MR Imaging in a Case of Postvaccination Myelitis

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Summary: We describe a case of acute transverse myelitis after the administration of the recombinant form of hepatitis B vaccine. Abnormal enhancement on MR imaging accompanied residual neurologic deficit.

Index terms: Myelitis; Drugs, reaction

Acute transverse myelitis is an inflammatory disorder of the spinal cord associated with many disease processes. The pathogenesis is unclear, but the probable mechanism involves an autoimmune phenomenon (1). Myelitis has rarely been reported secondary to vaccinations, including rabies (2), smallpox (3), influenza (4), rubella (5), and the plasma-derived form of hepatitis B (6). We present a case of acute transverse myelitis, which seems secondary to the administration of the recombinant form of hepatitis B vaccine. The magnetic resonance (MR) findings are described and correlated with the residual neurologic deficit.

Case Report

A 40-year-old male health care worker presented with a 6-week history of progressive lower-extremity numbness and difficulty walking. Symptoms began 2 weeks after receiving the first dose of the recombinant form of hepatitis B vaccine. After administration of the second dose, 1 month later, the sensory disturbance ascended to the nipple level, and the patient had difficulty walking. Physical examination revealed markedly impaired proprioception and vibration sense, minimal weakness, hyporeflexia in the lower extremities, and a T-4 sensory level.

MR was performed at admission on a 1.5-T magnet. T1-weighted (500/11/4 [repetition time/echo time/excitations]) and T2-weighted (2000/30,80/2) spin-echo pulse sequences revealed a swollen edematous cord extending from C-3 to T-9. On T1-weighted images there was diffuse hypointense signal relative to the spinal cord, whereas on T2-weighted images there was diffuse increased signal in the same regions. Postgadolinium images showed extensive enhancement isolated to the posterior columns from C-6 to T-8 (Fig 1A–D). His residual deficit consists of moderate posterior column dysfunction corresponding to the area of gadolinium enhancement.

Discussion

Neurologic complications after vaccination are well known but rare. They include seizures, Guillain-Barré syndrome, peripheral neuropathy, cranial nerve palsies, transverse myelitis, and encephalopathy (2, 3, 4, 6). In this case of transverse myelitis, although pathologic proof is not possible, the striking temporal relationship between symptoms and the two doses of hepatitis B vaccine strongly suggests that the vaccine was the cause.

The MR findings in postvaccination transverse myelitis have not been described but are probably similar to those changes seen secondary to transverse myelitis of other causes. MR findings described in acute transverse myelitis include cord expansion (7), increased signal on T2-weighted images (7, 8), and an enhancement pattern that can be normal, diffuse, peripheral, or slightly nodular (8). T1-weighted images are usually of normal or slightly increased signal intensity, possibly secondary to petechial hemorrhages, although decreased signal has been described (8). In our case, diffuse hypointense signal on T1-weighted images and diffuse hyperintense signal on T2-weighted images was seen throughout the cord from C-3 to T-9, as well as diffuse cord swelling. The abnormal enhancement was localized to the posterior columns, and this corresponded to his predominant and persistent deficit. A previous report also showed a relationship between location of enhancement and persistent deficit (9). In another instance, progressive decrease in en-
enhancement corresponded to clinical improvement (8). A larger series would be required to determine whether enhancement characteristics or temporal changes are predictive of residual deficit. It is possible that the MR appearance may have been different in our patient had he been imaged more acutely. It is not known whether posterior column enhancement and/or white matter tract involvement is a characteristic of postvaccination myelitis or a unique finding in this patient.

Currently, the recombinant form of hepatitis B vaccine may be given to people at high risk for exposure, as well as to certain people who have been acutely exposed. Hepatitis B vaccine has obvious benefits outweighing the possible rare complications. However, although associated severe neurologic complications are extremely rare, they may be underreported, because there is usually a delay in symptom occurrence, and an idiopathic or postviral cause of symptoms would be difficult to disprove. The administration of the recombinant form of hepatitis B vaccine usually consists of multiple doses. Although it is impossible to identify those who may eventually develop neurologic complications, it is important to identify early signs of an untoward reaction. If, as in our patient, symptoms occur after the initial dose, administration of further doses should be carefully considered.

References

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