Fluorine-18-Fluorodeoxyglucose Positron Emission Tomography/Computed Tomography in a Giant Left Pectoral Muscle Plasmacytoma and Multiple Myeloma Case

Abstract

Extramedullary plasmacytoma is an unusual manifestation in multiple myeloma (MM). It can present as a solitary bone lesion and/or soft-tissue mass. Plasmacytoma can be presented at any location, but it is more common in the head and neck, usually without systemic involvement. The presence of plasmacytoma in MM is a predictor of rapidly progressive disease. The value of fluorine-18-fluorodeoxyglucose (FDG) positron emission tomography (PET)/computed tomography (PET-FDG) is increasing, in the diagnosis, detection of occult lesions, and therapeutic monitoring. We describe a patient with rapidly-progressive, refractory, left pectoral muscle plasmacytoma and MM. A PET-FDG guided the therapy and allowed to identify the presence of disease relapse.

Keywords: *Extramedullary plasmacytoma, fluorine-18-fluorodeoxyglucose positron emission tomography/computed tomography, intramuscular plasmacytoma, multiple myeloma, soft-tissue plasmacytoma*

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A 69-year-old male was admitted to our hospital referred from another center with the diagnosis of diffuse large B cell lymphoma. The patient complained of chest pain and a left hemithorax mass that grew rapidly for 1 month. Physical examination identified a large, solid, and painful mass at the left chest wall extending from the sternum to the axillary region, attached to deep planes.

A whole-body computed tomography (CT) (images not available) showed a presternal mass that extended into the left pectoral muscles and subcutaneous tissue associated with mediastinal and left axillary lymph nodes. A fluorine-18-fluorodeoxyglucose (18F-FDG) positron emission tomography/CT (PET/CT) (PET-FDG) was requested for staging [Figure 1a and b]. PET-FDG showed a giant soft-tissue mass that involved the left chest wall, extending down to the sternum and ribs (maximum standardized uptake value [SUVmax] 14.17), associated with multiple hypermetabolic mediastinal and axillary lymph nodes. In addition, hypermetabolic bone lesions in the spine, ribs, pelvis, and extremities were also observed.

In view of the PET/CT imaging findings, a core needle biopsy of the left pectoral muscle mass and bone marrow aspiration was done, revealing infiltration by plasmatic cells (52%). Immunohistochemical staining of the tumor cells was positive for CD138, CD38, and CD56 and negative for PAX-5, EMA, CD20, and CD30 [Figure 2].

Laboratory findings revealed decreased serum albumin 3.2 g/dl (normal range, 3.5–5.2 g/dl) and hemoglobin 9.7 g/dl (normal range, 13.5–17.5 g/dl) and increased lactic acid dehydrogenase 229 U/L (normal range, 105–215 U/L), beta 2 microglobulin 5.7 mg/L (normal range, 1.2–2.5 mg/L), serum IgG 6.3 g/dl (normal range, 0.7–1.5 g/dl) and kappa monoclonal protein 5.3 g/dl.

Electrophoresis analysis was positive for immunoglobulin free light chains Kappa and negative for Lambda. Ki67 proliferation index of the tumor cells was 90% and ALK gene rearrangement in tumor tissue was negative. Amplification at chromosome band 1q21 by fluorescence *in situ* hybridization (FISH) exhibited cytogenetic abnormalities with 1q21 gain,

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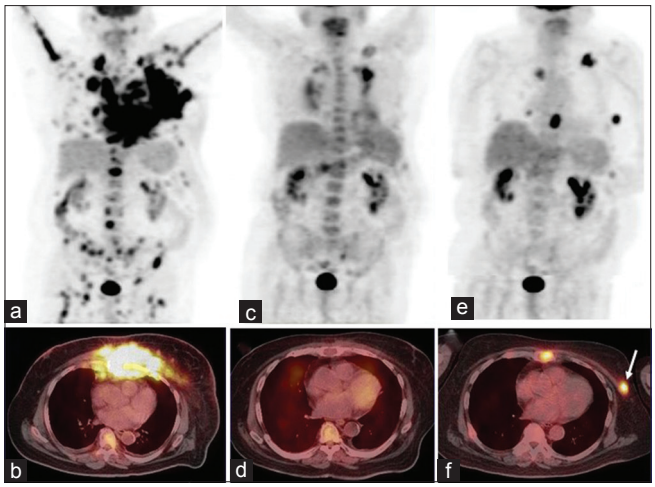

Figure 1: Maximum intensity projection images (above) and axial fluorine-18-fluorodeoxyglucose positron emission tomography/computed tomography fused slices (bottom). (a) At staging, images showed numberless hypermetabolic lesions in axial and appendicular bony, associated with mediastinal lymph node involvement and huge uptake of fluorodeoxyglucose in the left anterior thorax wall (maximum standardized uptake value 14.17). (b) Large tumor that involves the left pectoral region extending to the anterior chest wall and sternum. (c) An interim positron emission tomography-fluorodeoxyglucose demonstrated good metabolic response with persistence of low-intensity metabolic activity in clavicles, left costal arch, and left acetabulum. Increased metabolic activity in both lungs was related to pneumonitis by chemotherapy (Bortezomib). (d) Metabolic response of the left pectoral mass and sternum lesion (maximum standardized uptake value 0.88–1.10). (e) At the end of treatment, images showed early relapse, with reappearance of pathological metabolic activity in clavicles, bilateral costal arches, and sternum. (f) Focal lesion in the left pectoral muscle (arrow, maximum standardized uptake value 11.12). The relapse of soft-tissue plasmacytoma was confirmed by biopsy

an indicator of aggressive clinical course in multiple myeloma (MM).

With the diagnosis of MM IgG Kappa and left pectoral muscle plasmacytoma a CyBorD regimen was started. After the third cycle of treatment, an interim PET-FDG [Figure 1c and d] demonstrated good metabolic response (SUV_{max} between 0.88 and 1.10). Chemotherapy regime was completed. At the end of treatment, a PET-FDG showed a new hypermetabolic left pectoral mass (SUV_{max} 11.12) and focal hypermetabolic skeletal lesions [Figure 1e and f]. Large core needle biopsy confirmed the relapse of plasmacytoma in the left pectoral muscle.

Despite local radiotherapy and Len/Dex and VBMCP/VBAD chemotherapy regimens, the chest wall mass progressed with clinical worsening and severe infectious complications. The patient died 7 months after the last PET-FDG. Autopsy revealed cutaneous tumor dissemination in the thoracic and abdominal walls, visceral plasmacytomas, medullary aplasia, and signs of multiple organ failure.

The abnormal production of clonal plasma cells in the bone marrow causes as a result a MM, which represents about 10% of hematological cancers.[1] The classical clinical characteristics include hypercalcemia, renal failure, anemia,

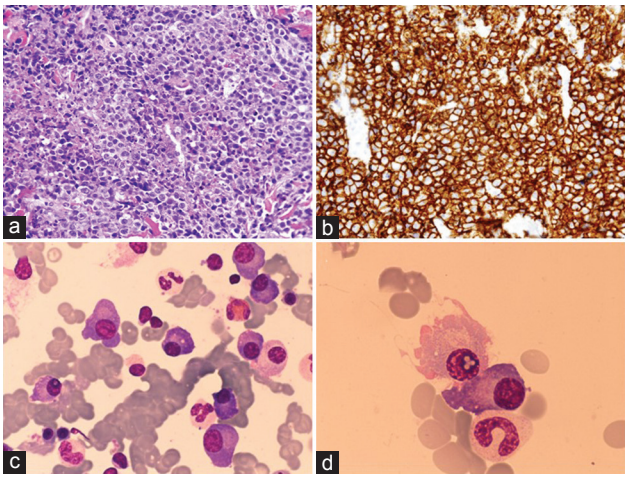

Figure 2: (a and b) Core needle biopsy of the left pectoral muscle mass. (a) Plasma cell infiltration with permeation and destruction of muscle fibers, H and E stain, original magnification x 300. (b) Tumor cells expressed CD138 on their cell membranes. Immunohistochemical staining, original magnification x 400. (c and d) Bone marrow aspirate. (c) Neoplastic plasma cell characterized by the presence of blue cytoplasm, perinuclear pale zone, and eccentric nucleus. (d) Intranuclear inclusions of immunoglobulin (Dutcher bodies)

and bone lesions. However, other forms of plasma cell neoplasia can be present, and signs and symptoms may be unlike that include plasma cell leukemia, bone solitary plasmacytoma, extramedullary plasmacytoma, and MM with extramedullary involvement.[2]

Extramedullary involvement is an uncommon presentation in MM, with an incidence of 2.2%–13% at the time of diagnosis.[2,3] Nevertheless, the evidence shows that the extramedullary MM is a factor of rapidly progressive disease and poor prognostic. The development of different laboratory techniques as gene rearrangement and FISH has provided valuable predictive information about progression of MM to aggressive disease. In our case, the presence of cytogenetic abnormalities with 1q21 gain was an indicator of worse course of disease, lower response rate to treatments, and opposite predictor of progression-free survival.

The role of PET-FDG in the evaluation of MM is increasing. It represents a relevant procedure for the diagnosis, showing greater sensitivity (80%–100%) than conventional radiological imaging.[4] In addition, PET-CT provides information about the presence of extramedullary disease, allowing the detection of lesions clinically not suspected. PET-FDG plays a role for the evaluation of the therapeutic response, the assessment of patients with minimal residual disease, and the diagnosis of early tumor relapse, especially in patients who are eligible to receive autologous stem-cell transplantation.[5] Its potential role as a predictor of progression of solitary extramedullary plasmacytoma to MM is under debate. Recently, Albano *et al*. have reported that avid PET-FDG plasmacytoma showing elevated SUV seemed to be correlated with a higher risk of transformation in MM.[6]

The case presented here shows the usefulness of PET-FDG for the diagnosis of medullary and extramedullary disease, the assessment of metabolic response to therapy, and the detection of early relapse that causes a change in the therapeutic approach. The morphofunctional assessment of the plasma cell disease plays a significant role as a predictor of progression, as in our case with fatal course.

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Conflicts of interest

There are no conflicts of interest.

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