

Acute Polyradiculoneuropathy Associated with *Salmonella* Gastroenteritis

Hideaki KONDO, MD¹⁾, Kunihiko NAGASATO, MD¹⁾, Toshiro YOSHIMURA, MD¹⁾, Tatsufumi NAKAMURA, MD¹⁾, Mitsuhiro TSUJIHATA, MD²⁾ and Shigenobu NAGATAKI, MD¹⁾

1) The First Department of Internal Medicine, Nagasaki University school of Medicine

2) The school of Allied Medical Sciences, Nagasaki University

We reported a case of acute polyradiculoneuropathy associated with *Salmonella* gastroenteritis. A 68-year-old man developed progressive motor weakness and areflexia following the febrile illness and diarrhea caused by a strain of *Salmonella* species O8 group. He showed a rapid and complete recovery from the illness. This is the first report in which *Salmonella* gastroenteritis might be etiologically related to an acute polyradiculoneuropathy.

Key words: *Salmonella* species O8 group, acute polyradiculoneuropathy, Guillan-Barré syndrome

Introduction

Acute inflammatory demyelinating polyradiculoneuropathy (AIDP) is an acutely or subacutely evolving paralytic disease of undetermined etiology. Two-thirds of patients give a history of antecedent acute infectious illness. Most common antecedent illnesses are viral infections, especially an influenza-like upper respiratory infection^{1,2)}. Some AIDP patients are associated with *Campylobacter jejuni*^{3,4)} and *Mycobacterium pneumoniae*⁵⁾ infections, but only scattered reports have appeared in which AIDP has occurred against a background of other bacterial infections such as typhoid fever^{6,7,8)}, paratyphoid⁹⁾ tularemia¹⁰⁾, tuberculosis¹¹⁾ and so on.

We experienced a patient with acute polyradiculoneuropathy (PRN) associated with *Salmonella* species O8 group gastroenteritis. This is the first case of acute PRN suggesting the contribution of this organism to acute PRN.

Case report

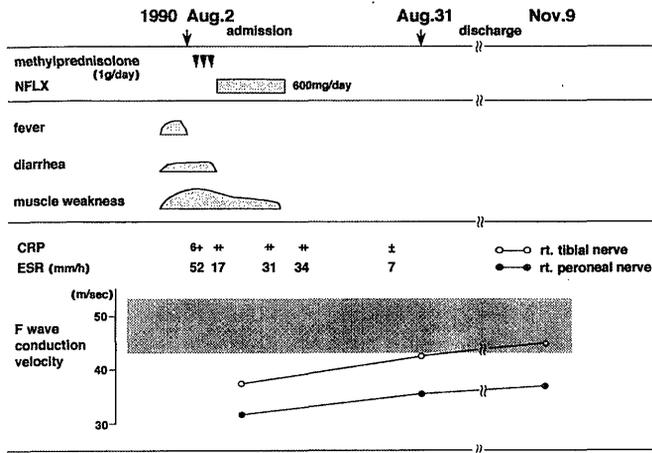
A 69-year-old man was admitted to our hospital on August 2, 1990, complaining of difficulty in walking and a rapidly progressive weakness of all limbs of 4 days' duration. There was no history of exposure to insecticides or heavy metals. He had had no vaccinations. He had eaten half-roasted chicken 4 days before the onset of weakness. Three days later, he had suffered from fever followed by watery diarrhea dark brown in color. On the following day, weakness of the legs with minimal paresthesia developed from the distal portions of the extremities and progressed proximally until he was unable to stand up, and felt paresthesia in the hands and fingers. There was no history of incontinence or urinary retention.

On examination he was febrile (38.5°C) and blood pressure was 120/78mmHg. Tenderness was observed at the right lower abdomen. Neurological examination revealed weakness of the lower extremities that was prominent in the distal muscles and minimal weakness in the hands. Glove and stocking type paresthesia was observed in the extremities. Ankle jerks were somewhat diminished. Pathologic reflexes were not elicited.

Investigations revealed a normal hemogram except for a considerable increase of polymorphs (5460/mm³) and decrease of lymphocytes (360/mm³). Erythrocyte sedimentation rate was 52mm/hour. Serum electrolytes were normal. Cerebrospinal fluid (CSF) examination disclosed 2 mononuclear cells per cubic millimeter and an elevated protein level of 113mg/dl. The stool culture was positive for a strain of *Salmonella* Sp. O8 group. Nerve conduction studies showed that F-wave conduction velocities (FWCV) were slow in the tibial and peroneal nerve; 37.3m/sec and 31.5 m/sec, respectively. Motor and sensory nerve conduction studies, however, showed no abnormality in velocities and amplitudes of the evoked potentials.

We treated the patient by steroid pulse therapy (methylprednisolone 1000mg daily for 3 days) combined with an administration of norfloxacin 600mg daily for 9 days.

Reprint requests should be sent to Dr. Shigenobu Nagataki, the First Department of Internal Medicine, Nagasaki University School of Medicine, 1-7-1, Sakamoto-machi, Nagasaki 852, Japan



Figure

His muscle strength improved rapidly and returned to almost normal on the 13th day of hospitalization, but FWCV remained slow in the tibial and peroneal nerve; 40.0m/sec and 34.2m/sec, respectively (figure). Motor and sensory nerve conduction velocities were in the normal range during these period.

Discussion

The rapid onset of symmetrical ascending motor weakness, areflexia, slowed FWCV and albuminocytological dissociation in CSF satisfy the diagnostic criteria of AIDP^{1,2)}. This case, however, is atypical for AIDP in some points. Onset of weakness was so fast as the next day of the triggering event (*Salmonella* gastroenteritis) or 4 days after eating half-roasted chicken. The interval between the antecedent infection and the onset of AIDP symptoms varies from 3 to 36 days; most frequently it is 1 to 3 weeks¹⁾. Several mechanisms for the pathogenesis of AIDP have been postulated and the most have in common an immunological basis¹⁾. A direct attack by infectious agent on the Schwann cell seems unlikely and it is difficult to envisage how *Salmonella* could find its way to nerves. Many of the viruses implicated in AIDP incorporate host cell membrane into their coats and such membrane components might conceivably be converted from haptens into immunogens. This postulate, however, will not explain the

cases in AIDP occurring after bacterial infection. The possibility that AIDP depends on molecular mimicry between antigens of infectious organisms and components of peripheral nerve myelin. This mechanism is proposed in the cases of AIDP occurring after *Campylobacter jejuni* infection^{3,4)}. There is, however, no evidence in the case of *Salmonella* infection. Another possible mechanism proposed in the AIDP following *Campylobacter jejuni* and *Salmonella typhi* infection^{3,4)} is that endotoxins produced by these organisms bind to nerve roots and induce demyelination and finally immune reactions resulting AIDP. If we consider any mechanisms on immunological basis, the timing of the onset of AIDP has to correspond to the interval, one to three weeks after an antecedent illness, required to mount an immune response. However, the interval of the onset of PRN and antecedent illness in this case is too short to consider the mechanisms mentioned above. Finally we postulated the mechanism in this case that an endotoxin produced by *Salmonella Sp. O8* group induced an acute PRN resembling AIDP as a direct toxic action on the nerve root rather than immune response.

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