

Computed Tomography Image of a Mediastinal Mass-Producing Cardiac Tamponade

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A 64 year old female presented with 2 weeks of severe dry cough. For the previous 5 months she had suffered from dyspnea on exertion and diffuse chest pain, as well as a 10 kilogram weight loss. Her history was significant for hyperlipidemia and a 45 pack per year history of smoking. Physical examination revealed distant heart sounds and distended neck veins, which are common signs of Beck's triad, which is associated with cardiac tamponade [1].

There was also decreased air intake to the right lung. Electrocardiography detected low voltage QRS complexes with electrical alternans. Computed tomography (CT)-guided angiogram revealed a space-occupying mass (11 × 7 × 9 cm) in the frontal mediastinum, which was compressing the upper border of the heart, accompanied by several adjacent glands enlarged to up to 2 cm in diameter. Pericardial effusion [Figure 1], signs of right ventricular strain [2], stasis of contrast material, and reflux to the hepatic veins [Figure 2] were found. Echocardiogram demonstrated a large pericardial effusion with signs of cardiac tamponade [3]. Pericardiocentesis drained 400 cc of fluid. During the subsequent week, 100 cc of pericardial fluid was

drained each day. A CT-guided biopsy later identified the mediastinal mass as a small cell carcinoma of the lung.

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Figure 1. Coronal reconstruction of contrast-enhanced computed tomography showing a huge mediastinal mass (blue arrow) compressing the upper border of the heart. Also visible are a pericardial effusion (gray arrow) and a small pleural effusion (white arrow)

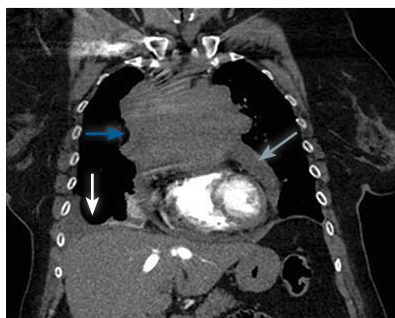


Figure 2. A maximum intensity projection reconstructed image from contrast-enhanced computed tomography shows a pericardial effusion, as well as signs of right ventricular strain, contrast pooling within the superior vena cava (blue arrow), and reflux of contrast into inferior vena cava (gray arrow) and hepatic veins (white arrow)



Capsule

Taking an active interest in HIV latency

Human immunodeficiency virus (HIV) cure efforts have been thwarted by an inability to target the latent reservoir, which is thought to be largely composed of resting CD4 T cells. A recent report suggested that the Fcγ receptor CD32 might be a marker of latently infected CD4 T cells. **Abdel-Mohsen** and colleagues meticulously examined T cells from treated HIV patients across

the world. CD32+ HIV-infected T cells had an activated phenotype and HIV RNA, indicating active HIV transcription. In contrast, the majority of HIV DNA resided in CD32- cells. Thus, targeting CD32+ cells is unlikely to hit the latent HIV reservoir.

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