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Two foci of FDG-avid secondary tumoral calcinosis incidentally noted in a patient with small-cell lung carcinoma after PET/CT

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This case report describes intense F-18 fluorodeoxyglucose (FDG) uptake within two foci of secondary tumoral calcinosis, incidentally noted during the workup of small-cell lung cancer. The patient had insulin-dependent diabetes mellitus and secondary hyperparathyroidism as a result of IgA nephropathy.

Case report

A 48-year-old male had an extensive past medical history that included insulin-dependent diabetes mellitus, hypertension, and coronary artery disease with previous myocardial infarction, congestive heart failure, and stage IV chronic renal failure secondary to IgA nephropathy. He presented to the emergency department with a comminuted fracture of his left proximal humerus after falling at his workplace (Fig. 1). Due to a complication of anemia, the fracture was initially treated nonoperatively with a coaptation splint. Although the plan was for internal fixation, his multiple comorbidities resulted in repeated hospitalizations that delayed the surgery. He was subsequently diagnosed with secondary hyperparathyroidism as a result of IgA nephropathy. The patient was reluctant to start hemodialysis despite the advice of his nephrologist. The humeral fracture went on to nonunion.

Four years after his initial injury, the patient presented to his primary care physician with a firm left shoulder mass as well as a similar mass in his right wrist. Radiographs demonstrated amorphous calcifications at the extensor surface of his right wrist and in the soft tissues adjacent to the left shoulder (Fig. 2). The shoulder radiograph also demonstrated the appearance of permeative destruction of the proximal humeral diaphysis at the previous nonunion fracture site. Further workup of this lesion was recommended to exclude malignancy. He was referred to an orthopedic oncologist, who ordered magnetic resonance imaging (MRI) of the shoulder (Fig. 3). The MRI demonstrated extensive metastatic calcification and a large joint effusion.

One month after the shoulder MRI, the patient underwent computed tomography (CT) of the chest for symptoms of pneumonia. The CT revealed a left hilar mass,

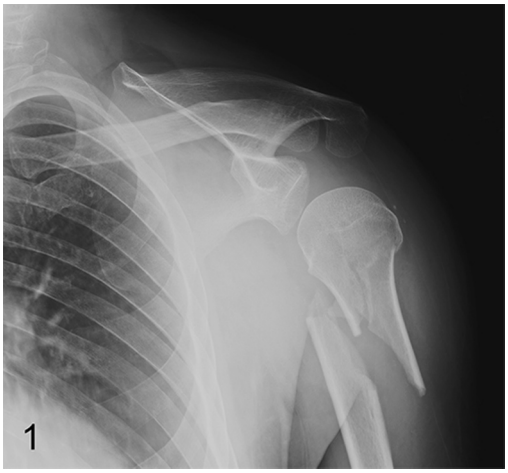

Figure 1. AP radiograph of the left shoulder demonstrates a comminuted fracture of the left proximal humerus with medial displacement. This was the patient's original injury.

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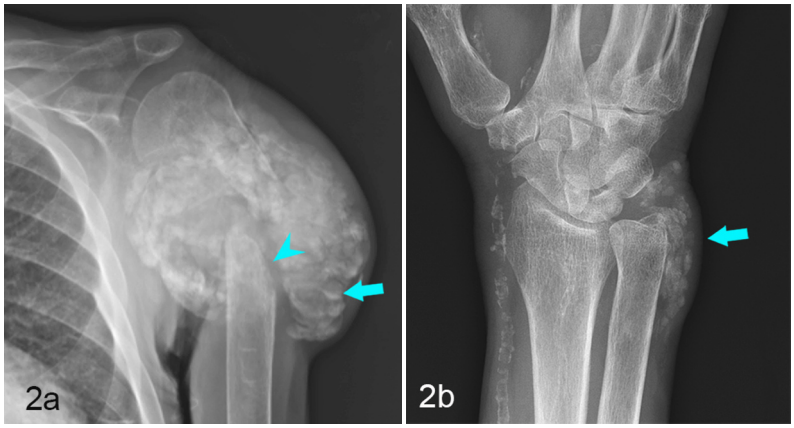

Figure 2. Four years following the initial injury, the patient presented to his primary care clinician with complaints of a firm left shoulder mass. (A) AP radiograph shows large amorphous calcification surrounding the fracture nonunion of the left proximal humerus. Notice the layering fluid level (arrow). The worrisome finding was the appearance of permeative destruction of the proximal humeral diaphysis (arrowhead). (B) AP wrist radiograph reveals amorphous calcification along the distal ulna (arrow) and extensive vascular calcifications.

which was biopsied and found to be small-cell lung carcinoma. During the workup for this malignancy, PET/CT revealed intense F-18 fluorodeoxyglucose (FDG) uptake within the extensive calcification surrounding the fracture nonunion of the left shoulder (Figs. 4A-C). The CT (Fig. 4B) also revealed an amorphous, cystic, and multilobulated calcified mass at the shoulder, which was determined to be a focus of secondary tumoral calcinosis related to the patient's chronic renal disease. In addition, an FDG-avid focus of tumoral calcinosis was noted adjacent to the right lesser trochanter (Figs. 4D, E).

Discussion

Few cases in the literature describe tumoral calcinosis with FDG avidity (1). In our case, the patient was undergoing workup for a primary lung cancer, which included a PET/CT examination, which in turn demonstrated intense metabolic activity of not only his primary lung neoplasm but also the secondary tumoral calcinosis of his left shoulder and right hip.

Primary tumoral calcinosis (also known as Teutschlaender disease in the European literature [2]) is a familial condition with multiple painless periarticular foci of amorphous calcification. A secondary (or metastatic), tumoral, calcinosis-like condition is seen in patients with chronic renal failure on hemodialysis. This is most likely due to secondary hypoparathyroidism with vitamin D deficiency. The diseased kidneys cannot effectively hydroxylate 25-hydroxyvitamin D to 1-25 dihydroxyvitamin D (cacitriol), which is the active form of vitamin D in the body. This results in hypocalcemia and increased release of parathyroid hormone, thereby increasing circulating serum calcium. This increase in calcium, along with the kidney's

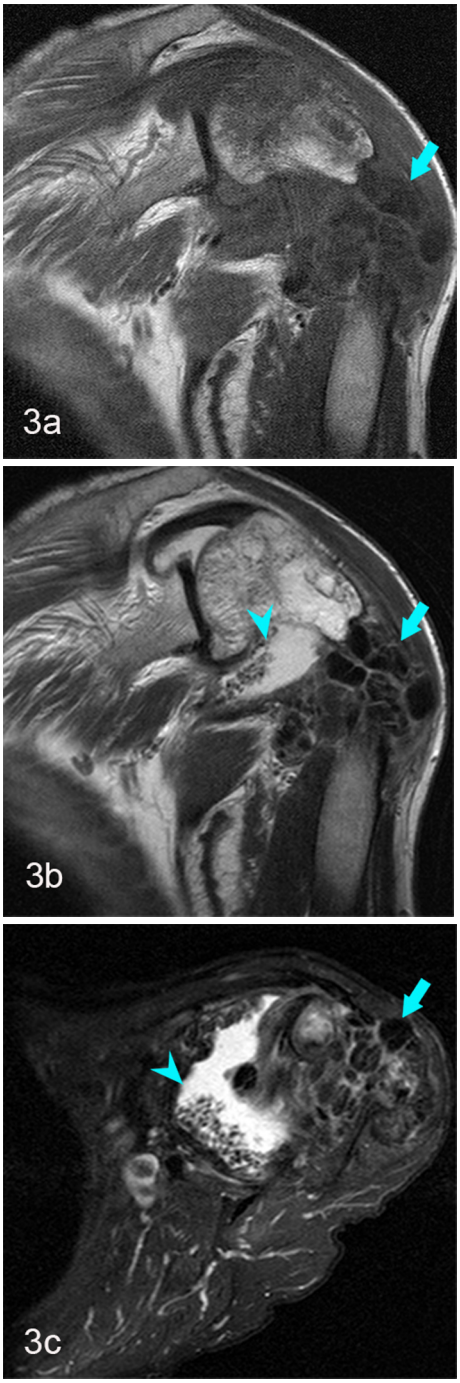

Figure 3. (A) Coronal T1-weighted (TR533/TE17), (B) coronal T2-weighted (TR2500/TE70), and (C) axial T2-weighted with fat saturation (TR3186/TE70) sequences (b) of the left shoulder demonstrate a large mass with low T1 and T2 signal (arrows) corresponding to mineralized areas on the prior radiograph. A large joint effusion with proliferative synovitis is also present (arrowheads).

inability to excrete a normal load of phosphate, causes an elevation in this calcium-phosphate product that then ac-

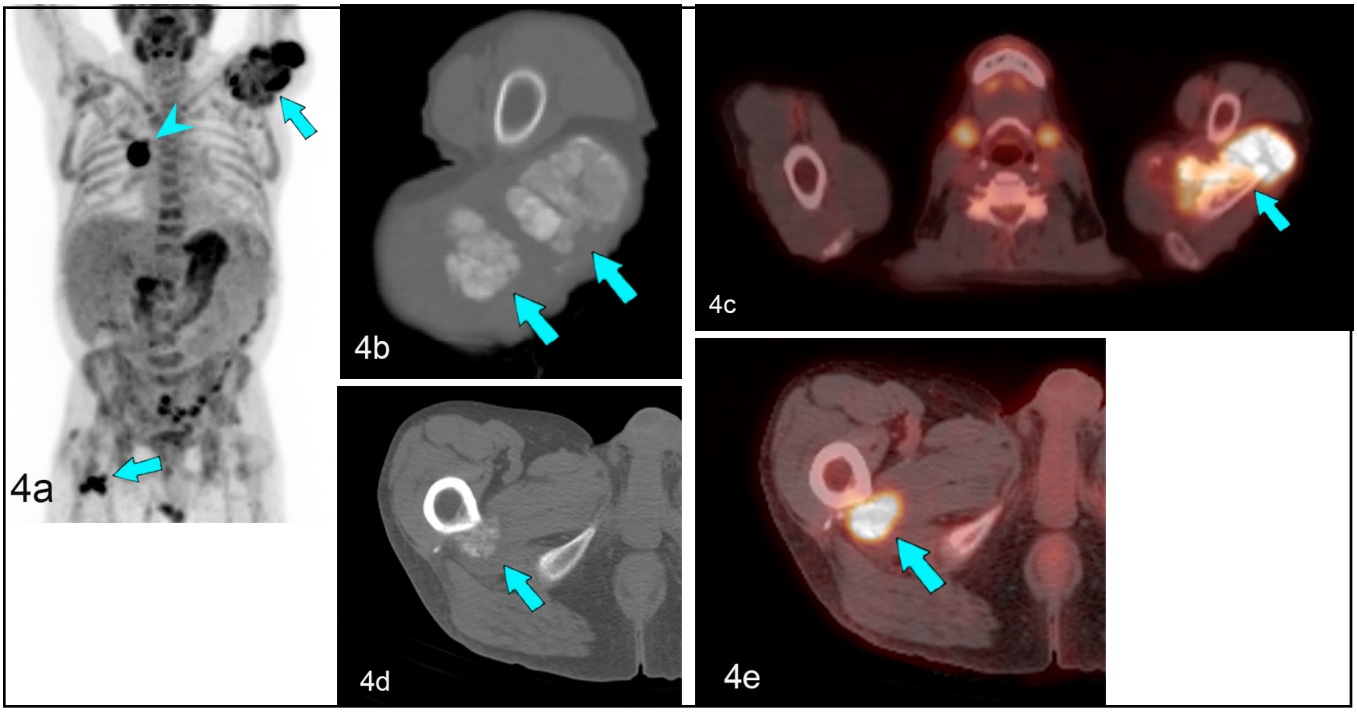

Figure 4. (A) Anterior projection from the PET maximum-intensity projection (MIP) image reveals intense FDG avidity of the mineralized foci at the left shoulder and right hip (arrows). Also present is a right hilar small-cell lung carcinoma (arrowhead). (B) Axial CT image demonstrates the amorphous calcifications at the left shoulder distal to the fracture nonunion. (C) Axial fused image from an F18-FDG PET CT demonstrates avid FDG uptake within the mineralized component. Axial CT (D) and fused image (E) at the right hip shos a mineralized focus at the right lesser trochanter with intense FDG avidity (arrows).

cumulates in soft tissues (3). Theories of pathogenesis include lesions occurring in areas of repetitive trauma, leading to a dysfunction in repair mechanisms around joints (4). Both the primary and secondary variations of tumoral calcinosis result in a radiologically similar condition. The most common locations of tumoral calcinosis are the hip, elbow, shoulder, foot, and wrist (4).

Classical radiographic findings of tumoral calcinosis are a deposition of amorphous, lobulated calcification in a periarticular distribution, often along the extensor surfaces of tendons (5). CT better delineates the calcific mass and shows either solid calcifications or cystic, rim-calcified structures with fluid-fluid levels with layering calcium precipitate. On T1-weighted MRI sequences, tumoral calcinosis is of heterogeneously low signal intensity. On T2-weighted sequences, two patterns have been described: a diffuse low-signal pattern and a nodular high-signal pattern (4). Positive radiotracer uptake during bone scintigraphy has been well documented with technetium-labeled diphosphonates (most often methylene diphosphonate) in these periarticular lesions (6). Reports of metabolic activity of tumoral calcinosis using PET are scarce in the literature. Okuyama et al described a case of idiopathic tumoral calcinosis demonstrating intense F-18 fluorodeoxyglucose (FDG) accumulation (1).

During PET, administered F-18 fluorodeoxyglucose is transported across the cell membrane, along with glucose, via glucose transporters. Glucose transporters are highly expressed in the brain, tumor cells, and other metabolically active tissues. Hexokinases then phosphorylate the glucose to glucose 6-phosphate; however, this step is blocked with FDG, and the product remains "trapped" in the cell, allowing ample time for imaging (6). This case report demonstrates the high metabolic activity of secondary tumoral calcinosis evidenced by intense uptake of FDG glucose within the lesion.

Initial treatment of secondary tumoral calcinosis includes administration of phosphate binders, dietary changes, and optimization of dialysis. If initial treatment fails, subtotal parathyroidectomy can be performed. Generally, surgical resection of the tumoral calcinosis is performed as a last resort. There is significant recurrence if the underlying metabolic imbalances are not corrected (7).

To our knowledge, there have been few documented cases of secondary tumoral calcinosis that exhibit FDG avidity on PET studies. This is most likely due to the distinctive appearance of tumoral calcinosis on radiography and CT, which negates the need for further evaluation. Nevertheless, tumoral calcinosis is an important condition to consider in the differential diagnosis for an incidental FDG-avid lesion on PET performed for other reasons.

References

1. Okuyama C, Kubota T, Matsushima S, Ushijima Y, Nishimura T. Intense F-18 FDG accumulation in idiopathic tumoral calcinosis. *Clin Nucl Med*. 2009;34:230–2. [PubMed]
2. Barrière H, Welin J, Lenne Y, Visset J. *[Teutschlaender lipo-calcino-granulomatosis or tumoral calcinosis of Inclan (author's transl)]. … de dermatologie et de …*. 1977. [PubMed]
3. Petscavage JM, Richardson ML. Tumoral calcinosis mimicking recurrent osteosarcoma. *Radiology Case Reports*. 2009;4:1–4. DOI: http://dx.doi.org/10.2484/rcr.v4i4.336
4. Olsen KM, Chew FS. Tumoral calcinosis: pearls, polemics, and alternative possibilities. *Radiographics*. 2006;26:871–85. [PubMed]
5. Martinez S, Vogler JB, Harrelson JM, Lyles KW. Imaging of tumoral calcinosis: new observations. *Radiology*. 1990;174:215–22. [PubMed]
6. Mettler FA, Guiberteau MJ. *Essentials of nuclear medicine imaging*. Elsevier Health Sciences; 2012.
7. Möckel G, Buttgereit F, Labs K, Perka C. Tumoral calcinosis revisited: pathophysiology and treatment. *Rheumatol Int*. 2003;25:55–9. [PubMed]