Bleeding news

Treating periprocedural bleeding in patients with cirrhosis

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Reduced levels of factors, both coagulation factors and anticoagulants produced in the liver, in cases of liver failure, lead cirrhotic patients to a situation difficult to balance, with an increased thrombotic and bleeding risk. Additionally, the most advanced cases include low platelet count, in different degrees. It is hard to assess this situation with lab tests, and so viscoelastic tests (VETs) can play a significant role. In this context, the authors offer, as an expert opinion, a perioperative—and peri-transplant—bleeding management based on the principles of Patient Blood Management (PBM).

In the preoperative assessment of cirrhotic patients, the medication is considered, as well as any comorbidities that may appear and be potentially optimised. The platelet count and lab coagulation values must also be known at baseline, even though they are not predictive of bleeding. A significant factor in the bleeding of cirrhotic patients is portal hypertension, which should always be assessed in the preoperative setting, particularly in surgeries or procedures with a high bleeding risk. In this regard, prophylactic correction of the coagulopathy with frozen fresh plasma is not recommended—on account of the volume overload it can cause. Additionally, in order not to increase portal pressure, the patient benefits from a strongly controlled fluid therapy—restrictive, yet not hypovolemic,—as well as a restrictive transfusion with hemoglobin thresholds of 7-8 g/dl, during the whole procedure. Current guidelines recommend preoperative optimisation of anemia (iron, folic acid, B6, B12)

However, there are individual recommendations to assess VET and consider correcting the platelet count or fibrinogen, or the supply of factor concentrate, according to procedure, since it has proven to decrease further need for transfusion. In fact, it is recommended to base the management of bleeding, both during and after surgery, on VET, which also studies fibrinolysis. Routine use of antifibrinolytic agents is not recommended—only in cases of bleeding with clinically-suspected or VET-diagnosed hyperfibrinolysis.

Regarding liver transplant, hemostasis must be assessed through VET, ideally at baseline, after reperfusion and before would closure, as well as if there is bleeding and after administering the treatment. Close monitoring of hemostasis will be maintained after surgery due to the risk of bleeding, thrombosis, liver dysfunction. As discussed, a careful management strategy of the patient's volemia should be adopted, avoiding overload and congestion, yet ensuring optimal hemodynamics and perfusion, maintaining hemoglobin levels around 7-8 g/dl.

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In conclusion, cirrhotic patients require close monitoring of hemostasis—if possible, through viscoelastic tests, with which transfusions have been significantly reduced. However, higher-quality studies are required to base recommendations for the management of anemia and bleeding in this scenario.

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"COAGULATION": a mnemonic device for treating coagulation disorders following traumatic brain injury – a narrative – based method in the intensive care unit.

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TBI-related coagulopathy is a frequent complication associated with a worse prognosis, due to its role in the development and progression of haemorrhagic brain lesions. The aim of the study was to design an acronym as a mnemonic device to facilitate the treatment of this complication.

This study is based on a narrative analysis arising from the question "Can you identify a factor associated with coagulopathy in patients with severe TBI?," asked to 33 physicians specialized in Intensive Medicine, experienced in the frequent treatment of TBI. Factors identified were then compared against published scientific evidence. Because all the factors identified (eleven) had strong scientific support, they were included in the acronym design: "COAGULATION":

C: "Cerebral computed tomography". Brain CT is fundamental to classify the severity of TBI and to determine its monitoring.

O: "Oral anticoagulant and antiplatelet use," as a high alert. Current scientific evidence shows how using these drugs leads to a 3-fold higher mortality rate and increases poor functional prognosis at 6 months after TBI.

A: "Arterial blood pressure". Hypotension is the most dreaded latent threat for the traumatised brain. Maintaining an appropriate cerebral blood flow is one of the primary goals in the management of TBI.

G: "Goal-directed haemostatic therapy". Knowledge of pathophysiology of coagulopathy in TBI is essential. It is characterised by an initial hypocoagulable state consisting of platelet dysfunction, increased consumption of platelets and coagulation factors, disseminated intravascular coagulation (DIC), and hyperfibrinolysis, followed by a hypercoagulable state, both local (cerebral microcirculation-ischemic lesions) and systemic (deep vein thrombosis).

U: "Use of fluids cautiously". A resuscitation with over 2 L fluid therapy is an independent factor for the development of coagulopathy in TBI. Colloids should be avoided since they favour a hypocoagulable state, compromising platelet function and fibrin formation, decreasing the activity of coagulation factors, and increasing fibrinolysis.

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L: "Low calcium levels". Calcium is an essential co-factor for the activation of coagulation, specifically K-Vitamin dependent factors and factor XIII, which is involved in the structure and strength of the clot. Additionally, it plays a central role in platelet activation. It is known that ionic calcium < 1.1 mmol/L, on the third day after a moderate to severe TBI, is predictive of worse functional outcomes and higher mortality. The main mechanism for the decrease in calcium are chelation phenomena, primarily through the action of inflammatory mediators released by astrocytes and damaged neurons.

A: "Anaemia-transfusion". Transfusion in TBI may be beneficial or harmful. Currently, there is not enough scientific evidence to recommend a transfusion threshold in TBI. O2 transport capacity drops by half with Hb values < 7 g/dL, and its availability does not increase with Hb values >12 g/dL. Additionally, Hb values >12 g/dL may increase blood viscosity and decrease cerebral brain flow. Therefore, it seems reasonable to maintain Hb values between 7 and 9 g/dL in TBI.

T: "Temperature". Hypothermia or hyperthermia, both are detrimental. Hypothermia favours a hypocoagulable state and increases the affinity of Hb for O2. Hyperthermia is much more frequent in TBI, exacerbates oedema and inflammation, favouring an increase in intracranial pressure. In cases of extreme elevation, the consumption of factors may increase, as well as fibrinolysis, and a DIC state may be generated.

I: "International normalized ratio-monitoring". Interpreting coagulation tests in essential. The current scientific evidence recommends assessing coagulopathy in TCE using viscoelastic tests. Thus, coagulopathy treatment algorithms based on viscoelastic tests are more sensitive than conventional coagulation tests, and they optimise transfusion in patients with haemorrhagic shock associated with polytrauma.

O: "Oral antithrombotic reversal". Anticoagulation reversal must be urgent/emergent, because the anticoagulation scenario leads to a progression of TBI-related brain lesions, mainly hemorrhagic.

N: "Normal acid-base status". During the evolution of a TBI, it is essential to achieve a balanced microenvironment. Both acidosis and alkalosis are dangerous. On the one hand, acidosis causes cerebral vasodilation and an increase in intracranial cerebral pressure, and it also reduces platelet aggregation, causes alterations in thrombin formation, and accelerates the degradation of fibrinogen. On the other hand, alkalosis increases affinity of Hb for O2 and it causes cerebral hypoxia.

In **summary, "COAGULATION"** is an easy mnemonic device to facilitate the treatment of TBI-related coagulopathy.