

## Pseudolesion in segment IV A of the liver from vein of Sappey secondary to SVC obstruction

Dharshan Vummidi, MBBS, MRCP, FRCR; Puneet Bhargava, MBBS, DNB; Jonathan R. Medverd, MD; Jeffrey B. Virgin, MD, PhD; George R. Oliveira, MD; and Sandeep Vaidya, MD

Pseudolesions in the liver are caused by unusual/altered hemodynamics of the liver and can be confused with a true hepatic mass. In superior vena cava (SVC) obstruction, there is recruitment of the cavo-mammmary-phrenic-hepatic-capsule-portal pathway, and the venous blood follows the internal mammary vein, the inferior phrenic vein, the hepatic capsule veins, and the intrahepatic portal system, causing a hypervascular pseudolesion in segment IV A of the liver. Recognizing the classic appearances of this hypervascular pseudolesion from the vein of Sappey in a CT study of the abdomen has prognostic implications in directing further evaluation of the chest for SVC obstruction. We present a case of a 54-year-old HIV-positive male smoker in whom identification of the hypervascular pseudolesion from the vein of Sappey on abdominal CT led to the diagnosis of SVC syndrome.

### Case report

A 54-year-old HIV-positive male smoker on antiretroviral therapy presented to the emergency room with upper abdominal pain and mild shortness of breath. On physical examination he had mildly distended chest veins. Contrast-enhanced CT of the abdomen performed for the evaluation of abdominal pain revealed a hypervascular perfusion abnormality without associated mass effect in the cephalic hepatic segment IV A from a vein of Sappey (Fig. 1, A-C). Frontal and lateral chest radiographs performed subsequently revealed a right upper-lobe mass with right paratracheal lymphadenopathy (Fig. 2, A, B). Contrast-enhanced CT of the chest demonstrated a mediastinal mass with SVC obstruction (Fig. 3, A, B) with cutaneous,

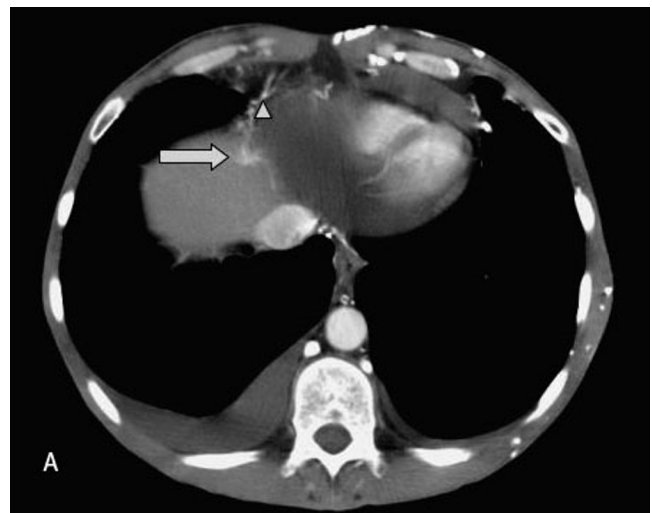


Figure 1A. 54-year-old HIV-positive man with hypervascular pseudolesion in segment IV A and vein of Sappey from SVC obstruction. Axial CT image demonstrates hypervascular pseudolesion in the segment IV A with enhancing vein of Sappey (arrowhead). Also note collateral recruitment of azygos and hemiazygos veins in addition to cutaneous collaterals. There are bilateral, right-greater-than-left pleural effusions. (See also Figure 1B.)

**Citation:** Vummidi D, Bhargava P, Medverd JR, Virgin JB, Oliveira GR, Vaidya S. Pseudolesion in segment IV A of the liver from vein of Sappey secondary to SVC obstruction. *Radiology Case Reports*. [Online] 2010;5:394.

**Copyright:** © 2010 The Authors. This is an open-access article distributed under the terms of the Creative Commons Attribution-NonCommercial-NoDerivs 2.5 License, which permits reproduction and distribution, provided the original work is properly cited. Commercial use and derivative works are not permitted.

The Department of Radiology, University of Washington School of Medicine, Seattle WA, includes Drs. Vummidi, Bhargava, Medverd, Oliveira, and Vaidya. The VA Puget Sound Health Care System, also in Seattle, includes Drs. Bhargava, Medverd, and Virgin. Contact Dr. Bhargava at [bhargp@u.washington.edu](mailto:bhargp@u.washington.edu).

**Competing Interests:** The authors have declared that no competing interests exist.

DOI: 10.2484/rcr.v5i3.394

## Pseudolesion in segment IV A of the liver from vein of Sappey secondary to SVC obstruction

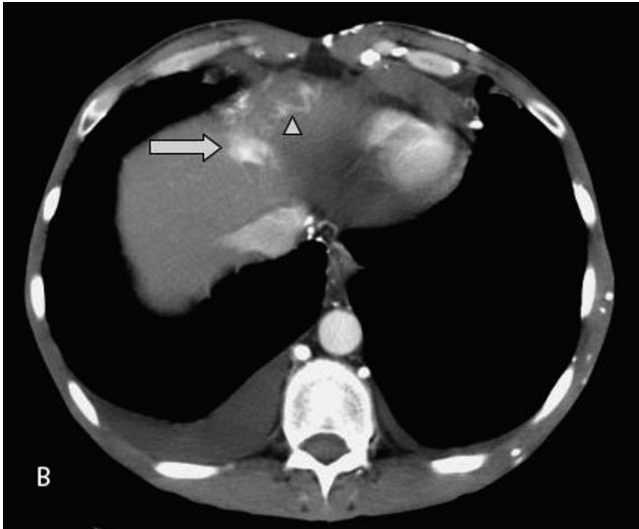


Figure 1B. 54-year-old HIV-positive man with hypervascular pseudolesion in segment IV A and vein of Sappey from SVC obstruction. Axial CT image demonstrates hypervascular pseudolesion (arrow) in the segment IV A with enhancing vein of Sappey (arrowhead). Also note collateral recruitment of azygos and hemiazygos veins in addition to cutaneous collaterals. There are bilateral, right-greater-than-left pleural effusions. (See also Figure 1A.)

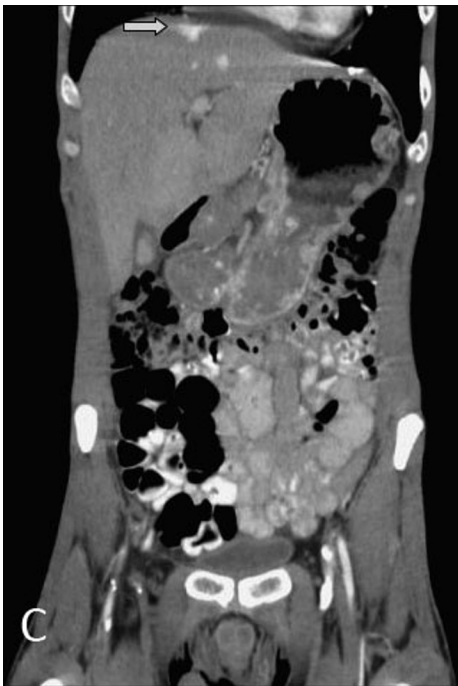


Figure 1C. 54-year-old HIV-positive man with hypervascular pseudolesion in segment IV A and vein of Sappey from SVC obstruction. Coronal CT image demonstrates hypervascular pseudolesion (arrow) in the segment IV A. The vein of Sappey was best visualized on the axial images.

thoracic, and abdominal collateral recruitment (left internal mammary vein, azygos vein, hemiazygos vein, superior epigastric vein, and vein of Sappey). A percutaneous biopsy

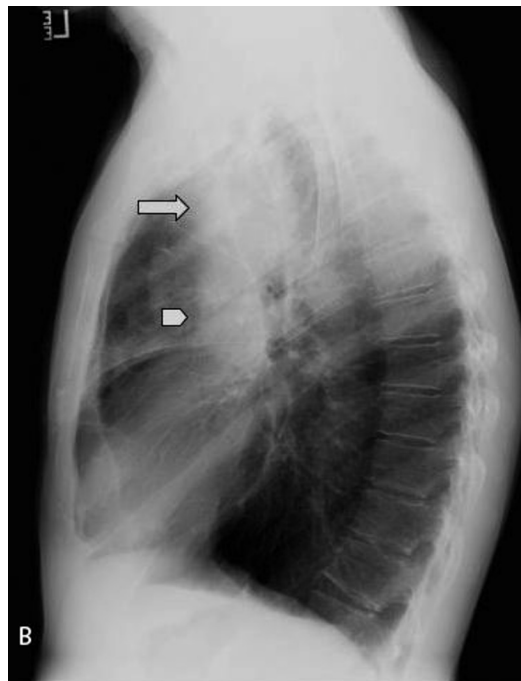
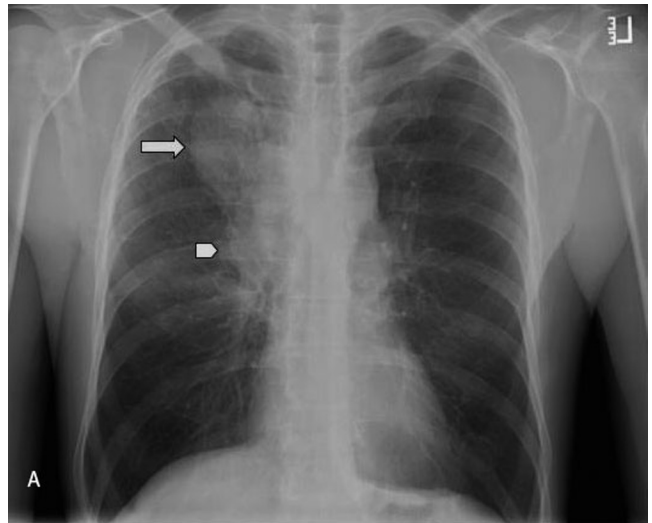


Figure 2A, B. 54-year-old HIV-positive man with hypervascular pseudolesion in segment IV A and vein of Sappey from SVC obstruction. Frontal (A) and lateral (B) chest radiographs demonstrate right upper lobe mass (arrow) with enlarged right paratracheal lymph nodes (arrowhead) and mass effect on the SVC.

of the mediastinal mass was then performed. On histopathology, numerous small highly atypical cells with high nuclear-cytoplasmic volume ratios consistent with small-cell carcinoma of the lung were seen on the hematoxylin and

## Pseudolesion in segment IV A of the liver from vein of Sappey secondary to SVC obstruction

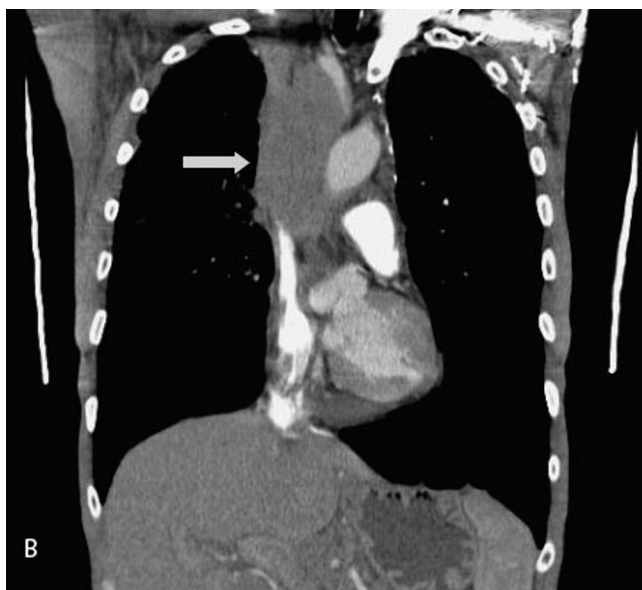


Figure 3A, B. 54-year-old HIV-positive man with hypervascular pseudolesion in segment IV A and vein of Sappey from SVC obstruction. Axial (A) and coronal (B) CT images through the chest demonstrate a mediastinal mass obstructing the superior vena cava (arrow) with extensive prominent collaterals in the left chest wall and scapular region.

eosin stain (Fig. 4). The patient was subsequently treated with chemotherapy.

### Discussion

SVC syndrome is an obstruction of the blood flow through the superior vena cava and is a medical emergency. It occurs in 5% to 10% of patients with a right-sided malignant intrathoracic mass lesion (1). Malignant causes account for 80% of cases, namely bronchogenic carcinomas, with most of these being small-cell carcinomas, sarcomas, and lymphomas (especially the large-cell-type Non-Hodgkin lymphoma) (2). Benign causes (20% of the cases)

include fibrosing mediastinitis, infections such as histoplasmosis and tuberculosis, aneurysms, vasculitis, arteriovenous fistulas, mass effect from benign mediastinal tumors, and thrombosis related to the presence of central venous catheters (3).

Understanding pseudolesions of the liver is important due to their close resemblance to primary liver cancers or metastases. Pseudolesions are caused by unusual/ altered hemodynamics of the liver. Recruitment of the hepatic subcapsular venous collaterals, portal vein obstruction, arterioportal shunts, liver compression, superior or inferior vena caval obstruction, hepatic vein obstruction, hepatic infarction, and intrahepatic vascular shunts result in pseudolesions (4).

Although the liver has a dual blood supply, the subcapsular portion of the liver is supplied by another venous system, a third hepatic inflow comprising the cholecystic, parabiliary, or epigastric-paraumbilical venous system (5, 6). The superior vein of Sappey drains the cranial component of the falciform ligament and the medial aspect of the diaphragm and enters the peripheral portal branches of the

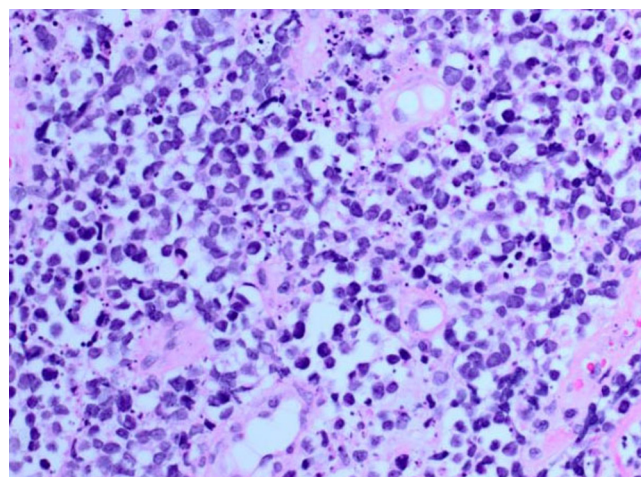


Figure 4. 54-year-old HIV-positive man with hypervascular pseudolesion in segment IV A and vein of Sappey from SVC obstruction. Hematoxylin & Eosin stain histopathology image demonstrates numerous small, highly atypical cells with high nuclear-cytoplasmic volume ratios consistent with small-cell carcinoma of the lung.

left hepatic lobe. It also communicates with branches of the superior epigastric or internal thoracic veins. The inferior vein of Sappey drains the caudal portion of the falciform ligament and enters the peripheral portal branches of the left hepatic lobe. It descends along the round ligament and communicates with branches of inferior epigastric veins around the umbilicus. The vein of Burow also communicates with branches of the inferior epigastric veins around the umbilicus. However, it does not enter the liver directly, but rather terminates in the middle portion of the collapsed umbilical vein, although some small communicating branches are present between it and the inferior vein of

## Pseudolesion in segment IV A of the liver from vein of Sappey secondary to SVC obstruction

Sappey, namely the intercalary veins (7). In SVC obstruction, there is recruitment of the cavo-mammary-phrenic-hepatic-capsule-portal pathway, and the venous blood enters the internal mammary vein, the inferior phrenic vein, the hepatic capsule veins, and the intrahepatic portal system, causing a hypervascular pseudolesion in segment IV A of the liver (8). The nuclear-medicine correlate of the same physiology is the focal hepatic hotspot sign observed on technetium-99m sulphur colloid scans of the liver and spleen, which represents the focal increased uptake of the radiopharmaceutical material in segment IV A of the liver (9).

With advances in scanner technology, we are increasingly noticing these enhanced collateral vessels causing pseudolesions, characteristically in the cephalic segment IV of the liver. These pseudolesions are best appreciated on dynamic-bolus, arterial-phase-enhanced CT. These subcapsular lesions commonly have a wedge shape, lack mass effect, and have normal vessels coursing through them (10). The characteristic finding would however be the enhanced superior vein of Sappey directly supplying the enhancing pseudolesion in the cephalic portion of segment IV (8). Normally, the blood arriving through the epigastric–paraumbilical venous system arrives in the late portal phase and rarely causes enhancement in the arterial phase, the exception being SVC obstruction due to the collateral flow (10).

In conclusion, recognizing the classic appearances of the hypervascular pseudolesion in the cranial aspect of segment IV of the liver from the vein of Sappey has prognostic implications in directing further evaluation of the chest for SVC obstruction.

### References

1. Salsali M, Clifton EE. Superior vena caval obstruction in carcinoma of lung. *N Y State J Med*. 1969;69(22):2875-80. [\[PubMed\]](#)
2. Escalante CP. Causes and management of superior vena cava syndrome. *Oncology (Williston Park)*. 1993;7:61-8. [\[PubMed\]](#)
3. Nieto AF, Doty DB. Superior vena cava obstruction; clinical syndrome, etiology and treatment. *Curr Probl Cancer*. 1986;10(9):441-84. [\[PubMed\]](#)
4. Lee JW, Kim S, Kwack SW, et al. Hepatic capsular and subcapsular pathologic conditions: Demonstration with CT and MR imaging. *Radiographics*. 2008 Sep-Oct;28(5):1307-23. [\[PubMed\]](#)
5. Matsui O, Takahashi S, Kadoya M, et al. Pseudolesion in segment IV of the liver at CT during arterial portography: correlation with aberrant gastric venous drainage. *Radiology*. 1994 Oct;193(1):31-5. [\[PubMed\]](#)
6. Yoshimitsu K, Honda H, Kaneko K, et al. Anatomy and clinical importance of cholecystic venous drainage: Helical CT observations during injection of contrast medium into the cholecystic artery. *AJR Am J Roentgenol*. 1997 Aug;169(2):505-10. [\[PubMed\]](#)
7. Martin BF, Tudor RG. The umbilical and paraumbilical veins of man. *J Anat*. 1980 Mar;130(2):305-22. [\[PubMed\]](#)
8. Dahan H, Arrive L, Monnier-Cholley L, et al. Cavo-portal collateral pathway in vena cava obstruction: imaging features. *AJR Am J Roentgenol*. 1998 Nov;171(5):1405-11. [\[PubMed\]](#)
9. Dickson AM. The focal hepatic hot spot sign. *Radiology*. 2005 Nov;237(2):647-8. [\[PubMed\]](#)
10. Yoshimitsu K, Honda H, Kuroiwa T, et al. Unusual hemodynamics and pseudolesions of the noncirrhotic liver at CT. *Radiographics*. 2001 Oct;21:S81-96. [\[PubMed\]](#)