Dear Editor;

Herpes zoster (HZ) is a common neurocutaneous disease resulting from reactivation of dormant varicella-zoster virus (VZV) in sensory ganglions. Factors weakening immune function may increase HZ and risk of neurological complications (1,2). Infrequent neurological complications such as transverse myelitis (TM), encephalitis, radiculoneuritis may develop more frequently in immune-compromised patients. Rarely, multiple neurological involvement can occur (1,2,3). Here, we report a case with TM and polyradiculitis related to HZ while he was under immunosuppression after liver transplantation.

A 54-year-old man was admitted to our clinic with pain and right leg weakness complaints. Twenty days ago, he developed burning, itchy vesicular eruption on his right leg, diagnosed with HZ, valacyclovir tablet and acyclovir cream was prescribed. Ten days after the rash, progressive weakness on right leg and numbness in below waist appeared.

He had chronic hepatitis B virus infection resulting in hepatocellular carcinoma and underwent liver transplantation fourteen months ago. He also had type-II diabetes mellitus for one year. His medications included prednisolone (10 mg/d), tacrolimus (20 mg/d), mycophenolate mofetil (2 g/d), insulin, entecavir (0.5 mg/d), ursodeoxycholic acid (1 g/d). High fever, crusted shingles rash on the right L5-S1 dermatome were determined in physical examination. There was muscle weakness in the right tibialis anterior 0/5, right gastrocnemius 3/5 and right quadriceps femoris 4/5. Diminished pain sensation and light touch was noted below T6 dermatome. Proprioception and vibratory sensation were determined to be lost to the knees. Deep tendon reflexes increased in the lower extremities. Laboratory findings disclosed as: ESR: 40 mm/h (<20); procalcitonin, 0.1 ng/ml (<0.05); white-blood-cells count, 15300/mm³ with normal cell differentiation. Liver function-tests slightly increased.

Right patella and Achilles reflexes were hypoactive during the neurologic examination 5 days after admission to our clinic. EMG was performed revealing reduced amplitudes of compound muscle action potentials with normal nerve conduction velocity and prolonged F-wave latencies on bilateral peroneal and posterior tibial nerves. Bilateral sural nerve conduction studies were normal. Needle EMG revealed abnormal spontaneous activity and reduced recruitment in right medial gastrocnemius. Lumbo-sacral magnetic resonance imaging (MRI) revealed no abnormality.

Cervical-thoracic MRI, performed 22-day after the rash, showed contrast enhancing T2-hyperintense intramedullary lesions extending caudally from C2-C3 consistent with myelitis (Figure 1). The cerebrospinal fluid (CSF) analysis showed normal opening pressure; 26 WBCs per mm³ of which 90% were mononuclear cells; 2950 red-blood-cells per mm³; protein 608 mg/L and glucose 71.5 mg/dL (serum glucose was 219). Acid-fast and gram stains, bacterial and mycobacterial cultures were negative. VZV IgG antibody was detected in CSF (VZVlgG:3.98 T.V.). Zoster
myelitis and polyradiculitis was diagnosed and he was treated with 10 mg/kg acyclovir intravenously 3 times daily for two weeks.

The spectrum of VZV myelopathy is broad, ranging from acute to chronic. HZ myelitis generally occurs at the same time or within the six-week of rash onset in immune-compromised patients (2). We observed myelitis at the third-week of the rash onset in our case. The pathogenesis seems to be abnormal immune response to infection rather than the direct effect of infectious agent (3). Motor neuron involvement, referred as segmental zoster paresis, occur in 0.5%–31% of HZ cases (4). Some electrophysiologic abnormalities were seen in the extremities that have no motor weakness (5). Although our patient had right leg weakness, bilateral findings were detected in nerve conduction study.

In liver transplant patients, HZ frequency has been reported as 1.2-18%. Most of the cases occurred within the first year (6,7).

Figure 1. Distribution of skin lesions due to herpes zoster. (A) Crusted rash on right L5 dermatomal trace. (B) Crusted rash on L5-S1 dermatomal traces.

Figure 2. Magnetic resonance image of the cervical (A) and thoroal (B) spinal cord. Sagittal T2 fast-recovery fast-spin echo sequence showing abnormal longitudinal serpiginous contrast enhancing hyperintense lesions.

Underlying malignancy is found 3 times more frequently in patients with zoster motor paresis than patients with purely cutaneous zoster (8). We also detected hepatocellular carcinoma recurrence in our patient.

We would like to emphasize that HZ in post-transplant patients should be considered as a medical emergency. In severe immunodeficiency with extended skin lesions or disseminated central-nerve system, treatment with high dose intravenous acyclovir and continuous monitoring of renal function are required (1,2). Despite the delay of systemic acyclovir treatment for 3 weeks following rash onset in our case, improvement appeared consistent with a clinical response to this therapy.

In conclusion, neurological complications related to HZ seems to be increased in patients after organ transplantation and prolonged steroid treatment, the clinical manifestations may be unusual and multiple as in our case.

References