Title:

Non-Cirrhotic Portal Hypertension: A possibly benign but complicated disease.

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In the last Baveno Consensus Conference held in 2015 a dedicated section described the management of patients with portal vein thrombosis, in presence or absence of cirrhosis (1). In this specific collective of patients few studies have described their natural history and therefore new studies are urgently needed in order to recommend evidence-based management. So far the recommendation regarding the treatment of complications of portal hypertension are aligned with the recommendation in the presence of cirrhosis (1, 2). However, these recommendations are based less on evidence and more on eminence. There are few published studies, which describe the natural history and management procedures in these patients, such as variceal management (3, 4), anticoagulation (5) or shunting procedures (6). The comparison to the cirrhotic patients
has not been done in a systematic manner, but the conclusions drawn have been stressing the similarities of NCPH with liver cirrhosis with portal hypertension. The paper by Gioia and colleagues (7) presents a step in the right direction to fill in this gap. They nicely describe that the outcome of patients with non-cirrhotic portal hypertension (NCPH) is not more favorable than in patients with a compensated Child A cirrhosis. This is clearly seen in these patients when these two groups are compared with their risk of variceal progression. Also, the risk of development of portal vein thrombosis and variceal bleeding was higher in NCPH patients compared to the Child A cirrhotic patients, while the development of ascites was higher in the cirrhotic patients. This study suggests that the complications of portal hypertension in the patients with NCPH are not due to the same mechanisms as in cirrhosis. Especially interesting is the fact that NCPH are more associated with thrombosis and less with ascites. This indicates that the pathophysiological mechanisms of portal hypertension seem to be more related to pure increase in the portal pressure, rather than the hyperdynamic circulation with consecutive decrease in effective arterial blood volume ultimately affecting the kidney function and leading to ascites (8, 9). These findings suggest that non-selective betablockers might not be the right treatment in these patients. Moreover, the effects one would desire in this situation would be a vasodilating drug to decrease the resistance to portal blood flow and thereby improve the flow and prevent thrombosis, rather than decreasing the portal blood flow and increasing the chance of developing portal vein thrombosis. These thoughts would ultimately change the approach in the patients with NCPH and require further investigations and research in this neglected field. In consequence, the development of treatments should also not be aligned to the treatment of cirrhotic portal hypertension, as it is currently (1, 2).
Therefore, new therapeutic strategies are required for these patients. Due to the small number of cases international initiatives such as EASL-VALDIG-registry will be helpful in the future to perform the correct comparisons and elaborate on the right procedures for these patients. Another issue is the matter of definition, which has not been unified until recently. Therefore, the definitions of NCPH, as well as the concept of advanced chronic liver disease have been introduced (1), which should be used in a consequent manner in future studies.

In summary, the recommendation for the management of NCPH patients should be elaborated independently of the cirrhotic portal hypertension, due to the different course of disease, which is ultimately demonstrated in the present study.

CONFLICT OF INTEREST

This author declares the following financial disclosures:

The author is supported by grants from the Deutsche Forschungsgemeinschaft (SFB TTR57 to P18), Cellex Foundation (PREDICT) and European Union’s Horizon 2020 research and innovation program (No 668031).

References:

