Pancolonic varices and portal venous overload

Colonic arterio-venous malformation, although a rare disease, merits to be known as possible cause of severe lower digestive haemorrhage, which often constitutes the first clinical symptom. Colonoscopy usually represents the first diagnostic tool, demonstrating grossly enlarged vessels in the colon mucosa, still bleeding or with signs of recent rupture. A more precise diagnosis of this disease usually requires a contrast-enhanced computed tomography and a selective angiography, which can also demonstrate the basis of this disease, which consists in congenital vascular malformations, involving the entire colon or one segment and the adjacent mesenteries (Fig. 1).

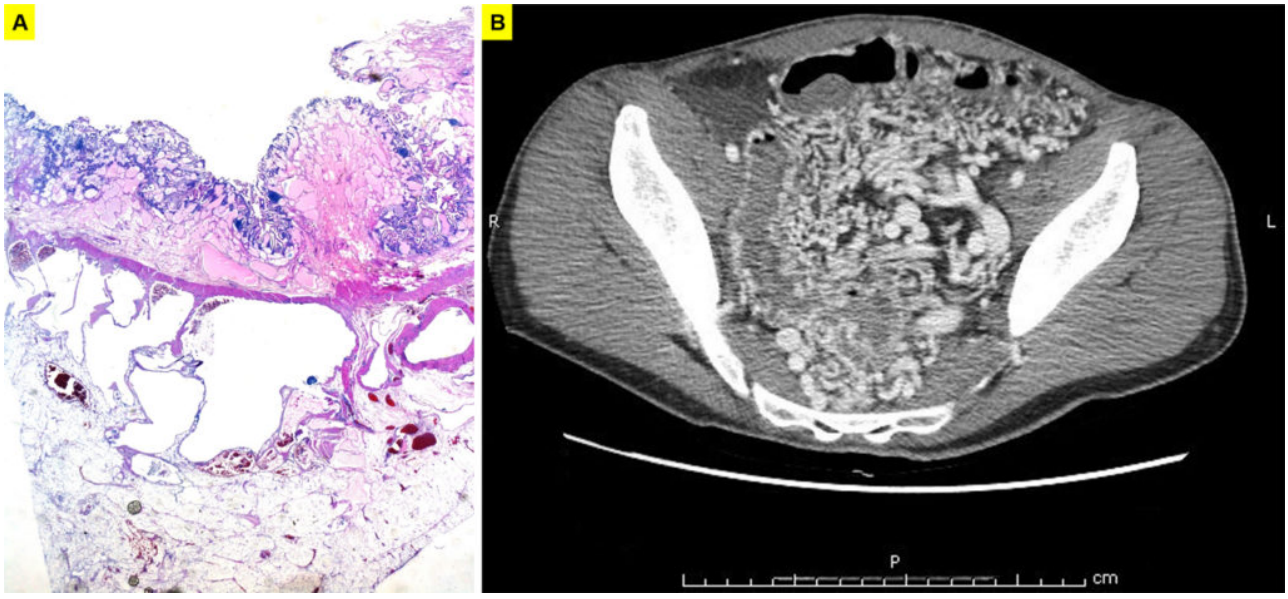

Fig. 1. A) Histology: arterio-venous malformations in the colonic wall and mesentery. B) Contrast-enhanced computed tomography axial scan: diffuse arterio-venous malformations in the sigmoid mesentery, with a cirsoid aspect.

The arterio-venous communications favour an overload of the corresponding venous system and secondary varices, which can rupture inside the colon. This increased venous return causes an augmented velocity in the portal blood stream, and a dilatation of the porto-mesenteric trunk, of the portal branches and of the hepatic veins (Fig. 2). In absence of concomitant liver diseases, the hepatic vein-portal gradient continues to be normal and this condition is not followed by signs of portal hypertension, as gastro-oesophageal varices or splenomegaly.

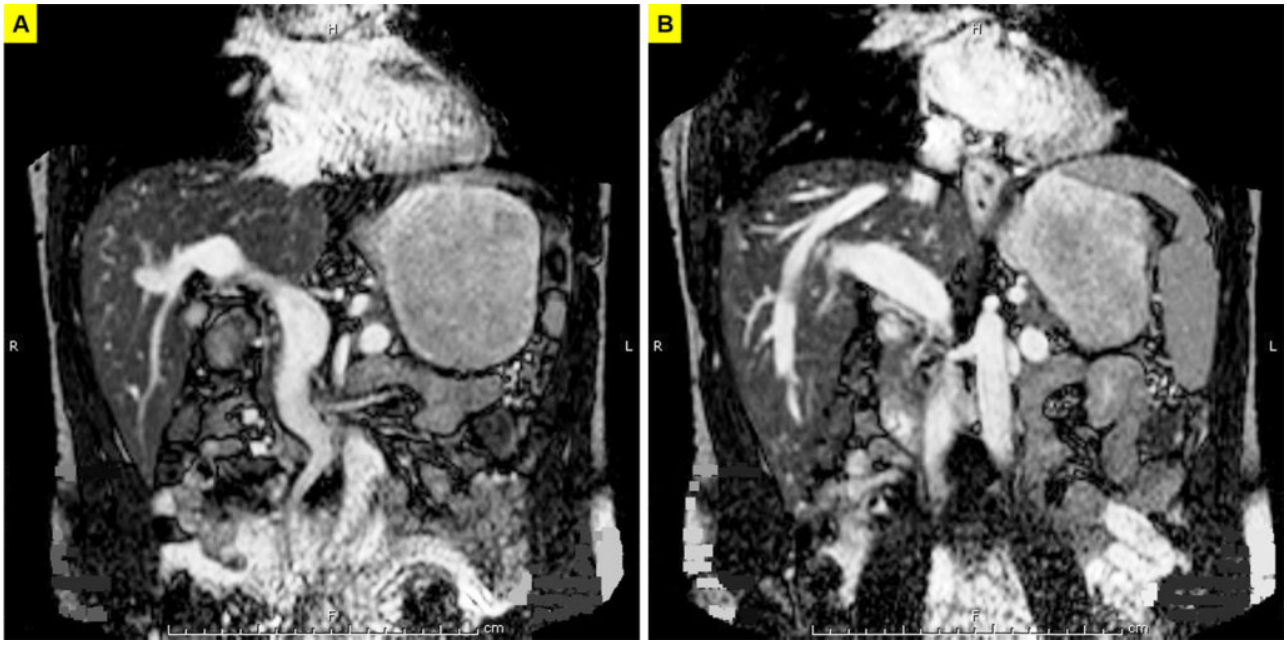

Fig. 2. Magnetic resonance coronal reformatted scans. A) Evident dilatation of the portal vein and of its right main branch. B) Dilatation of the right hepatic vein.

All this proves the great hemodynamic capacity of the hepatic vascular system and represents a typical model of portal “hyper-afflux”. Its evolution towards a portal hypertension seems not impending or likely, considering the long latency of this condition, as observed in adult patients. Inversely, in arterio-portal fistulas, that involve larger vessels, the portal over-inflow is at high pressure, with damage of the liver sinusoids and subsequent increase in their resistance, so determining a real portal hypertension. In Budd-Chiari syndrome, hypertension in the centrilobular veins have effect back directly on sinusoidal network with subsequent development of portal hypertension.

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