AngioJet assisted transvenous-transhepatic mechanical thrombectomy in the Portal Vein

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Disclosure

I do not have any potential conflicts of interest
Portal vein thrombosis (PVT) is not uncommon. Prevalence in patients with cirrhosis is up to 15%*. Clinical manifestation depends on the extent of the obstruction and the speed of development. Possible symptoms include colicky abdominal pain, diarrhea and variceal bleeding. However, acute PVT may also be clinically silent.

Management of Acute PVT

» **Primary management**: Anticoagulation and treatment of predisposing conditions (when possible).

» **Goal of therapy**: Prevent extension of clot, intestinal infarction and chronic portal hypertension. Allow for recanalization of the PV.

» **Therapy**: I.V. low molecular weight heparin to achieve rapid anticoagulation. Switch to an oral anticoagulant when possible.
Management of acute PVT

- PV recanalization success-rate with heparinization ~ 30-50%

- Recanalization rate is substantially lower in cases of extensive PVT (especially in cases including the SMV). Persistent PVT may lead to increasing abdominal pain, varicose hemorrhage and chronic portal hypertension.

- Catheter-directed lysis of acute extensive PVT using Urokinase or rtPA is an increasingly favored 1,2,3.

1: Blum U et al. Radiology, 1995
2: Hollingshead M et al. J Vasc Interv Radiol, 2005
Add-on therapies to anticoagulation

» **Systemic lysis**
  (i.v.)
  risk of bleeding

» **Indirect lysis**
  (via SMA)
  risk of bleeding
  risk of injuring the SMA

» **Direct lysis**
  (transhepatic or percutaneous)
  risk of bleeding
  technically more difficult
Methods

Three patients (mean age: 41.3 years) with extensive, therapy-refractory PVT underwent transvenous-transhepatic PV thrombectomy between 2015 and 2016, in order to re-establish PV patency.

Patients were followed clinically and with imaging (82, 484, 682 days, mean 349 days)
Clinical examination: All patients had progressive abdominal tenderness and reduced bowel activity, one patient had bloody diarrhea.

Laboratory: All patients had elevated C reactive protein (CRP) of 120 / 43 / 29 mg / l. Lactate was not increased in any of the cases.
Patients were referred to intervention after progression of symptoms under heparinization. PV access was achieved with the following steps:

- Ultrasound guided jugular access
- Insertion of a 10 F sheath
- Catheterization of right hepatic vein
- Transhepatic access to the portal venous system with the use of a 16 G needle set (ultrasound guided)
- Manipulation through occluded PV with guidewire and 4-F catheter
Mechanical thrombectomy was performed in the main portal vein (1), splenic vein/IMV (2) and the SMV (3) with an AngioJet thrombectomy device (Boston Scientific, USA) 15 min after using the power-pulse spray technique with 250,000 IU of Urokinase.
Following mechanical thrombectomy control-angiography was performed to assess portal venous flow and residual thrombus-load.
Multiple side hole infusion catheters were inserted into the SMV and/or spenic vein/IMV over which continuous thrombolysis was applied for at least 24 hours with a high dose of Urokinase (100,000 IU/h).
Control-angio following 24 hour catheter thrombolysis
Thrombophilia screening

» Patient 1: Thrombophilia screening inconspicuous
   Prior DVT

» Patient 2: JAK2-V617F-Mutation- Myeloproliferative Syndrome

» Patient 3: Protein S deficiency
To preserve PV patency polytetrafluoroethylene-covered stent-grafts (Viatorr; W.L. Gore and A.) were implanted to establish portalvenous-systemic shunts

Anticoagulation was continued with Phenprocoumon in all patients
Portalvenous-systemic shunt increases splanchnic flow, thus decreases the risk of re-thrombosis*

Patients with severe and chronic PVMT profit from a portosystemic shunt*

Results

- Success rate for recanalization of the PV was 100%. PV remained patent in all patients to the present day.

- No major complications.

- 1 patient developed a case of clinically non-relevant hematuria.

- 1 patient developed an episode of hepatic encephalopathy after discontinuing his laxatives which could be resolved with medication.
Conclusion

Transvenous-transhepatic mechanical thrombectomy using the AngioJet device can efficiently reduce thrombus-load in the PV system.

Portalvenous-systemic shunt should be considered in cases of severe PVT.
Thank you for your attention
Anticoagulation is generally recommended for at least 3-6 months.

Long-term anticoagulation is recommended for patients with permanent thrombotic risk factors that cannot be corrected and for patients with acute PVT that extends into the mesenteric veins.

In patients with cirrhosis, non-warfarin based therapy may be preferable since the INR may not reflect the patient's level of anticoagulation.
Conclusion

- Identification of predisposing conditions — Patients with PVT who do not have cirrhosis or who have compensated (Child A or B) cirrhosis should be evaluated for conditions that may have predisposed to thrombosis, such as prothrombotic states. Because PVT is common in patients with decompensated cirrhosis, it is reasonable not to pursue an evaluation for other predisposing conditions in such patients. The approach to the evaluation of patients with established venous thrombosis is discussed in detail elsewhere.
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