

gen, and alpha fetoprotein, were negative. The x-ray films of the chest and computed tomography scan of the thorax and abdomen were normal. The patient was treated with subcutaneous heparin and diclofenac, and fever and migratory thrombophlebitis subsided.

Because the patient had been working with manure several days before his initial symptoms, Q fever serologic testing was requested. The antibody levels measured by complement fixation (CF) against phase II *Coxiella burnetii* antigen was 1:512. By indirect immunofluorescence, the titers of IgM and IgG against phase I and II were 1:64 and 1:512 and 1:256 and >2,048, respectively. Antibody titers against *Mycoplasma*, *Chlamydia*, *Legionella*, enterovirus, and influenza were negative.

Recovery was uneventful and the patient was asymptomatic during a follow-up visit 3 weeks later. Antiphospholipid antibodies were negative. Three months after the acute phase of the infection, new titers of antibodies (CF) against *C. burnetii* were 1:128. Two years after the episode the patient was asymptomatic.

This patient is unique in that he had acute Q fever with migratory thrombophlebitis. A diagnosis of Trousseau's syndrome associated with an occult malignancy was considered on admission, but it was excluded soon. The recent history of exposure to manure was the key for the clinical diagnosis. Although specific anti-coxiella treatment was not given, the patient followed a self-limited course, and both clinical and laboratory abnormalities promptly subsided.

Microscopic vasculitis and thrombosis are commonly found in patients with other rickettsial infections (5), but vascular phenomena must be considered an exceptional event in patients with Q fever. However, thrombophlebitis and pulmonary embolisms have been occasionally reported (6–8). These unusual mani-

festations have been associated with aPL during the course of acute Q fever (7,8).

Antibodies to phospholipids have been found in 80% of patients in a large series of acute Q fever (9). None of the patients in the study showed thrombotic events or cardiac valve involvement in contrast to patients with lupus or primary aPL syndrome in whom clinical manifestations attributed to aPL developed (9). This observation could be explained by the fact that aPL found in patients with lupus and primary aPL syndrome are directed against a complex antigen that includes a lipid-binding inhibitor of coagulation, which has been characterized as a  $\beta$ 2-glycoprotein I (apolipoprotein H). This glycoprotein seems to inhibit the activation of the contact phase system of the intrinsic pathway of blood coagulation (10). On the other hand, apolipoprotein H is not necessary for the aPL activity observed in patients with Q fever and other infectious diseases (10).

According to these studies, the observation of low titers of aPL in the serum of our patient during the acute phase of Q fever must be seen as a finding of uncertain importance not necessarily associated with migratory thrombophlebitis. In short, migratory thrombophlebitis (Trousseau's syndrome) should be added to the ever-growing list of unusual manifestations of Q fever.

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## West Nile Poliomyelitis

**To the Editor:** In the July 2003 article, "Acute Flaccid Paralysis and West Nile Virus Infection" (1), Sejvar et al. reported seven patients with acute onset of asymmetric weakness and areflexia but no sensory abnormalities. The authors also referenced three previously reported cases of West Nile virus (WNV)-associated flaccid paralysis and argued that all of these symptoms could be explained by anterior-horn cell loss. The two

cases of spinal cord pathologic findings published to date demonstrated focal loss of anterior-horn neurons (2,3). We report a case of West Nile poliomyelitis with preserved deep-tendon reflexes, diminished sensory nerve action potentials, and pathologic findings which do not localize to the anterior horn.

An 83-year-old woman sought treatment at the hospital on September 12, 2002, with 3 days of fever, acute confusion, nausea, vomiting, and profound weakness. Computed tomography (CT) of the head was unremarkable. Her examination was notable for dysarthria, tremors, and global weakness with rightsided predominance. Cerebrospinal fluid contained 75 leukocytes/mm<sup>3</sup> (84% neutrophils), glucose 88 mg/dL, and protein 97 mg/dL. On the second hospital day, respiratory failure developed, requiring mechanical ventilation. Electrodiagnostics performed on hospital day 7 demonstrated reduced motor and sensory amplitudes on right median and ulnar nerves, reduced motor amplitudes, and mildly reduced conduction velocities in the right peroneal nerve and right posterior tibial nerves. These findings suggested a predominantly axonal polyneuropathy involving both sensory and motor nerves. No myopathic process was demonstrated. Serum antibodies for WNV (immunoglobulin [Ig] M capture enzyme-linked immunosorbent assay [ELISA] 73.8; IgG capture ELISA 0.738) and cerebrospinal fluid antibody titer (IgM capture ELISA 200.5) were both positive. Weakness, respiratory failure, and preserved deep-tendon reflexes persisted.

On hospital day 15, the patient died after withdrawal of support. Sections from the postmortem medulla and spinal cord were positive for WNV RNA by reverse transcription-polymerase chain reaction testing at the state laboratory. The brain demonstrated characteristic microglial nodules and perivascular lymphocytic infiltrates (4). Spinal cord sections showed leptomeningeal and parenchymal chronic inflammatory infiltrates, often perivascular in location. Patchy cellular infiltration was found throughout the spinal cord, without a predilection for the anterior-horn. Spinal nerve roots demonstrated focal lymphocytic inflammation within the endoneurial compartment. No evidence of a demyelinating process was found.

Our data contradict the thesis proposed by Sejvar et al. that West Nile poliomyelitis is restricted to the anterior-horn (1). The electrodiagnostics showing axonal polyneuropathy and the spinal cord pathologic findings, which did not demonstrate focal loss of anterior-horn neurons, suggest a broader spectrum of the clinical-pathological syndrome of West Nile poliomyelitis than previously described (2,3). Our findings conform to the hypothesis outlined by Jeha et al., which favors a more widespread myelitis (5). We also confirm the findings of preserved deep-tendon reflexes in West Nile poliomyelitis first reported by Glass et al. (6).

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**In Reply:** The letter by Holman, et al., (1) in this edition of *Emerging Infectious Diseases* continues to broaden the scope of illness attributable to West Nile virus (WNV) infection. However, we would like to take the opportunity to clarify the conclusions drawn from our article, "AFP and West Nile virus infection" (2). In this article, we reported on seven patients, all of whom had asymmetric weakness without sensory loss, which evolved acutely over the course of several hours. Electrodiagnostic studies of all patients displayed markedly reduced compound motor axon potentials with preserved sensory nerve action potentials; the results were interpreted as consistent with a process primarily localized to the motor axons or, far more likely, given the clinical scenario and pathologic data from WNV-infected animals, the anterior horn cells of the spinal cord. These findings were later substantiated by Li et al. (3). Subsequently, Jeha et al. expanded the scope of illness producing WNV-associated flaccid paralysis by describing several patients with predominant myelodradiculitis (4).

The patient presented by Holman clearly seems to have a condition that, as shown by electrodiagnostics and pathology, localizes to other areas, in addition to spinal anterior horn cells.

However, rather than suggesting that a single case report “contradicts” the anterior horn cell hypothesis, we suggest that WNV-associated flaccid paralysis be viewed as having a spectrum of causes, one of which certainly is poliomyelitis like illness.

The neuropathologic findings in poliomyelitis (due to poliovirus, WNV, or other viruses) are not restricted focally to the anterior horn cells (5). Demonstrating pathologic changes as well as focal anterior horn cell loss in the patient referenced by Holman is in keeping with neuropathologic findings in poliomyelitis. Additionally, the presence of a diffuse axonal polyneuropathy cannot be concluded from Holman’s data. Reduced compound motor axon potentials and slowing of conduction velocity could certainly be seen in pathologic conditions affecting anterior horn cells or spinal nerve roots. In addition, reduced sensory nerve action potentials in the median and ulnar nerves alone, without documentation of neuropathy in additional nerves, cannot be used as evidence of a diffuse axonal polyneuropathy, since both of these nerves are commonly prone to entrapment neuropathies. Finally, the context in which the preservation of this patient’s reflexes is observed remains unclear. Preserved “normal” deep-tendon reflexes, in the setting of disease that interrupts the reflex arc at any point, are incongruous with established physiologic and clinical concepts.

The seven patients observed by our group clearly had a distinct clinical syndrome with similar clinical findings and electrodiagnostic results. However, as demonstrated by prior reports (3,5–7), multiple mechanisms may lead to WNV-associated flaccid paralysis. In fact, we acknowledge a spectrum of cord, root, and nerve involvement with WNV flaccid paralysis.

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## Typhus Group Rickettsiae Antibodies in Rural Mexico

**To the Editor:** In 2002, the risk of transmission of epidemic typhus in the

state of Mexico was assessed by analyzing serum specimens from 393 residents of previous typhogenic areas for immunoglobulin (Ig) G antibodies against *Rickettsia prowazekii*. Louse-borne typhus has been a historic scourge in Mexico. In 1576, in a population of 9 million, 2 million deaths were attributed to epidemic typhus (1). These illnesses primarily affected indigenous peoples, who called the illness *cocolixtle* and *matlazahuatl* (2).

In 1951 a national campaign against louseborne typhus was begun by using newly developed technological approaches, antibiotics, and insecticides, resulting in decreases in the incidence and case-fatality rate. In 1951 >1,000 cases and 737 deaths caused by epidemic typhus were reported in 18 states, and 6,781 localities were identified as at risk (3). By 1965, only 36 cases and no deaths were reported from 12 states with 4,841 localities at risk. Most cases occurred during the cold months of November–April. One third of cases occurred in persons 19–29 years of age with nearly 40% of the deaths in patients aged 15–44 years. In 1979, 10 years had passed without any cases of epidemic typhus reported in Mexico. In the 1980s, three outbreaks of typhus occurred in rural communities, two in Chiapas and one in the state of Mexico (4).

In the state of Mexico, during the period 1893–1907, 7,353 epidemic typhus deaths were reported (annual mortality rate, 52.4/100,000 population); from 1939 to 1943, 1,220 cases were reported with 707 deaths (annual mortality rate, 12.1/100,000 population); and from 1959 to 1963, 64 cases were reported with 14 deaths (annual mortality rate, 0.1/100,000 population) (3). In 1967, Atlacomulco, a county in the state of Mexico that had been free of typhus for 5 years, experienced an outbreak of louseborne typhus associated with a case of Brill-Zinsser disease in a 76-year-old man who had a history of