Acute Hepatic Failure Associated with Stevens-Johnson Syndrome Induced by Carbamazepine Treatment in a Patient with Transverse Myelitis

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Carbamazepine-induced liver injury is less common, but the consequences of the side effects can be very serious leading to death or a need for liver transplantation. We report a case of a 60-year-old female transverse myelitis patient with fulminant hepatic failure and Stevens-Johnson syndrome induced by carbamazepine who successfully underwent deceased donor liver transplantation. The patient, a 60-year-old female, was admitted to our service due to acute liver insufficiency and a drowsy mental state attributable to carbamazepine. She had been treated with carbamazepine to control transverse myelitis. Fifty days after the use of carbamazepine, she developed jaundice, erythematous papules and bullae, and decreased consciousness. The diagnosis of Stevens-Johnson syndrome was considered. She underwent deceased donor liver transplantation. She was discharged with normal graft functions 5 months after transplantation. Thus, liver transplantation can be a feasible therapy for patients with carbamazepine-induced hepatic failure associated with Stevens-Johnson syndrome.

Key Words: Carbamazepine, Stevens-Johnson syndrome, Fulminant hepatic failure, Liver transplantation, Transverse myelitis

INTRODUCTION

Transverse myelitis (TM) is a focal inflammatory disorder of the spinal cord. Carbamazepine is widely used to relieve the neuropathic pain, but it is frequently associated with neurologic adverse events, Stevens-Johnson syndrome (SJS), and hepatic side effects. Carbamazepine-induced liver injury is less common, but the consequences of the side effects can be very serious, leading to death or a need for liver transplantation. We report a case of a 60-year-old female with fulminant hepatic failure (FHF) and SJS induced by carbamazepine who successfully underwent deceased donor liver transplantation (DDLT). This is the first report in which neurologic and graft function in a patient with TM, FHF, and SJS recovered completely after DDLT.

CASE REPORT

The patient, a 60-year-old female, was admitted to a neurologic department in another hospital for sensori-motor weakness of lower extremities. T2-weighted magnetic resonance imaging of the spine revealed diffuse and ill-defined high signal intensities at the T8-10 level compatible with the neurologic deficit (Fig. 1). With the results of other laboratory tests, she was diagnosed as idiopathic TM and was treated with intravenous high-dose steroid. After the pulse therapy, the motor power of the lower extremities had improved significantly but she complained of intermittent lancinating pain in the legs. The patient...
had been treated with carbamazepine (200 mg twice daily) in another hospital to control her sensory symptom. Fifty days after starting carbamazepine, she developed jaundice, erythematous papules, bullae, and skin erosions (Fig. 2). The patient was transferred to our liver unit because of acute liver insufficiency and a slowing of mentation, which was attributable to the carbamazepine.

On the initial physical examination, she was somnolent, but arousable. She was jaundiced and covered with erythematous macules and papules. The carbamazepine was stopped immediately. The neurologic examination revealed a motor weakness in both lower legs, graded as 1/5 according to the Medical Research Council grading system for muscle strength below the knee. The maximum bilirubin concentration in the blood was 22.5 mg/dl and the peak AST, ALT, and ALP were recorded to be 583 U/L, 738 U/L, and 615 U/L, respectively. The blood ammonia level was elevated to 393 μg/dl (normal range, 19 ~ 87 μg/dl). The serologic tests for hepatitis were negative. The level of consciousness decreased progressively and she became comatose. She was transferred to the intensive care unit and placed on mechanical ventilation. On the initial abdominal computed tomography (CT), a large amount of ascites and splenomegaly were noted. On brain CT, cerebral edema was not noted. She was placed on the national emergency liver transplant waiting list. Her condition deteriorated rapidly and the model for end stage liver disease score was 39. Despite continuous renal replacement therapy, she did not recover, but deteriorated and developed pulmonary edema. Fortunately, 5 days after listing, a suitable deceased donor became available, and she underwent DDLT. The pathologic findings of the explanted liver showed massive hepatic necrosis (Fig. 3). Immunosuppression was based on induction therapy with steroids and FK 506. On post-operative day (POD) 5, the SJS gradually improved. Seven days after DDLT, she was able to open her eyes spontaneously. She was extubated 11 days after liver transplantation with full awakening and ability to expectorate. Her neurologic examination showed a quadriplegia in the immediate post-operative state, but she improved and gradually recovered the power of her extremities; by post-operative day 120 she was able to walk with a walking stick. On post-operative day 45, a fungal pneumonia occurred, but was controlled by aggressive physiotherapy and an antifungal agent. The patient underwent prolonged hospitalization for rehabilitation due to acute TM and post-operative disuse syndrome. The patient was discharged with normal graft functions 5 months after DDLT. At the 12 month follow-up, the patient had no significant pathology and was ambulating independently.

DISCUSSION

TM is a rare neurologic syndrome caused by inflammation of the spinal cord, resulting in motor, sensory, and autonomic dysfunction.(1,2) TM is an interruption of spinal cord function not caused by macrotrauma.(3) The initial symptoms usually include lower back pain, abnormal sensations, such as burning, tickling, prickling, or tingling in the legs, and paraparesis of the legs. The paraparesis...