A Case of Acute Sensory Neuropathy Associated with Contrast Enhancement of the Cauda Equina on Magnetic Resonance Imaging

Toshinari Kobayashi, Makoto Shiraishi, Shinichiro Nemoto, Yoji Nomura, Munefumi Hotta, Takaaki Suzuki, Tsutomu Kamo, Yoichi Takahashi, and Mamoru Tadokoro

(Received for Publication: January 24, 2005)

Abstract

A 46-year-old woman experienced dysesthesia and pain in the lower limbs one week after cold-like symptoms. On admission, she presented with superficial sensation disturbance without deep sensation disturbance. Bilateral tendon areflexia was observed, but muscle strength was maintained at the normal level, and no dysautonomia including vesicorectal disturbance was observed. Her cerebrospinal fluid (CSF) showed albuminocytologic dissociation. Peripheral nerve conduction velocity was normal in motor nerves, while sensory conduction velocity was absent in the peroneal and sural nerves. T1-weighted lumbar spine magnetic resonance imaging with gadolinium revealed enhancement of the cauda equina. From these findings, the patient was diagnosed as having acute sensory neuropathy. Her symptoms subsequently progressed; sensory disturbance extended to her upper limbs after four weeks from onset. Following the administration of steroids and intravenous immunoglobulin, sensory disturbance and CSF abnormalities improved. The enhancement also disappeared within two months. In this case, blood-nerve barrier of the cauda equina was extensively disrupted. Abnormal immunological mechanisms may be involved in the pathogenesis of the disease.

Key Words

Acute sensory neuropathy, MRI, Cauda equina

Introduction

Acute sensory neuropathy (ASN) is a sensory disturbance with poor prognosis. Following episodes of infections, deep sensation disturbance without motor paralysis occurs relatively rapidly and leads to ataxia of extremities and trunk. We report our experience of an ASN case with enhanced cauda equina on magnetic resonance imaging (MRI).

Case Report

Patient: a 46-year-old woman.
Chief Complaints: numbness and pain in lower limbs.
Past History: nothing significant.
Family History: nothing significant.
History of Present Illness: First, fever of 39–40°C and headache developed. On the following day, a rash developed mainly on the lower body. The patient was seen by a physician, who prescribed...
cold medicine. The numbness of the lower limbs persisted, and 4 days later, the patient started to have difficulties in walking because of the pain. Although her fever resolved in a few days, symptoms of the lower extremities did not improve. She was referred to us and admitted to our hospital.

**Physical Examination:** There were no abnormal findings on general physical examination. Neurological examination revealed complete loss of deep tendon reflexes but no decrease in muscle strength of the extremities. The patient had a tingling sensation (dysesthesia) from the thighs (L1 level) to the toes. Deep sensation as well as the autonomic nervous system was normal.

**Laboratory Studies:** Blood and urine analyses were normal. Cerebrospinal fluid (CSF) showed albuminocytologic dissociation with a cell count of 28/mm$^3$ and protein at 300 mg/dl. Myelin basic protein and immunoglobulin G (IgG) levels in CSF were increased, 7.0 ng/ml and 70 mg/dl, respectively. In the serological examination, tumor markers, antineuronal antibodies, anti-GM1 ganglioside, and autoantibodies including anti-SS-A antibody and anti-SS-B antibody were negative. Titers of antibodies including those against cytomegalovirus, influenza virus, and herpes virus were within normal ranges.

**Electrophysiological Studies:** Motor nerve conduction velocities and motor amplitudes were normal in upper and lower limbs. Sensory nerve conduction velocities were normal in upper limbs but absent in the peroneal and sural nerves.

**Imaging Studies:** MRI performed two weeks after the onset (Fig. 1). Thickening with enhancement was noted in the spinal cord from L1 to L5 on sagittal T1-weighted image (Fig. 1b, d).

**Pathological Studies:** A biopsy from the sural nerve was performed. The specimen stained with the Luxol fast blue and Bodian methods showed severe deficit of myelinated nerves of various sizes as well as fibrosis and myelin-like structures (Fig. 2a). Osmium staining of unwinding nerve fibers revealed many myelin spherules along the length of the fibers (Fig. 2b).

**Clinical Course** (Fig. 3): Based on the preceding infectious episode, acute onset of a sensory distur-

---

Fig. 1. a, Sagittal T1-weighted MRI. b, Abnormal enhancement was noted at the spinal cord from level L1 to L5 on sagittal T1-weighted image with gadolinium (Gd) contrast (arrow). c, Axial T1-weighted image of the spinal cord at L1 level. d, Axial T1-weighted MRI with Gd contrast (the same level as c). Thickening with enhancement was noted in the posterior column (arrow).
bance at and below the L1 level without reduction of muscle strength, and the highly increased levels of protein and IgG in the CSF, the patient was diagnosed as having ASN. Although steroid pulse therapy transiently alleviated the dysesthesia in the lower limbs and reduced the protein and IgG levels in the CSF, the symptoms recurred. Therefore, this suggested that the ASN in this case involves axonal degeneration as well as demyelination.

Generally, the main etiology of ASN is the damage to dorsal root ganglion cells. It is, however, difficult to identify from electrophysiological findings whether the main lesions are on dorsal-root ganglion cells or axons of sensory nerves. Taking advantages of recent advanced diagnostic imaging technology, Wada M\(^6\) revealed that the dorsal root of the spinal cord was enhanced on cervical MRI and suggested demyelination caused by immune response-associated breakdown of the blood-nerve barrier as the mechanism of ASN. In our case, lumbar MRI demonstrated extensive enhancement of the cauda equina. Spinal roots that reach out of the epidural space to dorsal root ganglion are normally enhanced due to the lack of blood-nerve barrier. On the contrary, enhancement of the cauda equina in the subarachnoid space is hindered by blood-nerve barrier; only a subtle enhancement, if any, is noted compared with the plain T1-weighted images\(^8\). The cauda equina in this case was apparently enhanced, indicating the destruction of blood-nerve barrier at the enhanced levels. Midori G\(^9\) has reported that enhancement of the cauda equina on MRI was found in 11 out of 16 chronic inflammatory demyelinating polyradioneuropathy (CIDP) cases, and the MRI findings in the present case resembled those of CIDP cases.

Discussion

ASN is characterized by abrupt onset of sensory disturbance. Based on the electrophysiological and pathological findings, Windebank AJ\(^2\) and Kondo M\(^4\) have attributed its pathogenesis to primary axonal damage due to impaired dorsal root ganglion. On the other hand, Oh S\(^5\) and Dawson DM\(^6\) have reported that clinical signs and their electrophysiological studies indicated involvement of demyelination in the proximal and distal sensory nerves including nerve roots. Oh S\(^5\) investigated eight cases of neuropathy, known as the sensory Guillain-Barre syndrome, and presented eight criteria to show the association of the demyelination mechanism. Our case differed from those in previous studies in that: The patient’s symptoms continued to progress for longer than four weeks, nerve conduction velocities were absent, and the symptoms recurred. Therefore, this suggested that the ASN in this case involves axonal degeneration as well as demyelination.

Generally, the main etiology of ASN is the damage to dorsal root ganglion cells. It is, however, difficult to identify from electrophysiological findings whether the main lesions are on dorsal-root ganglion cells or axons of sensory nerves. Taking advantages of recent advanced diagnostic imaging technology, Wada M\(^6\) revealed that the dorsal root of the spinal cord was enhanced on cervical MRI and suggested demyelination caused by immune response-associated breakdown of the blood-nerve barrier as the mechanism of ASN. In our case, lumbar MRI demonstrated extensive enhancement of the cauda equina. Spinal roots that reach out of the epidural space to dorsal root ganglion are normally enhanced due to the lack of blood-nerve barrier. On the contrary, enhancement of the cauda equina in the subarachnoid space is hindered by blood-nerve barrier; only a subtle enhancement, if any, is noted compared with the plain T1-weighted images\(^8\). The cauda equina in this case was apparently enhanced, indicating the destruction of blood-nerve barrier at the enhanced levels. Midori G\(^9\) has reported that enhancement of the cauda equina on MRI was found in 11 out of 16 chronic inflammatory demyelinating polyradioneuropathy (CIDP) cases, and the MRI findings in the present case resembled those of CIDP cases.

Fig. 2. a. Cross section of the biopsied sural nerve (100×, A section stained by the Luxol fast blue and Bodian methods). Note marked deficit in myelinated fiber density, some degenerated fibers. b. Teased-fibers by Osmium staining (100×) revealed many myelin spherules along the length of the fibers.
The prognosis of ASN is usually poor\(^1\). One of the reasons for poor response to treatment may be that dorsal root ganglion lacks tight junctions between vascular endothelial cells and is vulnerable to humoral factors\(^3\). However, a concrete definition or established criteria have not yet been offered.

Considering the treatment of the present case, in which intravenous administration of steroids and immunoglobulin alleviated symptoms and improved findings on the follow-up MRI, treatment for peripheral demyelinating diseases may be appropriate for cases of ASN.

In conclusion, the clinical course and neuroimaging findings in this case suggested that ASN is a disease entity that involves a wide variety of pathologies and abnormal immunological mechanisms.

**References**

6) Dawson DM, Samuels MA, Morris J. Sensory
MRI にて馬尾の造影効果を認めた急性感覚性ニューロパチーの 1 例

抄 録
症例は 46 歳女性である。感冒様症状出現の 1 週間後、両下肢に異常感覚が出現した。当院受診時に深部感覚障害を伴わない感覚障害と両側深部腱反射の消失を認めた。筋力低下はなく、膀胱直腸障害などの自律神経障害も認めなかった。脳脊髄液所見上、蛋白細胞解離を認め、末梢神経伝導速度では、運動神経は正常範囲、感觉神経は腓骨および腓腹神経で導出不能であった。腹部 MRI では、ガドリニウム造影 T1 強調像で馬尾に造影効果を認めた。これらのことから急性感覚性ニューロパチーと診断した。本症例の経過は、発症 4 週以降にも上肢に異常感覚が出現するなどの進行を認めたが、ステロイドと免疫グロブリン大量投与により異常感覚と髄液所見が改善し、2 ヶ月後には造影効果が消失した。本症例の発症機序として、広範囲に馬尾の blood-nerve barrier が破壊される免疫異常の関与が考えられた。

小林 優成 1 白石 真 1 根本慎一郎 1 野邑 陽二 1
深田 宗文 2 鈴木 孝昭 2 加茂 力 2 髙橋 洋一 2
田所 衛 3

1 聖マリアンナ医科大学横浜市西部病院 内科学（神経内科）
2 聖マリアンナ医科大学 内科学（神経内科）
3 聖マリアンナ医科大学 病理学