Case Report

Bronchogenic Carcinoma with Subacute Cerebellar Degeneration and Eaton-Lambert Syndrome; An Autopsy Case.

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We report an autopsied patient who initially presented signs of cerebellar ataxia. After seven months, small cell carcinoma of the lung was revealed. Electromyography on repetitive stimulation and autopsy findings proved that the neurological disorders were due to Eaton-Lambert syndrome and subacute cerebellar degeneration, as forms of carcinomatous neuromyopathy.

Key Words: Carcinomatous neuromyopathy, Small cell carcinoma

Malignant neoplasm sometimes causes neurological disturbance without distant metastasis or direct invasion to the nervous system. This condition is known as carcinomatous neuromyopathy. We report an autopsy case of small cell carcinoma of the lung in which the initial sign was cerebellar ataxia, and which was complicated by two forms of carcinomatous neuromyopathy, subacute cerebellar degeneration and Eaton-Lambert syndrome.

CASE REPORT

A 37-year-old policeman suddenly developed vertigo. After one week, it became difficult for him to walk and dysarthria became evident. Diagnosis of acute cerebellar ataxia was made at a hospital, and prednisolone therapy was started. However, the neurological disturbances did not improve. Seven months after onset, coughing and expectoration of sputum were presented. The chest radiograph at this time showed a tumor shadow in the left hilus and mediastinal widening (Fig. 1). Chest computed tomography (CT) more apparent presented the tumor shadow surrounded the left lower lobe bronchus (Fig. 2).

The patient was then transferred to our hospital, and bronchofiberscopy was performed.

Fig. 1. Chest roentgenogram taken on admission to our hospital, showing a left hilar tumor shadow and mediastinal widening.

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Fig. 2. Chest computed tomography at the level of left lower lobe bronchus. Tumor shadow (arrow head) surrounding the bronchus is shown apparently.

Fig. 3. The biopsied material from the tumor revealed the typical findings of small cell carcinoma, small rather uniform cells with scanty cytoplasm and nuclei containing finely stippled chromatin and inconspicuous nucleoli (HE stain, original magnification x440).

Fig. 4. Brain computed tomography could not detect the metastatic lesion in the cerebellum and cerebrum. But slightly atrophic change of the cerebellum might be suspected because of mild enlargement of superior cistern.

showing the left lower bronchus were obstructed by the tumor. Tumor biopsy through the fiberscope revealed small cell carcinoma (Fig. 3). Neurological examination showed nystagmus, dysarthria, and diplopia. Dysmetria and truncal ataxia were marked. These findings were compatible with cerebellar ataxia. In addition, muscle weakness was present and deep tendon reflexes were absent. Brain CT showed no metastatic lesion, but slight atrophy of the cerebellum was suggested because of enlarged superior cistern (Fig. 4).

Cerebellar abnormalities were thought due to subacute cerebellar degeneration, a kind of carcinomatous neuromyopathy, since he did not have metastasis to the central nervous system, familial, alcoholic or infectious factors apparently responsible for the ataxia. In order to evaluate the extent of muscle weakness, electromyography was performed (Fig. 5). A repetitive stimulation test at 3 Hz showed a waning pattern, whereas stimulation at 50 Hz showed a marked waxing pattern. These results were compatible with Eaton-Lambert syndrome.

Chemotherapy and irradiation therapy for the carcinoma were performed, producing slight improvement in the neurological disturbances. Twenty-five months after the initial onset, the patient died of cancer. Postmortem examination showed widespread small cell carcinoma of the lung with multiple metastases, but not in the
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EMG
1 Repetitive stimulation test
3Hz Waning
0.2mV
1sec.

50Hz Waxing
2mV
1sec.

Fig. 5. Electromyogram taken on repetitive stimulation test. Stimulation at 3 Hz shows waning, with marked waxing at 50 Hz. These findings are compatible with Eaton-Lambert syndrome.

Fig. 6. Section of cerebellar cortex showing marked loss of Purkinje cells. The molecular layer is thin, and hyperplasia of Bergmann astrocytes and glial fibers is present (HE stain, original magnification x10).

cerebellum. The cerebellum was atrophied and histological examination showed marked loss of Purkinje cells from the cortex, although no inflammatory cells were found (Fig. 6) and the deep cerebellar nuclei were almost fully preserved. These pathological findings were consistent with subacute cerebellar degeneration.

DISCUSSION

Satoyoshi\(^4\) first reported a case of bronchogenic carcinoma accompanied by two forms of carcinomatous neuromyopathy, subacute cerebellar degeneration and Eaton-Lambert syndrome. As far as we have been able to determine, there were only six reported cases\(^4, 5\) (three of which were Japanese), including our present case, in which these three conditions were present and for which precise autopsy findings were recorded.

The etiology and pathogenesis of subacute cerebellar degeneration and Eaton-Lambert syndrome are unclear. But autoimmune etiology has suggested in each conditions\(^6, 7\). The presence of auto-antibodies to Purkinje cells in patients of subacute cerebellar degeneration\(^6\) and auto-antibodies to nerve terminal determinant in Eaton-Lambert syndrome\(^7\) were documented. These may mean that auto-antibodies to bronchogenic carcinoma neoantigen was cross reacting with Purkinje cells and determinants on nerve terminal. And, recently plasmapheresis has been tried as the treatment\(^8\).

The occurrence of the both two conditions in cases of bronchogenic carcinoma is rare, but in the category of carcinomatous neuromyopathy, they have had a greater tendency to occur in cases of bronchogenic carcinoma than in cases of carcinoma at other organs\(^2, 9\). There have also been cases in which the neurological abnormalities occurred prior to the discovery of bronchogenic carcinoma, as in our case. Thereby, it is important to note that it can be the first clinical symptom of bronchogenic carcinoma.

REFERENCES


