THE NEW ZEALAND MEDICAL JOURNAL
Journal of the New Zealand Medical Association

An unusual complication in a patient with Graves' disease

Yasmeen Khalid, Raashda Sulaiman, Rasheed Zahir, Varadarajan Baskar, Harit N Buch

We report a case of Graves' disease with an unusual complication of pericardial and pleural effusion with a possible underlying immunological mechanism.

Case report

A 68-year-old Caucasian lady presented with lethargy, weight loss and palpitations. She had tremors and was tachycardic ; a provisional diagnosis of hyperthyroidism was made. The diagnosis was confirmed by thyroid function tests with fT4 72.0 pmol/L (reference range (RR): 12.0–22.0 pmol/L) and TSH <0.01 mU/L (RR: 0.27–4.20 mU/L).

Aetiology of hyperthyroidism was confirmed to be Graves' disease with diffusely increased uptake on radio-nuclide scan and a high TSH-receptor antibody level of 13 U/L (normal <1.0 U/L).

She was commenced on 40 mg/day of carbimazole and 80 mg/day of propranolol. Ten days later she was admitted to hospital with shortness of breath and atrial fibrillation with a fast ventricular rate. She had a loud pericardial rub and raised jugular venous pressure.

Chest X-ray (Figure 1) showed cardiomegaly and mild left-sided pleural effusion. An urgent echocardiogram (Figure 2) confirmed moderately large global pericardial effusion with no features of cardiac tamponade and normal left ventricular systolic function. Blood film revealed polymorphonuclear leucocytosis of 20.5×10^9/L and inflammatory markers were raised with CRP 187 mg/L and ESR 75 mm/hr.

Figure 1. Chest X-ray

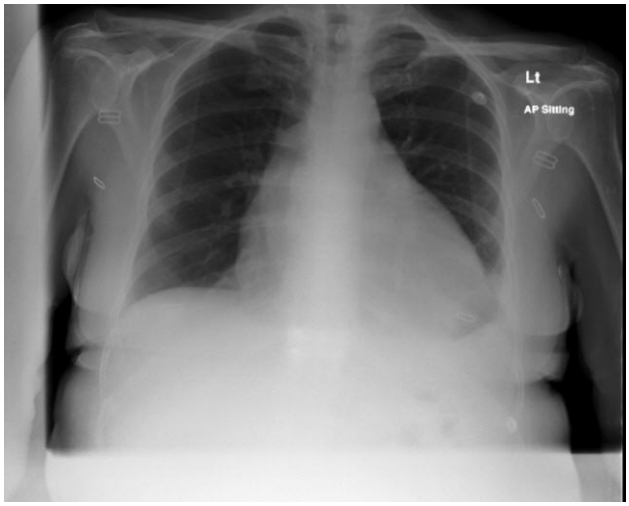
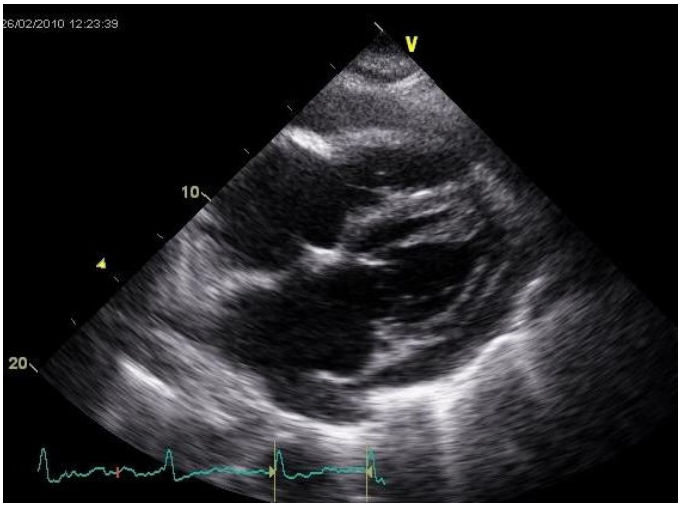


Figure 2. Echocardiogram



Anti-nuclear antibodies and anti-neutrophil cytoplasmic antibodies (ANCA) were negative and complement levels were normal. Pleural fluid was confirmed to be an exudate with pleural fluid to serum protein ratio of 1.4.

Pleural biopsy showed benign inflammatory changes. Ventricular rate was controlled with an increase in the dose of propranolol to 160 mg/day and as she remained clinically stable she was discharged home. Over the next 6 weeks stable euthyroidism was maintained with "block and replacement" therapy consisting of 40 mg carbimazole and 100 µg thyroxine (fT4 21.3 pmol/L, fT3 5.6 pmol/L, TSH 0.02 mU/L) and a repeat echocardiogram at this stage confirmed complete resolution of pericardial effusion and inflammatory markers returned to normal.

Six months later, she remains euthyroid on antithyroid medication with no serous membrane involvement.

Discussion

There are several possible explanations for pericardial and pleural effusions in this patient. Heart failure in patients with hyperthyroidism and atrial fibrillation can cause effusions although normal ventricular function on echocardiogram did not support this diagnosis. Primary or secondary malignancy, lymphoma and chronic infections like tuberculosis were excluded by pleural biopsy and her subsequent clinical course. Immune-mediated conditions like systemic lupus erythematosus is another potential explanation in a patient with autoimmune thyroid disease but this was excluded by the absence of clinical features and negative autoantibody screen.

Our patient developed serous membrane involvement 10 days after commencement of carbimazole which has been linked with systemic vasculitis.[1] However there were no other clinical features to support systemic vasculitis, ANCA was negative and despite the continuation of carbimazole there was a resolution of serous effusion which makes this possibility unlikely.

We believe that pericardial and pleural effusions were secondary to immunological epiphenomenon related to Graves' disease similar to the more common manifestations like ophthalmopathy and dermopathy. There was a strong temporal association between her presentation with Graves' disease and the diagnosis of serous membrane effusions.

There are case reports in the literature which have described association of pleural and pericardial effusions with Graves' hyperthyroidism.[2–5] None of the patients required any treatment other than antithyroid drugs and after the initial aspiration in some cases there was no recurrence of the pericardial effusion.[2]

Conclusion

Pleural and pericardial effusions are rare but recognised complications of Graves' disease and should be considered in patients who present with dyspnoea, chest pain or pericardial rub.

Author information: Yasmeen Khalid ,Clinical Fellow Diabetes; Raashda Sulaiman, Registrar Clinical Chemistry; Rasheed Zahir, Consultant in Acute Medicine; Varadarajan Baskar, Consultant Diabetes and Endocrinology; Harit N Buch, Consultant Diabetes and Endocrinology; Departments of Diabetes, Endocrinology and Acute Medicine, New Cross Hospital, Wolverhampton, UK

Correspondence: Yasmeen Khalid, Diabetes Centre, New Cross Hospital, Wednesfield Road, Wolverhampton WV10 0QP, UK. Email: Yasmeenkhalid@nhs.net

References:

1. Schocket AL, Lain D, Kohler PF. Immune complex vasculitis as a cause of ascites and pleural effusions in systemic lupus erythematosus. J Rheumatol 1978;5:33-8. http://www.ncbi.nlm.nih.gov/pubmed/147941
2. Clarke NR, Banning AP, Gwilt DJ, Scott AR. Pericardial disease associated with Grave's thyrotoxicosis. QJM 2002;95(3):188-189. http://qjmed.oxfordjournals.org/content/95/3/188.full
3. Ovadia S, Lyssy L, Zubkov T. Pericardial Effusion as an expression of thyrotoxicosis. Tex Heart Inst J2007;34(1):88-90. http://www.ncbi.nlm.nih.gov/pmc/articles/PMC1847910/
4. Teague EC, O'Brien JN, Campbell PS. Pericardial effusion and tamponade complicating treated Graves` thyrotoxicosis. Ulster Med J 2009;78(1)56-58. http://www.ums.ac.uk/umj078/078(1)056.pdf
5. Nakata A, Komiya R, Ieki Y, et al. A patient with Graves' disease accompanied by bloody pericardial effusion. Internal Medicine. 2005;44(10):1064–1068. http://www.ncbi.nlm.nih.gov/pubmed/16293918