Contrast-enhanced fluorodeoxyglucose positron emission tomography/contrast-enhanced computed tomography in mediastinal T-cell lymphoma with superior vena cava syndrome

Sampath Santhosh, Arun Kumar Reddy Gorla, Anish Bhattacharya, Subhash Chander Varma[1], Bhagwant Rai Mittal

Departments of Nuclear Medicine and [1]Internal Medicine, Postgraduate Institute of Medical Education and Research, Chandigarh, India

ABSTRACT

Positron emission tomography-computed tomography (PET/CT) is a routine investigation for the staging of lymphomas. Contrast-enhanced computed tomography is mandatory whenever parenchymal lesions, especially in the liver and spleen are suspected. We report a rare case of primary mediastinal T-cell lymphoma evaluated with contrast-enhanced PET/CT that showed features of superior vena cava syndrome.

Keywords: Fluorodeoxyglucose, non-Hodgkin's lymphoma, positron emission tomography/contrast-enhanced computed tomography, primary mediastinal lymphoma, superior vena cava syndrome

A 28-year-old male patient presented with a history of a dry cough for a year and neck swelling (1-month). Multiple large mediastinal lymph nodes were detected on computed tomography (CT) of the thorax. Biopsy suggested T-cell lymphoblastic lymphoma (LBL) type of non-Hodgkin's lymphoma (NHL). Contrast-enhanced ^{18}F-fluorodeoxyglucose positron emission tomography/contrast-enhanced CT (FDG PET/CECT) was done for initial staging. Maximum intensity projection [Figure 1a] and coronal fused PET/CT [Figure 1b] images showed an intensely FDG-avid (SUVma × 12.0) heterogeneously enhancing conglomerate nodal mass measuring 15.3 cm × 14.2 cm × 13.5 cm with central necrosis (arrow) in the superior mediastinum and right hemithorax, extending from the D3–D9 vertebrae, with collapse of the upper lobe of the right lung. Physiological FDG uptake was seen in the marrow and rest of the visualized organs. The axial CECT image [Figure 1c] showed the lesion encasing the right main bronchus causing left mediastinal shift, displacing and compressing the right brachiocephalic vein, distal azygous vein and superior vena cava (SVC) (arrows). An axial CECT image at a higher level (d) showed multiple collateral veins in the subcutaneous plane in the anterior thoracic and abdominal wall bilaterally (right >left) and right upper limb. A volume-rendered image (e) clearly depicted the subcutaneous collaterals (arrows), suggesting SVC obstruction. A bone marrow biopsy was negative for lymphomatous infiltration. The final diagnosis was Stage I (X) NHL with SVC syndrome.

SVC syndrome is caused by obstruction of the SVC either by extrinsic compression or internal thrombus. Clinical signs include cyanosis, plethora distention of subcutaneous vessels, and edema of the arms, head and neck. Edema may compromise the function of the larynx or pharynx causing dyspnea, stridor, cough, hoarseness, and dysphagia. Cerebral edema can occur as serious sequelae causing headaches, confusion, and possibly coma.[1] SVC syndrome is most commonly associated with metastatic pulmonary or mediastinal malignancy.[2] Benign causes

Address for correspondence:
Dr. Sampath Santhosh, Institute of Nuclear Imaging and Molecular Medicine, Tamil Nadu Government Multi Super Specialty Hospital, Omandurar Government Estate, Anna Salai, Chennai - 600 002, Tamil Nadu, India.
E-mail: santhosh610@yahoo.com

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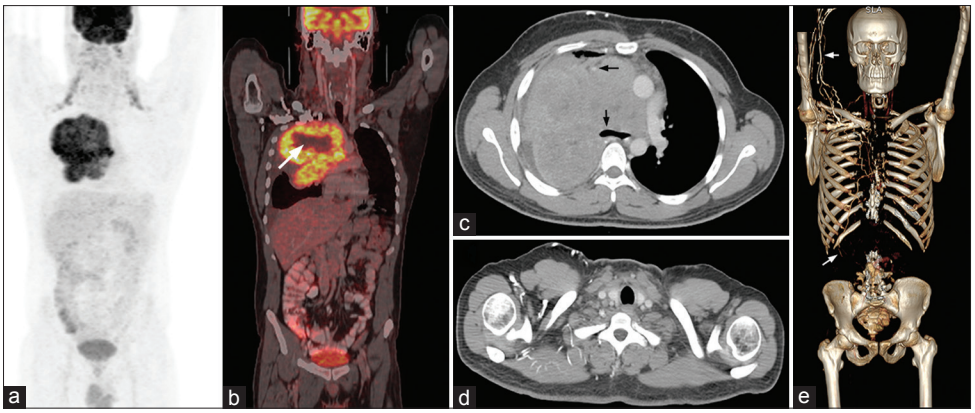

Figure 1: Maximum intensity projection (a) and coronal fused positron emission tomography-computed tomography (b) images showed an intensely fluorodeoxyglucose-avid heterogeneously enhancing conglomerate nodal mass with central necrosis (arrow) in the superior mediastinum and right hemithorax. The axial contrast-enhanced computed tomography image (c) the lesion compressing the distal azygous vein and superior vena cava (arrows). An axial contrast-enhanced computed tomography image at a higher level (d) multiple collateral veins in subcutaneous plane in the anterior thoracic wall bilaterally. A volume-rendered image (e) clearly depicted the subcutaneous collaterals (arrows), suggesting superior vena cava obstruction

include infection, idiopathic mediastinal fibrosis, retrosternal thyroid, aortic aneurysm, benign tumors, mediastinal hematoma, sarcoidosis, radiation fibrosis, and iatrogenic causes like thrombosis of the SVC following central venous catheter placement.[3] Our patient presented with a cough and there was distention of subcutaneous vessels in the neck on the right side with imaging findings of SVC obstruction caused by compression of SVC by enlarged mediastinal nodal T-cell lymphoma. LBL is a rare form of aggressive NHL, occurring in 1–2% of all NHLs.[4] T-cell LBL is typically seen in adolescence and young adults with a nearly three-fold male predominance. It presents with a mediastinal mass in 60–70% of the cases.[5] On the other hand, about 10% of lymphomas involving the mediastinum are primary, of which T-cell LBL is very rare.[6,7] In the NHL Classification Project, only 13% T-cell LBLs had Stages 1 or 2 disease.[4] Patients with T-cell LBL often present with shortness of breath due to either compression of the SVC or pleural or pericardial effusion.[8,9] The azygous-hemiazygous is the common collateral pathway that predominates in SVC obstruction; however, the internal-external mammary and lateral thoracic pathways were involved as collaterals in our case due to additional azygous vein obstruction.[3] Other unusual collateral pathways that may be seen with SVC obstruction include systemic-to-pulmonary venous pathway causing right to left shunt and cavoportal pathway causing unusual enhancement patterns in the liver.[3] PET/CT has been used for staging other types of mediastinal lymphomas.[10,11] This case highlights the role of PET/CT in staging a rare type of primary mediastinal T-cell NHL and suggests that contrast-enhanced FDG-PET/CT may be a useful investigation for the evaluation of mediastinal lymphoma with suspicion of SVC syndrome.

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Conflicts of interest

There are no conflicts of interest.

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