Adult T-cell leukemia blast crisis
in a patient with acute liver failure

Adult T-cell leukemia (ATL) is a peripheral T-cell type malignancy in which symptoms develop due to infection by human T-cell lymphotropic virus type-I (HTLV-I). After a long latent period, ATL occurs in about 5% of carriers in several regions of the world where HTLV-I is endemic, such as Kagoshima Prefecture in southwest Japan. We report an HTLV-I carrier who developed ATL blast crisis resulting from acute liver failure.

A 50-year-old man visited a local hospital with low-grade fever, loss of appetite, and jaundice that had continued for seven days. He did not have history of liver disease earlier. Laboratory studies revealed AST 614 IU/L, ALT 834 IU/L, total bilirubin 17.6 mg/dL, and prothrombin time 42%. The patient was transferred to our hospital for further treatment. At admission, ultrasonography and CT scan showed an atrophic liver and large amount of ascitic fluid. Hepatitis viruses A, B and C, cytomegalovirus, Epstein-Barr virus, and herpes simplex virus serology were negative. Autoimmune and alcoholic hepatitis were ruled out as well. Scintigraphy with Tc-99m galactosyl human serum albumin (GSA) showed poor accumulation of radioisotope in the liver. We made a diagnosis of acute liver failure because of complicating hepatic encephalopathy. He received conventional therapy, and ALT level and uptake of Tc-99m GSA by liver improved by day 46 after admission. Liver function tests showed a tendency toward improvement.

However, laboratory studies showed elevated serum lactate dehydrogenase (LDH) and serum calcium values. His birthplace was Kagoshima Prefecture, known to be an area where ATL is endemic. We tested for HTLV-I antibody (PROBLOT HTLV-I WB; Fujirebio, Japan), and found it positive. After day 50, atypical lymphocytes in the serum gradually increased. Laboratory tests showed calcium and LDH levels elevated. Bone marrow aspirate showed ATL cells. We diagnosed him as an HTLV-I carrier with ATL blast crisis. On day 54, he was started on chemotherapy for ATL; however, the treatment failed to be efficacious. Urine culture showed cytomegalovirus infection, and tuberculin reaction was negative, indicating a possible decline in cell-mediated immunity resulting from acute liver failure. The patient died on day 91.

At autopsy, the liver exhibited multiple regenerating nodules on the surface. On microscopy (Fig), there was no hepatic infiltration by ATL cells or viral or bacterial infections of unknown origin. There was marked enlargement of the portal canals, and the liver showed signs of recovery.

Fig: Photomicrograph showing marked enlargement of portal tract with proliferation of bile ducts (arrow) (H&E, 10x)

The ATL oncogenesis mechanism is still unclear. The clinical form of ATL is classified into four types: acute, chronic, smoldering, and lymphoma. Iroi et al reported a patient with ATL blast crisis after the onset of acute liver failure; this patient showed general decline in condition. This report is similar to our case who had smoldering-type ATL on admission. We believe the ATL blast crisis was caused by a decline in immunity due to acute liver failure.

Etsushi Kawamura, Daiki Habu,* Hiroko Kurooka, Takehiro Hayashi, Ai Oe, Jin Kotani, Shigeaki Higashiyama,** Hirohisa Nakamae,# Kenji Torii, Joji Kawabe, Susumu Shiomi
Departments of Nuclear Medicine, *Hematology, **Radiology and #Hematology, Graduate School of Medicine, Osaka City University, 1-4-3, Asahimachi, Abenoku, Osaka 545-8585, Japan

References

Correspondence to: Dr. Kawamura. Fax: +81 (6) 6646 0686. E-mail: etsushi-k@med.osaka-cu.ac.jp