

# Intracortical Bone Metastasis Mimicking Intracortical Osteoid Osteoma: A Case Report<sup>1</sup>

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Cortical metastasis usually occurs in the diaphysis of the long bones with the appearance of a cookie-bite pattern; this is associated with cortical destruction extending into the soft tissue as well as into the medullary cavity, or there can be a periosteal reaction. We report here on a 66-year-old woman who was diagnosed with intracortical metastasis in the proximal metaphysis of the right femur as an initial metastatic focus from primary lung cancer. CT detected an intracortical osteolytic lesion without cortical destruction or thickening. The MR images showed extensive peritumoral edema in the surrounding soft tissue and adjacent bone marrow edema, and this all mimicked osteoid osteoma.

**Index words :** Neoplasma metastasis  
Osteoma, osteoid  
Tomography

Skeletal metastases compose the majority of neoplastic processes involving bone. Most skeletal metastasis comes about from a hematogenous spread from an original tumor (1). The diagnosis of skeletal metastatic lesion is not generally difficult when multiple lesions are present and the clinical history is correlated with this. If the metastasis occurs in an unusual location as a single lesion, it is not easy to make a diagnosis. In some instances, skeletal metastases involve the cortex of long bones. Depending upon the size and exact location of the lesion, there may be either "endosteal" scalloping of the cortex from lesions seated in the medullary cavity, "subperiosteal" scalloping for periosteal metastases, or focal cortical defects from direct cortical metastases.

Even in the cases with focal cortical defects, the lesion usually settles in the diaphysis of a long bone and this shows focal cortical bone destruction with a poorly defined margin, which may suggest a metastatic lesion (2). We present here a case of the intracortical bone metastasis in the greater trochanteric area of the right femur as an initial metastatic focus, and this was associated with excessive peritumoral edema that mimicked intracortical benign bony lesion. We also discuss the diseases that can be included in the differential diagnosis of this malady.

## Case Report

A 66-year-old woman was admitted because of localized pain at the lateral site of the right hip; she had suffered with this pain for the previous 2 weeks. She had undergone right upper lobectomy for adenocarcinoma of lung 4 months ago and she had received three sessions of chemotherapy. The recent PET-CT scan done 1 month before showed no evidence of abnormal FDG

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uptake in the whole body after right upper lobectomy of the lung and chemotherapy. On physical examination she felt direct tenderness at the lateral site of the right hip and the pain worsened during external rotation and abduction. She fell down 1 week previously, but there was no presence of abnormality such as bruising, swelling or external wound at this area.

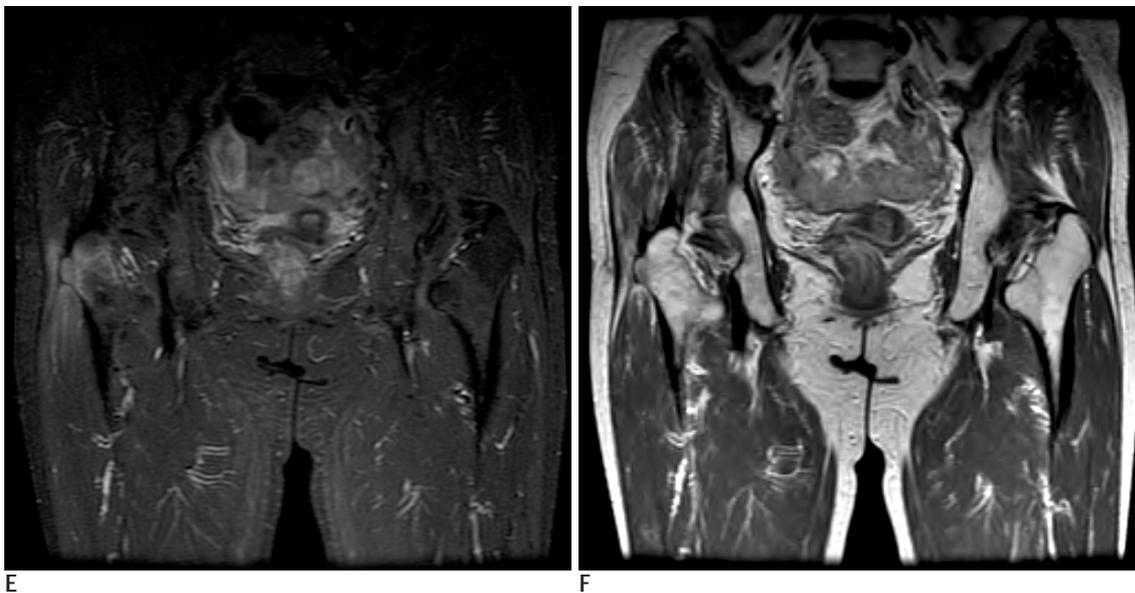
Plain radiograph showed an oval shaped, geographic osteolytic lesion without a sclerotic rim in the cortex of the greater trochanteric area of the right femur (Fig. 1A). On CT scan this lesion was localized in the cortex as a

well-defined osteolytic lesion with a preserved inner and outer shell. There was no cortical thickening or periosteal bone formation (Fig. 1B).

On MR image, this lesion was hypointense on the T1-weighted image and intermediately intense on the T2-weighted image. The fat suppressed T2-weighted image showed diffuse peritumoral edema in the surrounding soft tissue and the adjacent bone marrow, which was well enhanced on the contrast-enhanced image and the lesion was enhanced as well (Fig. 1C - F). An incisional biopsy and simple curettage were performed. Pathologic

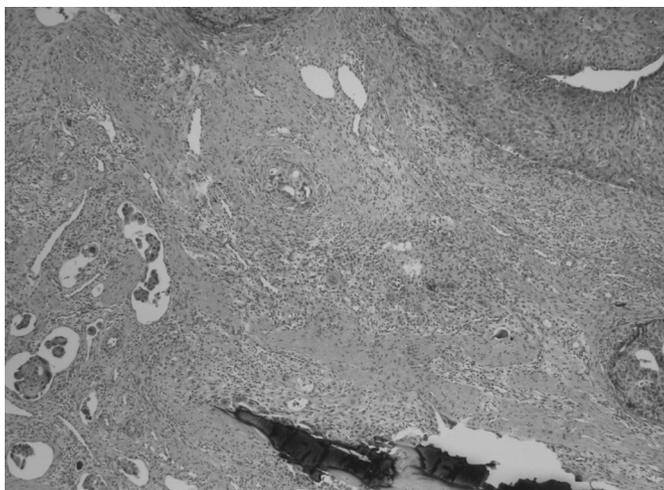


Fig. 1. A. The radiograph shows a well-demarcated, geographic osteolytic lesion in the cortex of the greater trochanteric area of the right femur.  
B. The axial CT scan shows an ovoid osteolytic lesion in the cortex with a preserved outer shell. There is no sign of definite cortical thickening or periosteal reaction.  
C. The coronal T1-weighted spin echo image shows an ovoid, hypointense mass in the cortex.  
D. The axial T2-weighted spin echo image shows intermediate intensity in the lesion with a low intensity rim.



E

F



G

**Fig. 1.** E. The fat suppressed coronal T2-weighted image shows peritumoral edema in the surrounding soft tissue and bone marrow. F. The contrast enhanced coronal T1-weighted image shows homogeneous enhancement in the lesion and in the surrounding bone marrow and soft tissue. G. The pathologic specimen shows tumor emboli in the lymphatics that have invaded into the cortical bone (H & E stain,  $\times 40$ ).

examination confirmed a poorly differentiated metastatic adenocarcinoma. Tumor emboli were caught in the lymphatic vessels adjacent the periosteum, and these had invaded into the cortex of the right femur (Fig. 1G). Two month after the operation for the bony lesion, there were newly developed multiple metastatic lymph nodes in the mediastinum and metastatic nodules in the lung and the liver on the follow-up CT scan.

### Discussion

Skeletal metastases are much more common than primary malignant tumor of bone and they usually show multiple lesions with an osteolytic, osteoblastic or mixed pattern (3). The majority of metastases to the long bone are located in the medullary cavity and they less commonly occur in the cortex. The distribution of cortical

metastases is usually in the diaphyses of the long bone as a result of tumor emboli being caught in the vascular networks of the cortex when tumor emboli are disseminated through the systemic artery (2, 4). Contrary to this, the medullary metastases are reported to have a distinct predilection for the proximal portions of the long bones. This may be because cortical metastases are more dependent on the cortical dimensions and blood flow, attaining a maximum in the midshaft, while medullary metastases are more dependent on the presence of red bone marrow (3, 4). Our case involved the cortex of the greater trochanteric area, and this was not a common manifestation of cortical metastasis. We speculated that this happened because the tumor emboli were not embedded in the vessels, but in the lymphatics. Anatomically, the deep inguinal lymph nodes are located near the termination of the saphenous vein. All

the lymph from the limb filters through them before reaching the iliac lymph nodes. The trapped tumor emboli in the lymphatics might have grown into the adjacent cortex of the femoral trochanter.

The cortical osteolytic metastatic lesions are categorized into 4 types; metastatic deposits confined to the cortex (type I), extension into the soft tissue mass (type II), extension into the marrow cavity (type III), and predominant cortical destruction with extension into the subperiosteum (type IV) (4). Even though the metastasis is confined to the cortex, it is usually shown as an ill-defined lesion or cortical destruction on radiographic images, suggesting malignant tumor. Our case demonstrated solitary geographic osteolytic bony lesion without periosteal bone formation, which was confined to the cortex and it had a preserved outer cortical shell on CT scan. The MR images revealed extensively peritumoral edema in the adjacent bone marrow and surrounding soft tissue. The differential diagnosis includes intracortical lesions as follows: benign lesions such as osteoid osteoma, fibrous cortical defects, Brodie abscess and aneurysmal bone cyst, and malignant lesions such as adamantinoma and intracortical osteosarcoma. In our case, intracortical lesion was associated with peritumoral edema, so the differential diseases are confined to several diseases. Osteoid osteoma in the long tubular bone is typically observed in the cortex and it appears as a radiolucent lesion that represents the nidus, and this usually has internal calcifications. The nidus is composed of vascular structures and it is well enhanced on the contrast-enhanced image. This tumor is usually associated with cortical thickening and edematous change in the surrounding bone marrow and soft tissue in the vicinity of the nidus on MR imaging (5, 6). Contrary to this, our case did not show reactive cortical thickening or internal calcification. Brodie's abscess is a form of subacute pyogenic osteomyelitis. The clinical features are the most important factor in making the diagnosis of Brodie's abscess, but the laboratory data may sometimes be within normal limits. Brodie's abscess appears as a sharply delineated, localized radiolucency in the cortex, and this is usually a metaphyseal lesion that's occasionally associated with bone marrow edema. Brodie's abscess usually has internal non-enhancement due to bone necrosis and pus, and it is associated with

the cortical thickening (3, 7). In contrast to this, our case showed the homogeneous enhancement and no cortical thickening. Even though it is extremely rare, osteosarcoma may develop as an intracortical tumor. For patients with intracortical osteosarcoma, the mean age at presentation is 22 years (age range: 10 - 43 years) and the predilection site is in the thickened cortex of the mid-shaft of the long bones, as compared to our case with an old age patient and a metaphyseal location (8). Radiologically, intracortical osteosarcoma appears as a localized intracortical lucency surrounded by a thickened cortex, which may extend into the surrounding soft tissue and it sometimes reveals tiny calcification in the adjacent soft tissue on CT (9, 10). Our case did not reveal any cortical thickening or tiny calcification in the overlying soft tissue on CT, and these features are usually displayed by intracortical osteosarcoma.

In conclusion, when intracortical metastasis occurs as a single initial focus that appears as a benign lesion in an unusual greater trochanteric area of the femur, and there is associated extensive peritumoral edema, the differential diagnosis should include skeletal metastasis from the original malignant tumor.

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