Review Article

Tumor thrombus: incidence, imaging, prognosis and treatment

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Abstract: Intravascular tumor extension, also known as tumor thrombus, can occur in many different types of cancer. Those with the highest proclivity include Wilm's tumor, renal cell carcinoma (RCC), adrenal cortical carcinoma (ACC) and hepatocellular carcinoma (HCC). The presence of tumor thrombus markedly worsens prognosis and impacts treatment approach. Imaging plays a key role in its diagnosis. Endovascular methods also play a large role in treatment.

Keywords: Tumor thrombus; renal cell carcinoma (RCC); hepatocellular carcinoma (HCC)

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Introduction

Intravascular tumor thrombus is defined as tumor extension into a vessel. Its presence changes stage, prognosis, and treatment. It occurs in a wide variety of malignancies, most frequently in renal cell carcinoma (RCC), Wilms tumor, adrenal cortical carcinoma (ACC), and hepatocellular carcinoma (HCC). Imaging plays a crucial role both in the detection of tumor thrombus and is essential in differentiating it from bland thrombus. The incidence, diagnosis and approach to treatment of tumor thrombus are discussed in this article.

RCC

RCC is the 9th most common type of cancer diagnosed in the US and is projected to result in 14,400 deaths in 2017. RCC has a known proclivity for vascular invasion, which occurs in approximately 10% of cases (1-3) (*Figure 1*). The presence of tumor thrombus changes staging, prognosis and surgical approach (4-8).

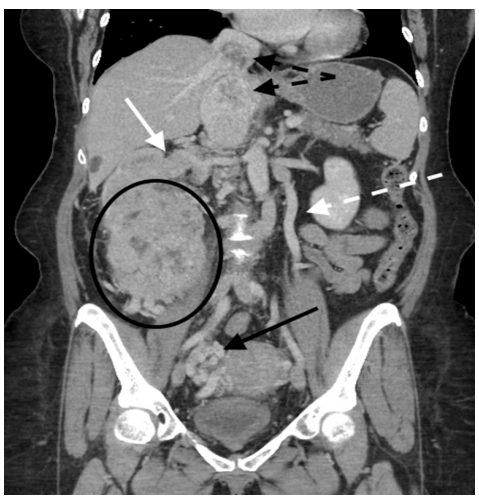

Figure 1 A 54-year-old woman with hematuria and a right renal mass (black circle). Tumor thrombus extends into the right renal vein (solid white arrow) and the IVC (dotted black arrow). Note the pelvic varicosities (solid black arrow) and enlarged left gonadal vein (dotted white arrow), which are secondary to IVC obstruction.

Table 1 T staging of RCC according to 2010 AJCC clinical staging

T stage	Description
T1	
T1a	<4, confined to kidney
T1b	>4 but <7 cm, confined to kidney
T2	
T2a	>7 but <10 cm, confined to kidney
T2b	>10 cm, confined to kidney
T3	
T3a	Tumor thrombus in segmental renal vein branches or main renal vein or tumor invades perineal fat but does not extend beyond Gerota's fascia
T3b	Tumor thrombus extends into IVC but remains below diaphragm
T3c	Tumor thrombus extends into IVC and above the diaphragm or tumor invades the wall of the IVC
T4	Tumor invades beyond Gerota's fascia; tumor invades ipsilateral adrenal gland

RCC, renal cell carcinoma.

Table 2 Mayo Clinic RCC tumor thrombus level classification system and potential surgical approach

Level	Description	Surgical approach to gain control central to tumor thrombus
0	Limited to renal vein or its tributaries	Renal vein ligation
I	Extends into IVC but <2 cm above renal vein orifice	Milking of IVC tumor thrombus into renal vein followed by renal vein ligation
II	Extends into IVC, >2 cm above renal vein orifice but below hepatic veins	Some mobilization of liver (ligation of accessory hepatic veins draining caudate lobe), clamping of intrahepatic IVC, clamping of infrarenal IVC and contralateral renal vein
III	Extends above hepatic veins but below diaphragm	Extensive mobilization of liver including ligation of diaphragmatic attachments, clamping of suprahepatic IVC with adjunctive venovenous or cardiopulmonary bypass
IV	Extends above diaphragm	Involvement of cardiothoracic surgery, potential thoracotomy and open-heart surgery

RCC, renal cell carcinoma.

Clinical symptoms, staging and prognosis

Tumor thrombus may be asymptomatic or may cause a variety of symptoms including varicocele, lower extremity swelling, cardiac dysfunction, pulmonary embolism, or Budd-Chiari syndrome (9-12). In the absence concurrent invasion of Gerota's fascia or the ipsilateral adrenal gland, RCC with tumor thrombus is staged as T3 (2) (*Table 1*). When the tumor thrombus is present only in a segmental renal vein branch or the main renal vein, it is staged as T3a. When the tumor thrombus extends into the IVC but below the level of the diaphragm, it is staged as T3b. When the tumor thrombus extends above the diaphragm or invades the wall of the IVC, it is staged as T3c. An alternative classification system of tumor thrombus level is the Mayo Clinic Classification, which is useful for surgical planning (*Table 2*) (13-16). While it is controversial whether higher-level thrombus is associated with increased cancer related mortality, surgical morbidity and mortality does increase. Perioperative complications of 78% and mortality of 13% have been reported for T3c tumors (16-20).

Table 3 Bland versus tumor thrombus

Feature	Bland thrombus	Tumor thrombus
Enhancement	–	+
Contiguity with mass	–	+
^{18}F-FDG avidity	–	+
Vessel size	Expanded in acute phase	Expanded by tumor
	Small/fibrotic in chronic phase	

Imaging

Both MRI and CT have high accuracy in detecting tumor thrombus, assessing its extension and distinguishing it from bland thrombus (21-24). Tumor thrombus is differentiated from bland thrombus by the presence of enhancement, vessel expansion and uptake of fluorodeoxyglucose on positron emission tomography (^{18}F-FDG-PET) (*Table 3*) (25). On angiography, the "streak and thread" sign may be seen (26). Venography will show filling defects within the affected vessel.

Invasion into and through the wall of the IVC occurs infrequently (27,28). A definitive sign of invasion of the IVC wall is presence of tumor on both sides of the caval wall. Vessel wall invasion is suggested when tumor thrombus causes complete occlusion and expansion of the IVC.

Treatment/surgical approach

Complete surgical resection of the parenchymal tumor and tumor thrombus results in a 5-year survival of greater than 50% (29-32). With incomplete tumor resection, 5-year survival drops to about 10% (10,31). In the presence of known metastatic disease, surgical tumor debulking not only improves symptoms but can also prolong survival, especially when targeted chemotherapy is subsequently employed (33-35). Surgery becomes more complex and has a higher morbidity and mortality rate the higher the tumor thrombus extends.

Prior to surgery, the patient should undergo anesthesia and/or cardiology assessment to evaluate if the patient is an appropriate surgical candidate given the relatively high rates of perioperative morbidity and mortality. Depending on the extension of tumor and/or need for vascular reconstruction, multidisciplinary discussion between urology, vascular surgery and cardiothoracic surgery may be necessary to determine optimal surgical approach.

If bland thrombus is present in addition to tumor thrombus, anticoagulation should be considered (36). IVC filter placement, however, should be avoided. The presence of an IVC filter can convert a challenging surgery to something nearly impossible. The presence of a supra-renal filter makes it difficult to obtain control cranial to the tumor thrombus at the time of surgery. Tumor thrombus may also become incorporated into the filter making removal of both the filter and the tumor thrombus very difficult (36).

Traditional neo-adjuvant chemotherapy and radiation are ineffective against RCC. Small studies of targeted molecular therapies (e.g., sorafenib, erlotinib and temsirolimus) have shown their ability to down stage tumor thrombus level to make subsequent surgery safer and easier (37-39). More investigation is needed to see if these targeted neo-adjuvant chemotherapy agents should be recommended in cases of tumor thrombus (20).

Pre-surgical trans-arterial embolization is sometimes performed. While it does decrease operative blood loss and shorten operative time, it has not been shown to improve overall survival (40-42). Therefore, its role and use is debated.

To prevent tumor thrombus from embolizing to the lungs during surgery, control central to the tumor thrombus must be gained (10,43). The approach to gain central control varies according to the level of intravascular tumor extension (*Table 2*). For level 0 tumor thrombus, the renal vein is ligated central to the tumor thrombus and *en bloc* nephrectomy is performed. For level I tumor thrombus, no clamping or control of the IVC is necessary; tumor thrombus is milked back into the renal vein while removing the kidney. The renal vein is then ligated and nephrectomy completed *en bloc* (20).

Surgery for level II tumor thrombus is more involved. Tumor thrombus extending >2 cm into IVC cannot be milked back into the renal vein. To gain control of the IVC cephalad to the tumor thrombus, the liver is mobilized by

Table 4 Classification for hepatocellular carcinoma with portal vein tumor thrombus proposed by Shi *et al*.

Type	Location of tumor thrombus
0	Found at microscopy
I	Extends up to segmental portal vein branches
II	Involves the right or left portal veins
III	Involves main portal vein
IV	Extends to involve superior mesenteric veins

ligating the accessory hepatic veins draining the caudate lobe. The intrahepatic IVC, below the level of the hepatic veins, is then clamped. Additionally, inflow control is necessary. Both the infrarenal IVC and contralateral renal vein need to be clamped. Fortunately, cardiopulmonary or venovenous bypass is not necessary for level II thrombus as clamping as the hepatic veins provide approximately 1/3rd of IVC flow. Therefore, adequate venous return can be maintained. After adequate proximal and distal control is obtained, an L-shaped cavotomy is made extending into the affected renal vein (16). Through this incision the tumor thrombus is removed from the IVC. The kidney and remaining tumor thrombus are removed *en bloc*.

Surgical resection for level III tumor thrombus, which extends above the level of the hepatic vein-IVC confluence but stays below the diaphragm, is complex. First, intraoperative transesophageal echocardiogram may be necessary to evaluate the precise cranial extent of the tumor. Once it is shown to be subdiaphragmatic, the liver needs to be extensively mobilized in order to gain control of the IVC above the hepatic veins. This is done by dividing the liver from its diaphragmatic attachments and ligating the accessory hepatic veins. This mobilization is often done in conjunction with a hepatobiliary or transplant surgeon (44). Cardiopulmonary bypass or venovenous bypass are necessary given that clamping the suprahepatic IVC obstructs all venous return from the IVC, which accounts for 2/3rds of all venous return.

Level IV tumor thrombus extends above the diaphragm into the right atrium (10). Involvement of cardiothoracic surgery and cardiopulmonary bypass are necessary. TEE helps predict adherence of the tumor to the myocardium and tricuspid valve, which helps one decide whether or not incision into the heart is necessary (45).

When tumor has invaded into the wall of the IVC, resection of the IVC is necessary to completely remove the tumor. When tumor has invaded the wall of the IVC, there is a high risk of local tumor recurrence. Bovine pericardium, autologous pericardium and expanded polytetrafluoroethylene (ePTFE) have all been used for IVC reconstruction.

HCC

HCC is the 6th most common cancer and is the 3rd leading cause of cancer related deaths (46,47). Macrovascular invasion is a well-recognized characteristic of HCC; portal vein tumor thrombus (PVVT) occurs in 35% of cases and hepatic vein tumor thrombus occurs in 2% of cases (48,49). Macrovascular invasion tends to affect younger patients, those with aggressive tumor biology and patients with poor underlying liver function (49). The prognosis of HCC with tumor thrombus is a poor and depends on the extent of PVVT (48,49). Of note, upwards of 25% of all patients with cirrhosis have bland portal vein thrombus (50,51). Bland and tumor thrombus can be distinguished by imaging characteristics (*Table 3*).

Staging, clinical symptoms and prognosis

The overall prognosis in HCC tumor is poor. Without PVTT, median survival is approximately 16 months. This prognosis drops to 6 months if PVTT is present (52). The presence of PVTT has been shown to have a more significant effect on prognosis than tumor size (53). The extent of PVTT is also very important in predicting survival (54-56). Patients with segmental PVTT are found to have twice the survival times as patients with main portal vein invasion (9 *vs.* 4.6 months) (54). Shi *et al.* proposed a PVTT classification system based on the extent of tumor involvement (*Table 4*) (56). Tumor thrombus may cause specific symptoms. PVTT may cause portal hypertension through obstruction of flow and/or due to arterioportal shunting. Hepatic vein tumor thrombus may lead to Budd-Chiari syndrome.

HCC patients with any macroscopic vascular invasion are classified as Barcelona Clinic Liver Cancer (BCLC) stage C (advanced stage) (*Table 5*) (57-59). Patients with extrahepatic spread and patients with reduced performance status (Eastern Cooperative Oncology Group 1 and 2) are also classified as BCLC stage C. BCLC stage C therefore encompasses a heterogeneous group of patients with wide ranging survival (54). Therefore, some suggest sub-classifying stage C (54,60).

Table 5 BCLC classification system

Stage	Tumor size	Liver function (Child-Pugh class)	ECOG performance status
Very early (0)	Single, <2 cm	A or B	0
Early (A)	≤3 nodules all <3 cm or single nodule <5 cm	A or B	0
Intermediate (B)	Single tumor >5 cm or more than 3 tumors or ≤3 tumors, one of which is >3 cm	A or B	0
Advanced (C)	Macrovascular invasion or extrahepatic metastases or size criteria greater than stage B	A or B	1 or 2
Terminal (D)	Severe HCC related tumor disability/symptoms	C	3 or 5

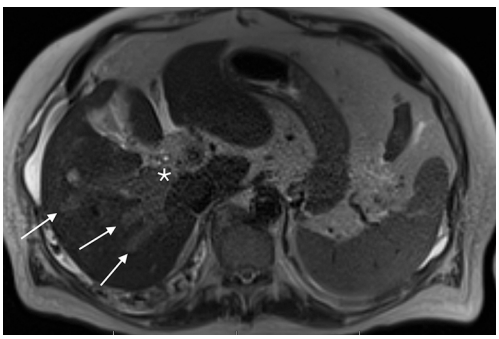

Figure 2 A 72-year-old man with cryptogenic cirrhosis found to have large right lobe of liver HCC with extensive portal vein invasion. Tumor thrombus extends into branches of the right portal vein (white arrows) as well as the main portal vein (white asterisk). This T2 weighted MRI shows relative hyperintensity of the portal veins. Normal patent portal vein would show hypointensity secondary to flow void. He underwent treatment with Y-90 embolization.

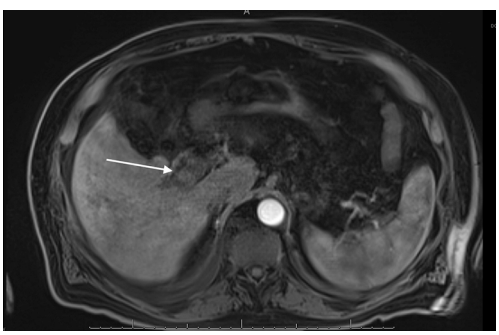

Figure 3 Different sequence from the same patient as *Figure 2*. Contrast MRI in the arterial phase shows early arterial enhancement in the thrombosed main portal vein differentiating it from bland thrombus. Main portal vein tumor thrombus is classified as Shi type III.

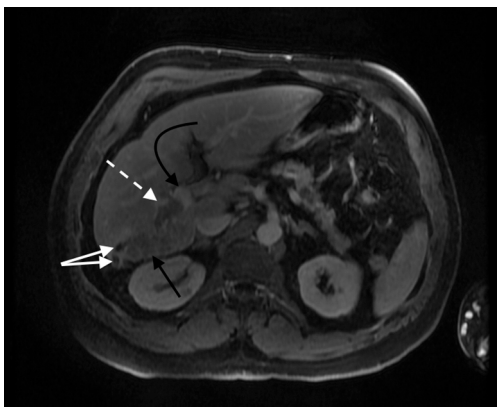

Figure 4 A 70-year-old man with alcoholic cirrhosis and previously ablated segment V lesion who on follow-up imaging was found to have an infiltrative lesion in segments VI/VII (solid black arrow) with portal vein tumor thrombus (dotted white arrow). Note the patent portal vein (curved black arrow). Bland tumor thrombus is present in the periphery of the right lobe (solid white arrows).

Imaging

PVVT can be diagnosed and differentiated from bland thrombus by many different imaging modalities, including conventional angiography, color Doppler ultrasound, contrast enhanced ultrasound, contrast enhanced CT scan, MRI, and FDG PET imaging (61-64) (*Table 3*, *Figures 2-4*). Contrast enhanced ultrasound is reported to be 100% sensitive and specific (65). Differentiating bland from tumor thrombus is necessary as the former occurs in up to 25% of patients with chronic liver disease (51). The key feature for diagnosing portal vein tumor thrombus is the presence of enhancement or color Doppler flow within the thrombus (63,64). Subtraction imaging may be helpful to detect subtle enhancement within the thrombus.

Diffusion imaging on MRI can be a useful adjunctive imaging finding (62). Tumor thrombus has an increased cellular density and nuclear to cytoplasm ratios and will show restricted diffusion, appearing bright on diffusion weighted imaging (DWI) and dark on apparent diffusion coefficient (ADC) mapping (66). Nonetheless, care must be taken not to evaluate diffusion sequences in isolation as bland thrombus may also demonstrate low ADC values. This is due to the high viscosity of thrombus as well as paramagnetic effects of intracellular deoxyhemoglobin and methemoglobin present in bland thrombus (67). Other imaging characteristics that favor tumor thrombus include expansion of the involved vessel and increased uptake on FDG-PET (63,64,68-70). On angiography, portal vein tumor thrombosis may be recognized by the characteristic streak and thread appearance due to parallel opacification of small vessels within the tumor and arterial venous shunting (26).

Treatment

Sorafenib is a multi-kinase inhibitor, targeting VEGFR, PDGFR and Raf family kinases (71). According to the American Association for the Study of Liver disease (AASLD), the European Association for the Study of the Liver (EASL), and the BCLC treatment guidelines, it is the recommended treatment for BCLC stage C patients (72,73). There are many potential side effects of this drug; one characteristic side effect is hand-foot syndrome, which consists of redness, swelling, numbness and peeling of the palms of the hands and soles of the feet (74). Survival of those treated with Sorafenib is improved by only a modest 2–3 months (75,76). Limiting patients with tumor thrombus to treatment with Sorafenib may be too conservative. In clinical practice, other more aggressive treatments are employed (54).

In western countries, surgical resection is rarely performed in the setting of HCC tumor thrombus given poor underlying liver function. In the east, cirrhosis is by and large due to Hepatitis B. These patients have better underlying hepatic function compared to alcohol or hepatitis C cirrhosis seen in the west. Therefore, surgical resection is performed for patients with branch of portal vein tumor thrombus (77). Reflecting this practice, the Hong Kong Liver Staging does not consider hepatic vascular invasion to be a contraindication for surgical resection (78).

Due to the high rate of recurrence after transplant, the presence of tumor thrombus is considered an absolute contraindication to liver transplantation (79,80).

Thermal ablation of portal vein tumor thrombus is rarely considered given the portal vein's the tumor's oblong shape as well as its proximity to critical structures, like the hepatic artery and bile ducts. However, one recent report has shown good results after endoluminal ablation followed by stenting (81).

Given the risk of hepatic necrosis and worsening of liver function, the presence portal vein tumor thrombus is considered a relative contraindication to transarterial chemoembolization (TACE) (82,83). EASL, AASLD and BCLC guidelines all discourage TACE in patients with macroscopic vascular invasion (72,73). However, in patients with non-occlusive thrombus, preserved liver function and small tumor burden, super-selective TACE can be safely and effectively performed with improvement in patient survival (83-85). The Asian Pacific Association for the Study of the Liver (APASL) guidelines recommend TACE in the setting of branch vessel thrombus in Child-Pugh A and B patients (86). While hepatic necrosis is not a worry in those with hepatic vein tumor thrombus, particulate embolization to the pulmonary vasculature is a potential sequela given hepatic arteriovenous shunting within the hepatic vein tumor thrombus (87).

Selective internal radio-embolization (SIRT) using Yittrium-90 (Y-90) is considered by many to be the preferred treatment of patients with HCC and portal vein tumor invasion (88,89). EASL guidelines state that that although SIRT appears to be safe and demonstrate promising results, more studies are needed before it can be recommended as standard of care (73). When treating patients with hepatic vein tumor thrombus, one should be cognizant of the propensity for hepatopulmonary shunting (90). The presence of hepatic vein tumor thrombus is associated with a 3–4× elevated lung shunt fraction, as detected by Technetium-99m microaggregated albumin (Tc-99m MAA) (91). As the limit for radiation exposure to the lungs is set at 30 Gy in a single session and 50 Gy in a cumulative dose reduction is often necessary (92).

There are emerging therapies for portal vein thrombus. External beam radiation therapy in combination with intra-arterial 5-FU and interferon-alpha infusion has shown positive results (93,94). Percutaneous portal vein stenting followed by TACE and external beam radiation has also shown promising results (95). Irradiation stents (self-expandable stent loaded with iodine-125 seeds) in combination with TACE has shown benefit in a small cohort of patients with partially obstructing portal vein

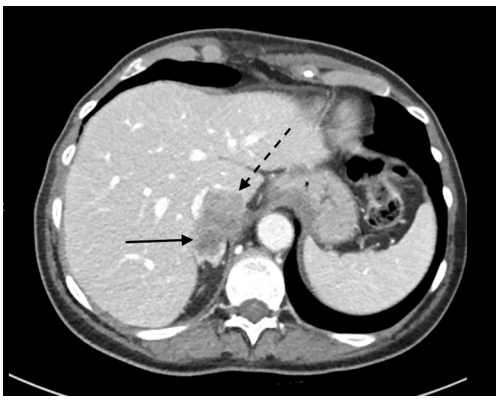

Figure 5 A 48-year-old female with new onset hypertension and palpitations who underwent CT abdomen was done which showed a right adrenal mass (solid black arrow) invading the IVC (dotted black arrow). She subsequently underwent an endocrinology work-up and was found to have elevated catecholamine's consistent with a pheochromocytoma.

tumor thrombus (96).

ACC

ACC is a rare and aggressive malignancy. ACC also has a propensity to lead to tumor thrombus which effects about 1/4th of patients (*Figure 5*). Similar to RCC with tumor thrombus, patients can present with a varicocele or lower extremity edema (97-99). Invasion of the wall of the IVC has been reported with ACC (100). Although there are some cases of long-term survival in cases of ACC with vascular invasion after complete resection, this is the exception rather than the rule (101-103). Systemic chemotherapy is the preferred treatment modality.

Wilm's tumor

Wilm's tumor is the most common kidney mass in children and accounts for 6% of all childhood cancers. Its peak incidence is 3–4 years old. It has a marked propensity for macrovascular invasion which occurs in up to 35% of cases. Extension into the IVC occurs in up to 10% of cases (104). Intracardiac extension is rare but does occur (105). While pre-operative chemotherapy is generally recommended, this is a decision that should be made on a case-by-case basis. If the tumor thrombus appears to be at high risk of embolizing, surgery prior to chemotherapy is preferred (106,107).

Other tumors

Many other tumors can develop tumor thrombus, but do so less frequently. These include intravenous leiomyomatosis, islet cell tumors of the pancreas, thyroid cancer.

Intravenous leiomyomatosis is a rare benign smooth muscle tumor of uterine origin that grows into veins pelvic veins. It often can cause lower extremity swelling but has been reported to cause death if they extend into the heart (108-110).

Islet cell tumors of the pancreas can develop tumor thrombus. This tumor thrombus can involve the splenic, the portal and/or the superior mesenteric veins (111-115). Splenic vein tumor thrombus can cause sinistral portal hypertension, with development of isolated gastric varices (116). As with other tumor thrombi, one will see heterogeneous arterial enhancement and expansion of the involved veins in contiguity with the primary mass. FDG avidity of the tumor thrombus may also be seen on PET scan (117).

While the incidence is unknown, there are reports of macrovascular invasion of thyroid cancer with extension into the jugular vein, SVC and right atrium (118-120). As with other tumor thrombi, proximal and distal control of the thrombus is necessary during surgical excision. This is illustrated in one case report which describes tumor thrombus embolizing to the pulmonary artery during surgery in the absence of adequate central control (121). Thyroid tumor thrombus can first be detected by ultrasound and may be further imaged with by contrast enhanced CT or MRI (122). Good long-term outcome can be achieved with complete excision.

Testicular cancer may rarely cause tumor thrombus. While autopsy reports have suggested the incidence of tumor thrombus to be as high as 11%, it is only seen in 1% of cases by imaging (123,124). In addition to tumor thrombus originating from the primary site and extending into the gonadal veins, retroperitoneal metastatic lymphadenopathy may grow directly into the IVC (125). There are variable approaches to treatment of testicular cancer in the setting of tumor thrombus; some advocate retrievable IVC filter placement prior to definitive therapy, others employ neoadjuvant chemotherapy, still others advocate chemotherapy alone and finally some support early primary surgical resection (126-128).

Lung cancer is the second most common cancer and is responsible for the most number of cancer related deaths both in US and worldwide (129). While rare, lung cancer tumor thrombus can occur. It may extend to involve the

pulmonary veins, extending towards and even into the left atrium. Tumor thrombus in the pulmonary veins may cause symptoms distinct from systemic venous tumor thrombus. Cases of embolization causing stroke, bowel infarction and an ischemic leg have been reported (130). Left ventricular obstruction even cause sudden death (131,132). Given these potentially devastating complications, surgery prior to chemotherapy is generally advocated (133).

Colorectal cancer is the 4th leading cause of cancer in the US (129). Venous tumor thrombus is a rare finding in colorectal cancer, seen in only 1–2% of cases (134). When it occurs, venous invasion into the portal venous system occurs from the cecum to the sigmoid colon. Given its dual venous drainage via the portal and internal iliac systems, tumor thrombus from rectal cancer can involve either the inferior mesenteric vein (portal venous system) or the internal iliac veins (systemic venous system) (135). Preoperative diagnosis of portal vein tumor thrombus is important, allowing the surgeon to ligate the involved vein above the thrombus can help one avoid tumor embolism to the liver. Open rather than laparoscopic approach may be indicated as the surgeon can directly palpate the involved vein to determine where to ligate and resect. The rate of liver metastasis occurs at a higher rate in the presence of tumor thrombus (134).

Conclusions

Intravascular tumor extension can occur in many different types of cancer. Those with the highest proclivity include Wilm's tumor, RCC, adrenal cortical carcinoma and hepatocellular carcinoma. Tumor thrombus occurs less frequently in a myriad of other tumor types, including benign and malignant histologies. Imaging plays a central role in its diagnosis. The presence of tumor thrombus markedly worsens prognosis and impacts treatment approach.

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None.

Footnote

Conflicts of Interest: The authors have no conflicts of interest to declare.

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