"Tumor immune microenvironment after locoregional treatment in hepatocellular carcinoma"

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Transcatheter hepatic arterial bland embolization (TAE), conventional transarterial chemoembolization (cTACE), and TACE with drug-eluting beads (DEB-TACE) remain the mainstays of catheter-based locoregional treatment for intermediate and advanced-stage hepatocellular carcinoma. These approaches offer survival benefit for selected patients, but recurrence rates remain high. Critical to the effectiveness of both TAE and cTACE/DEB-TACE is tumor hypoxia and resulting tissue necrosis secondary to acute vascular occlusion. This acute necrotic cell death is widely recognized as a potent activator of adaptive immunity. Indeed, several investigators have reported various alterations in circulating T-cell profiles after cTACE and TAE alone. In this lecture, we will discuss the tumor-immune cell landscape after transcatheter arterial bland embolization, including the most relevant T-cell subsets. We have demonstrated that TAE induces increased infiltration of Th17 cells in liver tumors, when compared with controls two weeks after treatment. A similar pattern was observed in the spleen, indicating both local and systemic effect. We did not observe significant differences in the percentage of other key immune cell populations, such as FoxP3+ Tregs, IFNγ producing CD4 T-cells, and CD8 T-cells. In conclusion, transcatheter hepatic arterial bland embolization induces local and systemic increased infiltration of Th17 cells and expression of their signature cytokine IL-17.