Acute Abdominal Pain Preceding Cutaneous Manifestations of Varicella Zoster Infection after Allogeneic Bone Marrow Transplantation

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ITOH, M., KAWAGUCHI, S., YAGO, K., SHIMADA, H. and MURO, H. Acute Abdominal Pain Preceding Cutaneous Manifestations of Varicella Zoster Infection after Allogeneic Bone Marrow Transplantation. Tohoku J. Exp. Med., 2001, 195 (1), 61–63 — The current communication describes clinical findings in two recipients of allogeneic bone marrow transplantation (BMT) with varicella zoster virus infection who complained of acute severe abdominal pain preceding cutaneous manifestations. Physical examination, laboratory data and gastroscopic findings were nonspecific. In these cases, acyclovir was very effective for the symptoms. Varicella zoster virus infection should be suspected in BMT recipients who have rebellant acute abdominal pain but no characteristic skin eruptions. bone marrow transplantation; varicella zoster; abdominal pain; acyclovir; skin eruption

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Varicella zoster virus (VZV) infection is one of the common complications of allogeneic bone marrow transplantation (BMT). Several authors reported acute abdominal pain as a presenting symptom of VZV infection preceding characteristic skin eruptions (Schiller et al. 1991; Perez-Oteyza et al. 1992; Verdonck et al. 1993; Hamanishi et al. 1998; Yagi et al. 2000). We report the cases of two recipients of allogeneic BMT with VZV infection who complained of acute severe abdominal pain preceding cutaneous manifestations. Acyclovir (ACV) was effective for the symptoms in both cases.

CASE REPORTS

Patient 1. A 17-year-old Japanese male recipient of HLA-matched unrelated BMT was admitted for acute severe epigastric and back pain that developed five months after the transplantation. He had been diagnosed as having severe aplastic anemia and underwent BMT. He had no history of varicella, and his VZV antibody was not measured. The VZV anti-
body status of his donor was unknown. His post-transplant period was only complicated by mild cutaneous acute graft vs. host disease (GVHD). He had been treated with cyclosporin A. Admission findings revealed an afebrile man without abdominal tenderness nor rigidity and normal bowel sound. His laboratory studies revealed mild liver dysfunction, but serum amylase level and complete blood cell count were normal. Abdominal ultrasound examination result was normal. Gastroscopy showed chronic gastritis. Histopathological findings of the gastric mucosa revealed nonspecific inflammation. The pain grew worse, and morphine hydrochloride was necessary. Five days later, papulae developed and the rash became vesicular and disseminated over his entire body. A Tzank test was positive for Varicella. Anti-viral therapy with ACV (15 mg/kg/day) was begun and abdominal pain promptly lessened.

Patient 2. A 40-year-old Japanese male recipient of HLA-matched sibling BMT had been treated with tacrolimus and corticosteroid for chronic GVHD and bronchiolitis obliterans organizing pneumonia. He had undergone transplantation 14 months previously for acute myelogenous leukemia with monosomy 7 in the first remission. The recipient's VZV antibody status was negative before BMT, but the donor was not tested for VZV antibody. The patient began to complain of acute severe epigastric pain. He was afebrile and physical findings showed abdominal tenderness without rigidity. His laboratory studies, including serum amylase level, revealed almost normal. A gastroscopy revealed esophageal and gastric ulcers. Histopathological analysis of biopsy material showed cells with intranuclear inclusions within gastric and esophagus mucosa. Omeprazole and pentazocine were begun, but they were not effective. Four days later, he developed vesicular eruptions characteristic of Varicella on his chest, which became widely dispersed thereafter. Varicella was diagnosed and anti-viral therapy with ACV (15 mg/kg/day) was started. Abdominal pain was markedly reduced.

DISCUSSION

VZV infection often occurs following allogeneic BMT. A previous report indicated that 35% of the recipients of allogeneic BMT experience VZV infection by one year after transplantation (Verdonck et al. 1993). The infection commonly manifests as cutaneous dissemination. However, visceral dissemination, including hepatitis, pneumonia, pancreatitis, gastroenteritis, and meningoencephalitis, occasionally occur and can be mortal. Sometimes, primary gastrointestinal symptoms may precede the characteristic skin lesions by 24–96 hours (Schiller et al. 1991) and a few authors reported abdominal presentation of VZV without skin eruption (Rogers et al. 1995; Yagi et al. 2000). In these previously reported cases as well as our cases, physical examination, laboratory studies and gastroscopic findings, including histopathological findings, are often nonspecific and nondiagnostic. There is a report that even laparotomy was performed because diagnosis was difficult (Yagi et al. 2000). In our two cases, neither ordinary antiulcer agents nor analgesics were effective for the pain, but empiric anti-viral therapy with ACV were very effective. We began anti-viral therapy with ACV 15 mg/kg/day. A dose of ACV 30 mg/kg/day was often chosen for disseminated visceral VZV infection, but this dose is not covered by the health insurance in Japan. We suspected VZV infection and started the standard dose of ACV in the early stage, which was very effective. We did not examine viral genetic study and VZV infection was diagnosed based on the clinical course and characteristic eruptions. Since early administration of ACV is preferable, it should be started promptly for such clinical symptoms before genetic data were obtained.

In conclusion, we should consider anti-viral
therapy for VZV infection in BMT recipients, who present with rebellant abdominal but no skin eruptions.

References


