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722.ALLOGENEIC TRANSPLANTATION: ACUTE AND CHRONIC GVHD, IMMUNE RECONSTITUTION

Correlation between Clinical Diagnosis and Histopathological Findings of Liver Dysfunction after Allogeneic-HSCT

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Liver dysfunction is one of the dismal complications after allogeneic hematopoietic cell transplantation (Allo-HCT). Although most frequently suspected complications are hepatic graft-versus-host disease (GVHD) and toxic hepatitis, precise diagnosis can be challenging. In order to accurately diagnose hepatic dysfunction after allo-HCT, liver biopsies are occasionally done. However, even after attaining pathology, discerning between overlapping pathologic findings among different hepatic complications, still remains to be a challenge. We tried to review the pathologic findings of liver biopsies of patients with elevated liver enzymes after allo-HCT.

We obtained 92 liver biopsy samples from 88 patients who underwent allo-HCT in Catholic Hematology Hospital from 2008 to 2023. All patients showed elevated liver enzymes presenting with either cholestatic pattern (hyperbilirubinemia and/or high alkaline phosphatase [ALP] or gamma glutamyl-transferase [GGT]) or hepatocellular pattern (elevating AST/ALT levels). Clinical course, laboratory and pathological findings were acquired on the date of liver biopsy. Acute hepatic GVHD was assessed according to the original Glucksberg criteria. Chronic hepatic GVHD was assessed by the National Institutes of Health criteria 2014. Hepatic GVHD was clinically diagnosed in patients showing elevated ALP or GGT with hyperbilirubinemia (typical cholestatic) or elevated aminotransferase (hepatic variant).

Median duration to the date of biopsy after allo-HSCT was 6.3 months (range, 0.8 to 61.3). Hepatic GVHD was divided into typical cholestatic pattern (n=30) and hepatic variant (n=28). Other clinical features were isolated hyperbilirubinemia (n=17), elevation of aminotransferase (n=8), HBV reactivation (n=5), toxic hepatitis (n=2), liver cirrhosis (n=1), and PTLD (n=1). Among 30 cholestatic hepatic GVHD cases, 20 biopsies revealed early pathologic features including bile duct damage and portal lymphocytic infiltration, 9 showed late features including loss of bile duct and fibrosis, and 1 showed non-specific findings. Among 28 hepatic-variant GVHD cases, 22 biopsies revealed early pathologic features, 5 showed late features including loss of bile duct and fibrosis, and 1 showed non-specific findings. All 8 isolated aminotransferase elevation cases showed typical GVHD findings (early feature 6, late feature 2), while 17 isolated hyperbilirubinemia cases showed 8 hepatitis pathology, 5 GVHD, 3 VOD, and 1 hemochromatosis. Among our final 36 hepatic variant GVHD cases, 35 (97.2%) patients showed GVHD improvement after treatment, while among final 35 cholestatic GVHD cases, 15 (42.8%) showed response ($p<0.001$).

Our clinical diagnosis for hepatic GVHD well correlated with pathologic findings. Clinical outcome of hepatic GVHD with cholestatic pattern was far worse than hepatic variant GVHD. Isolated aminotransferase elevation strongly correlated with pathology consistent with GVHD. As isolated hyperbilirubinemia was related with various pathologic findings, we recommend liver biopsy in this group of patients in order to discern between different etiologies.

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