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Relevance of ADAMTS13 in liver transplantation and surgery

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Abstract

ADAMTS13 specifically cleaves unusually-large von Willebrand factor (VWF) multimers under high shear stress, and down-regulates VWF function to form platelet thrombi. Deficiency of plasma ADAMTS13 activity induces a life-threatening systemic disease, termed Thrombotic microangiopathy (TMA) including thrombotic thrombocytopenic purpura (TTP). Children with advanced biliary cirrhosis due to congenital biliary atresia sometimes showed pathological features of TMA, with a concomitant decrease of plasma ADAMTS13 activity. Disappearance of their clinical findings of TTP after successful liver transplantation suggested that the liver is a major organ producing plasma ADAMTS13. *In situ* hybridization analysis showed that ADAMTS13 was produced by hepatic stellate cells. Subsequently, it was found that ADADTS13 was not merely responsible to development of TMA and TTP, but also related to some kinds of liver dysfunction after liver transplantation. Ischemia-reperfusion injury and acute rejection in liver transplant recipients were often associated with marked decrease of ADAMTS13 and concomitant formation of

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