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LEADING ARTICLE

Splanchnic vein thrombosis complicating severe acute pancreatitis

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Splanchnic vein thrombosis is a well-recognized complication of acute and chronic pancreatitis but little is known about the exact incidence, clinical relevance and optimal treatment strategy. In this issue of *HPB*, two new studies[1,2] have shed new light on this complication. In a systematic literature review, Butler *et al.* from Indianapolis described a pooled incidence of pancreatitis-induced splenic vein thrombosis of 23% in acute pancreatitis and 12% in chronic pancreatitis.[1] Only half the patients with splenic vein thrombosis developed splenomegaly and 12% experienced gastrointestinal bleeding. Gonzelez *et al.* from London also described a series of 127 patients with acute pancreatitis of whom 20 patients (15%) developed splanchnic vein thrombosis.[2]

Of all patients presenting with pancreatitis, 80–85% suffer a self-limiting disease without pancreatic necrosis or collections and hence are very unlikely to develop venous thrombosis. Therefore, the incidence of 15–23% from the aforementioned studies would appear to be an overestimate and suggest that all patients with severe acute pancreatitis develop splenic or splanchnic vein thrombosis. When looking in more detail at the two studies, the reasons for the reported high incidences become clear. First, the systematic review[1] included 52 case reports of splenic vein thrombosis, thus increasing the incidence. Additionally, as noted by Butler *et al.*,[1] the incidence will be significantly affected by time as a result of the changing use and accuracy of cross-sectional imaging over the past two decades. Second, in the study by Gonzelez *et al.*,[2] all 20 patients with splanchnic vein thrombosis were tertiary referrals. Furthermore, 19 of these 20 patients had peripancreatic collections clearly supporting the notion that splanchnic vein thrombosis is a complication of severe and not mild pancreatitis.[2] It seems fair to conclude that the high incidences do not reflect the 'true' incidence of splanchnic or splenic vein thrombosis that would be found in an unselected series of patients with acute pancreatitis.

Nonetheless, it can be assumed that in a tertiary referral centre for patients with severe acute pancreatitis, this complication will be encountered frequently. The increased recognition coincides with an increasing clinical relevance. As the optimal indications and interventions are acknowledged for infected necrotizing pancreatitis,[3,4] identifying pre-operatively the presence of splenomegaly, major collaterals or varices which may impede a left-sided minimally invasive approach is crucial. In the presence of splenomegaly, the retroperitoneal window between the spleen and kidney may be reduced significantly thus preventing a left-sided approach to the infected necrosis.[3] Similarly, any varices traversing this window should be avoided to prevent the potential for haemorrhage upon dilatation of the retroperitoneal tract.[3] In the era of endoscopic or laparoscopic approaches to walled-off necrosis, recognizing the presence of gastric or left-sided sinustral hypertension is vital if a catastrophic haemorrhage is to be avoided. Thus in the presence of significant gastric varices between the necrosis and the gastric lumen an infracolic approach via the avascular window of the left transverse mesocolon may be a safer option. The importance of a surgical-induced haemorrhage after necrosectomy should not be under estimated as it has been shown to be a significant independent predictor of mortality.[5] Unlike arterial bleeding, which can usually be controlled by radiological embolization, variceal bleeding from the pancreatic bed can be more problematic.[5] Techniques such as porto-systemic shunts, splanchnic vasoconstrictors or a tamponade used to control a haemorrhage from portal hypertension in patients with cirrhosis would appear to offer little in the patient with severe acute pancreatitis. In the setting of gastointestinal bleeding related to splenic venous thrombosis a highly effective treatment is a splenectomy.[6] In complicated situations, a two-step procedure has been described involving splenic artery embolization followed by delayed a splenectomy.[6] In patients unfit for surgery, embolization could be the sole treatment.[6]

Ascites from extensive splanchnic thrombosis is a further concerning symptom and although based on case reports[2,5] would appear to be associated with significant morbidity and mortality. For this reason, at least one author[2] recommends anticoagulation

in this setting or if there is propagation of thrombosis into the main or intra-hepatic portal vein.[2]

Given the likelihood that the majority of patients with splenic vein thrombosis will be asymptomatic[1] and the reported spontaneous recanalization rate is 30%,[2] it would seem sensible to reserve anticoagulation therapy for patients with progression of thrombosis; however, the evidence base for this remains poor. It would seem important to try and identify those who are at a higher risk of bleeding particularly if anticoagulation is being considered. Careful attention to the drainage of the coronary vein may help select those at an increased risk of oesophageal varices.[1] Routine endoscopy, which is the gold standard to identify oesophageal varices, should also be considered.[1] It should also be appreciated that splenomegaly is not present in all patients with splenic vein thrombosis and hence not a reliable marker of sinustral hypertension.[1] Thought should also be given to the presence of cirrhosis which may contribute to portal hypertension and be associated with an increased risk of bleeding.[1]

In the future, the use of complete, large, multicentre, collaborative research databases[4] will be required to determine the true significance of this increasingly recognized complication. Most importantly, identifying prognostic factors that then allow those patients at high risk of further complications to be concentrated into appropriately designed treatment studies will be needed if the outcome associated with this severe illness is to be improved.

Conflicts of interest

None declared.

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