Subacute Combined Degeneration of the Spinal Cord Concomitant with Gastric Cancer

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Abstract

We report a rare case of subacute combined degeneration of the spinal cord concomitant with gastric cancer. A 67-year-old man was admitted because of posterior column symptoms, pyramidal tract sign and peripheral neuropathy with severe hyperchromic anemia. He was treated with mecobalamin 1 mg IM, after which his anemia and neurological signs recovered. He was diagnosed as having subacute combined degeneration with pernicious anemia. Subsequent stomach biopsy revealed gastric cancer, and the patient underwent gastrectomy. It is a well known association that chronic atrophic gastritis is associated with gastric cancer or subacute combined degeneration. Our findings suggest that in this case subacute combined degeneration and gastric cancer are independent of each other; rather, both resulted from chronic atrophic gastritis.

Key words: subacute combined degeneration, pernicious anemia, gastric cancer

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Introduction

It is well known that pernicious anemia occurs due to a cobalamin deficiency after gastrectomy; however, it also occurs in cases of chronic atrophic gastritis without gastrectomy. Pernicious anemia is the end-stage of type A atrophic gastritis affecting the fundus and body of the stomach (1). Many chronic atrophic gastritis patients have autoantibodies to parietal cells and an intrinsic factor, and the pathogenetic mechanism of pernicious anemia is therefore thought to be an autoimmune mechanism (2). Infrequently, pernicious anemia is accompanied by gastric cancer or persistent carcinoid tumor (3-5). A number of epidemiological studies have addressed these conditions (6-9). Here, we report a case with subacute combined degeneration of the spinal cord and gastric cancer.

Case Report

A 67-year-old man presented with ulcer and phlegmone in the right lower extremity. He underwent surgery for phlegmone in another hospital in July 2003. After discharge, he noticed hand tremor. He had difficulty writing, sometimes dropped cups, and experienced paresthesia at the anterior tip of his tongue. In August, he noticed gait disturbance and was unable to walk without a cane. During the 2 weeks before admission, he experienced urinary incontinence. He was admitted to our hospital in September 2003. The patient’s past and family histories were unremarkable.

On admission, neurological examination revealed that he was alert, but had slightly dystarthric speech. The cranial nerves were within the normal limit. Manual muscle strength test revealed 4+/5-5, with the exception of the right lower extremity due to surgery. Deep tendon reflexes were diminished and plantar response was extensor on the left side. Gait was atactic and tandem gait was impossible. Romberg sign was positive. Finger to nose test revealed bilateral ataxia, in addition to bilateral dysmetria, and adiadochokinesis. Heel to knee test was also atactic. Sensory examination revealed hypesthesia to touch and pain in the distal portion of extremities. Position and vibratory sensations were notably decreased in the left lower limb. Athetotic movements were observed when the patient lifted his arms anteriorly and then closed his eyes.

Laboratory findings: WBC 4,400 μl, RBC 143×10^6 μl, Hb 875
6.2 mg/dl, Ht 17.5%, MCV 122 fl (normal range 83-98), ESR 42 /h, LDH 1,792 IU (80-170), serologic test for syphilis (-), cobalamin 66 pg/ml (233-914), folic acid 8.0 ng/ml (3.6-12.9), anti-parietal cell antibody 40x (<10x), intrinsic factor (-), gastrin 1,750 pg/ml (<200). The sensory and motor conduction velocities of the median and ulnar nerves were within normal limits, but in the lower extremities there was no electrical response of bilateral peroneal and tibial nerves in both sensory and motor nerve conduction studies.

Spinal MRI imaging: T2-weighted sagittal MRI images revealed a high intensity area in the posterior portion of the cord from the cervical to lumbar cord which was continuous without enhancement (Fig. 1). These high intensity areas faded slightly 3 weeks after beginning mecobalamin treatment. Brain MRI images revealed diffuse cerebral atrophy.

Hospital course: This patient was diagnosed with pernicious anemia; subsequently mecobalamin 1 mg IM was started every other day. After 3 weeks, neurological symptoms gradually improved and he was able to walk without any assist. Then, gastrofiberoscopy was performed to investigate the possibility of gastric cancer. This revealed chronic gastritis with 5 polyps and pathological findings compatible with adenocarcinoma. In November, gastrectomy was performed after recovery from anemia and neurological symptoms. Pathological findings confirmed tubular adenocarcinoma of moderately poorly differentiated type.

**Discussion**

Neurological findings suggested posterior column symptoms, pyramidal sign and peripheral neuropathy with severe hyperchromic anemia due to subacute combined degeneration in the spinal cord with pernicious anemia. While pernicious anemia is often accompanied by gastric cancer (6-9), we have seen only a report having a subacute combined degeneration and gastric cancer (10). In the present case, the patient possessed antiparietal cell antibody and hypergastrinemia: these data and gastrofiberscopic findings suggest atrophic gastritis A, which carries a risk of gastric cancer.

The pathognomotic mechanism of gastric cancer accompanied by pernicious anemia is thought to begin with inflammation associated with chronic atrophic gastritis seen in pernicious anemia that destroys parietal cells, thereby impairing gastric acid secretion. The lack of acid disables the negative feedback control of gastric acid on gastrin secretion by antral gastric cells. The resulting high gastrin levels stimulate proliferation and hyperplasia of both neuroendocrine cells of the fundic mucosa and nonspecialized epithelial cells, promoting the development of gastric carcinoid tumors and carcinomas (11).

MRI findings show intramedullary hyperintensity on T2-weighted images, which is thought to be mainly due to demyelination. By contrast, axonal loss is thought to occur later and to a lesser degree (12). There was no enhancement of the lesion in contrast studies, except for only a few cases (13, 14).

These results suggest that concomitant with subacute combined degeneration and gastric cancer is not a complication, but both that diseases develop from the same condition as chronic atrophic gastritis. We neurologists must carefully consider the possibility of gastric cancer when we encounter subacute combined degeneration.

**References**


Figure 1. A. Cervical cord T2-weighted sagittal MRI image (TR: 2,879, TE: 120). B. T2-weighted axial image (TR: 4,895, TE: 90). MRI images show a high intensity area in the posterior spinal cord from C-1 to L-1 (→).

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