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Acquired familial amyloid polyneuropathy after domino liver transplantation

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Domino Liver Transplantation (DLT) started in 1995, using a donor liver with familial amyloid polyneuropathy (FAP), then increasing the pool of donors. Our Center started DLT in 2001; however cases of acquired amyloid neuropathy are increasingly being recognized following this procedure. Also our Center experience showed that the onset of FAP symptoms occurs earlier than expected. Several papers described clinical cases of acquired FAP developing 5 to 9 years after DLT. From 2001 to 2011 we have done 1078 liver transplants; we carried out 262 domino transplantations. All receptors transplanted between 2001 and 2011, were evaluated. All of them were observed and had clinical, histopathologic (salivary gland biopsy) and electrophysiologic evaluation. The symptomatic group started with sensory complaints involving their feet 4 to 10 years after DLT; three of those patients have been retransplanted to halt FAP progression and their clinical and paraclinical improvement is described. Five other patients are in the waiting list for retransplant. Patients with FAP acquired by transplantation are candidates for liver retransplantation to minimize the progression of symptoms. Liver retransplantation is considered to be a high-risk procedure but so far the results have been favorable.

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Viewing cellular origin of intrahepatic cholangiocarcinoma from perspective of tumor heterogeneity

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Intrahepatic cholangiocarcinoma (ICC) is an extraordinarily heterogeneous malignant disease among the tumors that have so far been identified. The cancer cell-of-origin has important implications for tumor cell fate and cancer phenotype. Recent data suggest that multiple cellular origins of ICC including differentiated hepatocytes, intrahepatic biliary epithelial cells (IBECs)/cholangiocytes, pluripotent stem cells such as hepatic stem/progenitor cells (HPCs), biliary tree stem/progenitor cells (BTSCs), and within peribiliary glands (PBGs). Both somatic mutagenesis and epigenomic features are highly cell-type-specific, that is, multiple cellular origins may profoundly influence genomic landscapes, key signaling pathways, driving phenotypic variation and pose significant challenges to personalized medicine, drug response and patient outcome. Specifically, the cellular origin of ICC can be accurately determined based on the distribution of mutations along its genome through Roadmap Epigenomics Program. Understanding ICC heterogeneity of cellular origins and molecular mechanism may contribute to establish hierarchical model of carcinogenesis and improve anatomical-based classification. The advent of personalized medicine for ICC treatment may enable the actual origin of the cellular and molecular mechanism of ICC to be determined to improve diagnosis, therapies and prognosis.

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