



Splenomegaly in Portal Hypertension

Li Yan and Hai-Bo Chu*

Department of General Surgery, 89th Hospital of PLA, China

Commentary

The traditional concept suggests that the splenomegaly of Portal Hypertension (PHT) is a passive congestive splenomegaly, and stasis secondary congestion is its pathologic feature [1,2]. The modern concept suggests that splenomegaly not only involves splenic congestion, but is also accompanied by congestion of splenic red pulp, vascular proliferation, fibrosis, lymphoid hyperplasia and activation [3-8]. In PHT, spleen angiogenesis can regulate the portosystemic collateral circulation and increase the spleen blood flow, which is an important pathologic and physiologic sign of the spleen [9]. If a spontaneous portosystemic shunt exists, it will not appear as splenomegaly. Thus, splenomegaly was hemodynamic compensatory personalization of PHT. Hypersplenism will occur after splenomegaly decompression [10,11]. In addition, the collateral vessels of splenic perisplenic ligaments (lienorenal ligament, splenophrenic ligament, and splenocolic ligament) are abundant in splenomegaly, thus a wide collateral circulation can be established in retroperitoneal and pericardial locations, and the spleen may be called a "shunt bridge", which was used in Warren surgery to divert blood flow from gastro-splenic region to achieve selective shunt depressurization [12].

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*Correspondence:

Hai-Bo Chu, Department of General Surgery, 89th Hospital of PLA, Weifang 261021, China,

E-mail: haibochuwf@163.com

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