Portal vein thrombosis following endoscopic treatment for gastric varices with N-butyl-2-cyanoacrylate: Management with TIPS

Key words: Portal vein thrombosis. N-butyl-2-cyanoacrylate. Gastric varices. TIPS.

Dear Editor,

Gastric varices (GV) are present in approximately 20% (1) of patients with portal hypertension; the management of GV-related bleeding remains a challenge (2): Prognosis is worse (3) as compared to esophageal varices (EV), with rebleeding and/or mortality rates of up to 30%.

Case report

We report the case of a 71-year-old woman with histologically unconfirmed cryptogenic liver cirrhosis (MELD 6; Child-Pugh A6) who had several variceal bleeding events during the previous 2 years, which were attributed to EV with typical subcardial prolongations. A previous endoscopy managed to eradicate varices by sequentially combining band ligation and sclerosis using N-butyl-2-cyanoacrylate (NBC). Under optimized medical treatment a hepatic venous pressure gradient of 15 mmHg was measured. The splenoportal axis remained always patent on Doppler sonograms every 6 months.

She developed an additional hemorrhagic event, now secondary to the rupture of a previously undescribed GV, which received emergency treatment with NBC (2 cc) prior to delayed transjugular intrahepatic portosystemic shunt (TIPS) insertion (Fig. 1). During the procedure a radiopaque partial portal vein thrombosis (PVT) was identified both with the radioscopy and in indirect splenoportography, which was then confirmed with direct portography; TIPS allowed to resolve this PVT and the pressure gradient dropped to 8 mmHg. With regular US follow-up the average flow velocity in the portal vein (40 cm/sec) and inside the TIPS (100 cm/sec) remained normal after 12 months.

Discussion

The endoscopic (subjective) differentiation between the two varieties of “cardiofundal” varices in Sarin’s classification (1), which may occasionally coincide, is challenging; this differentiation is not arbitrary but results from anatomic peculiarities with therapeutic implications. GV development after intermittent endoscopic treatment for EV—and vice versa—is well documented (4), particularly in the setting of suboptimal drug prophylaxis, as in our case.

NBC is the endoscopic treatment of choice (5), with a success rate nearing 90%, but may result in severe, fortunately rare complications (6); it also is a potential cause of PVT, particularly following repeated injections (7). Complications decrease when technical recommendations are strictly complied with (8). The recommended amount of NBC (with lipiodol on a 1:1 basis) is 1 cc per action and varix. TIPS is an alternative for refractory bleeding (9) and the rescue therapy after conventional secondary prophylaxis failure (10).

The development of PVT in our patient is highly peculiar: Unusual, incidental, direct endothelial injury with an unequivocal temporal relationship with a prior NBC treatment. Lipiodol, a radiopaque compound, fully delineated the complete splenoportal axis, maybe because an amount greater than recommended was used. TIPS, as usual when “running out” of endoscopic options, induced in monotherapy a complete, instantaneous response with thrombosis resolution, a fact to our knowledge not previously described in the medical literature.
Fig. 1. Radioscopic visible gastric varices (A) with artifacts (embolization material remnants) from a previous endoscopic therapy with N-butyl-2-cyanoacrylate (arrow) in the portal vein and both intrahepatic portal branches. Direct portogram via a suprahepatic communication through the transjugular route (B) showing thrombosis-related repletion defects in the portal vein and left portal branch (arrows). Portosystemic shunt (C) using a specific covered stent (black arrow) between the right suprahepatic and portal veins, prolonged with a bare self-expandable prosthesis (white arrow) to go past the thrombosis in the portal trunk in order to extend and preserve its trajec (TY: Transjugular).

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