Inferior vena cava-syndrome

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Summary: Inferior vena cava syndrome (IVCS) is caused by agenesis, compression, invasion, or thrombosis of the IVC, or may be associated with Budd-Chiari syndrome. Its incidence and prevalence are unknown. Benign IVCS is separated from malignant IVCS. Both cover a wide clinical spectrum reaching from asymptomatic to highly symptomatic cases correlated to the underlying cause, the acuity, the extent of the venous obstruction, and the recruitment and development of venous collateral circuits. Imaging is necessary to determine the underlying cause of IVCS and to guide clinical decisions. Interventional therapy has changed the therapeutic approach in symptomatic patients. This article provides an overview over IVCS and focuses on interventional therapeutic methods and results.

Keywords: Inferior vena cava syndrome, tumor compression, inferior vena cava thrombosis, Budd-Chiari syndrome, venous stenting, catheter-directed thrombolysis, thrombaspersion

Introduction

The inferior vena cava-syndrome (IVCS) is caused by agenesis, compression, invasion, stenosis, or thrombosis of the inferior vena cava (IVC). It may also be associated with Budd Chiari syndrome (BCS).

IVCS comprises a broad clinical spectrum [1–3]. The exact incidence and prevalence of IVCS are unknown [1, 2], but from the clinician's point of view it is seen less frequent than superior vena cava syndrome (SVCS) indicating that it is more often asymptomatic or oligosymptomatic [1].

In contrast to SVCS overviews addressing IVCS are scarce. The following article describes the clinical aspects and spectrum of the syndrome to enable the reader to suspect IVCS and to support diagnostic and therapeutic decisions. It will also focus on interventional techniques in the treatment of IVCS as these modalities have substantially changed the therapeutic approach in symptomatic patients.

Etiology, anatomy, and pathophysiology

The venous backflow from the lower half of the body, the pelvis, the genitals, and the lower extremities usually drains through the right and left iliac veins into the IVC. The IVC feeds about two third of the total venous return to the heart. Blood flow in the IVC is supported by the respiration cycle

(increased flow during expiration) and by changes of the tricuspid valve level and atrial geometry associated with the cardiac cycle.

The IVC can be divided in different segments, which develop in a complex process early in gestation: (1) the suprahepatic IVC originates from the embryonic vitelline venous system (2) the suprarenal infrahepatic IVC from the embryonic subcardinal venous system (3) the infrarenal IVC from the embryonic supracardinal venous system (4) the iliac veins and the confluence region from the embryonic posterior cardinal vein [4]. Abnormal regression or persistence of these embryonic veins lead to congenital anomalies of the IVC [4, 5]. Most important, failure of the conjunction between the hepatic and prerenal segments results in infrahepatic interruption of the IVC with azygos continuation [3, 4, 6]. In this anomaly infrarenal IVC is intact, whereas the suprarenal portion of the IVC is absent. Blood is shunted through the retro-crural azygos vein, which becomes consecutively dilated [4–6]. The prevalence of the infrahepatic interruption of the IVC with azygos continuation is approximately 0.6 % [4, 5] and is sometimes accompanied by congenital heart defects, asplenia, or polysplenia syndromes [4, 5, 7]. In contrast, absence of the infrarenal IVC with preservation of the suprarenal segment of the IVC is very rare and it has been assumed that this condition is acquired by intrauterine or perinatal thrombosis of the IVC and not truly embryologic in origin [4].

Inferior vena cava anomalies have been recognized as risk factors for deep venous thrombosis (DVT) in children

and young adults [8–10]. IVC anomalies occur in about 0.5% to 1% of the general population, and in 2% to 3% of patients with congenital heart defects [4, 9, 10]. Chee YL et al. and Ruggeri M et al. reported a prevalence of about 5% in younger DVT-patients [9, 10] and Bass JE et al. identified 5 (16%) anomalous IVC in 31 younger patients with ilio-femoral DVT assessed by venography and magnetic resonance angiography [5]. Furthermore, analysis of the RIETE-registry revealed that patients with IVC-abnormalities are more likely to develop severe post-thrombotic syndrome after their first DVT than patients without IVC-anomalies [8]. Thrombosis of the IVC was otherwise identified to be associated with primary thrombophilia, hormonal contraception, estrogen replacement therapy, Behcet's disease, paroxysmal nocturnal hemoglobinuria, and malignancies [11, 12]. IVC-filters increase the risk of IVC-thrombosis especially when anticoagulation therapy is withheld [11–13].

IVCS may also develop in Budd-Chiari syndrome (BCS), a rare occlusive disease affecting the hepatic veins (HV) and/or the IVC [14–16]. French, Italian, and Swedish studies reported incidences of 0.69 to 2.2 per million inhabitants [17–19]. In Europe, BCS most often occurs in primary and secondary thrombophilia including hormone intake, Behcet's disease, paroxysmal nocturnal hemoglobinuria, and myeloproliferative syndromes [14, 16, 19, 20], whilst in Asia it is often caused by membranous or segmental stenoses or occlusions of the HV and/or the IVC [21]. Rarely, liver tumors or extrahepatic tumors compress or invade the IVC and/or the HV [14–16].

According to the location of the obstruction a truncal type (I) with occlusion of the hepatic segment of the IVC $\pm$ HV, a radicular type (II) with occlusion of the hepatic veins, and a veno-occlusive type (III) with obstruction of small centrilobular veins are differentiated [14, 15]. Types of IVC-involvement in BCS have been characterized in more detail as the exact location and extend of these lesions influences interventional strategy. In type I and II-lesions there are segmental stenoses or occlusions of the IVC below the orifice of the HV, while in type III-lesions the stenoses or occlusions extend above the HV junction [14].

Symptoms and signs of BCS are unspecific and consist of abdominal pain, hepatomegaly, ascites, lower extremity swelling, and jaundice [14–16]. Diagnosis is made by duplex ultrasound, CE-CT with portal phase imaging, and /or properly timed CE-MRI [14, 16]. BCS might lead to liver cirrhosis, portal hypertension, and hepatocellular carcinoma [14, 15]. The overall prognosis of the patients is reduced, but improved significantly during the last decades by the introduction of anticoagulation therapy and interventional procedures like recanalization of the stenosed or occluded VCI and hepatic veins, transjugular or direct intrahepatic porto-systemic shunt placement (TIPS, DIPS), or by orthotopic liver transplantation if all other modalities have failed [14, 15, 18, 19, 21].

When the IVC becomes significantly obstructed venous pressure below the lesion rises including the pelvic and

lower extremity veins. A pressure gradient > 3 mm Hg correlating with a Doppler peak velocity ratio of > 2.5 across the lesion was defined to characterize a $> 50\%$ stenosis [22]. Nevertheless, in patients selected for interventional therapy pressure gradients in the IVC often reach double-digit values [see literature in the interventional therapy sections].

Venous congestion in the lower body usually leads to anasarca, ascites, genital edema, and lower extremity swelling [1, 3, 11]. On the other hand, anatomically preformed collaterals enlarge under increased pressure conditions supporting venous backflow [2]. Thus, the development and the severity of symptoms in IVCS is correlated with the acuity and extent of the venous obstruction and is inversely correlated with the recruitment or development of venous collateral circuits [2, 11].

In humans, there are preferred collaterals from the IVC to the superior vena cava (SVC) and the right heart, which can be divided into four major pathways [2]: (1) the deep pathway comprising the intravertebral, paraspinal, extra-vertebral venous plexus, and the ascending lumbar veins, which anastomose with the azygos-hemiazygos-venous system (2) the intermediate pathway including the peri-ureteric venous plexus, the gonadal veins and the renal veins, which drain into the IVC (3) the superficial pathway via the inferior and superior epigastric veins and the mammary veins to the subclavian veins and/or through the circumflex iliac veins, the lateral thoracic veins, and the axillary veins [2]. The most important of these collateral circuits is the deep pathway, which is connected to the azygos-hemiazygos system.

Cardiac output in IVCS is usually preserved. Nevertheless, cardiac preload and cardiac output reserve was shown to be reduced in severe IVC stenosis under exercise conditions [23]. Only in acute complete IVC thrombosis or thrombosis of collaterals, in cases of preexisting chronic heart failure, and in mass compression of the central veins or the heart itself cardiac output might already be compromised under resting conditions [11, 24–26].

Renal vein outflow obstruction might develop by renal vein thrombosis or obstruction, leading to renal proteinuria, hematuria, impaired renal function, or even pelvic congestion syndrome [1, 2].

Malignant IVCS was described in patients with renal carcinomas, adrenal carcinomas, pheochromocytomas, pancreatic carcinomas, hepatocellular carcinomas, ovarian carcinomas, cervical carcinomas, prostatic cancer, gastric cancer, retroperitoneal sarcomas, metastatic malignant disease involving the pelvic and retroperitoneal lymph nodes, and primary lymphomas [11, 12, 27–32]. Rarely, primary tumors of the IVC like endotheliomas and leiomyosarcomas were found [33, 34].

Benign causes of IVCS are enlarged caudate lobes in patients with liver cirrhosis, giant liver and renal cysts, granulomatous diseases, Crohn's disease, aortic aneurysms, retroperitoneal hematomas, retroperitoneal fibrosis, and even blunt abdominal trauma [35–39].

Clinical symptoms

Symptoms in IVCS vary greatly [1]. Patients may be asymptomatic or oligosymptomatic despite significant IVC-obstruction or otherwise present with edemas of the lower body, the genitals, and the lower extremities. Ascites or jaundice may also be present. Patients with acute or subacute IVC-thrombosis or tumors often complain of abdominal pain, back pain, and/or pelvic pain radiating to the groin [1, 11]. In more chronic courses dilated and tortuous superficial veins on the abdomen, the thoracic wall, and the lower extremities indicate collateral formation and signs of chronic venous disease with leg swelling, discolorations, ulcers, and venous claudication may be present [1, 3]. Renal vein involvement is indicated by flank pain, proteinuria, and hematuria resembling nutcracker syndrome, which might otherwise develop independently of IVC-obstruction or compression. Pelvic congestion syndrome might be seen in patients where the intermediate collateral pathway is generated [2].

Establishing diagnosis

The diagnosis of IVCS is based on clinical grounds [1, 3]. As the etiology covers a broad spectrum of pathologies imaging is necessary to determine the underlying cause and guide therapeutic decisions. Diagnostic instruments are duplex ultrasound, contrast enhanced ultrasound (CEUS), intravascular ultrasound (IVUS), contrast-enhanced MRI and CT, and FDG PET-CT.

Diagnosis of IVC pathologies is challenging with duplex ultrasound (DUS) for some reasons: (1) IVCS is rare and may be ignored, when exclusion of a lower extremity thrombosis is in the focus of the examiner (2) direct visualization of the iliac veins and the IVC is often hampered by bowel gas or massive abdominal adiposity (3) diagnosis highly depends on the experience of the examiner.

Regardless of its limitations, DUS is the modality of choice for an initial screening examination [11, 12, 14–16, 40, 41]. As has been shown by Lin et al. monophasic waveforms in the common femoral veins or the external iliac veins are reliable indicators of proximal venous obstruction [40], as in case of endoluminal obstruction or external compression respiratory variation of the Doppler waveform is dampened, resulting in a monophasic constant flow curve (see Figure 1). If this flow pattern is present in both femoral veins involvement of both iliac veins and/or the IVC is signified [41]. Furthermore, reversed flow in the superficial epigastric veins indicate for iliaco-caval occlusion or compression [42]. Thus, a monophasic flow-pattern in both femoral veins and/or a reversed flow in the superficial epigastric veins inevidently indicates a closer examination of the proximal venous tracts [40–42].

If good ultrasonographic views can be obtained the exact diagnosis (IVC-thrombosis, stenosis, tumor compression, IVC-anomaly) may be established by DUS alone [41]. Contrast-enhanced ultrasound (CEUS) is useful for the detection of IVC-thrombosis and in differentiating bland

thrombus from tumor invasion [43]. Intravascular ultrasound (IVUS) reliably detects pathologies affecting the IVC wall and the luminal space [44, 45]. Moreover, IVUS is the method of choice for quantifying venous stenoses and guiding venous interventions [44].

Pressure measurements are also helpful to determine the hemodynamic significance of a stenotic IVC-lesion. According to Labropoulos threshold pressure gradient > 3.0 mm Hg indicates a $\geq 50\%$ stenosis [22]. Thus, if there is a pressure gradient clearly above this level a significant stenotic IVC segment is signified.

Appropriately timed CE-CT and CE-MRI (resp. MR-venography) are essential for better overview and the planning of an venous intervention [11, 12, 46–51]. Nevertheless, there are some potential pitfalls, which are critical to avoid misdiagnosis (f.e. flow-related pseudo-filling defects on the level of the renal veins, ferro-magnetic artefacts due to IVC filters and stents) [11, 46–48, 50, 52], while findings such as a flat IVC and early enhancement of the IVC limit differential diagnoses [46]. CE-CT is also recommended for the identification of complications related to IVC filters [52].

Differentiating tumor invasion in the IVC from bland thrombus formation is of special clinical interest. Comparing DUS, MRI, and CT highest sensitivity was found for MRI (77.8%), highest specificity for CE-MRI and CE-CT (97.4%) [53]. Other authors advocate CEUS, IVUS, or FDG PET-CT [43, 53, 54], which is otherwise extremely sensitive for the detection of retroperitoneal and intraabdominal malignancies and metastases.

Sonography is the diagnostic screening method of choice in BCS [14, 16, 55]. Common findings in B-mode sonography include IVC thrombi and webs, IVC stenosis, hepatic venous thrombosis or stenosis, enlarged caudate lobe, intrahepatic lesions, ascites, and intrahepatic or extrahepatic collaterals, while in DUS monophasic to absent flow in the hepatic veins and in the IVC, and/or high flow velocities in stenotic lesions can be detected [14, 16, 55, 56]. The combination of specific signs including thrombosis, stenosis, fibrotic cords, or insufficient recanalization of the hepatic veins or the IVC in combination with an enlarged caudate lobe offered the highest predictive value to identify patients with BCS (specificity 100%) [55]. In a study performed by Faraoun et al. a total of 176 patients with primary BCS were diagnosed by DUS, CE-CT and CE-MRI. Hepatic vein obstruction was a constant finding associated with IVC-abnormalities in 29% patients, obstruction of the three hepatic veins was present in 89.8% of the cases, and longstanding thrombus in the hepatic veins was observed in more than 92% of patients with all three imaging methods. Agreement between DUS, CT, and MRI was perfect in the identification of hepatic vein thrombus ($\kappa = 0.9$), but was only slight to moderate in revealing the type of hepatic vein abnormality [57]. Thus, DUS in combination with MRI or CT seems to represent the best imaging strategy in BCS [14, 16, 55, 57].

Venography is nowadays restricted to patients selected for interventions. If performed, at least two projections

Figure 1. Monophasic constant flow curve in the right common femoral vein due to an extended retroperitoneal mass compressing the right iliac vein and the IVC.

(posterior-anterior and lateral) are necessary. Things might appear normal in one projection but turn out to be pathologic from another perspective [58].

Establishing tumor diagnosis

IVCS is associated with a wide spectrum of malignancies. Imaging data usually enables radiologists and oncologists to narrow the differential diagnoses and to define staging strategies [59–66]. Nevertheless, histology and molecular genetics are important for an exact tumor diagnosis and often determine therapeutic decisions and over-all prognosis. Sonography- or CT-guided biopsy, endoscopy, endosonography, and video-assisted laparoscopy may be indicated depending on the suspected tumor entity, location, and local spread, while additional bone marrow aspiration is indicated in patients with suspected lymphomas.

Therapeutic strategies

Therapeutic decisions in IVCS are determined by the patient's clinical condition, the underlying etiology, the balance between risk and benefit and the availability of therapeutic procedures in the institution.

Patients with IVCS usually present in a stable clinical condition which allows elective diagnostic imaging and interdisciplinary treatment decisions.

In case of VCI-thrombosis anticoagulation should be initiated and an interdisciplinary team of vascular physicians should decide about additional invasive procedures like thrombaspiration and/or thrombolytic therapy [11, 12, 67].

Obtaining a histologic diagnosis is essential for oncologic decision making but implicates some time delay. Stent therapy in tumor patients provides rapid symptomatic relieve within hours to days and does not interfere with biopsy [1]. Thus, in a symptomatic patient with unknown histologic diagnosis stenting is a prior-ranking option, while

patients with known radio- or chemotherapy-sensitive tumors like lymphomas may be primarily treated with these modalities [1].

In BCS therapeutic intervention is based on the acuity and extend of symptoms in an established stepwise approach [14, 15, 21].

Interventional therapy in patients with malignancy

Stenting in malignant IVCS is indicated: (1) in patients with severe symptoms (2) in case of persistency of symptoms despite radio- and/or chemotherapy, and (3) in patients in whom chemotherapy and radiation are contraindicated or of minor effectiveness [modified according to 1].

The literature regarding stent placement in malignant IVCS is limited [1]. Table I summarizes technical and clinical results of interventions in patients with malignancies published between 1998 and 2020 [68–72]. Obviously, the Wallstent and the Cianturco-Z-Stent are most frequently used. Increased post-stent placement pressure gradients indicating residual stenosis correlated with worse outcomes [70]. The Stanford group also correlated technical problems and complications to the use of Wallstents and described decreased stent adherence to the IVC-wall as a result of inadequate mechanical properties indicating that inadequate stent design, insufficient stent diameters, and inadequate radial force of the stents might be critical issues [70]. Indeed, almost all stents used in the published series were not primarily designed and/or approved for venous stenting, which might have negatively influenced the results [73]. On the other hand, in the only series published with a relevant quota of venous dedicated stents (53%) technical success rates and patency rates did not exceed those performed with arterial stents [69].

In the hands of experienced interventionalists working in specialized centers stenting of malignant IVCS is associated with acceptable complication rates (see Table I). Nevertheless, stenting in the IVC implies a high degree of technical

Table 1. Technical and clinical results of stent placement in malignant IVCS

Authors	n	Tumor entity	Stents used	Technical success	Complications	Primary patency	Secondary patency	Clinical success
Epelboym Y et al. 2020 [68]	17	Variable primary and metastatic entities	Gianturco-Rosch-Z stent n = 17	100%		94%		58% edema resolved
Kuetting et al. 2017 [69]	19	Variable primary and metastatic entities	Gianturco-Rosch-Z-stent n = 4; Sinus XL n = 9, Zilver Vena n = 1, Wallstent n = 7	100%	n = 1 stent migration; n = 6 stent compressions during follow-up	68%	100%	79% improved
Devic Z et al. 2016 [70]	57	Hepatic metastases (n = 22; 39%), primary hepatic malignancy (n = 16; 28%), retroperitoneal metastases (n = 16; 28%), other primary malignancy (n = 5; 9%)	Wallstent n = 47; Wallflex stent n = 18; others n = 17; SMART stent n = 15	96%				80–89% ascites or edema improved
Brountzos EN et al. 2004 [71]	50	Variable primary and metastatic malignant entities	Gianturco-Rosch-Z stent n = 45; Wallstent n = 5	100%	4% (n = 2 stent migrations to the right atrium)	92% after 3 mo; 59 % after 18 mo	100% after 18 mo	86.4% improved; 66.7% until last clinical visit during follow-up
Fletcher WS et al. 1998 [72]	28	Variable tumor entities, hepatic metastatic disease (n = 28; 100%)	Gianturco-Rosch Z-stent n = 27; Wallstent n = 1	100%	7.1% (n = 1 thrombus formation requiring reintervention and additional stent placement; n = 1 pulmonary embolism)	96.4%	96.4%	Edema and ascites improved in all patients available for follow-up.

Figure 2. A) Color-coded ultrasound showing compression of the iliaco-caval junction by a retroperitoneal tumor mass. B) (same patient as in figure 2 a) partial tumor compression of the IVC and high-grade tumor compression of the iliac vein. Partial compression of the venous dedicated stent due to the tumor mass and final result.

complexity. IVC tears and rupture, stent thrombosis, stent migration into the heart, pulmonary embolism, retroperitoneal and groin hematomas, and hemopericardium are potential threats [68–72].

Stent placement in malignant IVCS often is a palliative procedure. Median survival rates in the reported series are low. In the series of Fletcher et al. including patients with hepatic metastatic disease the median survival was only 27.5 days, while in the series of Brountzos et al. and Devic a mean survival of 75 resp. 104 days was reported [70–72].

There are no randomized trials addressing antiplatelet or antithrombotic therapy after successful interventional therapy in malignant IVCS. Nevertheless, most centers prefer at least a short-term antiplatelet and/or anticoagulation treatment regime [68–72]. Figures 2A–2B show an extended retroperitoneal mass (retroperitoneal sarcoma) compressing the IVC and the right iliac vein treated by venous stenting and PTA.

Interventional therapy in IVCS of benign origin

Benign IVCS most often results from chronic obstructions or stenoses of the IVC due to congenital IVC abnormalities and/or previous IVC thrombosis [4, 8, 9, 11, 12, 67, 74].

In contrast to chronic iliac vein obstruction only smaller series or individual cases have been published. Table II summarizes technical and clinical results published more recently in collectives with benign IVCS unrelated to IVC-filters [75–80].

As was reported by Neglén et al. and Delis et al. successful IVC stenting not only translated into clinical improvement, but also into improvement of venous hemodynamic parameters like venous outflow fraction, calf muscle pump function, venous pressure, and venous reflux [81, 82].

In the hands of experienced interventionalists overall complication rates are low (see Table II). Nevertheless, life threatening complications like bleeding associated with IVC tears, stent migration into the heart, pulmonary embolism, and hemopericardium may occur [44, 75–80].

Patients with thrombotic IVCS successfully treated with stents are at increased risk of subsequent stent thrombosis. Stent thrombosis correlated with younger age (< 40 y), extensive stenting especially below the common femoral vein, and structurally altered inflow veins in the Swiss registry [83, 84].

Filter-related IVC-occlusions pose special technical problems for the interventionalist as the obstructed filter must either be removed, balloon-dilated, or stented [11, 13, 85–90]. Filter removal bears significant risks, especially in case of wall perforation, stent extravasation, or stent fracture requiring advanced removal techniques with increased

Table II. Technical and clinical results of endovascular therapy in benign IVCS

Authors	n	Type of lesion	Stents used	Additional interventional techniques	Technical success	Complications	Primary patency	Assisted primary or Secondary patency	Clinical success
Barrette LX et al. 2020 [80]	37	IVC occlusion	n = 37 Palmaz stent		100%		66 after 24 mo and 48 mo	84 (ass. primary p.) after 24 mo and 48 mo	
McDervitt JL et al. 2019 [79]	69	39 IVC post-thrombotic occlusion and 30 IVC atresia	Wallstent; Gianturco-Rosch Z-stent; Palmaz stent; S.M.A. R.T stent; AneuRX stentgraft; Viabahn stentgraft;	29% (n = 20) catheter-directed thrombolysis; 14% (n = 10) sharp recanalization	100%	1.4% severe (stent migration into right heart with surgical removal); 5.8% moderate (n = 2 IVC pseudoaneurysms requiring covered-stent placement in both cases and blood transfusion in one case; n = 1 stent migration; n = 1 iliac vein stenosis requiring additional stenting); 5.8% minor adverse events	91.88, and 62% at 6.12, and 24 mo	98%, 95%, and 81% at 6.12, and 24 mo (ass. primary p.)	76%, 85%, 87%, and 100% from 14d up to 24 mo
Erben Y et al. 2018 [78]	67 (1 asymptomatic; 66 symptomatic; 89% history of VTE; 38% IVC filter; 30% thrombophilia)	IVC occlusion +/- iliac occlusion	Wallstent; Protegé; Gianturco-Rosch Z stent; Luminex		90%		78% after 36 mo	87 (ass. primary p.); 91% (secondary p) after 36 mo	83%
Sebastian T et al. 2018 [77]	62 (53% post-thrombotic syndrome, 27% acute thrombosis, and 19% for non-thrombotic IVC	IVC occlusion	Nitinol self-expanding stents		98%		57% at 24 mo	76% (ass. primary p.); 87% (secondary p.) after 24 mo	No new ulcers; ulcer healing in all 8 pts. with ulcers; 48% showed significant improvement, and another 26 (43%) were completely free of symptoms

(Continued on next page)

Table II. (Continued)

Authors	n	Type of lesion	Stents used	Additional interventional techniques	Technical success	Complications	Primary patency	Assisted primary or Secondary patency	Clinical success
Grøtta O et al. 2017 [76]	20	IVC occlusion n = 12; IVC stenosis n = 8	Wallstent	n = 1 catheter-directed thrombolysis	84%	No major complications, 10% transient back pain without morphological correlate in CT	67% after 24 mo	83% (secondary p.) after 24 mo	68% sustained and significant clinical improvement
Raju S et al. 2006 [75]	111 (10 asymptomatic or oligo-symptomatic; 4 acute symptomatic with leg thrombosis; 97 chronic symptomatic)	IVC occlusion n = 21; IVC stenosis n = 90	Wallstent	n = 4 catheter directed thrombolysis; n = 12 endovascular saphenous ablation	83% overall; 100% in IVC stenosis, 66% in IVC occlusion	n = 1 contrast-induced renal failure; 20% transient back pain	58% after a median of 11 mo	82% (ass. primary p.) after a median of 11 mo	51% free of swelling; 61% free of pain; 63% free of ulcers

major complication rates in up to 5% of the cases even in the hands of experienced venous interventionalists [86, 87]. Risk might be minimized by a second access with a stiff guidewire placed in the superior vena cava. If extravasation occurs a 18 or 20 mm balloon can then be placed quickly over the Amplatzer wire and inflated for sealing. As an alternative to filter removal recanalization and stenting of a chronically obstructed filter has been advocated [88–90]. In the largest series published by Chick et al 2017 comprising data from 120 patients with 123 IVC filters as well as in smaller series technical success was high and primary and secondary patency did not significantly differ from those reported in non-filter-related IVC lesions [88–90].

For the prevention of filter related complications IVC filters should be retrieved once the patient can be adequately anticoagulated. Figures 3A–3C illustrate a rare case of a restenosis after implantation of a IVC-prosthesis treated by venous stenting and PTA.

Unfortunately, evidence-based recommendations addressing the kind and the duration of antiplatelet and/or anticoagulation therapy after interventional procedures in benign IVCS do not exist. In IVCS related to a previous thrombosis long-term anticoagulation may be preferred.

Interventional therapy in acute IVC thrombosis

Although anticoagulation therapy remains fundamental in the treatment of acute IVC thrombosis additional endovascular options including transcatheter thrombolysis, vacuum assisted or mechanical thrombectomy, or a combination of these techniques are increasingly used as acute IVC thrombosis is associated with an excess mortality and morbidity under conservative treatment [11, 12, 67].

Catheter directed thrombolysis and interventional thrombectomy techniques have widely replaced open surgical thrombectomy [11, 67]. In the U.S.A. catheter-directed thrombolysis (CDT) steadily increased according to the Nationwide Inpatient Sample database [91]. However, based on this registry and a propensity-matched control group, no differences in the hospital stay and over-all mortality were found between the CDT and the anticoagulation groups, but higher rates of pulmonary embolism (12.1% versus 7.8%), intracranial hemorrhage (1.6% versus 0.2%), and acute renal failure (13.9% versus 9.4%) were reported in the CDT group [91]. Unfortunately, randomized head-to-head comparisons are not available.

Only anecdotal evidence exists for beneficial effects of ultrasound-enhanced thrombolysis in acute IVC-thrombosis or iliaco-caval thrombosis [92, 93]. Given the large diameter of a thrombosed IVC it is questionable whether the technical principle of ultrasound-enhanced thrombolysis provides an advantage over conventional CDT. Moreover, a recently published Dutch trial failed to demonstrate significantly lower rates of post-thrombotic syndrome in patients with iliaco-femoral thrombosis occasionally ascending into the

Figure 3. A) Medium grade stenosis at the distal end of the IVC-prosthesis treated by balloon dilation. B) Same patient as figure 3a. High-Grade Stenosis at the proximal end of the IVC-prosthesis with marked post-stenotic dilation. Predilation and final result after implantation of a dedicated venous stent. C) CT-A one year after IVC-Stenting.

IVC treated with ultrasound-enhanced thrombolysis [94]. A lack of evidence also exists for percutaneous mechanical, rheolytic, and vacuum thrombectomy [95–98]. Compared to CDT these techniques are timesaving and may help to avoid bleeding complications. They proved useful and safe in acute iliaco-femoral thromboses occasionally ascending into the IVC [95–101].

Although anticoagulation is the mainstay for patients with acute IVC thrombosis [11, 12, 67], there is no evidence-based recommendation how long anticoagulation therapy should be maintained after a successful interventional therapy [11]. Again, in IVCS related to thrombosis long-term anticoagulation may be preferred at least in cases without identifiable trigger-mechanisms.

Table III. Interventional therapy in Budd Chiari Syndrome with IVC involvement

First author	n	Kind of lesion	Intervention	Stents used	Technical success	Complications	Primary patency	Secondary patency	Clinical success/survival rate
Ding PX et al. 2019 [103]	456	Obstruction of IVC with different degrees of HV involvement	Balloon angioplasty n = ; stenting n =	Cianturco Z stent	99.8%	Major complications 2.8%, minor complications 2.2%	93.6%, 99.8%, 98.2%, 97.2% after 1, 2, 5, and 10 y	Not reported	98.4%, 95.8%, 91.2%, and 76.5% after 1, 2, 5, and 10 y
Cheng DL et al. 2019 [105]	162	Obstruction of IVC and HV in all patients, n = 25, 61, and 76 had obstructions of 1, 2, and 3 hepatic veins overall 69.8% membranous lesions	n = 109 (67.3%) IVC recanalization; IVC and HV intervention n = 53 (32.7%) balloon angioplasty n = 32 HV and n = 113 IVC; balloon angioplasty and thrombolysis n = 14 HV and n = 19 IVC; balloon angioplasty, thrombolysis, and stent n = 3 HV and n = 14 IVC; balloon angioplasty and stent n = 3 HV and n = 11 IVC	Cianturco-Z-stent; bare metal stent if venous drainage of HV was blocked	96% in HV and IVC interventions; 97% in IVC interventions	Not reported	Not reported	Not reported	Among 157 patients successfully treated, 146 were cured and 11 showed clinical improvement. Clinical symptoms were relieved in 82.4% after the initial intervention, and 94.2% after the second intervention (at 2 y)
Meng X et al. 2016 [106]	55	Membranous complicated by thrombosis (n = 55)	TIPS n = 5 (if recanalization had failed) Balloon angioplasty n = 55; stent placement n = 47; thrombus aspiration n = 23; catheter-directed thrombolysis n = 13; TIPS for clinical non-responders n = 5	Cianturco-Z-stent; bare-metal stents or fluency stentgraft for TIPS creation	96.4%	No major complications; n = 1 heparin-induced hematuria; n = 2 femoroinguinal hematoma	94%; 89%; 66% after 1, 5, and 10 y	Not reported	90%; 86%; 86% survival after 1, 5, and 10 y
Huang Q et al. 2016 [107]	265	Membranous (n = 136) (MO) and segmental obstruction (SO) (n = 129) of IVC	Balloon angioplasty n = 265; stent placement n = 56	Cianturco-Z-stent	99.2%	Major complications n = 3 (1.1%) (n = 2 pericardial tamponades; n = 1 IVC rupture); no reporting of minor complications	93.5%, 85.2%, and 76.1% at 1, 3, and 5 y (98.3%, 90.7%, and 83.8% in the MO group; 88.3%, 79.1%, and 67.9% in the SO group)	Not reported	clinical improvement depending on symptom 87.3–100% in MO-group and 75.9–100% in SO-group

(Continued on next page)

Table III. (Continued)

First author	n	Kind of lesion	Intervention	Stents used	Technical success	Complications	Primary patency	Secondary patency	Clinical success/survival rate
Ding PX et al. 2016 [108]	93	65 isolated hepatic vein obstruction; 28 combined hepatic vein and IVC obstruction; membranous n = 91, segmental n = 2	HV balloon angioplasty in all patients; stent placement n = 2	Not reported	100%	Major complications n = 6 (6.45%); intraperitoneal bleeding n = 3; intrahepatic hematoma n = 1; intrathoracic bleeding n = 1; aV fistula n = 1; 1 bleeding complication was fatal	Cumulative 1, 2, 2–5, 5–8 y primary patency 97%, 92.9%, 90%, and 86.5%	Cumulative 1, 2, 2–5, 5–8 y secondary patency 100%, 98.6%, and 97.3%	Cumulative 1, 2, 2–5, 5–8 y survival rates 98.75%, 98.6%, 100%, and 100%
Zhang CQ et al. 2003 [109]	115	IVC stenosis n = 54; IVC occlusion n = 48; HV stenosis n = 11; HV occlusion n = 19	Angioplasty and stent placement in all patients	Cianturco Z stents	94%		96.6% after a mean follow-up of 58 mo (12–84 mo)		

Interventional therapy in Budd-Chiari syndrome

Recanalization procedures in BCS are restricted to cases with obstruction and/or thrombosis of the IVC and the larger hepatic veins, while in diffuse HV-obstruction and in veno-occlusive type interventional recanalization is impossible [14, 15]. These cases are either treated conservatively, with TIPS/DIPS creation, or with liver transplantation in case of progressive liver failure [14, 15].

While BCS in western countries is almost exclusively caused by thrombosis BCS in Asia is usually a consequence of membranous or segmental lesions in the IVC and/or the HV [21, 102–104]. Thus, interventional approaches differ between the orient and occident with much more recanalization procedures being performed in Asia [14, 21, 102–104].

Table III summarizes technical and clinical results of balloon dilation and stenting (often in combination with sharp recanalization) in larger series from Asia [103, 104]. As can be seen, technical and clinical success rates of balloon angioplasty and/or stenting are high and have considerably contributed to an improved prognosis of the patients [21, 103–109].

Minor and major complication rates of the highly experienced centers are low (see Table III) [21, 102–110]. The rate of HV or IVC-rupture was reported to be < 1% in a mono-center report focusing on bleeding complications associated with balloon angioplasty in BCS patients [111]. Higher complication rates were reported in transhepatic angioplasty of the HV and in sharp (needle) recanalization procedures [112–114].

Comparing recanalization techniques in of HV and IVC with direct intrahepatic portosystemic shunt creation, Mukund et al. found improved liver synthesis functions in recanalization compared to patients treated with DIPS. Nevertheless, mortality did not differ between the groups [115].

BCS-Patients with thrombotic lesions are either managed conservatively with long-term anticoagulation or receive pre-interventional treatment with anticoagulant drugs to reduce clot burden [116, 117]. Alternatively, primary interventional strategies with thrombus aspiration, thrombus retrieval, and/or catheter-directed thrombolysis followed by stent placement have been advocated [118–121]. Technical success, mid-term, and long-term result of these intervention do not seem to differ substantially from recanalization procedures in non-thrombotic lesions [118–121].

Long-term anticoagulation after interventional therapy of BCS is recommended to avoid re-thrombosis and stent occlusion [21].

Conclusions

IVCS is a complex condition best managed by an interdisciplinary team approach. In the last decades percutaneous

interventional techniques have fundamentally changed therapeutic strategies in symptomatic patients in benign and malignant IVCS as well as in IVCS associated with BCS. Interventions in IVCS and BCS are complex procedures, which should best be concentrated in specialized centers. Despite increasing knowledge many clinically important questions in the management of IVCS patients remain unsolved including optimized patients' selection strategies for interventions, differential procedural considerations, optimized devices, and antithrombotic and antiplatelet treatment regimens to avoid restenosis and -thrombosis.

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