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TEST YOURSELF: ANSWER

Multiple disappearing spinal lesions

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Diagnosis

Vertebral enhancement secondary to retrograde contrast filling of vertebral venous collaterals as a consequence of superior vena cava syndrome.

Discussion

Superior vena cava (SVC) syndrome is characterized by abnormal venous return to the right atrium from the upper body, which may cause vertebral body enhancement and mimic sclerotic lesions. Altered venous return typically results from obstruction of the SVC, either intraluminal, seen in the setting of thrombus, or via extrinsic compression, caused by pathologies such as mediastinal lymphadenopathy, a lung mass, or fibrosing mediastinitis [1, 2]. SVC syndrome often has an insidious onset as the body adapts to increasing SVC pressures by recruiting collateral vessels for venous return. Multiple collateral pathways can occur depending on the location of the obstruction and these pathways may change over time as the cause of the obstruction evolves, and as such, the imaging findings may be variable [3–6]. Other factors, including the site of contrast administration, timing of image acquisition, and native venous anatomy, may also impact the appearance of SVC syndrome on CT.

The internal vertebral plexuses lie within the vertebral canal anterior and posterior to the spinal cord, while the external vertebral plexus overlies the anterior vertebral body. Connecting the internal and external venous plexuses are the basivertebral veins, which are valveless vessels travelling horizontally through the vertebral bodies [7]. In this case of a patient with non–small cell lung cancer, arterial phase restaging CT thorax demonstrates mediastinal soft tissue infiltration and stenosis of the SVC (Fig. 1a). Additionally, there are lobulated areas of increased attenuation of multiple lower cervical and upper thoracic vertebral bodies (Figs. 1b, c) which was initially attributed to metastatic disease. On subsequent CT pulmonary angiogram (CTPA) 1 month later when the patient presented to the ER, these vertebral bodies demonstrated normal attenuation (Fig. 2a, b) and the dense "lesions" were no longer seen. This can be explained by the 35-s delayed acquisition on the initial imaging, which permitted enough time for contrast to traverse the peripheral venous return system and encounter the obstructed SVC with subsequent retrograde filling of the venous collaterals. In this specific patient's case, the collateral pathway of least resistance included retrograde flow through the azygos vein and into the vertebral and subscapular plexuses. With the earlier image acquisition used for CTPA (approximately 5–10-s delay), there is insufficient time for contrast to accumulate in the basivertebral veins and the vertebral bodies exhibited normal attenuation. The distended contrast-filled dilated capillaries at the basivertebral anastomotic sites are thought to account for foci of increased attenuation within the bone marrow seen in this patient's initial arterial phase CT. Previous reports of this rare phenomenon are few [8–10].

This case demonstrates transient vertebral enhancement in the setting of SVC obstruction. This phenomenon is important to recognize as these dense vertebral foci can mimic osseous metastases particularly in the setting of known primary malignancy. Consideration should be given to CT image acquisition timing and routes of possible collateral venous flow when assessing for abnormal osseous attenuation in patients who have findings of SVC obstruction.

The case presentation can be found at doi:10.1007/s00256-019-03357-z.

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Compliance with ethical standards

Conflict of interest The authors declare that they have no conflicts of interest.

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