has been described,³ prompt and effective surgical and drug therapy is essential.

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Myasthenia gravis complicated by sarcoidosis

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Myasthenia gravis (MG) complicated by sarcoidosis is rare. Whether this complication represents the association between the two diseases or a simple coincidence is unclear. We encountered a patient in whom MG seemed to have induced sarcoidosis.

A 32-year-old woman visited our department because of blepharoptosis in February 1990. The blepharoptosis was improved by the tension test. X-ray examination and computed tomographic scanning of the chest showed no abnormalities. Results of the serum acetylcholine receptor antibody test were positive. Electromyography showed the typical waning phenomenon after continuous peripheral nerve stimulation. On the basis of these findings, a diagnosis of MG was made. She was followed up without administration of anticholinesterase drugs.

In November 1992, she noticed fatigability of the skeletal muscles, difficulty in speech and swallowing, and frequent choking. Because of the exacerbation of MG, pyridostigmine bromide (60 mg) was administered daily. In July 1993, she was admitted to our department because of an abnormal shadow on chest X-ray films. Physical examinations revealed no palpable lymph nodes in the neck, axilla, or inguen. Examination of the eyes showed no uveitis.

Examination of peripheral blood samples showed no abnormalities in the leukocyte, erythrocyte, or platelet counts but an increase in the CD₄/CD₈ ratio among lymphocyte subsets. Urinary findings were within normal limits. The aspartate aminotransferase, alanine aminotransferase, alkaline phosphatase, and γ -guanosine triphos-

phate levels were within normal limits, but the thymol turbidity test level was 14.6 U, and the zinc turbidity test level was 24.2 U. The total protein concentration was 8.8 gm/dl with 51.0% α_2 -globulin and 29.8% γ -globulin. The serum level of angiotensin-converting enzyme was 23.4 U/ml, and the lysozyme level was 14.3 μ g/ml. The erythrocyte sedimentation rate was 56 mm/hr. The serum levels of triiodothyronine, thyroxine, and thrombin-stimulating hormone were within normal limits. Tests for thyroglobulin and mitochondria antibodies were negative. Rheumatoid factor and antinuclear antibody tests were negative, as was the positive protein derivative reaction.

Chest X-ray examination showed bilateral hilar lymphadenopathy. Chest computed tomographic scanning also showed marked bilateral hilar lymphadenopathy but no enlargement of the thymus or thymoma. Gallium 67 scintigraphy revealed accumulation-positive images on the bilateral hilar areas. A diagnosis of sarcoidosis complicated by MG (Osserman IIA) was made.

An operation was performed on October 19, 1993. After a T-shaped skin incision and longitudinal sternotomy had been done, the mediastinum was reached and extended thymectomy and biopsy of the hilar lymph nodes were performed. Histopathologic examination of the hilar lymph nodes demonstrated noncaseous granuloma, supporting the diagnosis of sarcoidosis.

Four months after the operation, she showed improvement in symptoms of MG with decreased blepharoptosis and diplopia and no fatigability of the skeletal muscles without administration of pyridostigmine bromide. Sarcoidosis has been observed after the operation without treatment, but no substantial changes have occurred in serum levels of angiotensin-converting enzyme or lysozyme.

MG is an autoimmune disease induced by the appearance of antiacetylcholine antibody. T cell abnormality is observed in the peripheral blood,¹ and germinal center formation is frequently observed in the thymus tissue. MG is closely associated with thymus abnormalities. On the other hand, sarcoidosis is a systemic disease characterized by formation of epithelioid cell granulomatous lesions not accompanied by necrosis, and its pathogenesis is unclear. However, recent information has suggested the following mechanism of the establishment of epithelioid cell granulomatous lesions, although the details are still unknown²: (1) T cells are activated after nonspecific stimulation or stimulation as an immune response to unknown cause, and activated T cells accumulate in the lesion; (2) accumulated T cells produce lymphokine; (3) lymphokine promotes the differentiation of monocytes/macrophages to epithelioid cells, resulting in the formation of epithelioid granuloma. Because the thymus plays an important role in the differentiation and growth of T cells, it is speculated that MG as "an unknown cause" activates the T cell line, inducing the formation of sarcoidosis lesions.

Some case reports of MG complicated by sarcoidosis have been reported. Sarcoidosis developed during the remission of MG in some cases³ but during the exacerbation period in others,⁴ showing no consistent tendency. Regression of MG and sarcoidosis after thymectomy was also reported.⁴ In our patient, MG became less severe after thymectomy, but sarcoidosis did not.

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Mediastinal vagal neurilemmoma causing tracheal stenosis

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Neurogenic tumors of the mediastinum are common and constitute the majority of neoplasms of the posterior mediastinum.¹ Usually such tumors arise from the intercostal nerve or the sympathetic chain. However, tumors originating from the vagus nerve are rare. A review of the literature revealed no patient with vagal neurilemmoma causing severe tracheal stenosis. We report here a case of vagal neurilemmoma in which hemoptysis resulted from compression of the membranous trachea.

A 68-year-old man was admitted with a 6-month history of feeling pressure on swallowing and recent onset of frequent hemoptysis. He had retired from his post as a public official 8 years previously, did not smoke, and had no history of serious illness. Physical examination showed no abnormalities, and results of laboratory findings including urinalysis, complete blood count, blood chemistry, arterial blood gases, and tumor makers were within normal limits. A spirogram also showed no abnormalities. Posteroanterior and lateral roentgenograms of the chest showed a mass compressing the trachea from behind. A computed tomographic scan revealed a well-circumscribed, heterogeneously enhanced mass, which was causing tracheal stenosis. Magnetic resonance imaging was performed for further anatomic definition of the mass (Fig. 1). Examination with a fiberoptic bronchoscope disclosed severe extrinsic pressure on the distal trachea and mucosal injection (Fig. 2). Transtracheal biopsy was impossible because the mucosa had a tendency to bleed. It was difficult to establish a definite preoperative diagnosis

Fig. 1. Magnetic resonance imaging showing a well-circumscribed mass compressing the membranous trachea.

computed tomography and magnetic resonance imaging suggested a benign tumor, such as neurilemmoma or neurofibroma, but we could find no case report of benign mediastinal tumor associated with frequent hemoptysis, and a pathologic diagnosis could not be obtained because of bleeding from the tracheal mucosa.

With the preoperative diagnosis of mediastinal neurogenic tumor, which was most likely a neurilemmoma but possibly a malignant tumor, a right thoracotomy incision was made. A smooth, encapsulated tumor with the dimensions of 4 × 4 × 3 cm was encountered behind the trachea, and careful observation revealed that it was originating from the right vagus nerve. Inasmuch as examination of frozen sections of the mass showed that the tumor was a neurilemmoma originating from the vagus nerve, we decided to resect the tumor completely. Because the tumor could not be dissected from the nerve, the tumor was extirpated with proximal and distal ligation and division of the vagus nerve. At that time, a 2 cm defect in the membranous trachea of the carina occurred, and a double-lumen endobronchial tube was placed into the left

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