Parathyroid Carcinoma – A Malignant Cause of Metabolic Skeletal Super Scan on Fluorodeoxyglucose Positron Emission Tomography-Computed Tomography

Abstract

Excessive abnormal tracer uptake in active tracer avid organ(s) with the suppression of physiological background tracer distribution is termed as super scan. Herein, we present an ^{18}F-fluorodeoxyglucose positron emission tomography-computed tomography (FDG-PET/CT), where the metabolic pattern of skeletal super scan with coexistent parathyroid tumor was seen giving rise to the suspicion of primary hyperparathyroidism. It was subsequently diagnosed as a case of parathyroid carcinoma. Very high levels of serum parathormone in parathyroid carcinoma lead to accelerated bone turn over resulting in metabolic skeletal superscan in FDG-PET/CT which is seldom observed in parathyroid adenoma.

Keywords: *^{18}F-fluorodeoxyglucose positron emission tomography-computed tomography, metabolic bone disease, parathyroid carcinoma, primary hyperparathyroidism, super scan*

Santhosh Sampath, Godwin Jeeva

Division of PET/CT, Gemini Scans, Chennai, Tamil Nadu, India

A 35-year-old male presented with diffuse body ache and unexplained weight loss. ^{18}F-fluorodeoxyglucose positron emission tomography-computed tomography (FDG-PET/CT) scan showed extensive FDG avid (maximum standardized uptake value [SUVmax] 8.0) mixed lytic-sclerotic lesions in the skull, mandible [arrows in Figure 1a and b], maxilla, sternum, and all the ribs [arrows in Figure 1c and d correspond to posterior left 10th rib]. Diffuse FDG uptake was visualized in all the vertebrae showing generalized sclerosis [arrow heads in Figure 1c and d]. Multiple small and large osteolytic FDG avid (SUVmax 8.7) and non-FDG avid [arrows in Figure 1e and f] lesions were visualized in bilateral pelvic bones. FDG avid cortical thinning, tunneling [arrowhead in Figure 2a], and endosteal scalloping were visualized in the appendicular skeleton [arrows in Figure 2a and b]. Except for the physiological uptake in the brain, there was very low background FDG distribution. These findings together with mandibular involvement [arrow in Figure 2c] gave rise to METABOLIC SKELETAL SUPER SCAN (MSSS) pattern that would be otherwise seen in whole body skeletal scintigraphy of metabolic bone disease.

A mixed solid-cystic lesion measuring 36 mm × 32 mm was seen in the posteroinferior aspect of the left lobe of thyroid [arrows in Figure 3a and b] with very faint FDG uptake (SUV_{max} 2.2) in the enhancing solid components [arrows in Figure 3c and d]. With the background of metabolic bone disease, this lesion was suggested to be a parathyroid tumor. The FDG avid lytic lesions were considered as brown tumors. Further evaluation revealed very high serum calcium (16 mg/ml) and parathormone (2831 pg/ml). The patient was subsequently treated with left inferior parathyroidectomy. The parathyroid specimen weighed 25 gram (measuring of 4.5 cm × 3.5 cm × 1.5 cm) and was diagnosed with parathyroid carcinoma due to lymphovascular invasion, stage pT1. With the absence of nonosseous lesions in the PET/CT, the stage was concluded as T1N0M0.

Parathyroid carcinoma is a rare malignancy generally diagnosed after the surgery for primary hyperparathyroidism (PHPT). Those patients have significantly higher serum parathyroid hormone and calcium levels compared with other causes of

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Address for correspondence:
Dr. Santhosh Sampath, Division of PET/CT, Gemini Scans, Vadapalani, Chennai - 600 026, Tamil Nadu, India.
E-mail: santhosh610@yahoo.com

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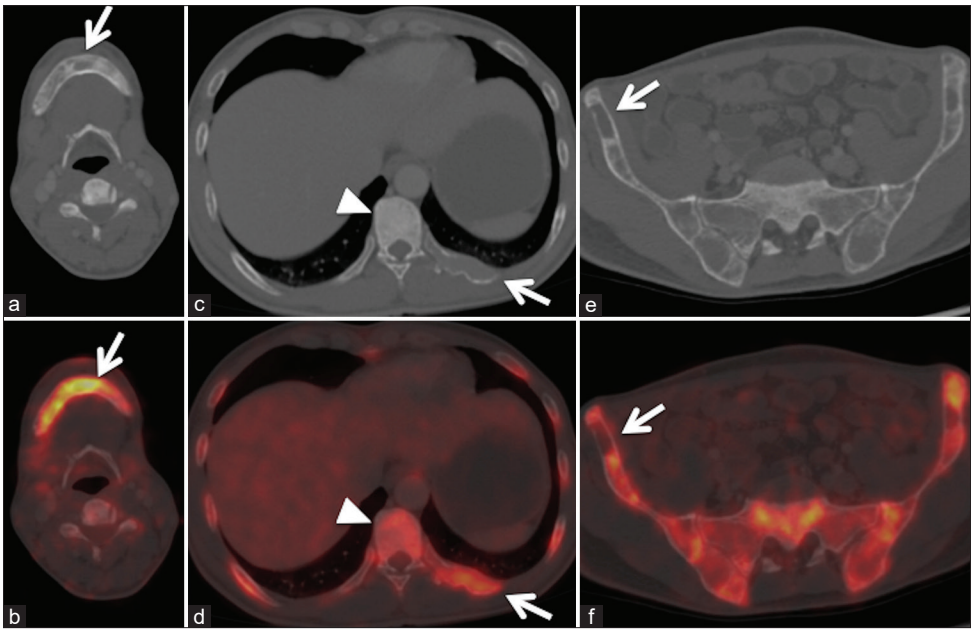

Figure 1: Axial computed tomography and fused positron emission tomography-computed tomography images of ^{18}F-fluorodeoxyglucose positron emission tomography-computed tomography show extensive fluorodeoxyglucose avid (maximum standardized uptake value 8.0) mixed lytic-sclerotic lesions in mandible (arrows in a and b), and posterior left 10th rib (arrows in c and d). Diffuse fluorodeoxyglucose uptake is visualized in D10 vertebra showing generalized sclerosis (arrow heads in c and d). Multiple small and large osteolytic fluorodeoxyglucose avid (maximum standardized uptake value 8.7) and non fluorodeoxyglucose avid (arrows in e and f) lesions are visualized in bilateral pelvic bones

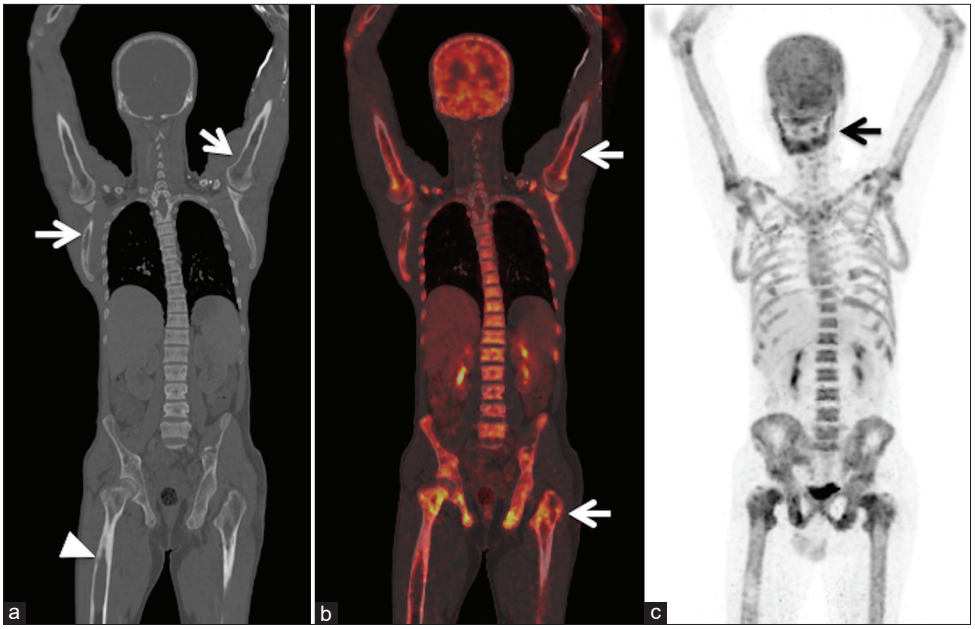

Figure 2: Coronal computed tomography (a), fused positron emission tomography-computed tomography (b) and maximum intensity projection (c) images show fluorodeoxyglucose avid cortical thinning, tunneling (arrowhead in a) and endosteal scalloping in the appendicular skeleton (arrows in a and b). Except for the physiological uptake in the brain, there is very low background fluorodeoxyglucose distribution (c)

PHPT.[1] Only two cases of parathyroid carcinoma presenting as MSSS in PET/CT have been reported in the literature.[2,3] In both cases, PET/CT was done for the evaluation of recurrent parathyroid carcinoma. In our case, a provisional diagnosis of PHPT was considered based on the PET/CT findings. Marrow infiltrative disorders such as leukemia[4] and skeletal metastases from poorly differentiated solid malignancies have also been reported to cause SSS in FDG-PET/CT.[5,6] In metastatic superscan of PET/CT, the distal appendicular skeleton is usually spared in adults due to the absence of red bone marrow, which is a prerequisite for primary hematological spread of tumor cells to the bone extracellular matrix.

In metabolic bone disease, the increased FDG uptake represents elevated metabolism associated with increased bone

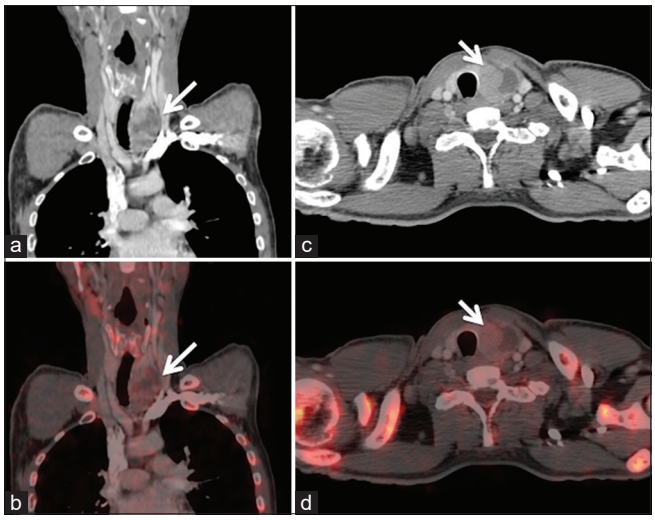

Figure 3: Coronal computed tomography (a) and fused positron emission tomography-computed tomography (b) show a mixed solid-cystic lesion in the posteroinferior aspect of the left lobe of thyroid (arrows in a and b). Axial computed tomography (c) and fused positron emission tomography-computed tomography (d) show very faint fluorodeoxyglucose uptake in the enhancing solid components (arrows in c and d)

turn over (osteoclastic and osteoblastic activity) and hence the entire axial and appendicular skeleton show hypermetabolism. Parathyroid carcinoma is known to be poorly FDG avid.[7-9] A MSSS pattern coupled with parathyroid lesion should raise the suspicion of parathyroid carcinoma and appropriate curative treatment should be provided. Almost complete resolution of FDG uptake with improvement in cortical mineralization is observed after parathyroidectomy.[2]

Declaration of patient consent

The authors certify that they have obtained all appropriate patient consent forms. In the form the patient (s) has/have given his/her/their consent for his/her/their images and other clinical information to be reported in the journal. The patients understand that their names and initials will not be published and due efforts will be made to conceal their identity, but anonymity cannot be guaranteed.

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Conflicts of interest

There are no conflicts of interest.

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