

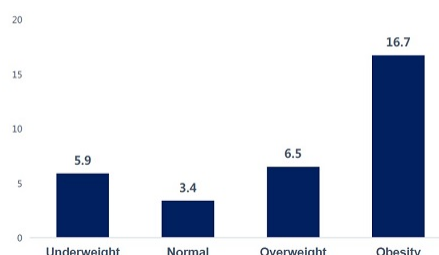
De novo fatty liver following pancreaticoduodenectomy ; incidence and risk factor

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Background: Development of fatty liver after pancreaticoduodenectomy (PD) has been increasingly recognized nowadays. Pancreatic exocrine insufficiency has been regarded as a major contributor in this setting, where intensifying pancreatic enzyme supplementation is suggested. We investigated incidence of de novo fatty liver following PD. **Methods:** A total of 161 patients who had PD between 2010 and 2012 who had regular abdominal CT evaluation before and after 1 year of PD were reviewed. Fatty liver was diagnosed using the liver-to-spleen attenuation ratio and difference between hepatic and splenic attenuation. **Results:** At one year, de novo fatty liver was noticed at 10 of 161 patients (6.2%). One hundred forty nine patients (92.5%) were receiving pancreatic enzyme supplementation. De novo fatty liver rate did not differ significantly by pancreatic enzyme supplementation (8.3% vs. 6.0% for pancreatic enzyme supplementation (-) vs. (+), $p=0.55$). In multivariate model, the body mass index was the only factor associated with the development of fatty liver. The de novo fatty liver was noticed in 5.9%, 3.4%, 6.5% and 16.7% of underweight, normal, overweight and obese patients. Compared to normal weight patients, obese patients were at increased risk of de novo fatty liver [odd ratio (OR): 5.17, 95% confidence interval (CI): 0.95-27.9, $p=0.056$]. **Conclusions:** Development of fatty liver was noticed for patients who received PD. Obesity was a risk factor for fatty liver when patients were under intensified pancreatic enzyme supplementation after PD.

Incidence of de novo fatty liver according to the baseline BMI category



Decreased FoxP3 and PD-1 but Sustained CTLA-4 during 1 Year Tenofovir Therapy in Chronic Hepatitis B

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Background: Immune regulatory molecules such as forkhead box P3 (Foxp3), programmed death-1 (PD-1) and cytotoxic T-lymphocyte-associated protein-4 (CTLA-4) on T cell are associated with antiviral effector T cell dysfunction, which influences on T cell exhaustion and persistent viral infection in patients with chronic hepatitis B (CHB). These FoxP3, PD-1 and CTLA-4 are known to up-regulated in chronic viral infection but there is few report about the phenotypic changes of these molecules during antiviral therapy, especially, tenofovir in CHB infection. We investigated the expression of FoxP3, PD-1 and CTLA-4 during 1 year tenofovir treatment in 28 patients with CHB. **Methods:** 28 patients with CHB under tenofovir treatment were enrolled for detection of intrinsic inhibitory molecules of T cell signals (PD-1, CTLA4) and extrinsic inhibitory molecule, FoxP3. Peripheral blood mononuclear cells (PBMC) were isolated from these subjects before tenofovir treatment (T0), 1 month (T1), 3 month (T3), 6 month (T6) and 12 month (T12) during tenofovir treatment. The expressions of FoxP3, PD-1, CTLA-4, CD8, CD4 on T cells were monitored by flow cytometry. **Results:** T cells from patients with CHB before tenofovir treatment (T0) showed dramatically increased expression of FoxP3, PD-1 and CTLA-4 compared to healthy control (% FoxP3 5.45 vs 0.85 $p<0.0001$, % PD-1 5.56 vs 2.23, $p<0.05$, % CTLA-4 6.08 vs 4.49 $p<0.05$). However, T cells from patients with CHB under tenofovir treatment showed decreased expression of FoxP3 at T12 compared to T0 significantly (%FoxP3/CD4, 6.22 vs. 3.83, $p<0.0001$). In case of PD-1, the expression of PD-1, especially in CD8T cells was statistically decreased at T12 compared to T0 (%PD-1/CD8, 5.4 vs. 3.2, $p=0.02$). Interestingly, the expression of CTLA4 was sustained at T12 through T0 ((%CTLA4/CD8, 2.03 vs. 1.72, $p=0.14$). **Conclusions:** In chronic hepatitis B, PD-1 as inhibitory T cell molecule and FoxP3 as regulatory T cell marker are down-regulated during 1 year tenofovir therapy, which could restore HBV-specific T cell function during tenofovir antiviral therapy. However, CTLA-4 as another T cell inhibitory molecule, the expression in CD8T cells was sustained.