

Hemorrhagic Pleural Effusion – An Unusual Presentation of Grave’s Disease

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ABSTRACT

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The most common manifestation of thyrotoxic cardiac disease is cardiac failure. Presentation of hemorrhagic pleural effusion in a thyrotoxic cardiac failure, which resolved with treatment of Grave’s disease, is a very rare presentation. We are reporting a case of hemorrhagic pleural effusion in a Grave’s disease.

Keywords: Hemorrhagic Pleural Effusion, Grave’s Disease

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INTRODUCTION

Cardiac failure is a most common manifestation of thyrotoxic cardiac disease. Presentation of exudative pleural effusion in a thyrotoxic cardiac failure is very rare presentation. Here, we are reporting one such uncommon presentation of Grave’s disease.

CASE REPORT

A sixty years old male was admitted to our hospital with history of exertional breathlessness for 6 months with acute aggravation of breathlessness, non-exertional palpitations for 2 months and cough for 2 months duration. Patient gave a history of transient left sided weakness which improved later. He had history of weight loss since 6 months. After admission, patient developed nasal regurgitation and hoarseness of voice.

He was a known case of diabetes mellitus since last 6 years, chronic obstructive pulmonary disease since 3 years, not a hypertensive. No past history of tuberculosis, bronchial asthma, seizures, cerebrovascular accident or transient ischemic attack. He is a chronic smoker, non- alcoholic.

On examination, the patient was conscious and oriented. Pallor was present, pretibial myxedema noted in both legs. The patient had pulse rate of 108/min,

irregular and blood pressure of 150/80 mm of mercury in the right upper limb in supine position, respiratory rate of 26/min, temperature was 98.6^oF. Periorbital edema noted, exophthalmos seen, lid lag present. On examination of eyes; Stellwag’s sign, Von Graefe’s sign, Dalrymple sign was present. Diffuse enlargement thyroid with bruit was present; gland was non tender, firm in consistency.

The respiratory system showed: barrel shaped chest, stony dullness on right inframammary, infraaxillary, infrascapular area & bilateral basal crepitations. Cardiovascular system examination showed: JVP elevated 10cm: ‘a’ waves absent & irregular ‘v’ waves, mild cardiomegaly, varying intensity of heart sounds & ejection systolic murmur in 3rd left sternal border.

The central nervous system examination showed higher mental functions normal. Bilateral palatal weakness was present, gag reflex was absent, nasal twang was present, nasal regurgitation present. Tongue flabby on palpation, bulk was normal, no wasting and no fasciculation. Tongue protrusion, side to side and all other tongue movements reduced. No motor or sensory deficits; reflexes – generalized hyperreflexia, no cerebellar signs, gait – normal. Proptosis present (Axial length in Hertel’s meter showed 21.5 mm in right eye & 22 mm in left eye), optic fundi normal, pupil normal. Patient had diminished eye movements in all directions.

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INVESTIGATIONS ON ADMISSION

Hemoglobin – 8.8gm% (Normocytic normochromic anemia); Total count- 9700 cells/cmm, P86 L11 E1 M2, ESR- 32 mm/hr; Blood urea - 29 mg/dl; S.creatinine – 1.0 mg/dl; S.Calcium – 8.8 mg/dl; Urine routine –within normal limits; Serum iron – 52 mcg/dl. Liver function tests were within normal limits. TFT showed T3- 2.63 ng/ml (0.80 – 2.0), T4- 15.57 mcg/ml (5.1–14.1), TSH- <0.005 microIU/ml (0.27– 4.2); TPO Ab – 34IU/ml (Ref range < 35 IU/ml); Acetylcholine receptor antibody– absent.

Chest X-ray PA view revealed emphysema with right sided pleural effusion; USG chest demonstrated moderate to gross right pleural effusion, collapse of right lower lobe. USG abdomen revealed no significant intra-abdominal pathology. USG thyroid showed diffusely enlarged gland with increased vascularity without any focal lesions. CT Chest showed moderate pleural effusion with collapse of right lower lobe, few enlarged lymph nodes in pretracheal and paratracheal regions, no mass lesion. Echo showed: normal LV systolic function and ejection fraction, no evidence of vascular lesion and no diastolic dysfunction.

MRI Brain was within normal limits. MRI Orbit showed fatty infiltration and string like appearance of extra-ocular muscles, sparing tendinous attachment at the ring of Zinn suggestive of Grave's ophthalmopathy. Technetium thyroid scan revealed thyroid enlargement, increased perfusion and trapping suggestive of Graves disease, Sputum culture grown was normal flora.

Pleural fluid was blood stained, analysis revealed cell count: WBC - 631 cells/cu.mm, neutrophils – 10%, lymphocytes – 90%, RBC – numerous/HPF, sugar: 55 mg%, protein: 4.1 g%. Pleural fluid ADA was 10.2 micro/l (<30); cytology showed lymphocytosis, no malignant cells.

Considering the presentation of high output congestive heart failure with atrial fibrillation, bulbar muscle weakness, diffuse goiter with bruit, ophthalmopathy, elevated T3, T4 & very low TSH, high uptake in technetium scan diagnosed to have Grave's disease with a rare manifestation of hemorrhagic pleural effusion

The patient was treated with Carbimazole 40 mg, Propranolol 40 mg, Torsemide 20mg bid and supportive measures. His cardiac failure improved, repeat chest x-ray showed complete resolution of pleural effusion after 14 days of treatment, nasal regurgitation and nasal twang of voice also decreased. On follow up bulbar myopathy is better.

DISCUSSION

Cardiac failure is a common presentation in Grave's disease. Effusions (pleural and pericardial) are rare in hyperthyroidism and predominantly reported in patients with Grave's disease.^{1,4} Heart failure in patients with hyperthyroidism and atrial fibrillation can cause transudate pleural effusion, bilaterally or on right side.

There are case reports like carbimazole induced exudative pleural effusion⁵ and propylthiouracil-associated eosinophilic pleural effusion.⁶ Hemorrhagic pericardial effusion in Grave's disease has been reported earlier.⁷ Hemorrhagic pleural effusion in Grave's disease is a very rare presentation. A similar immunological mechanism to ophthalmopathy and pretibial myxedema has been proposed for these effusion.^{1,2}

Basic immunological abnormality in Grave's disease is Type V immune reaction,³ where antibodies (Thyrotropin receptor antibody) are cyto stimulant to the thyrocyte. Lymphocytic infiltration and edema is seen in infiltrative ophthalmopathy. In pretibial myxedema, the characteristic histologic feature consists of deposition of mucin (glucosaminoglycans) throughout the reticular dermis and with attenuation of collagen fibres. Both ophthalmopathy and pretibial myxedema can occur without hyperthyroidism known as euthyroid Grave's.

Similar to above manifestation, presentation of hemorrhagic pleural effusion is secondary to immunological epiphenomenon related to Grave's disease.^{1,2} It is a rare manifestation unlike the more common immunological manifestation like ophthalmopathy and dermopathy. Treatment of hemorrhagic pleural effusion consists of the treatment of thyrotoxicosis.^{1,2} Hemorrhagic pleural effusion is one of the rarest presentations which used to resolve itself on treatment of Grave's disease.

END NOTE

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Conflict of Interest: None declared

Editorial Comment: The case is reported for a rare presentation of a disease. The awareness about this is needed for the general practitioner and specialist alike.

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