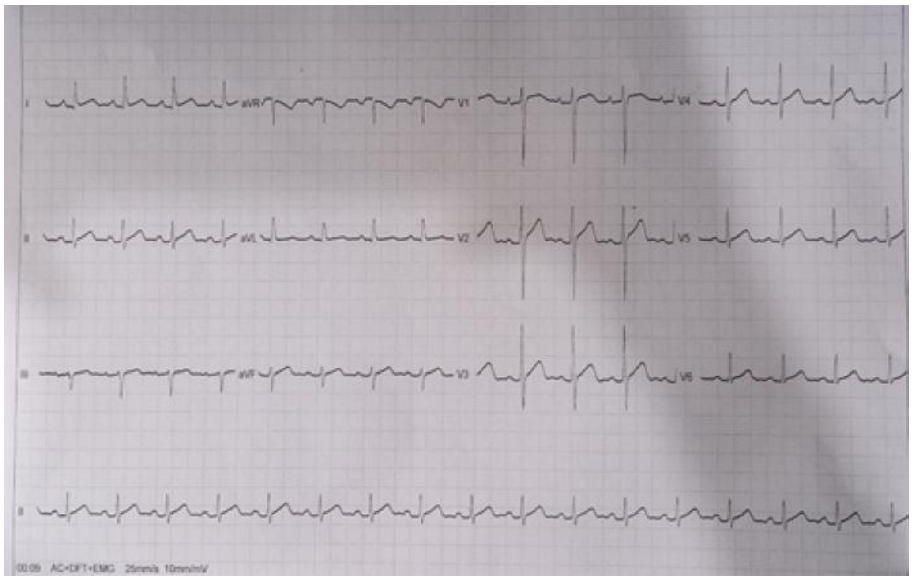
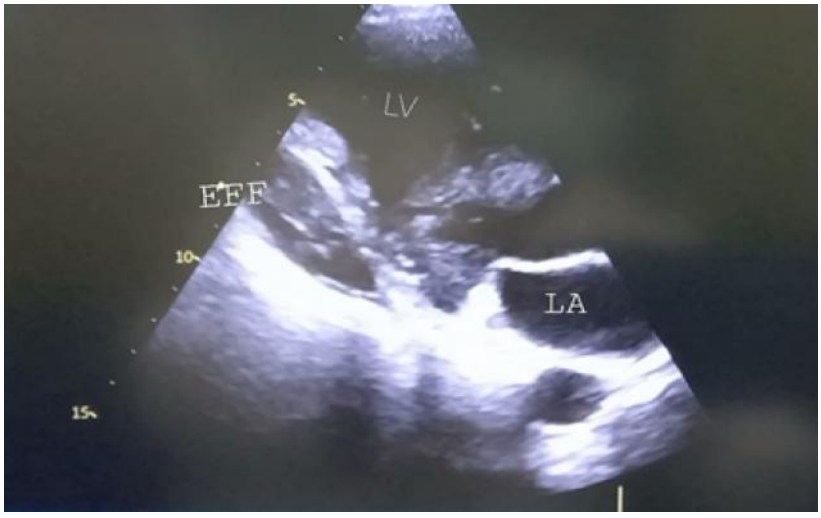


Figure 1: 12-lead ECG of the patient

Table 1: The laboratory panel of the patient.

Blood	Reference Range	In this hospital
Haemoglobin(g/dl)	12.0-16.0	10.9
White cell count (mm^3)	4,000-11,000	11,720
Neutrophils (%)	37-72	49.5
Lymphocytes (%)	20-50	39.2
Platelet count (/mm3)	150,000-400,000	313,000
Sodium (mmol/L)	135-145	137
Potassium (mmol/L)	3.5-5.0	3.4
Chloride (mmol/L)	95-110	97
Bicarbonate (mmol/L)	20-30	25
Urea (mg/dl)	15-45	16
Creatinine (mg/dl)	0.5-1.5	0.3
Thyroid Function Test		
TSH (mlU/L)	0.270-4.20	0.056
T4 (pmol/L)	12-22	96.8
T3 (pmol/L)	3.9-6.7	31.8
Urine		
Glucose		Negative
Protein		Negative
Blood		Negative
Bilirubin		Negative
Ketones		Negative
Urobilinogen		Normal
Nitrite		Negative
SG	1.010-1.035	1.015
pH	5.0-9.0	6.0

chest. No history of headaches, blurring of vision, feeling of impending doom or syncopal attacks. She however reported malaise, easy fatigability and dyspnoea on exertion which later progressed to dyspnoea even at rest. She denied any report of cough, paroxysmal nocturnal dyspnea, orthopnoea or recurrent pedal oedema. She had no preceding symptoms of upper respiratory tract infection or background anxiety disorder. She also has unintentional weight loss, early satiety and loss of appetite. There was associated recurrent effortless postprandial vomiting with vague abdominal pain and hyper-defecation. She reported tremors, heat intolerance and scanty menses. She did not have dysphagia or change in her voice.



EFF= Effusion, LA= left atrium, LV= Left ventricle

Figure 2: Two-dimensional echo showing the pericardial effusion with fibrinous materials.

At presentation, she was restless, diaphoretic and mildly dehydrated. She was not febrile, not pale, anicteric, not cyanosed and had no digital clubbing. She had no significant peripheral lymphadenopathy and no pedal edema. There was an anterior neck swelling measuring 4 x 6cm but more to the right side. The swelling was firm in consistency with irregular edges, non-tender and no differential warmth. There was no exophthalmos or lid lag. There were fine tremors. She was dyspneic, respiratory rate was 36 breaths per minutes, trachea was central and breath sound were vesicular bilaterally. Her pulse was 112/min and regular, BP 120/70mmHg, JVP was not elevated and apex beat was difficult to localize. First and Second Hearts sounds were heard with pericardial friction rub. She was conscious, alert, oriented in time place and person, pupils 3mm equal, round, bilaterally reactive to light, no cranial nerve deficit, no sign of meningeal irritation, normal muscle bulk, tone and reflexes.

She was admitted for evaluation. Imaging studies revealed a relatively normal chest with a normal mediastinal contour. Table 1 shows the investigation results. The ECG (Figure 1) showed sinus tachycardia. Echocardiography (Figure 2) revealed a pericardial effusion over the left ventricle with fibrinous strands in the pericardial cavity.

She was commenced on oral medications which includes prednisolone 40mg daily, propranolol 20mg b.d., rabeprazole 20mg daily and carbimazole 15mg b.d. She had made significant improvement on follow up clinic appointments.

DISCUSSION

Atrial fibrillation, thyrotoxic cardiomyopathy, pulmonary arterial hypertension, systemic hypertension and high or normal output heart failure are known cardiac complications of hyperthyroidism. The patient discussed had normal left ventricular function on echocardiography and ECG did not show any features of atrial fibrillation. An ongoing malignancy and infection especially tuberculous pericarditis was also considered but this was excluded by the absence of suggestive clinical picture and laboratory profile.

The possibility of pericardial effusion/pericarditis being a possible complication of hyperthyroidism was entertained after review of some case reports suggesting a rare relationship between pericardial disease and hyperthyroidism.[4] The earliest case to have been reported was in 1958. Koo *et al.*[3] reported a case of acute recurrent pericarditis that was presumed to be associated with Graves' disease based on pathological background of the disease and resolution of symptoms following treatment of hyperthyroidism. There have been different arguments as to whether it is a mere coincidence or a cause- effect relationship. Some authors believe that pericardial involvement in hyperthyroidism may present with pericarditis without effusion, myopericarditis or cardiac tamponade[5]**.** The general consensus is that, the immunological mechanism which explains pre-tibial, periorbital myxedema and ophthalmic myopathy could be at play in pericardial effusion caused by hyperthyroidism especially in patients with Grave's disease.[6] Autoantibodies or viral infection (Epstein Barr Virus in most cases) involved in Graves' disease can initiate an inflammatory process within the pericardium by interacting with its receptors. [7-10]

Clarke *et al.*[6] reported four middle-aged patients with Graves' disease associated with pericardial disease. They opined that pericardial disease could be a rare complication of Graves' disease and may have similar mechanism of action with that of Grave's associated ophthalmomyopathy and dermopathy. Most case reports presented typical acute pericarditis who on further evaluation were found to have thyrotoxicosis. On the other hand, some cases reported the development of acute pericarditis in patients with established diagnosis of Graves' disease on treatment. However, it was suggested that single episodes of pericarditis with Graves' thyrotoxicosis could not explain the pathophysiology, as you would expect that autoimmune forms of pericarditis should be recurrent but this was not the case in most reports.[2] Acute thyrotoxicosis may directly impact pericardial fat metabolism making it a possible alternate cause of pericarditis.[11] In some clinical scenarios, there were cases of drug-induced pericarditis following commencement of propylthiouracil for treatment of thyrotoxicosis. Drug induced vasculitic syndrome was singled out as a possible mechanism[12]. Pericarditis has also been described after the initiation of other anti-thyroid medications like iodine and carbimazole. In patients with pericardial effusion/pericarditis, there is need to rule out hyperthyroidism as a possible aetiology, especially in patients with clinical features that are suggestive. In our case, symptoms resolved following treatment for hyperthyroidism.

REFERENCES

1. **Tsai A.** Thyroid Woes. Diabetes Forecast. 2016;69(1):58-62.
2. **Ertek S,** Cicero AF. Hyperthyroidism and cardiovascular complications: a narrative review on the basis of pathophysiology. Archives of medical science: AMS. 2013; 9(5):944.
3. **Koo EH,** Kim SM, Park SM *et al.* Acute Recurrent Pericarditis Accompanied by Graves' Disease. Korean circulation journal. 2012; 42(6):419-422.

4. **Kortekaas K,** van der Lienden B, Jong S *et al*. Graves' disease as an uncommon cause of acute pericarditis. Case Reports. 2014; 2014: bcr2013203 342.
5. **Gupta P,** Chhabra L, Hiendlmayr B *et al*. Thyrotoxic pericarditis: An underappreciated phenomenon. International journal of cardiology. 2015;198:32-33.
6. **Clarke N,** Banning A, Gwilt D *et al*. Pericardial disease associated with Grave's thyrotoxicosis. Qjm. 2002; 95(3):188-189.
7. **Nagata K,** Nakayama Y, Higaki K *et al*. Reactivation of persistent Epstein-Barr virus (EBV) causes secretion of thyrotropin receptor antibodies (TRAbs) in EBV-infected B lymphocytes with TRAbs on their surface. Autoimmunity. 2015; 48(5):328-335.
8. **Nagata K**, Kumata K, Nakayama Y *et al*. Epstein-Barr Virus Lytic Reactivation Activates B Cells Polyclonally and Induces Activation-Induced Cytidine Deaminase Expression: A Mechanism Underlying Autoimmunity and Its Contribution to Graves' Disease. Viral Immunol. 2017; 30(3): 240-249.
9. **Nagata K,** Higaki K, Nakayama Y *et al*. Presence of Epstein-Barr virus-infected B lymphocytes with thyrotropin receptor antibodies on their surface in Graves' disease patients and in healthy individuals. Autoimmunity. 2014; 47(3):193-200.
10. **Nagata K,** Fukata S, Kanai K *et al*. The influence of Epstein-Barr virus reactivation in patients with Graves' disease. Viral Immunol. 2011;24(2):143-149.
11. **Chhabra L,** Khalid N, Spodick DH. [Letter on the article "Pericardial effusion: an unknown expression of Graves' disease"]. Presse Med. 2014; 43 (12 Pt 1):1407.
12. **Colakovski H,** Lorber DL. Propylthiouracil-induced perinuclear-staining antineutrophil cytoplasmic autoantibody-positive vasculitis in conjunction with pericarditis. Endocrine Practice. 2001; 7(1):37-39.