CASE REPORT

Vanishing bone metastases — a pitfall in the interpretation of contrast enhanced CT in patients with superior vena cava obstruction

N THOMAS, MBChB, FRCR, T B OLIVER, MBChB, FRCR and T SUDARSHAN, MBChB, FRCR

Department of Clinical Radiology, Ninewells Hospital and Medical School, Dundee, UK

ABSTRACT. We describe a previously unreported case of vertebral marrow enhancement owing to collateral circulation in a patient with superior vena cava obstruction. Failure to recognise this phenomenon led to the misdiagnosis of sclerotic bone metastases.

Received 5 August 2010
Revised 27 October 2010
Accepted 18 November 2010

DOI: 10.1259/bjr/50676625

© 2011 The British Institute of Radiology

We describe a patient with superior vena cava (SVC) obstruction who had retrograde filling of the thoracic and vertebral venous collateral systems, which caused enhancement of the thoracic vertebrae and was mistaken for metastatic infiltration.

Several other venous collateral imaging patterns have been reported in patients with SVC obstruction, but we are not aware of any previous description of the above phenomenon, which we believe to be of general interest to radiologists.

Case report

A 60-year-old male smoker presented with weight loss and clinical features suggestive of SVC obstruction. A chest radiograph showed a superior mediastinal mass and a contrast enhanced CT (CECT) of the chest and abdomen was performed using the following parameters: 64 slice Lightspeed VCT (GE Medical Systems, Milwaukee, WI), 0.625 mm source image thickness, 100 ml iodinated contrast (Ultravist 300, Schering, Berlin, Germany), via iv cannula in left antecubital fossa injected by pump at 3 ml s^{-1} with a 26 s delay.

CT confirmed a large anterior mediastinal mass. This occluded the left brachiocephalic vein and the SVC, proximal to the azygocaval junction. Contrast filled an extensive collateral circulation involving the mediastinum, left chest wall and vertebral venous plexus, passing into the distal SVC via the azygos vein.

High attenuation was observed within the vertebral bodies and left posterior elements of several upper

thoracic vertebrae. This was ascribed to metastatic disease (Figure 1a,b).

Following biopsy diagnosis of squamous cell carcinoma, a CT positron emission tomography (PET) staging study was performed. The non-contrast enhanced CT study showed that the upper thoracic vertebrae were normal (Figure 2). On re-examination of the initial CECT, it was noted that much of the vertebral abnormality was centred around the opacified basivertebral and the anterior vertebral body veins and it was realised that retrograde opacification of the capillary spaces within the vertebrae had been mistaken for sclerotic metastatic disease (Figure 3).

Discussion

SVC obstruction is due to malignancy in 80% of cases, but is also a recognised complication of iatrogenic or non-iatrogenic trauma and fibrosing mediastinitis owing to granulomatous infection, autoimmune disease or drugs [1].

When SVC obstruction develops, blood is diverted via venous collaterals. Four classic collateral pathways involving the azygos and hemiazygos, internal mammary and lateral thoracic, superficial thoraco-abdominal and vertebral venous plexuses have been described and proven by venography [2–4]. In practice, several collateral pathways may be involved together and the specific pattern in a given patient will depend on the cause, exact location and duration of venous obstruction, along with individual variations in venous anatomy.

Imaging findings of several less common anatomical variations have been reported. These include infraphrenic and hepatic parenchymal collaterals and systemic to pulmonary venous shunting [5–7]. Infraphrenic and hepatic parenchymal collaterals are said to be more likely if there is obstruction of the azygoatrial portion of the SVC [8]. Focal pericardial enhancement has also been

Address correspondence to: Dr Naveena Thomas, Department of Clinical Radiology, Ninewells Hospital and Medical School, Ninewells Avenue, Dundee, DD1 9SY, UK. E-mail: naveenathomas@nhs.net

(a)

(b)

Figure 1. (a) Coronal oblique reformatted contrast enhanced CT (CECT) image demonstrating patchy enhancement of thoracic vertebral bodies which was mistaken for metastatic disease. (b) Left parasagittal reformatted CECT image demonstrating increased density within upper thoracic vertebral bodies and posterior elements. Multiple opacified mediastinal, prevertebral and neural foraminal collateral vessels are also apparent along with an anterior mediastinal mass.

reported [9], but to our knowledge focal vertebral marrow enhancement has not been previously described.

In the above case, opacified blood in the left subclavian vein flowed via mediastinal and left chest wall collaterals into the vertebral venous plexus. Venous pressure must have been sufficiently high to allow opacification of intravertebral veins and capillary spaces, manifested by a patchy increase in vertebral density, which was

mistaken for bony metastases. This error was disclosed by a subsequent non-CECT PET study that showed, in the absence of contrast, vertebral density was normal.

The vertebral venous plexus is a thin-walled, valveless network of veins within and surrounding the spinal column, extending from cranium to sacrum. The internal portion of the plexus (venous plexus of Batson) is epidural

Figure 2. Non-enhanced midline sagittal reformatted positron emission tomography (PET) CT image demonstrating normal upper thoracic vertebral density.

Figure 3. Midline sagittal reformatted image from contrast enhanced CT at comparable position to Figure 2, demonstrating increased density within thoracic vertebral bodies due to enhancement around opacified anterior vertebral body veins.

and receives blood from vertebral bodies via the basi-vertebral veins. It communicates with an external para-vertebral venous plexus via multiple intervertebral veins that pass through the spinal foramina. The paravertebral plexus forms multiple anastomoses via segmental veins, communicating with the left brachiocephalic, azygos-hemiazygos and left renal veins or inferior vena cava [10]. The vertebral venous plexus is thought to be a common route for the spread of vertebral metastatic disease but also offers a route by which blood returning from the head and upper limbs can bypass an obstructed SVC.

Conclusion

This case illustrates vertebral marrow enhancement, a previously unreported manifestation of retrograde contrast opacification of the vertebral venous plexus, in a patient with SVC obstruction. Awareness of this phenomenon is important as failure to recognise it could result in a false-positive diagnosis of bony metastatic disease.

References

1. Wilson ES. Systemic to pulmonary venous communication in the superior vena caval syndrome. *AM J Roentgenol* 1976;127:247–9.
2. Eckenhoff JE. The vertebral venous plexus. *Can J Anaesth* 1971;18:487–95.
3. Stanford W, Jolles H, Ell S, Chin LC. Superior vena cava obstruction: a venographic classification. *Am J Roentgenol* 1987;148:259–62.
4. Sheth S, Ebert MD, Fishman EK. Superior vena cava obstruction evaluation with MDCT. *Am J Roentgenol* 2010;194:W336–W346.
5. Ho HT, Horowitz AL, Ho AC. Systemic to pulmonary venous communications (right to left shunt) in superior vena cava obstruction demonstrated by spiral CT. *Br J Radiol* 1999;72:712–13.
6. Hutchins WW, Kirchner PT, Macmahon H. Perfusion lung scan in superior vena cava obstruction: demonstration of venous collaterals and systemic – pulmonary venous shunt. *Am J Roentgenol* 1982;138:754–6.
7. Kapur S, Paik E, Rezaei A, Doan NV. Where there is blood, there is a way: unusual collateral vessels in superior and inferior vena cava obstruction. *Radiographics* 2010;30: 67–78.
8. Rastogi R, Thulkar S, Garg R, Gupta A. Infraphrenic collaterals in malignant superior vena cava obstruction. *Clin Imaging* 2007;31:321–4.
9. Holemans JA, Howlett DC, Rankin SC. Case report: superior vena cava obstruction: unusual CT findings due to venous collaterals. *Clin Radiol* 1997;52:559–60.
10. Drake R, Vogl W, Mitchell AW, Tibbits R, Richardson P. *Gray's Atlas of Anatomy*. 40th edition. London, UK: Churchill Livingstone, 2008.