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Commentary

Collateral Circulation in Portal Hypertension

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Commentary

One of the most common complications in portal hypertension (PHT) is portosystemic collateral formation, including variceal bleeding and hepatic encephalopathy; the former is also one of the most common causes of death among adults worldwide [1]. The regulatory mechanism of collateral vessels was unclear, but it was still the key to the compensatory mechanism of a high dynamic circulation of PHT. The degree of the formation in the portosystemic collateral circulation reflects the ability of the body's compensatory protection [2]. The study showed that vasodilation is related to increased vasodilation and low reaction vasoconstriction, which is referred to as a "low blood vessel contraction state" [3]. Therefore, increased portal pressure appears before variceal hemorrhage clinically [4,5]. The collateral circulation is abundant in extrahepatic PHT, including gastroepiploic veins, short gastric veins, the gastric vein, left gastric vein, left colonic veins, and spontaneous splenorenal shunts [6]. The collateral circulation becomes a sign in vitro, reflecting high resistance and high flow of the portal vein [7,8]. The gastric posterior vein can form a shunt with the kidneys, which is a common cause with encephalopathy. According to statistics, the incidence of the gastric posterior vein and renal shunt were significantly greater than the left gastric vein and the gastric short vein [9]. Esophageal varices were detected in approximately 50% of patients with cirrhosis, and approximately 5% – 15% of patients with cirrhosis formed varices or worsening of varices each year [10]. Thus, understanding the pathogenesis of collateral circulation and compensatory mechanisms is important to understand the nature of PHT. At the same time, the portosystemic collateral circulation can inspire the innovative thinking of surgeons and lead to new methods of surgical treatment.

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