The many faces of venous thrombosis in the cancer patient: MDCT imaging

Poster No.: C-1001
Congress: ECR 2011
Type: Educational Exhibit
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Keywords: Cardiovascular system, Oncology, Veins / Vena cava, CT, Embolism / Thrombosis, Neoplasia
DOI: 10.1594/ecr2011/C-1001

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Learning objectives

The goal of this exhibit is:

- to highlight the intense relationship between cancer and thrombosis
- to review the MDCT features of venous thromboembolic disease in the cancer patient
- to consider the MDCT pitfalls and clues to diagnosis.

Images for this section:

**Fig. 1:** Fig.5 - Schematic drawing of benign, malignant, and combined thrombosis. In malignant thrombosis the thrombus is in closer relationship to the tumor, enhances to a similar degree, and may cause vessel dilation and collaterals.
Background

Cancer patients are at increased risk for developing thromboembolic disorders because of several concurrent factors (Fig.1 on page 3) (1, 2). Thrombi can develop in close anatomic relation to the affected organ but can also be encountered in totally different and unpredictable body areas. The thromboembolism frequently represents a complication of a known, frequently advanced cancer disease or of its treatments. Nevertheless, thrombosisis may also represent the first symptom of a clinically occult tumor, acting as a paraneoplastic syndrome. Finally, the thrombotic event can be asymptomatic and may represents and incidental finding during cancer patient imaging. In this case, the main interest of the radiologist is pointed toward the tumor process but dramatic complications may develop from an overlooked thromboembolism.

Images for this section:

Fig. 1: Fig.1 - Our summarization of the Virchow triad in the cancer patient.
MDCT features of thrombosis

CT plays a relevant role in the assessment of various thrombotic sites and is the imaging modality of choice in patients suspected for pulmonary embolism (PE). Scanning each vessel at its peak enhancement is preferable, since an excessively "early" or "late" acquisition may cause false positive or false negative results. From this point of view, the high speed of modern scanners can paradoxically represent a limitation.

The key finding is recognition of a filling defect within the lumen (3, 4). Thrombi are usually isoattenuating on unenhanced scans and can be missed. Contrast-enhanced acquisition allows detecting the thrombus as a hypoattenuating defect, filling completely or partially the vessel lumen. In the latter case the thrombus will appear as a defect in the partially enhanced lumen. Recent thrombi may float within the lumen while long-standing or chronic thrombi are adherent to the vessel wall and may show calcium deposits. Since the venous walls are supplied by arterioles arising from the adjacent artery (vasa vasorum), a parietal enhancement of the vein can be frequently seen. This increases the difference between the vessel wall the non-enhancing luminal defect (rim sign) (Fig.2 on page 7) (3, 4).

Indirect signs of thrombosis include: dilation of the thrombosed segment, vascular ectasia above the obstruction, development of collateral pathways (including the portal vein cavernomatous transformation), infarction of the drained organ, changes in parenchymal perfusion due to compensatory increase in arterial supply (such as the transient hepatic attenuation difference - THAD) (Fig.3 on page 8, Fig.4 on page 9).

Benign versus malignant thrombosis

Distinguishing benign from neoplastic thrombosis is fundamental, because of different management and prognosis (Fig.5 on page 10). Benign thrombi are homogeneous and do not enhance after contrast medium injection, appearing as markedly hypoattenuating (Fig.6 on page 11). Malignant thrombi are due to the progressive endoluminal growth of a tumor. Tumor thrombus is usually seen in direct contiguity with the tumor mass and adherent to the vessel walls, the involved venous segment is frequently dilated, and collateral pathways are common (Fig.7 on page 12). The normal enhancement of the venous wall is lost. MDCT will identify thrombus enhancement, to a degree similar to the primary tumor. Hypervascularized tumors such as hepatocellular carcinoma (HCC) and renal carcinoma will show intense, arterial-dependent blush of the thrombus (Fig.8 on page 13) (4, 5). Sometimes benign and
malignant thrombi are seen together, with the latter closer to the tumor (Fig.9 on page 14, Fig.10 on page 15).

**Lower limb deep venous thrombosis**

Cancer patients have an incidence of venous thromboembolic disease being 5-6 times higher than the general population (1). Subjects presenting with deep venous thrombosis (DVT) and/or PE have an incidence of previously undiagnosed cancer of 4-6.5% (1). Consequently, cancer should always be considered in a patient presenting with thromboembolism and having not risk factor for abnormal coagulation. DVT is seen in up to 43% of subjects treated because of cancer, depending on the type of neoplasm and chemotherapy agent (6). Tumors most frequently combined with DVT include hematological malignancies and solid tumors of the brain, lungs, pancreas, and gynecologic organs.

In some patients, DVT is a subtle occurrence, with small thrombi detected within the calf veins. This requires a careful color Doppler exploration. Nevertheless, in many cases, the DVT is much more extended, involving unilaterally or bilaterally the calf veins, the popliteal vein, and the superficial and common femoral vein. In most instances, the thrombus stops at level of the ligamentum inguinale, without extending to the iliac vein. Consequently we suggest including always the proximal part of the thigh in a whole-body CT scan, to detect both inguinal lymphadenopaties and femoral DVT (Fig.2 on page 7, Fig.11 on page 16).

**Other venous thromboses**

An obstruction of the superior vena cava has a malignant etiology in 97% of cases (particularly, lung cancer and lymphoma) and can be due to venous compression, infiltration, or thrombosis (7). The impaired venous return from the head, neck, and upper limbs (superior vena cava syndrome) can determine airsways compromising, hemodynamic instability, and cerebral edema. The diagnosis of superior vena cava syndrome is clinical but MDCT plays a major role in detecting and characterizing the underlying disease (Fig.12 on page ... , Fig.13 on page ... ) (8).

Malignant inferior vena cava thrombosis is mostly related to the endoluminal growth of HCCs, adrenal carcinomas, or renal carcinomas (Fig.14 on page ...).

Pelvic veins thrombosis is frequently seen associated to female reproductive system malignancies, also because of the compression caused by bulky masses.

Malignant portal vein thrombosis is associated with a variety of tumors including extra-hepatic malignancies, liver metastasis and cholangiocellular carcinoma but is typically
found in patients with HCC (needing appropriate differentiation from benign, chronic liver disease-related thrombosis) (Fig.9 on page 14, Fig.15 on page ).

In the cancer patient, the Budd-Chiari syndrome can be due to blood hypercoagulability (lymphoproliferative disorders) or to mechanical compression of the hepatic vein by hepatic or perihepatic masses (10). Imaging may detect a filling defect in one or in multiple hepatic veins as well as in the retrohepatic inferior vena cava. Additionally, the liver will show typical changes in shape (caudate lobe hypertrophy) and attenuation (Fig.16 on page ).

**Pulmonary embolism**

Pulmonary emboli are detected incidentally in up to 5% of patients with known malignancy undergoing CT imaging (versus 1.5% of the general population and 2.2% of unselected outpatient population) (10, 11, 12) and 70% of the nonemergent patients with incidental detection of pulmonary emboli on routine MDCT have a cancer (13) (Fig.17 on page , Fig.18 on page ). Oppositely, occult malignancies are discovered incidentally in 13% of hospitalized patients undergoing pulmonary CT angiography and CT venography because of suspected thromboembolic venous disease (7). Cancers with the highest relative risk include brain tumors, pancreatic tumors, lymphomas, and gynecologic malignancies. The D-dimer has a high negative predictive value and sensitivity for PE in oncologic patients (14). Since many cases of PE are combined with DVT in the cancer patient, a comprehensive CT protocol is advisable, extending the pulmonary artery scan to the lower limbs (in the venous return phase) (6).

**Iatrogenic thromboses**

Cancer patients bearing a central venous device (CVD) are at increased risk of developing venous thrombosis and pulmonary embolism. Thrombi have been reported in 12-74% of cancer patients with indwelling CVD, depending on the type of malignancy, type of chemotherapy, type of CVD, insertion site, and tip location (1, 2). CVD-related thrombosis increases the risk of infection and causes postphlebitis syndrome in 15-30% of cases and pulmonary embolism in 11% of cases (half asymptomatic) (1, 2). MDCT allows the depiction of most CVD-related complications (Fig.19 on page ) (15, 16).

Thrombosis can be seen in strict temporal relationship with percutaneous ablation procedures. In percutaneous ethanol injection the intravasated alcohol causes a chemical thrombus while in thermal therapies (radiofrequency, microwave, or laser) it is the heat to damage the vessel wall determining the thrombosis. Thrombosis is usually seen in vessels close to the ablated mass and usually resolves spontaneously (17).
Fig. 2: 57-year-old woman with metastatic breast cancer and known deep venous thrombosis. Filling defect within the femoral vein (arrow). The venous walls enhance normally.
**Fig. 3:** Fig.3 - 66-year-old woman with metastatic lung cancer. Left kidney metastatic deposits, with venous thrombosis (yellow arrow) and dilatation of the spermatic vessels (secondary varicocele, red arrows).
**Fig. 4:** Fig. 4 - 60-year-old woman with colon cancer and infarction. Left colonic angle carcinoma (yellow arrow) seen combined with portal venous thrombosis (red arrows) and diffuse thickening of the large bowel walls due to venous infarction.
**Fig. 5**: Fig.5 - Schematic drawing of benign, malignant, and combined thrombosis. In malignant thrombosis the thrombus is in closer relationship to the tumor, enhances to a similar degree, and may cause vessel dilation and collaterals.
Fig. 6: 15-year-old boy with right humerus osteosarcoma and benign venous thrombosis. Wide infiltration of the shoulder soft tissues (red arrows) and a non-enhancing defect within the subclavian vein (yellow arrow).
Fig. 7: 60-year-old man with recurring sarcoma of the thigh. Direct tumor infiltration of the superficial femoral vein.
Fig. 8: 76-year-old woman with left renal clear-cell carcinoma (red yellows) and venous thrombosis (yellow arrows). Inhomogeneously enhancing, malignant thrombus, extending into a dilated renal vein and inferior vena cava. The presence and extent of thrombosis influences the tumor stage in the TNM classification of renal cancer.
**Fig. 9:** Fig.9 - 64-year-old woman with intrahepatic cholangiocellular carcinoma (not shown) and portal vein thrombosis. The thrombus has an enhancing, malignant appearance at level of the portal vein bifurcation (arrows in A) and a non-enhancing, benign appearance in the proximal tract of the portal trunk (arrow in B).
**Fig. 10**: Fig.10 - 61-year-old man with multifocal HCC and portal vein thrombosis. The thrombus has an enhancing, malignant appearance at level of the portal vein bifurcation (yellow arrows) and a non-enhancing, benign appearance in the proximal tract of the portal trunk (red arrows).
Fig. 11: Fig.11 - 59-year-old woman with ovarian cancer (not shown) and peritoneal carcinomatosis. Coronal reformatted view showing unsuspected right femoral vein thrombosis (yellow arrow). Also note right subphrenic peritoneal deposits (red arrows) and peritoneal effusion.
Conclusion

Venous thromboembolism in cancer patient may have a variety of locations and clinical presentations, ranging from dramatic, life-threatening complications to subtle, eventually overlooked changes. Radiologists should be better aware of the strict correlation between cancer and thrombosis, particularly because of the relevant consequences on patient work-up, treatment, and prognosis of both the diagnosed or overlooked the tumor and the diagnosed or overlooked thromboembolism.

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References

