폐암절제 후 발생한 기엥-바레 증후군

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=Abstract=

Guillain-Barre Syndrome After Resection of Lung Cancer

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A 68-year-old man with Guillain-Barre syndrome after the resection of right upper lobe for squamous cell lung cancer is presented. He developed a sudden, symmetric, extremity weakness, respiratory insufficiency, and sensory ataxia on postoperative day 6. He was intubated emergently and placed on a ventilator. Electrodiagnostic studies were performed on days 2, 20, and 40 following the onset of weakness. Motor nerve conduction abnormalities were the predominant findings. Prolonged motor distal latencies, temporal dispersion, and partial motor conduction blocks were present and formed the diagnostic features of Guillain-Barre syndrome. With supportive care and additive use of intravenous immunoglobulin, the illness resolved 6 weeks later after the onset of weakness.

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Key words: 1. Guillain-Barre syndrome

2. Squamous cell lung cancer

3. Intravenous immunoglobulin

Case

A 68-year-old man with chest pain was admitted to our institute. In his forties he underwent an anterior fixation of cervical spine after traffic accident and he has a history of smoking. His chest X-ray revealed a

right upper zone opacity measuring 2 × 3cm and a subsequent Computed tomography of the thorax reported a mass on the right upper lobe consistent with lung cancer. Bronchoscopic cytology results indicated that the tumor was squamous cell lung cancer. Mediastinoscopy showed no metastatic disease. The patient underwent a

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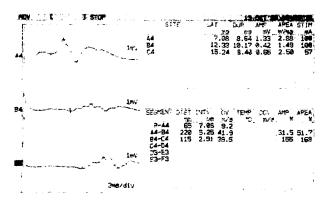


Fig. 1. The finding of electrophysiologic study performed on day 2 following the onset of weakness, showing the prolonged motor distal latencies, temporal dispersion, and partial motor conduction blocks.

NCV, Nerve conduction velocity

right upper lobe lobectomy with minithoracotomy Pathologic analysis disclosed a well unevenfully. -differentiated squamous cell carcinoma. All lymph node were negative for metastatic disease. Pathologic TNM staging was stage Ia(T1N0M0). He took a typical stable postoperative course and no sign of systemic inflammation. On the evening of postoperative day 6, he noted a sensation bilaterally in the toes and feet on walking. Next morning, his respiratory efforts became shallow with poor chest excursion suddenly. Emergent bronchoscopy showed no abnormal findings. He was intubated, transferred to the intensive care unit and placed on a ventilator. Lumbar puncture produced clear, colorless fluid with a cerebrospinal fluid(CSF) protein of 168 mg%(normal $15 \sim 45 \text{ mg\%}$), glucose mg/dl(serum 206 mg/dl), and cell counts of 3 red blood cells/mm³ and 2 white blood cells/mm3. Electrophysiologic studies were performed on days 2, 20, and 40 following the onset of weakness(Fig. 1). Motor nerve conduction abnormalities were predominant findings. Prolonged motor distal latencies, temporal dispersion, and partial motor conduction blocks were present and form the diagnostic features of GBS. His motor power was recorded as 1/5 in all four limbs with absence of tendon reflexes. Abnormalities in sensory nerve conductions and blink reflexes were also present. In histocompatibility(HLA) molecular study, HLA-DRB1*1301 allele was identified. Tracheostomy was performed 10 days later after application of ventilator. Our patient received 0.4 mg/kg/day of intravenous immunoglobulin (IVIg) for 5 days after confirmation of GBS and booster IVIg was administrated 3 weeks later as same dosage. He showed rapid improvement of muscular weakness after booster injection of the IVIg. He was weaned off ventilator throughout next 5 weeks, transferred out of intensive care unit, and discharged from the hospital with walking on postoperative day 42 in good medical condition. He is well 24 months after operation.

Discussion

Guillain-Barre syndrome(GBS) is an immune mediated peripheral neuropathy characterized by acute onset of symmetric limb weakness and areflexia. Patients with typical GBS had greater leg weakness than arm weakness, with ascending progress.

Neuropathy involved with disseminated malignancy (paraneoplastic GBS) is rare and heterogeneous disorders. Some of paraneoplastic GBS are part of complex syndrome involving simultaneously the central and peripheral nervous system, the most frequent of which is delayed or subacute sensory neuropathy/paraneoplastic encephalomyelitis.

Sometimes the clinical differentiation between GBS and paraneoplastic GBS may be difficult. Few of neuropathy associated with small cell carcinoma and adenocarcinoma of the lung is reported4), but there is none in acutely developed GBS with squamous cell lung cancer.

GBS is now recognized as a heterogenous disorder with many clinical manifestations and a diverse disorder, including both acute inflammatory demyelinating polyneuropathy(AIDP) and acute motor axonal neuropathy (AMAN)⁵⁾. The disease is of unknown etiology, although several lines of evidence suggest an immunologic pathogenesis. Approximately Two-thirds of the cases are preceded by an infection, most frequently of the respiratory or gastrointestinal tracts. Recently, the importance of host factors, associated with a certain MHC class II genes, was also suggested⁹⁾.

In association with the carcinoma, depending on diagnostic criteria, up to 50% of patients with carcinoma developed peripheral neuropathy⁶⁾ and Treatment toxicity, tumorous infiltration, metabolic disturbances, or terminal cachexia account for most cases⁷⁾. Some of them are part of complex syndromes involving simultaneously the central and peripheral nervous system, the most frequent

of which is subacute sensory neuropathy/ paraneoplastic encephalomyelitis⁸⁾. Jean-christophe Antoine et al. suggested that most of the neuropathies which occurred within 2.5 years with a carcinoma correspond to known inflammatory disorders suggest that despite the absence of specific antibodies or other known immunological markers, tumors have in some way induced the immunological perturbations underlying the neuropathies³⁾.

Dimitri S. Monos et al. showed that the two forms of GBS, AIDP and AMAN, are characterized by independent HLA associations and each of these forms has a different HLA association than the other⁸. In his studies, increased number of AIDP patients has the DRB1*1301 allele, and alleles DRB1* 1301-03 and DRB1*1312 were increased in AMAN patients. Our patient has a DRB allele.

Although we could not perform the anti-onconeural antibodies because of absence of specific antibody in korea, clinical manifestation, electrophysiologic study and HLA study make the diagnosis of GBS more likely than paraneoplastic syndrome.

Treatment of GBS has evolved over the past two decades from supportive care only to the additive use of plasmapheresis and, more recently, IVIg. IVIg therapy has been shown to be an effective treatment for GBS, other inflammatory and autoimmune disorders. M. K. Sharief et al. suggested the mechanism of action of IVIg is selective modulation of circulating proinflammatory cytokines and recommended the high dose one cycled IVIg therapy⁹. Our patient received 0.4 mg/kg/ day of IVIg for 5 days and one more cycle 2weeks later. He showed more rapid improvement of muscular weakness after booster injection, and completely recovered. We have experienced an effective two cycled IVIg therapy, and it has not been reported.

Respiratory failure requiring mechanical ventilation is a common complication of Guillian-Barre syndrome and up to one-occurs in 14~44% of patients¹⁾. Generally

early tracheostomy was recommended to a patient applied ventilator in GBS to avoid some of the complications related to prolonged endotracheal tube placement. We performed tracheostomy at 10 day from weakness and there was no respiratory complication

Although we could not find an exact cause of the illness, we have experienced an effective two cycled IVIg therapy in GBS after resection of squamous cell lung cancer and herein report.

We here report a case of GBS after resection of squamous cell lung cancer, presenting as an acute form, recovered completely with 2 cycled immunoglobulin therapy.

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=국문초록=

면평상피 세포암으로 우측 폐 상엽 절제술을 시행한 68세 남자 환자에서 나타난 기엥바레 증후군에 대하여 보고하고자 한다. 환자는 수술 후 6일 째 갑작스럽고 양측성의 하지의 근력약화 및 호흡부전과 감각실조를 호소했다. 응급으로 기관내 삽관 후 인공환기기를 거치했다. 근력 약화 후 2일, 20일, 40일째 전기진단적 검사를 시행하였다. 운동신경전도장애가 현저하게 나타났다. 지속적인 운동말단신경잠시, 전도 시간의 분산, 부분적인 운동신경전도차단이 나타났으며 이와 같은 것들은 기엥바레 증후군의 진단적 특징이다. 보조적인 치료와 함께 정주적 면역 글로불린의 부가적인 사용을 시행하였으며 병세는 근력 약화 후 6주만에 회복되었다.

중심 단어: 1. 기엥-바레 증후군

- 2. 편평상피세포암
- 3. 정주적 면역 글로불린