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REVIEW

Endovascular Treatment for Acute Portal Vein Thrombosis

Tatsuo Ueda, Hidemasa Saito, Sayaka Shirai, Fumie Sugihara, Ryutaro Fujitsuna, Taiga Matsumoto, Hiromitsu Hayashi and Shin-ichiro Kumita

Department of Radiology, Nippon Medical School Hospital, Japan

Abstract:
Acute portal vein thrombosis is characterized by nonspecific abdominal pain, causing severe morbidity and mortality. Prompt diagnosis is crucial to avoid short-term complications such as intestinal infarction, sepsis, and death. The therapeutic goal is to prevent thrombus extension into the mesenteric veins and intestinal ischemia complications. Systemic anticoagulation is the standard treatment. However, endovascular treatments such as thrombolysis, thrombectomy, balloon angioplasty, stent placement, and transjugular intrahepatic portosystemic shunt placement have been performed in patients who are refractory to anticoagulation therapy or at a high risk of intestinal ischemia. This review discusses the clinical and diagnostic considerations in acute portal vein thrombosis, focusing on current endovascular treatments that are effective and safe. However, prospective data are required to compare endovascular treatment techniques and assess their outcomes.

Keywords:
endovascular treatment, portal vein thrombosis, thrombolysis, thrombectomy, angioplasty

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Introduction

PV thrombosis (PVT) is defined as an occlusion of the PV system, including its branches, such as the mesenteric veins (MVs) and/or splenic veins (SVs) [1]. PVT can be classified according to the time of thrombus formation. Because PVT is often asymptomatic and the specific time of onset cannot always be determined, the American Association for the Study of Liver Diseases (AASLD) guidelines suggest subdividing PVT at diagnosis into recent (rather than acute), with a likelihood of onset of less than 6 months, and chronic, with a likelihood of onset of 6 months or more [2]. However, acute PVT generally occurs within 30 days of thrombus formation [3].

The incidence of PVT is approximately 0.7 per 100,000 people per year [4]. Acute PVT incidence rates are even lower, although specific figures are unavailable. Nonspecific abdominal pain is the most common manifestation of acute PVT [5], which can cause significant morbidity, including intestinal infarction, and mortality rates of up to 50%-75% [6, 7].

Systemic anticoagulation is the first-line therapy for PVT. However, insufficient dissolution of acute PVT was often found after systemic anticoagulation [8, 9]. Recently, endovascular treatments (EVTs) such as thrombolysis, thrombectomy, angioplasty, and transjugular intrahepatic portosystemic shunt (TIPS) have been reported to be effective for acute PVT treatment in addition to systemic anticoagulation [3, 6, 10-15]. Here, we reviewed the clinical and diagnostic considerations for PVT, focusing on current EVT techniques for managing acute PVT.

Causes

The risk factors for PVT may be categorized into local and systemic factors (**Table 1**). Regardless of the cause, the development of PVT is usually attributed to prothrombotic risk factors identified by Virchow, such as venous stasis, endothelial damage, and hypercoagulability [16]. Hepatopancreaticobiliary malignancies, cirrhosis, surgery, trauma, and inflammatory diseases of the abdomen (such as pancreatitis, cholecystitis, and cholangitis) are common local risk factors for PVT.

Hepatopancreaticobiliary malignancies contribute to ap-

Corresponding author: Tatsuo Ueda, s9015@nms.ac.jp
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Table 1. Local and Systemic Risk Factors for PVT [68].

Local risk factors	Systemic risk factors
Malignancy	Acquired
Hepatobiliary or any abdominal organ	Myeloproliferative disorders
Cirrhosis	Malignancy
Abdominal infection/inflammation	Antiphospholipid syndrome
Cholecystitis	Hyperhomocystinemia
Pancreatitis	Oral contraceptive use
Cholangitis	Recent pregnancy
Appendicitis	Inherited
Iatrogenic portal vein injury	Protein C deficiency
Splenectomy	Protein S deficiency
Cholecystectomy	Antithrombin deficiency
Abdominal surgery	Factor V Leiden mutation
Trauma	Prothrombin mutation

proximately 25% of PVT cases through extrinsic PV compression, increased hypercoagulability, or direct vascular invasion [17]. Decreased PV flux and dysfunction of the coagulation pathways may contribute to thrombus formation and the development of PVT in cirrhosis, depending on the degree of compensation [9, 16, 18].

Inherited thrombophilia (such as factor V Leiden mutation, protein C or S deficiency, and antithrombin deficiency), myeloproliferative disorders, pregnancy, and oral contraceptive use are common systemic factors that contribute to the development of PVT. A series of cases indicates that the most common systemic risk factors for PVT are myeloproliferative disorders and inherited thrombophilia [19].

Clinical Presentations

The clinical presentation of PVT depends on the degree and timing of thrombus development. Acute thrombosis with a complete occlusion of blood vessels causes visceral congestion and intestinal ischemia. Patients often suffer from abdominal pain, nausea, vomiting, gastrointestinal bleeding, fever, sepsis, and lactic acidosis [20]. A physical examination may reveal splenomegaly and abdominal distension. Failure to restore blood flow can result in intestinal perforation, peritonitis, septic shock, and death.

In cases of partial occlusion, symptoms may be minimal or absent. Even with chronic PVT, patients often remain asymptomatic owing to the adequate development of collateral circulation or cavernous transformation of the PV (cavernoma). In such cases, PVT is often discovered incidentally during the imaging of the abdomen. The clinical manifestations of chronic PVT are related to those of portal hypertension, including esophageal/gastric varices, gastrointestinal bleeding, portal hypertensive gastropathy, ascites, and splenomegaly with pancytopenia [21, 22].

Terminology and Classification

PVT refers to any obstruction of the intra- or extrahepatic PVs and PV branches. PVT is a heterogeneous disease in terms of its etiology, manifestations, natural history, and treatment options. Therefore, terminology and classification systems vary greatly between research articles. According to the AASLD guidelines [2], PVT should be described in terms of (a) site and extent, (b) time course, (c) degree of luminal occlusion, and (d) interval change. Site and extent are characterized by the involvement of the intrahepatic PV, main PV, PV branches (SVs and MVs), thrombus length, and contiguity of involvement between sites.

The time course of thrombosis is classified as "recent" and "chronic." The term "recent" implies a PVT presumed to be present for <6 months and is preferred to the term "acute" because the latter implies recent-onset thrombosis with clinical symptoms. However, not all patients with recent PVT develop symptoms. "Chronic" refers to PV obstruction lasting more than 6 months after suspected onset.

The AASLD guidelines propose the use of the following descriptors for luminal occlusion: (a) complete occlusion (100% of the lumen), (b) partial occlusion (>50% of the lumen), (c) minimal occlusion (<50% of the lumen), and (d) cavernous. Interval changes should be specified as progressive, stable, or regressive PVT.

Various classification systems for grouping PVT variants into broad categories have been proposed [23-31]. However, the most widely used classification is based on intraoperative findings at the time of liver transplantation in cases of liver failure [28], and the grade is related to the prognosis after transplantation. However, this classification is based solely on the anatomical localization of the thrombus. Therefore, new classifications that include the course of the disease and background liver disease have been proposed (**Table 2**) [24].

Imaging Findings

Doppler ultrasound (US) is the first-choice screening imaging modality for PVT diagnosis. US is noninvasive, simple, and repeatable, making it effective for monitoring PVT progression and determining treatment efficacy. A thrombus may be hypoechoic when acute or hyperechoic when

Table 2. Anatomical–Functional Classification of PVT in Cirrhosis as Proposed by Sarin et al. [24].

Site of PVT (Types 1, 2a, 2b, and 3)
Type 1: Only trunk
Type 2: Only branch: 2a, one branch; 2b, both branches
Type 3: Trunk and branches
Degree of portal venous system occlusion (O, NO)
O: Occlusive: No flow visible in the PV lumen on imaging/Doppler study
NO: Nonocclusive: Flow visible in the PV lumen through imaging/Doppler study
Duration and presentation (R, C)
R: Recent (first time detected in previously patent PV, presence of a hyperdense thrombus on imaging, absent or limited collateral circulation, dilated PV at the site of occlusion)
Asymptomatic: (As)
Symptomatic: (S), acute PVT features (with or without ABI)
Ch: Chronic (no hyperdense thrombus; previously diagnosed PVT on follow-up, portal cavernoma and clinical features of PHT)
Asymptomatic
Symptomatic: features of portal hypertension (with or without PHT)
Extent of PV system occlusion (S, M, SM)
Splenic vein, mesenteric vein, or both
Type and presence of underlying liver disease:
Cirrhotic, noncirrhotic liver disease, post-liver transplant, HCC, local malignancies, and associated conditions

ABI, acute bowel ischemia; PHT, portal hypertension; PVT, portal vein thrombosis. Procedure-related results.

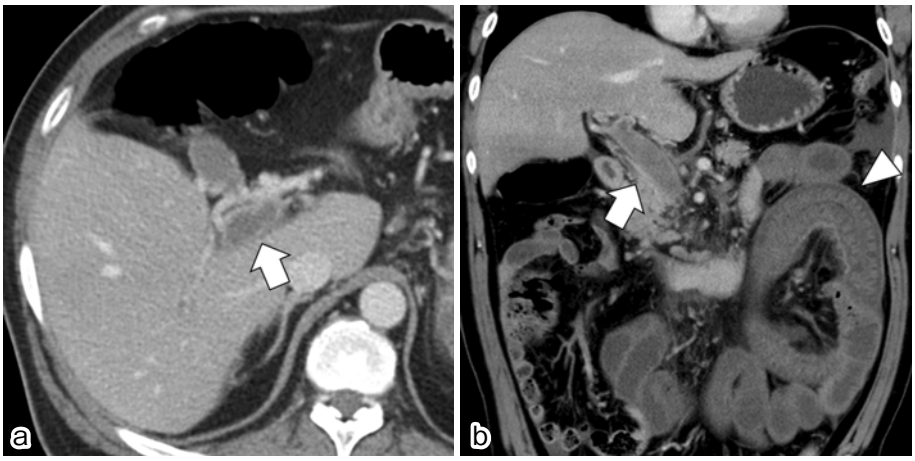

Figure 1. Acute portal vein thrombosis (PVT) in a 47-year-old male.
(a, b) Axial and coronal contrast-enhanced CT images in the PV phase demonstrate a large thrombus (arrow) within the main PV with bowel congestion (arrowhead).

chronic. When a thrombus occludes the PV, the main trunk and the right and left branches of the PV are not visualized and the PV distal to the occlusion is dilated. If the PV diameter does not change with respiration, acute PVT is strongly suspected. However, chronic PVT is characterized by collateral vessel formation around the hepatic portal area. Doppler US can be used to confirm the direction of blood flow in the portal lumen and collateral vessels. Color Doppler can be used to diagnose PVT with 94%-100% sensitivity and 96% specificity and is considered an effective examination method [32].

The diagnosis and extent of the acute portal occlusion should be confirmed by contrast-enhanced CT and MRI (**Fig. 1**). Acquisition of images at the correct time (portal phase) is essential to avoid misdiagnosis. PVT appears as a filling defect in the PV on contrast-enhanced CT. On non-contrast CT, it appears hypodense or isodense compared with adjacent tissues. It may appear hyperdense when the thrombus has recently formed (<1 month) (**Fig. 2**) [33]. Thus, an acute thrombus may be difficult to distinguish using contrast-enhanced CT alone (**Fig. 2**). Images acquired during the late arterial phase are not optimal for PVT diagnosis. Furthermore, in cases of low PV flow, delayed contrast arrival in the PV may be observed on CT as it may create the appearance of a filling defect, leading to a false positive diagnosis of thrombosis [34].

Portal phase CT scans show the absence of a visible lumen corresponding to the PV clot and provide additional information regarding thrombus spread to the MVs and arches, the presence of a local factor, or intestinal congestion and ischemia (**Fig. 1** and **2**). Distal thrombosis (obstruction of secondary SMV radicals), intestinal anomalies (ho-

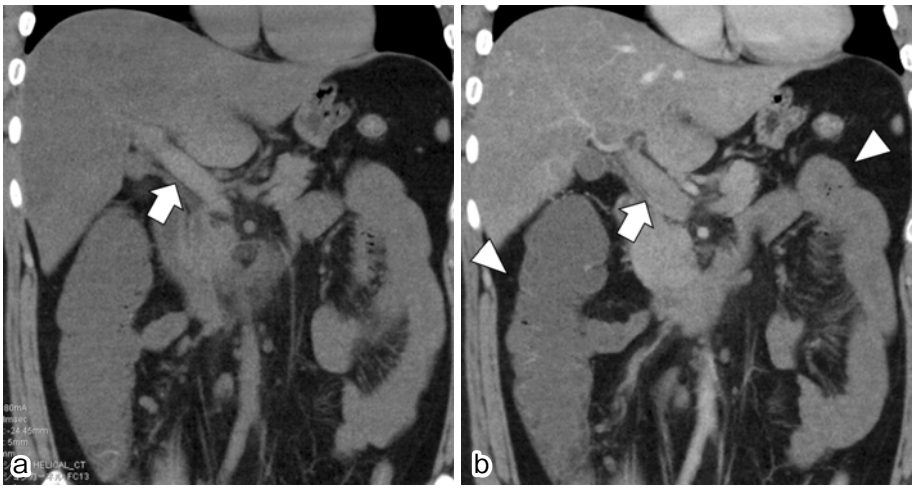

Figure 2. Acute PVT in a 22-year-old male.
(a) A coronal noncontrast CT image shows hyperdense areas (arrow) in the PV, which indicates a relatively acute thrombus.
(b) A coronal contrast-enhanced CT image in the PV phase demonstrates PVT as an unenhanced PV (arrow) with bowel congestion (arrowhead).

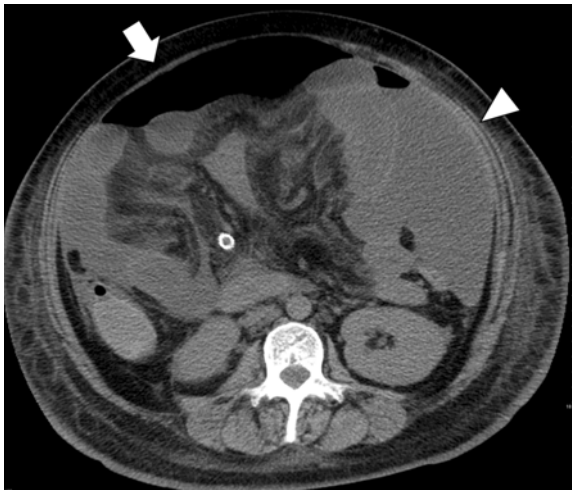

Figure 3. Acute PVT in a 53-year-old female.
An axial noncontrast CT image shows free air (arrow), which indicates intestinal perforation, hyperattenuating wall thickening, bowel dilatation, and large ascites (arrowhead).

mogeneous or heterogeneous hypoattenuating or hyperattenuating wall thickening, dilatation, or abnormal or absent wall enhancement), mesentery, mesenteric stranding, massive ascites, pneumatosis, and portal venous gas are more frequently observed in patients requiring intestinal resection (**Fig. 3**) [35].

Indications for Treatment

The therapeutic goals of acute PVT are to (a) prevent thrombus spread to the MVs, (b) prevent complications of bowel ischemia (MV infarction), and (c) achieve recanalization of the PV to prevent the development of portal hypertension.

Pharmacological Treatment (Anticoagulation Therapy)

The anticoagulation therapy of choice in patients with PVT has historically been limited to heparin and warfarin. However, the introduction of direct oral anticoagulants (DOACs) has added complexity to treatment decisions by providing more therapeutic options [36].

Studies investigating the role of anticoagulation in PVT as the primary indication for therapeutic anticoagulation are limited [37, 38]. In 2010, a large prospective study investigating the efficacy and safety of anticoagulants (heparin and warfarin) in 95 consecutive patients with acute PVT without cirrhosis was published [39]. Anticoagulation was successful in 38% of the patients who achieved complete recanalization. Two patients had thrombotic progression with intestinal infarction, and nine patients had bleeding during anticoagulation therapy. Data on the use of DOACs as a treatment for PVT are limited. However, DOACs are becoming an increasingly common treatment for patients with thrombosis.

Several retrospective small-scale studies have demonstrated the successful use of DOACs for treating acute PVT without cirrhosis [40, 41]. A large, retrospective, single-center cohort studied 330 patients with PVT without cirrhosis who were treated with warfarin (n = 108), low-molecular-weight heparin (n = 70), DOACs (n = 93), and no anticoagulation (n = 57) [42]. DOAC therapy had superior efficacy (thrombolytic rate) and less severe bleeding compared with warfarin in the cohort. DOACs offer several advantages over warfarin, including no requirement for monitoring and predictable anticoagulant effects. In addition, Hidaka et al. [43] reported that antithrombin III (AT-III) is safe and effective in patients with PVT and liver disease, achieving complete and partial recanalization in 55.6% of the patients. AT-III has been covered by insurance for PVT in Japan since 2017.

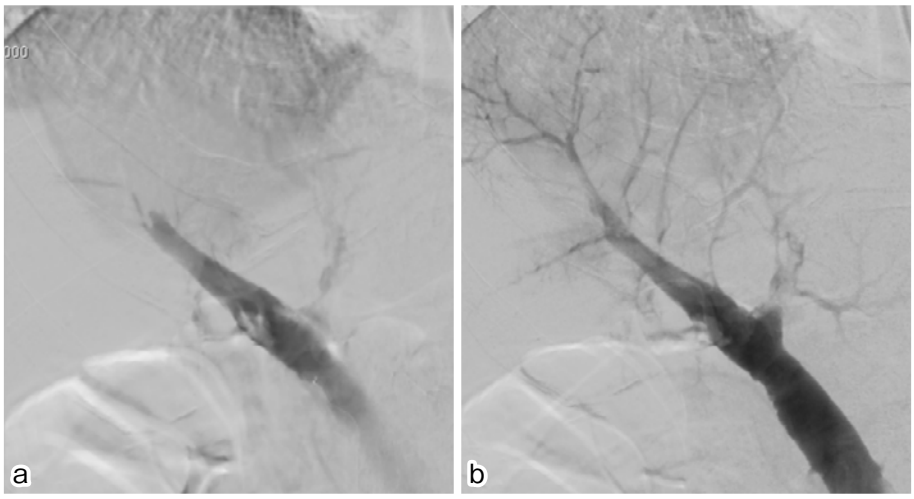

Figure 4. Acute PVT in a 47-year-old male.
(a) A pretreatment angiogram shows a filling defect of contrast in the peripheral PV. Catheter-directed thrombolysis (CDT) with urokinase is performed via direct PV access through a transileocolic approach.
(b) A post-CDT angiogram shows improved peripheral PV flow.

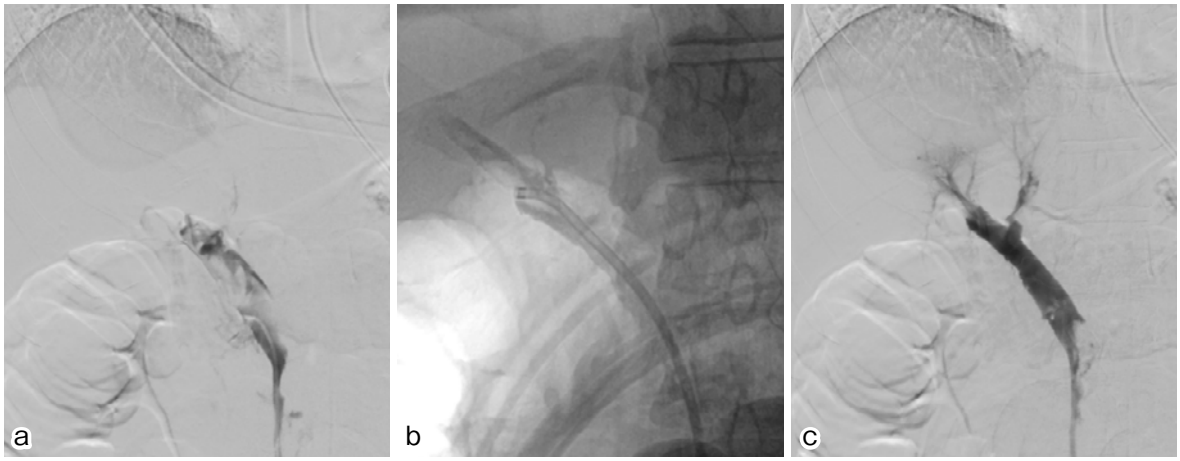

Figure 5. Acute PVT in a 47-year-old male.
(a) A pretreatment angiogram shows large filling defects of contrast in the main PV.
(b) Manual aspiration thrombectomy using an 8 Fr guiding catheter is performed via direct PV access through a transileocolic approach.
(c) A post-aspiration thrombectomy angiogram demonstrates debulking of the thrombus.

EVT

Systemic anticoagulation is the standard therapy for acute PVT. However, most thrombi are insufficiently dissolved by this therapy [8, 9]. Recently, EVTs such as thrombolysis, thrombectomy, angioplasty, and TIPS have emerged as effective treatment options for acute PVT in addition to systemic anticoagulation [3, 10-12]. EVT is performed in patients who are refractory to anticoagulation therapy or at a high risk of intestinal ischemia who require early revascularization.

Approach Site

Different approaches to accessing the PV include the percutaneous transhepatic, percutaneous transsplenic, and transjugular approaches (including TIPS) and the transileocolic approach under laparotomy (**Fig. 4**-**8**). The percutaneous approach is less invasive than the transileocolic approach, but the risk of bleeding is higher. The percutaneous transhepatic approach is the most performed technique because it is relatively easy to execute [44].

In a report of 46 patients who underwent the transsplenic approach, 3 patients (6.5%) experienced major bleeding and 6 patients (13%) experienced minor bleeding [45]. In addition, the transsplenic approach to access the thrombosed PV was superior to the transhepatic approach, with a higher success rate of PV recanalization (60 of 61 patients) and fewer side effects in a small retrospective series [46].

The transjugular approach allows access to the PV without violating the liver capsule [1].

The transileocolic approach, which requires laparotomy

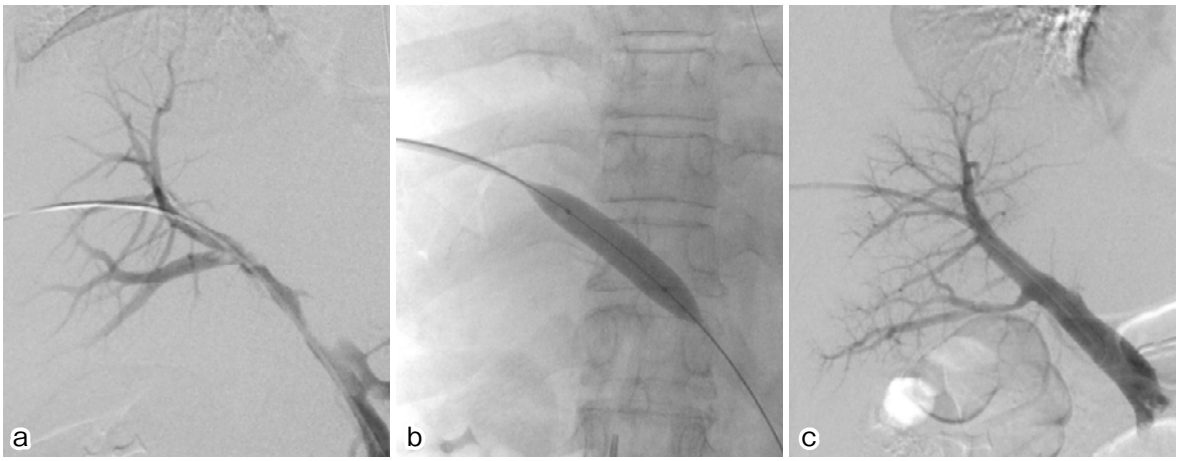

Figure 6. Acute PVT in a 58-year-old female.
(a) A pretreatment angiogram shows partial filling defects of contrast in the PV.
(b) Balloon angioplasty in addition to thrombolysis and thrombectomy is performed via a transhepatic approach.
(c) A post-treatment angiogram demonstrates the disappearance of PVT.

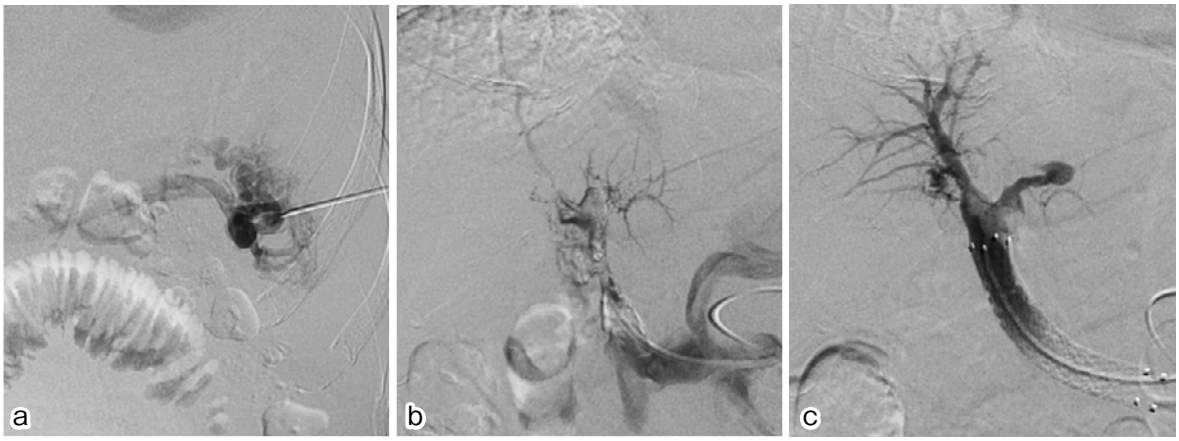

Figure 7. Acute PVT in a 54-year-old male.
(a) A transsplenic approach is performed to treat PVT because of large ascites surrounding the liver.
(b) A pretreatment angiogram shows a large filling defect of contrast in the PV. In addition to thrombolysis and balloon angioplasty, stent placement is performed via a transsplenic approach.
(c) A post-treatment angiogram demonstrates improved PV flow.

and an open abdominal approach with vacuum-assisted wound closure, is limited to cases requiring bowel resection [10]. However, this approach has some advantages over the percutaneous approach. First, the percutaneous approach may be difficult to perform in cases of massive ascites and complete obstruction of the intrahepatic PV. Second, EVT should be performed under laparotomy in cases requiring necrotic bowel resection. Third, the transileocolic approach is related to a lower bleeding risk compared with the other approaches because ileocolic vein puncture can be performed under direct observation. Lastly, the transileocolic approach allows the use of larger devices, such as those for aspiration or angioplasty [11].

EVT Techniques

Thrombolysis

Traditionally, thrombolysis has been widely used as an EVT for acute PVT and should only be considered in selected cases of persistent intestinal ischemia despite anticoagulation [2]. Systemic and local catheter-directed thrombolysis (CDT) can be performed with thrombolytic agents such as urokinase and tPA. CDT can be performed via direct PV access using a percutaneous transhepatic [6, 44, 47-50], percutaneous transsplenic [45, 46], transjugular intrahepatic [1], or transileocolic approach (**Fig. 4**) [10, 11]. Indirect intra-arterial approaches via the superior mesenteric artery have also been used [47, 50-52]. After an incomplete EVT session with a residual thrombus, the sheath and heparin-coated catheter remain in the thrombus, and in the interval between EVT sessions, an automated pump can be

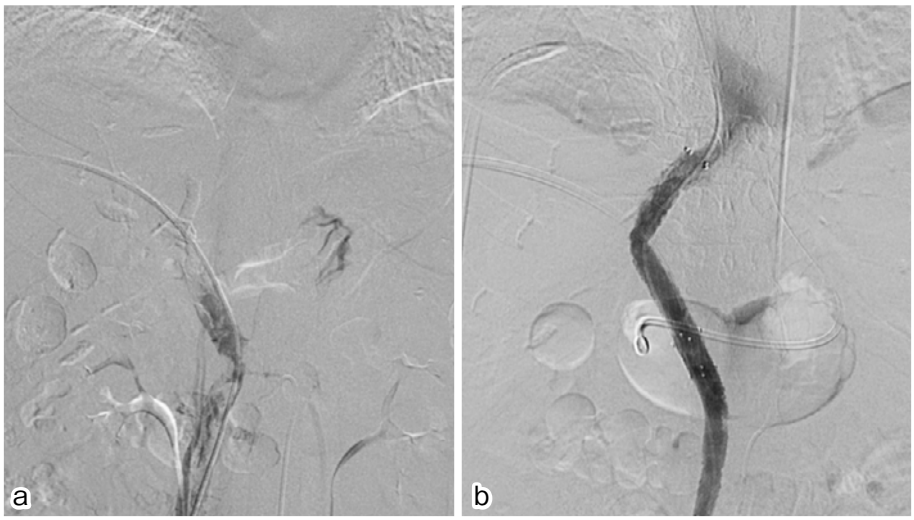

Figure 8. Acute PVT in a 53-year-old female.
(a) A pretreatment angiogram shows large filling defects of contrast in the superior mesenteric vein, main PV, and peripheral PV.
(b) In addition to thrombolysis, thrombectomy, and angioplasty, transjugular intrahepatic portosystemic shunt placement is performed via the jugular vein approach. A post-treatment angiogram demonstrates improved PV flow.

used to perform adjunctive continuous CDT with urokinase [10, 11].

In a study of 65 patients with noncirrhotic PVT comparing anticoagulation alone with anticoagulation plus pharmacomechanical thrombectomy and thrombolysis [53], there was no significant difference in the bowel resection rates, occurring in 15% of patients who received anticoagulation therapy alone and 10% of patients who received adjunctive thrombolysis and/or thrombectomy. However, the study [53] observed long-term complete or partial resolution of PVT at the final follow-up in 37% of patients treated with anticoagulation alone, compared with 71% in the anticoagulation plus intervention group.

According to a systematic review of thrombolysis for non-cirrhosis-related PVT, the recanalization rate following thrombolysis was 84% and the symptomatic improvement rate was 86%. However, direct and systemic thrombolysis approaches have demonstrated no significant recanalization rates [54]. Significant procedure-related morbidity and mortality have been reported with recanalization rates similar to those achieved using anticoagulation alone [47, 50, 55]. In addition, urokinase use is currently restricted owing to the coronavirus disease 2019 pandemic. Therefore, hybrid EVT, combining thrombectomy, angioplasty, and TIPS, in addition to thrombolysis, has recently been reported [3, 6, 10-15].

Thrombectomy

Thrombectomy restores flow in the main PV through balloon, rheolytic, and aspiration thrombectomy. The successful application of thrombectomy-assisted thrombolysis has been described in the literature, but there are no prospective data comparing the results of the different techniques [56].

In balloon thrombectomy, a balloon is inflated (Fogarty, Edwards Lifesciences, Irvine, CA, USA) over the thrombus and the inflated balloon is pulled back over a guide wire to draw the thrombus into a patent vein, which is subsequently washed away. This is usually followed by angioplasty to strengthen the lumen reinforcement and macerate any remaining thrombus. Rheolytic therapy uses high-velocity jets of saline to macerate the clot and promote passive clot expulsion based on the Bernoulli principle. Currently, there are no rheolytic thrombectomy devices available in Japan. Aspiration thrombectomy has evolved from the simplest setup of a large-volume syringe attached to the end of a large-bore catheter (**Fig. 5**) to the use of vacuum-driven thrombectomy tools. Aspiration thrombectomy uses a continuous aspiration device (Penumbra Indigo System, Alameda, CA, USA) primarily used for neurovascular and peripheral procedures. Unlike rheolytic thrombectomy, aspiration thrombectomy does not return the volume to the patient and can rapidly decrease the volume if the thrombus does not directly engage the end of the catheter.

A few studies on thrombectomy have not shown improved clinical or radiological outcomes [6, 44]. Although the combination of thrombectomy and direct thrombolysis remains controversial, some studies have advocated for its use in the initial debulking of a thrombus to minimize the duration of subsequent thrombolysis and enhance thrombus dissolution, which theoretically minimizes thrombolysis-related complications. Two studies [6, 44] demonstrated shorter mean therapy duration and fewer bleeding complications compared with other studies [47-52, 57]. EVT is used for acute limb ischemia, but the use of aspiration thrombectomy as a first-line treatment can reduce the need for CDT [58, 59].

The long-term effects of thrombectomy remain unclear. Uflacker [60] reported that thrombectomy could increase the injury risk to the vessel wall, facilitating further formation of thrombi in the medium and long terms. Among the two

aforementioned studies, only one patient who underwent thrombectomy and thrombolysis experienced a relapse and developed recurrent thrombosis of the PV [6, 44]. Benmassaoud et al. [48] used direct thrombolysis and thrombectomy with 100% recanalization, but this was significantly confounded by the initial systemic thrombolysis within 48-72 h.

The three aforementioned studies [6, 44, 48] suggests that directed thrombolysis and thrombectomy may be effective adjunctive therapeutic options rather than alternative therapies. Comparative data on the efficacy of specific endovascular interventions are unavailable. However, considering the high bleeding rate associated with thrombolysis, thrombectomy may be the most appropriate treatment in acute settings [1].

Angioplasty (balloon angioplasty and stent placement)

Stent placement in acute PVT enables the compression of the thrombus by the stent and fixation to the vessel wall, thereby preventing thrombus dislodgement. This technique allows rapid revascularization while reducing the distal embolization risk. However, stent placement is suitable only for larger vessels, including the main PV or SMV. In addition, angioplasty techniques, including balloon angioplasty and stent placement, can treat a residual thrombus and stenosis in cases where further thrombolysis or thrombectomy is not possible. However, balloon angioplasty should only be conducted after the completion of thrombectomy because distal embolization of angioplasty-induced thrombi occurs frequently, especially in residual thrombosis [10, 61].

Balloon angioplasty (**Fig. 6**) and/or stent placement (**Fig. 7**) without thrombolysis or thrombectomy may be safe and effective treatments for postoperative mainstream PV and SMV thrombosis [10, 13]. In general, the long-term outcomes in patients with chronic PVT are favorable (five-year survival rate > 70%) and primarily depend on the associated disease. Thus, the risk-benefit ratio of such invasive procedures should be considered [62].

TIPS placement

TIPS placement alone improves PVT due to improved portal hypertension and restored PV flow in liver cirrhosis (**Fig. 8**). Two meta-analyses [63, 64] reported post-TIPS PVT resolution rates of 73.7% [63] and 77.7% [64] and encephalopathy rates of 25.3% [63] and 16.4% [64], respectively. Although PVT alone is not an indication for TIPS placement under current guidelines because of the significant risk of hepatic encephalopathy, the AASLD guidelines recommend the assessment of other TIPS indications in patients with cirrhosis who have recently had PVT [2] and the European Association for the Study of the Liver guidelines recommend TIPS placement in liver transplant candidates who have PVT refractory to anticoagulation therapy [65].

EVT Endpoint

The goal of acute PVT treatment is to reconstruct the blocked veins. Establishing portal circulation, that is, blood flow from the PV to the hepatic portal vein via the sinusoids, is critical for maintaining patency [10]. Establishing portal circulation in some areas can maintain patency, even when partial recanalization is achieved. EVT techniques, including thrombectomy, balloon angioplasty, and stent placement, are effective in removing thrombi within the main lumen of the PV and SMV, but they cannot remove small peripheral thrombi. By contrast, CDT may be useful for small peripheral thrombi that are impossible to remove by EVTs. Therefore, repeated thrombectomy and CDT should be combined to establish portal circulation.

However, even when complete radiographic recanalization is achieved, reocclusion may occur in the early stages where reocclusion rates are particularly high [6, 15]. Therefore, anticoagulation therapy following EVTs is critical to maintain PV patency [19, 66].

Surgical Treatment

Surgical thrombectomy for PVT is generally challenging and is linked to substantial morbidity, but it can be indicated if anticoagulation or minimally invasive EVTs are unsuccessful [67]. If the thrombus is new and is confined to the SMV, which is relatively rare, surgical thrombectomy may be accomplished. However, recanalization is achieved in only 30% of patients and is related to a high rate of recurrence when performed >30 days after the apparent onset [51].

Conclusion

EVTs, including thrombolysis, thrombectomy, angioplasty, and TIPS, are effective in patients with acute PVT who are refractory to anticoagulation therapy or at a high risk of intestinal ischemia and require early revascularization. EVT procedures are performed using transhepatic, transsplenic, transjugular, and transileocolic approaches. Establishing portal circulation with sufficient inflow and outflow is important for maintaining PV patency. Removing or reducing the thrombus in the main PV and SMV is crucial, and removing minute peripheral thrombi is necessary to establish portal circulation. Prospective data are required to compare the management of EVT techniques and evaluate their outcomes.

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