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## Introduction

Graves' disease (GD) is associated with a number of cardiovascular complications, including rhythm disturbances, mitral valve prolapse, pulmonary hypertension and heart failure. Pericardial effusion is a rare complication of thyrotoxicosis and there are very few cases described in the literature. We present an unusual case of a patient who presented with pericardial effusion and was also found to have recurrence of GD.

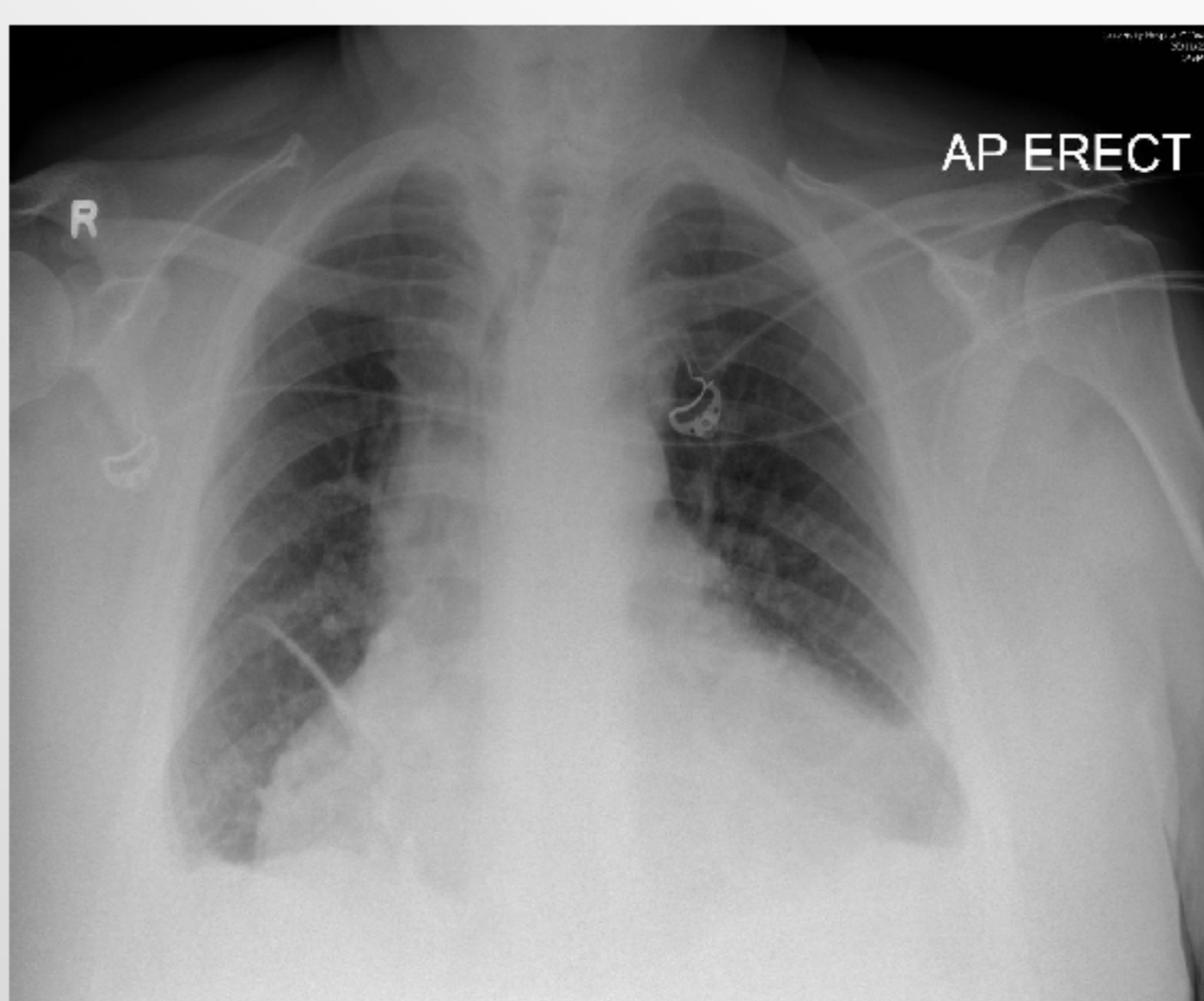
## Case

A 59-year-old gentleman presented with a 3-day history of dyspnoea and pleuritic chest pain. He had a history of GD previously treated with carbimazole for 12 months, pulmonary embolism (PE), asthma, hypertension, obesity and was established on warfarin, following a second episode of PE, 4 weeks prior to this clinical event. The rest of the patient's regular medications included: valsartan 150 mg daily, amlodipine 10 mg daily, aspirin 75mg daily, simvastatin 40 mg daily, isosorbide mononitrate 20 mg twice daily and budesonide and formoterol inhalers.

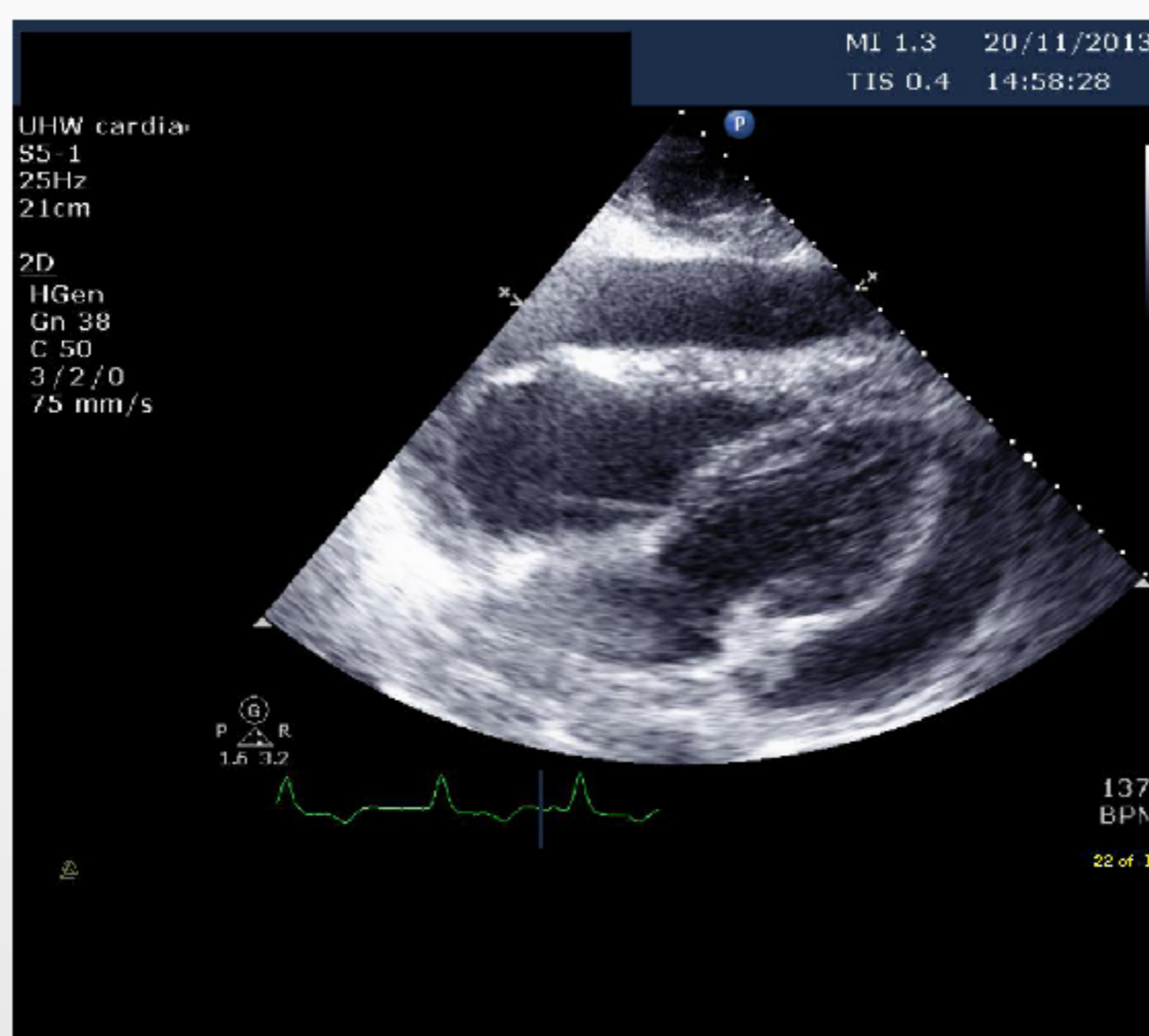
Clinical examination revealed tachypnoea, tachycardia, raised JVP, irregularly irregular pulse and bibasal crackles. Electrocardiography confirmed the presence of atrial fibrillation with fast ventricular response and a CXR showed marked cardiomegaly with bilateral pleural effusions (Figure 1). An urgent echocardiogram showed a 2.9cm pericardial effusion (Figure 2, 3), compromising the right ventricular filling. 10 days prior to this admission, a previous echocardiogram had shown a smaller size pericardial effusion.

TFTs revealed suppressed TSH, elevated free T4 (fT4) at 34.7pmol/l (reference range 9.2-21pmol/l) and positive TSH receptor antibodies, compatible with relapsed GD. Other laboratory investigations showed acute kidney injury (eGFR 38ml/min/1.73m<sup>2</sup>) and elevated INR at 17.2. Interestingly, 10 days prior to admission patient's renal function was normal.

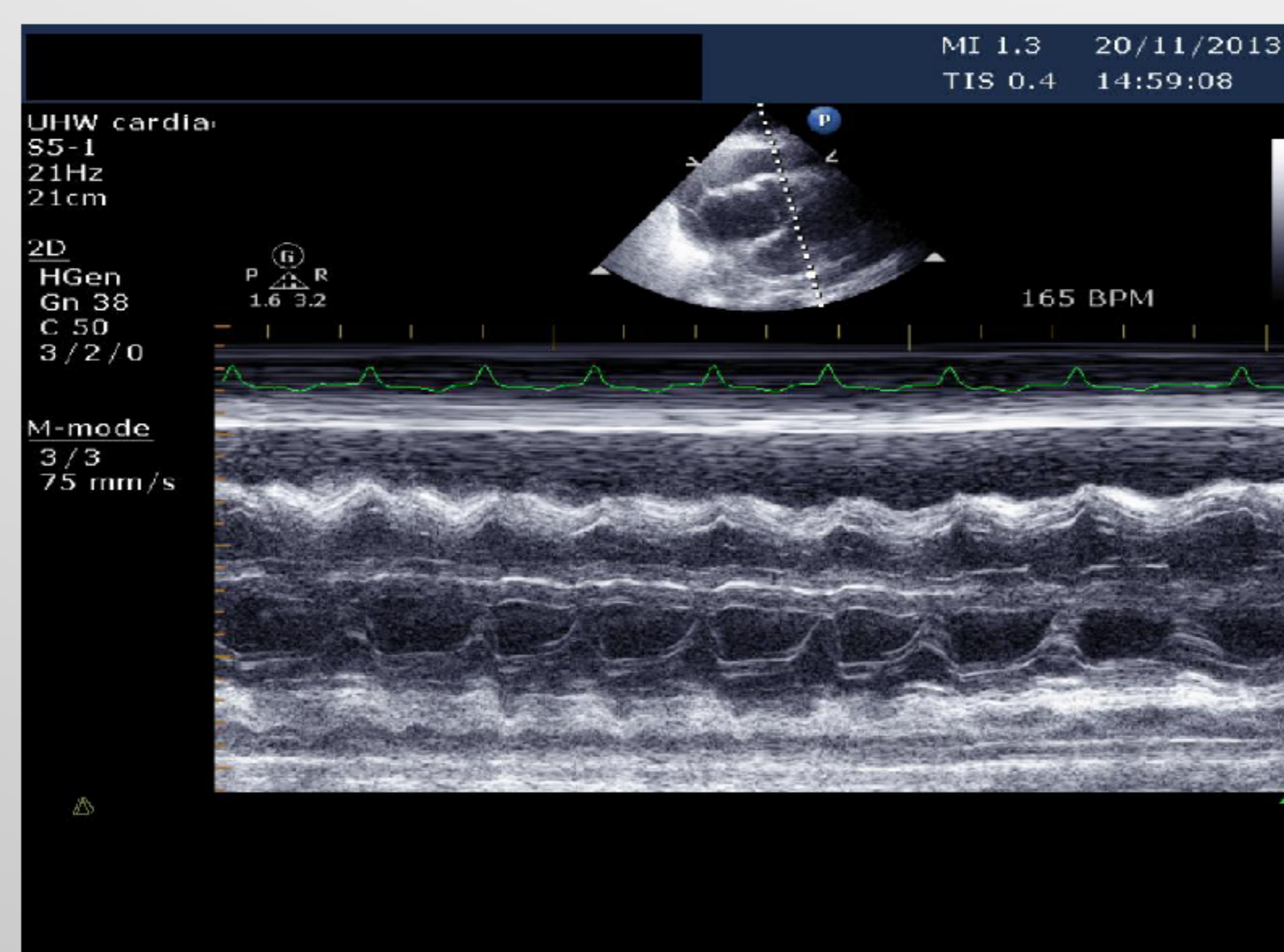
**Figure 1.** Chest xray, anterior-posterior view demonstrating marked cardiomegaly and bilateral pleural effusion with prominent pulmonary vascular markings.



**Figure 2.** Subcostal view showing large circumferential pericardial effusion.



**Figure 3.** M-mode in subcostal view across the right ventricle demonstrating a large pericardial effusion causing diastolic collapse of the right ventricle.



## Case (cont'd)

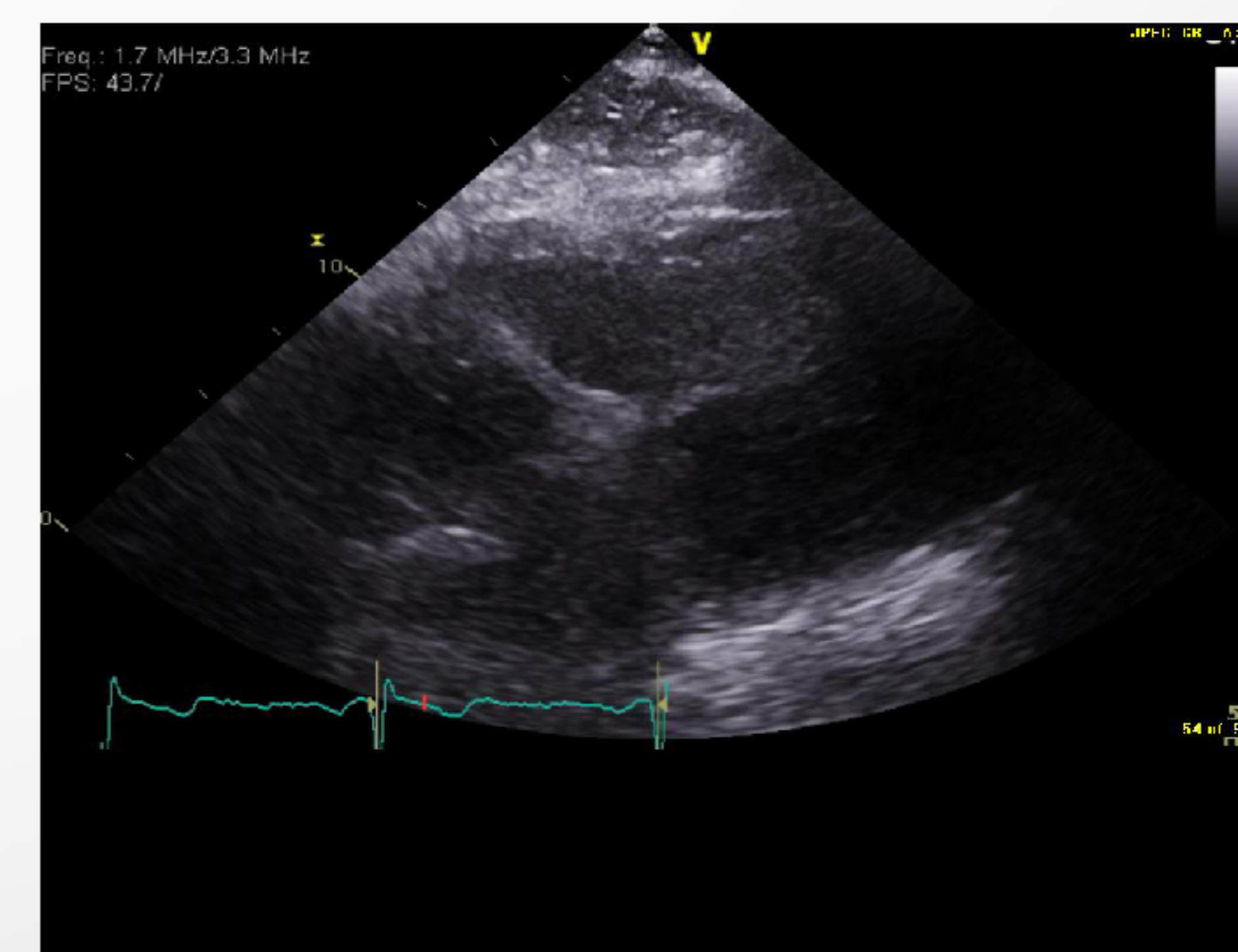
After pharmacological reversal of the patient's coagulopathy, pericardiocentesis was performed which resulted in immediate symptomatic relief and improvement of the patient's haemodynamic status. Analysis of the pericardial fluid showed inflammatory cells. Microbiology and cytology investigations were unremarkable. A full body CT scan did not show evidence of neoplastic process. The patient was subsequently discharged on Carbimazole 40mg daily with further outpatient follow-up.

## Discussion

Pericardial effusion in the context of GD has only been reported in a small number of cases [1, 2, 3]. In a study by Levy et al looking at the aetiological diagnosis of 204 pericardial effusions, a definite cause was found in 52.4% of cases and GD was not listed as a cause [4].

We have described an unusual presentation of GD. The patient was investigated extensively, but no alternative cause for pericardial effusion was found. The presence of inflammatory cells in the fluid, suggests an inflammatory pericarditis. In the absence of history suggestive of connective tissue disorder, acute infection or previous chest trauma, we believe that the most likely aetiology of the pericardial effusion is the relapse of GD. This is further supported by a repeat echocardiogram following improvement of his thyroid function (TSH 0.03 mU/l with fT4 15pmol/l) three months after the initial admission, which did not show any evidence of recurrence of the pericardial effusion (Figure 4). The elevated INR initially, may explain the haemorrhagic nature of the pericardial fluid.

GD could also have also been implicated in the pathogenesis of the patient's recent PE, given the thrombogenic tendency that hyperthyroidism is associated with, predisposing to a hypercoagulable and hypofibrinolytic state [5] with more compact clots [6].



**Figure 4.** Subcostal view after normalization of fT4 showing only a rim of pericardial fluid.

## Conclusions

This case shows that pericardial effusion can be a rare complication of GD. Therefore, we recommend that thyroid function tests should be performed in patients presenting with unexplained pericardial effusion and equally, patients with GD who present with dyspnoea or chest pain, should be investigated for the possibility of pericardial effusion.

## References

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