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Acute-Onset Sensory Ataxic Polyradiculopathy With Root Enhancement in Spine MRI

Ki-Han Kwon
Yoo-ha Hong
Min Jun Song
Soo-Jin Cho

Department of Neurology,
Dongtan Sacred Heart Hospital,
Hallym University College of Medicine,
Seoul, Korea

Dear Editor,

A 62-year-old male was admitted to the neurology department because of unsteady gait and numbness of the whole body that had persisted for 7 days. The patient presented no history of common cold or diarrhea. The first symptoms were stiffness and tightness in the buttocks. A few days later, the symptoms manifested as pain and tingling sensation in the fingertips and toes. He eventually experienced numbness throughout the legs and consequently walked with an unsteady gait. Ultimately, he could not walk without assistance. A neurologic examination revealed sensory deficits to all modalities throughout his body except for the face. The touch sensation was significantly reduced at his fingertips and trunk. On the 7th day after admission, he showed athetoid movements of the fingers when stretching the arms with eyes closed. Deep tendon reflexes were initially normal, but they gradually reduced and finally were absent. A routine nerve conduction study (NCS) of both the upper and lower extremities showed normal findings. The cerebrospinal fluid showed elevated protein (145.1 mg/dL) with pleocytosis (17 cells/mm^3). The serum was negative for antiganglioside antibodies such as GM1, GD1b, GQ1b, and paraneoplastic antibodies. Brain MRI was unremarkable, but whole-spine MRI revealed enhancement of the lumbar nerve roots (Fig. 1). The somatosensory evoked potential (SSEP) presented no specific abnormalities. We treated the patient with intravenous immunoglobulin, after which his neurologic symptoms gradually improved, and he was able to walk without assistance after 2−3 weeks. During the 18-month follow-up, three NCSs showed no definite interval changes and normal.

When a patient shows abrupt ataxia without weakness, other conditions should be explored in the differential diagnosis. We excluded Miller Fisher syndrome due to the lack of ophthalmoplegia and anti-GQ1b antibody, and sensory ataxic Guillain-Barré syndrome (GBS) or ganglionopathy due to lack of any abnormalities in serial NCSs.[1-3] Bae and Kim reported a case with cerebellar ataxia with acute-onset motor axonal neuropathy, anti-GM1, and anti-GD1b.[4,5] However, our patient tested negative for antiganglioside antibodies and exhibited sensory ataxia, including a positive Romberg's test and athetoid movements of the fingers. Therefore, the ataxia was due to a sensory dysfunction rather than cerebellar dysfunction.

Before conducting the NCS, we thought this case might be a kind of ganglionopathy due to non-length-dependent sensory impairment: sensory symptoms at the buttocks before more-distal involvement of the toes or fingertips. Ganglionopathy may have idiopathic, toxic, paraneoplastic, and immunologic etiologies. Furthermore, ganglionopathy can produce absent or decreased amplitudes of sensory nerve action potentials in NCSs. However, the present case did not exhibit matching NCS and laboratory findings.

Vazquez Do Campo et al.[1] reported on a possible case of acute immune sensory polyradiculopathy (AISP). Their patient showed areflexia, normal NCS findings, and abnormal SSEP findings. Even though they demonstrated proximal involvement with an abnormal SSEP, they did not find nerve root involvement in a radiologic study. Our patient had abnormal enhance-

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Correspondence
Ki-Han Kwon, MD
Department of Neurology,
Dongtan Sacred Heart Hospital,
Hallym University College of Medicine,
7 Keunjaebong-gil,
Hwaseong 18450, Korea
Tel +82-31-8086-2310
Fax +82-31-8086-2709
E-mail neurokkh@hallym.or.kr

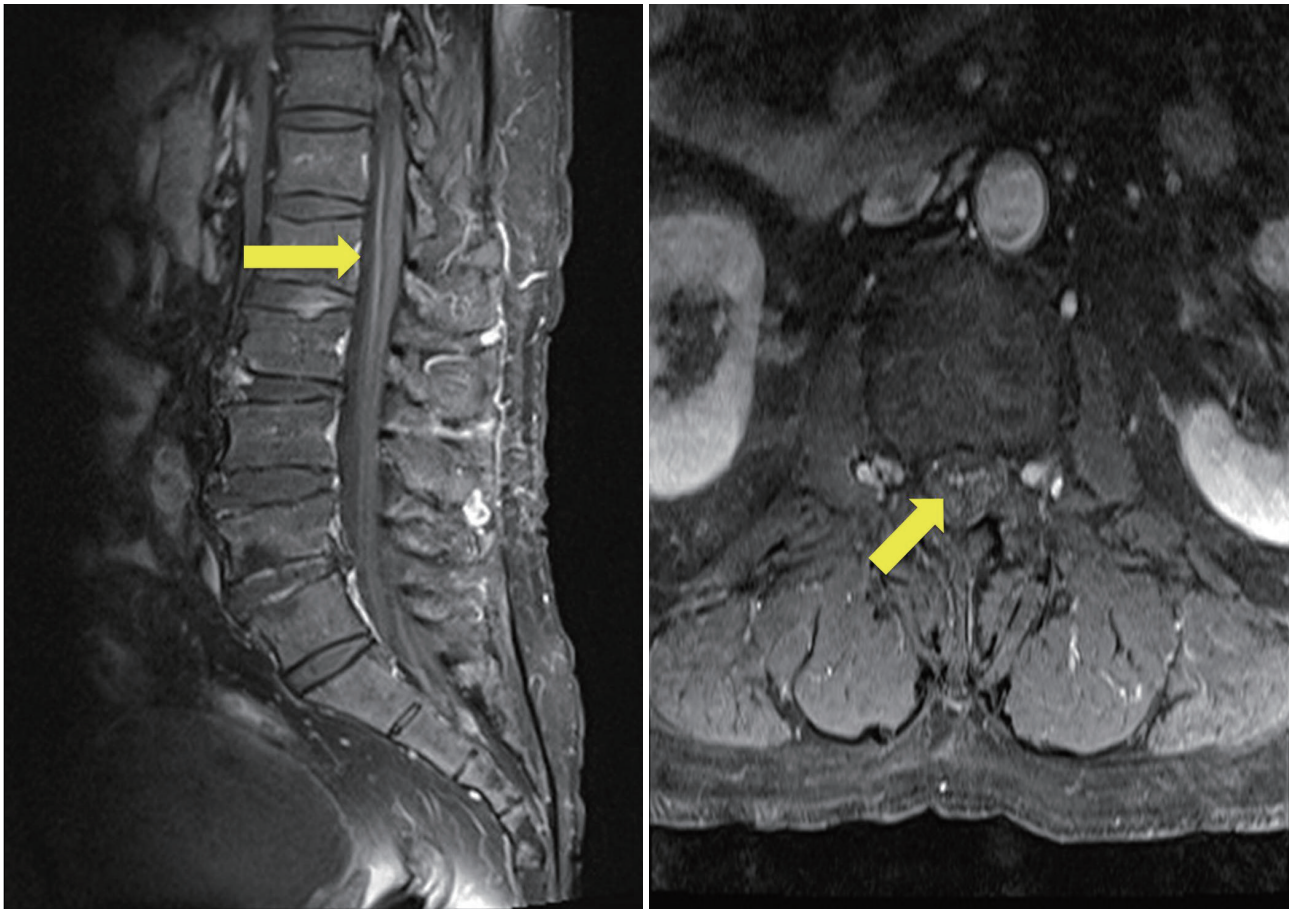

Fig. 1. T1 fat suppression-contrast spine MRI (sagittal and axial view). There is mild thickening and enhancement of nerve roots at cauda equine (yellow arrows).

ment of the nerve roots without a definitely abnormal SSEP test. We attribute the normal SSEP results to nerve impulses avoiding the lesion site due to tiny root enhancement. The complete clinical course of this patient was not consistent with chronic inflammatory demyelinating polyradiculoneuropathy, and follow-up studies did not show any progression or recurrence symptoms.[6]

We, therefore, consider the present patient to be the first case of AISP with strong radiologic evidence of root involvement. We cautiously suggest that like in a patient with chronic immune sensory polyradiculopathy, GBS also can involve only the very-proximal part of the entire length of nerve fibers, without the involvement of ganglionic or postganglionic sensory nerve fibers.

Ethics Statement

Written informed consent was obtained from the patient.

Availability of Data and Material

The datasets generated or analyzed during the study are available from the corresponding author on reasonable request.

ORCID iDs

Ki-Han Kwon https://orcid.org/0000-0001-6399-2236
Yoo-ha Hong https://orcid.org/0000-0002-8693-2831
Min Jun Song https://orcid.org/0000-0001-7416-9149
Soo-Jin Cho https://orcid.org/0000-0002-4053-3763

Author Contributions

Conceptualization: Ki-Han Kwon. Data curation: Min Jun Song. Formal analysis: Min Jun Song. Investigation: Ki-Han Kwon. Methodology: Ki-Han Kwon. Project administration: Ki-Han Kwon. Resources: Yoo-ha Hong. Supervision: Soo-Jin Cho. Validation: Ki-Han Kwon. Visualization: Yoo-ha Hong. Writing—original draft: Ki-Han Kwon. Writing—review & editing: Yoo-ha Hong, Soo-Jin Cho, Ki-Han Kwon.

Conflicts of Interest

The authors have no potential conflicts of interest to disclose.

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