Intrathoracic Goiter with Hyperthyroidism, Tracheal Compression, Superior Vena Cava Syndrome, and Horner’s Syndrome*

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Intrathoracic goiter is an important cause of mediastinal masses. We present a patient with hyperthyroidism, tracheal compression, superior vena cava syndrome (SVCS) and Horner’s syndrome due to intrathoracic goiter. To our knowledge, this is the first case in the literature with all of these combined findings. (Chest 1990; 97:1005-06)

SVCS = superior vena cava syndrome

Intrathoracic extension of goiters usually causes a benign neck and mediastinal mass with morbid potential. These goiters can develop slowly and patients may be asymptomatic for many years. They may have some problems about diagnosis and anesthesia during surgery. We present a patient with intrathoracic goiter complicated by hyperthyroidism, tracheal compression, SVCS, and Horner’s syndrome. We believe that this is the first published case with all of these findings.

CASE REPORT

A 36-year-old woman was admitted to the hospital with breathlessness, dysphagia and coughing. Her complaints had persisted for approximately six months and increased during the last month. She suffered from palpitations, heat intolerance, weight loss, sweating, dysphagia. These symptoms had been diminished after taking propylthiouracil in another hospital.

On physical examination, she was experiencing dyspnea, orthopnea, tachycardia, inspiratory stridor, and wheezing. The skin was thin and moist and the face was flushed. There were prominent venous collaterals on the neck and upper thoracic region (Fig 1). The left lobe of the thyroid gland was diffusely palpable and the lower margin could not be defined. There was right ptosis, meiosis, exophthalmos and the right pupil was smaller than the left by over 2 mm. Examination of the chest showed small and medium rales in the basal regions. Laboratory examination results from complete blood cell count, urinalysis, electrocardiography, and biochemical tests were within the normal limits. Thyroid function tests revealed hyperthyroidism; T₃ 2.14 ng/ml (0.5-1.75 ng/ml); T₄ 13.8 ng/dl (4.8-12.8); TSH: 0.9 mU/ml (1.0-5.5); free T₃ 5.3 ng/ml (1.3-3.6); free T₄ 2.7 ng/dl (0.8-2.0).

Chest roentgenogram revealed fibrotic changes on the basal regions that suggested bronchiectasis and widened upper mediastinum with no tracheal displacement. Ultrasonographic examination revealed a diffusely enlarged left lobe of the thyroid and its lower border could not be defined. Thoracic computed tomographic scans showed an enlarged left lobe of the thyroid with its lower margin extended to the eighth thoracic vertebral level and the right lobe was also enlarged to the anterior mediastinum and both the right and left lobes had compressed the trachea and esophagus (Fig 2). She was treated by surgery, but unfortunately, she died of respiratory failure during the operation.

DISCUSSION

Intrathoracic goiter is an important cause of mediastinal masses. It may be defined as any thyroid enlargement that

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Figure 1. Right ptosis, exophthalmos, enlarged thyroid gland and venous collaterals on the neck and sternal region.

Figure 2. Computed tomographic scan demonstrates mediastinal-located large thyroid tissue and bilaterally compressed but not deviated trachea (T).
has its greater mass inferior to the thoracic inlet.\textsuperscript{1} Seventy-eight to 80 percent of these masses descend into the anterior mediastinum while the remainder descend into the posterior mediastinum with the middle mediastinal goiters rarely encountered.\textsuperscript{1} The most common symptoms associated with intrathoracic goiters are dyspnea, stridor, dysphagia, hoarseness, coughing, wheezing, and cervical mass. Interestingly, in addition to mechanical findings, the presented case also had neurologic and biochemical findings.

The case presented here had a large intrathoracic goiter and most of the symptoms were due to compression on adjacent structures. Dyspnea and dysphagia were due to compression on trachea and esophagus, respectively. There was no tracheal deviation which is caused by bilateral compression.

Horner’s syndrome secondary to thyroid disease may be seen. It usually results from cervical sympathetic chain invasion by malignancies, but secondary to sympathetic chain compression with multinodular goiter has also been reported.\textsuperscript{4} We think that our patient’s neurologic symptoms were due to compression and would be resolved by resection of the mass.

Superior vena cava syndrome with intrathoracic goiter has been reported.\textsuperscript{5} It is seen with malignancy more frequently than with benign intrathoracic goiter. Positional dilated veins may simulate SVC syndrome and should be differentiated.

Hyperthyroidism with intrathoracic goiter has been reported in up to 20 percent of case reports,\textsuperscript{6} but it has been reported in recent series only occasionally.\textsuperscript{1} It is interesting that our patient had all these combined findings. Our patient had clinical manifestations of hyperthyroidism, confirmed by thyroid function tests, probably due to using propylthiouracil before admission to the hospital. She had mild symptoms and laboratory findings. If the patients have not had any compression symptoms, radioactive iodine treatment may be used. As in our patient who has compression symptoms, operative treatment should be required.

**References**


**Recurrent Pericarditis and Dermatitis Herpetiformis**

**Evidence for Immune Complex Deposition in the Pericardium**

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Recurrent pericarditis can be associated with many chronic illnesses. Dermatitis herpetiformis is a chronic papulovesicular eruption which is characterized by granular IgA deposits in the dermal papillary tips and associated with a gluten-sensitive enteropathy. We describe the first case of recurrent pericarditis in association with dermatitis herpetiformis. This supposition is supported by exclusion of other possible etiologies and pericardial biopsy which revealed the deposition of IgG, IgA and complement.

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**ANA = antinuclear antibody**

A 60-year-old white woman with a 30-year history of dermatitis herpetiformis developed recurrent pericarditis and malabsorption. Dermatitis herpetiformis is a chronic papulovesicular eruption characterized by granular IgA deposits in the dermal papillary tips. It is often associated with a gluten-sensitive enteropathy and a high occurrence of serum HLA-B8 and HLA-Dw3.

A biopsy of this patient’s skin lesions revealed IgA deposits in the dermal layer of the skin and HLA typing was positive for HLA-B8. Open pericardial biopsy revealed the presence of IgA, IgG and complement deposition in the pericardium. This is the first case of recurrent pericarditis in association with dermatitis herpetiformis and malabsorption suggesting a common immunologic denominator in a multisystem disorder.

**Case Report**

A 60-year-old white woman with dermatitis herpetiformis presented with sudden onset of pleuritic chest pain and a three-component friction rub in association with an exacerbation of her skin lesions. The patient had presented with three similar episodes in the previous seven years. She denied symptoms of upper respiratory infection; however, she had a tuberculous infection 33 years earlier for which she received a 12-month course of isoniazid and para-aminosalicylic acid. She first presented to us eight years ago with cardiac irritability (ventricular fibrillation, resuscitated twice) and chest pain. She was evaluated at that time with cardiac catheterization and angiography which demonstrated cardiomyopathy but no evidence of coronary artery disease. Since that time, she has had multiple episodes of pericarditis. Because of her extreme ventricular irritability, an endomyocardial biopsy was not attempted. Results of an ANA screen and titer, LE preparation, ESR, rheumatoid arthritis screen, extractable nuclear antigens and anti-DNA binding capacity were all within normal limits or negative. Computerized axial tomography of the heart revealed normal pericardial thickness with no evidence of calcification. Open peri-