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Report of the Baveno VI Consensus Workshop

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Article:

de Franchis R; Baveno VI Faculty. Expanding consensus in portal hypertension Report of the Baveno VI Consensus Workshop: Stratifying risk and individualizing care for portal hypertension. *J Hepatol* 2015; 63: 743-52.

Comments

The latest update of the Baveno consensus was held on April 10-11 of 2015 in Baveno, Italy. The meeting led by Professor Robert deFranchis started in 1986 and has been held approximately every 5 years with a publication following each meeting. This latest Baveno VI workshop was attended by the "who is who" in world of portal hypertension; mainly experts responsible for most data produced in the last decade. The goals of these meetings are to develop and update definitions of key events and concepts in portal hypertension. The proceedings of these meetings are considered by most Hepatologists the quintessential guidelines in portal hypertension. Since they are organized under the auspices of EASL they are therefore considered the EASL guidelines of portal hypertension. Other guidelines from the United States (AASLD), Asia (APASL) and the UK are also very popular, but do not use the format of the Baveno guidelines. A key issue of this consensus is that is it constantly evolving over prior definitions. In all meetings the experts review the evidence on the natural history, the diagnosis and the management of portal hypertension and make evidence-based recommendations not only on these topics but also recommend research agendas in the field. All these meetings are highly successful and produce a consensus statement on almost aspects related to portal hypertension in adults and children. As always not all topics are settled and several points remain unsettled due to lack of proper studies. This last meeting not only focused on all issues of natural history, diagnosis and management but also introduced new concepts. An important one was related to the different stages of cirrhosis and the different risks of developing complications and of dying. In fact the meeting was entitled "Stratifying risk and individualizing care for portal hypertension". There were discussions on invasive and non-invasive methods for diagnosing varices and portal hypertension, the role and impact of the underlying etiology of cirrhosis was discussed mainly in relation to new hepatitis C therapies , the primary prevention of decompensation, the management of the acute bleeding episode, the prevention of recurrent bleeding and other decompensating events, and vascular diseases of the liver in cirrhotic and non-cirrhotic patients. All areas were assigned a group of experts (around 6-10) and they issued a number of statements that were then discussed among the audience and agreed upon.

An important concept that was introduced was that of compensated advanced chronic liver disease (cACLD). This was term was proposed in order to illustrate that the range of severe fibrosis and cirrhosis may occur in a continuous fashion and teasing them apart is not easy relying only on clinical data. A clearer role of transient elastography (TE) was introduced and we now know that TE allows the early identification of patients with chronic liver disease who may develop clinically significant portal hypertension. In fact liver stiffness measured by TE is adequate to suspect cACLD. TE values < 10 kPa usually rule out cACLD and values between 10 and 15 kPa indicate cACLD and if they are > 15 kPa then this is very indicative of cACLD. If needed the diagnosis of cACLD can be confirmed with liver biopsy, hepatic venous pressure gradient (HVPG), or upper endoscopy. HVPG measurement is still considered the gold-standard method to define clinically significant portal hypertension (values > 10 mmHg). These patients do not have varices or ascites but should be monitored closely.

The issue regarding the avoidance of screening endoscopy was also brought up and new information

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indicates that individuals with TE <20 kPa and a platelet count > 150,000 have a low risk of having varices and could avoid screening endoscopy, but a drawback is that they would need a yearly follow-up with TE and platelet counts. Still today most physicians prefer screening endoscopy as this method can also determine if patients have other common conditions such as portal gastropathy, GAVE or gastric varices. The consensus for the first time focused on therapy of the etiology of cirrhosis, and it was concluded that cure of the etiology of liver disease could improve liver function and reduce fibrosis thereby reducing portal pressure.

Some of the recommendations did not change much from the ones in Baveno V, in particular those related to surveillance of esophageal varices, patients with no varices or small varices and those with large varices. In those with no varices and ongoing liver injury and it was recommended that an upper endoscopy (EGD) should be repeated in 2 yrs, otherwise in 3 years. Those with small varices with ongoing liver injury need a repeat EGD in 1 yr, otherwise in 2 years. Those with large varices need therapy with beta blockers or endoscopic band ligation. This should be based on expertise, local resources and patient preference. In regards to the bleeding episode, most recommendations in regards to resuscitation, airway management, blood volume restitution, antibiotic prophylaxis and early vasoactive therapy remained unchanged. It was agreed that endoscopic therapy should be performed within 12 h of admission and that band ligation was the preferred endoscopic therapy. Risk stratification is of key importance and given the results of high quality studies showing that the early placement of transjugular intrahepatic portosystemic shunts TIPS (within 72 h) for patients AVB with Child B actively bleeding or C cirrhosis (≤ 13 points) is associated with a significant reduction in rebleeding and mortality. Thus an early TIPS with PTFE-covered stents should be considered in patients bleeding from varices that are at risk high risk of treatment failure.

In regards to secondary prophylaxis, not much changed and beta blockers and band ligation are recommended until eradication of varices. The issue of safety of beta blockers in patients with advanced liver disease was addressed and the recommendations were that in patients with cirrhosis and refractory ascites both propranolol and nadolol should be used with caution and close monitoring of blood pressure, serum sodium and serum creatinine. These drugs should be discontinued in those with refractory ascites that have a low systolic blood pressure < 90 mmHg, hyponatremia (< 130 mEq/L) or acute kidney injury. If there was a clear trigger for these events (e.g. spontaneous bacterial peritonitis), re-staring beta blockers should be considered after resolution of the precipitant. Many other issues regarding research agendas in different areas and the topic of vascular diseases of the liver in cirrhotic and non-cirrhotic portal hypertension are thoroughly discussed.

In summary this consensus keeps getting better and has more data driven recommendations than before. There is no question that the recommendations published in this consensus have all been thoroughly discussed and agreed upon by the most respected physicians around the globe in this area and this gathering of experts is due in great part by the unrelenting effort of Professor de Franchis who has been at the forefront of this endeavor for many years. I highly recommend that these guidelines be implemented as quality indicators in all GI and Liver and units around the world.

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