

Case report

Bone Scintigraphy Reveals Pulmonary Microcalcification Secondary to Coronary Stent Thrombosis

Sang-Hyeong Kil

Department of Nuclear Medicine, Pusan National University Yangsan Hospital, Yangsan, Korea

***Corresponding Author:** Sang- Hyeong Kil, Department of Nuclear Medicine, Pusan National University Yangsan Hospital, 20, Geumo-ro, Mulgeum-eup, Yangsan-si, Gyeongsangnam-do, 50612, Republic of Korea, Tel: +82-55-360-1901, E-mail: rlf1007@naver.com

ABSTRACT

Purpose: This case report describes an unusual presentation of diffuse pulmonary uptake of Tc-99m dicarboxypropane diphosphonate (Tc-99m DPD), attributed to pulmonary microcalcification resulting from coronary stent thrombosis. **Materials and Methods:** A 64-year-old man with hypertension, hyperlipidemia, and a previous drug-eluting coronary stent placement underwent Tc-99m DPD bone scintigraphy for suspected avascular necrosis of the femoral head. **Results:** Bone scintigraphy revealed focal uptake in the femoral head, indicative of avascular necrosis, as well as unexpected diffuse pulmonary uptake of Tc-99m DPD. These findings were interpreted as evidence of pulmonary microcalcification secondary to coronary stent thrombosis, likely due to microembolic vascular inflammation and subsequent calcium deposition. **Conclusion:** This case highlights the diagnostic utility of detecting extraosseous radiotracer uptake and emphasizes the need to correlate nuclear imaging results with cardiovascular history to identify systemic disease manifestations.

Key words: Bone Scintigraphy, Pulmonary Microcalcification, Coronary Stent Thrombosis, Tc-99m DPD, Extraosseous Radiotracer Uptake

Introduction

Technetium-labeled phosphate bone scintigraphy is a well-established imaging modality for detecting skeletal pathologies, as it reflects osteoblastic activity and regional blood flow through radiotracer binding to hydroxyapatite crystals. Although primarily intended for osseous imaging, bone scintigraphy can occasionally reveal non-skeletal radiotracer uptake, which may offer crucial diagnostic clues[1–4].

Diffuse pulmonary accumulation of bone-seeking radiopharmaceuticals has been documented in conditions such as metastatic pulmonary calcification, chronic kidney disease, and parathyroid abnormalities[5].

In rare instances, such pulmonary uptake may also be linked to cardiovascular procedures, including percutaneous coronary interventions (PCI), where stent-associated microemboli can incite vascular inflammation and calcification[6, 7].

This report describes a case of diffuse pulmonary uptake of Tc-99m DPD in a patient undergoing scintigraphy for suspected avascular necrosis, with findings suggestive of pulmonary microcalcification secondary to coronary stent thrombosis.

Case report

A 64-year-old man with a history of cardiovascular comorbidities, including hypertension and hyperlipidemia, underwent Tc-99m DPD bone scintigraphy for evaluation of right hip pain. Imaging was conducted 2 hours after intravenous administration of 740 MBq of Tc-99m DPD, using a dual-head gamma camera (Symbia E; Siemens, Germany). A complementary pelvic MRI (MAGNETOM Avanto 1.5T; Siemens, Germany), enhanced with gadoterate meglumine (Dotarem®; Guerbet, France), was additionally performed.

The scintigraphy revealed focally increased radiotracer uptake in the right femoral head, hip, and sacroiliac joint, suggestive of avascular necrosis with post-traumatic changes. Moreover, diffuse pulmonary uptake of Tc-99m DPD was observed, which was an unexpected finding. MRI confirmed bilateral avascular necrosis, accompanied by bone marrow edema and synovitis in the right hip (Fig. 1). Chest radiography was unremarkable. The patient remained asymptomatic, without evidence of renal, parathyroid, or vitamin D dysfunction.

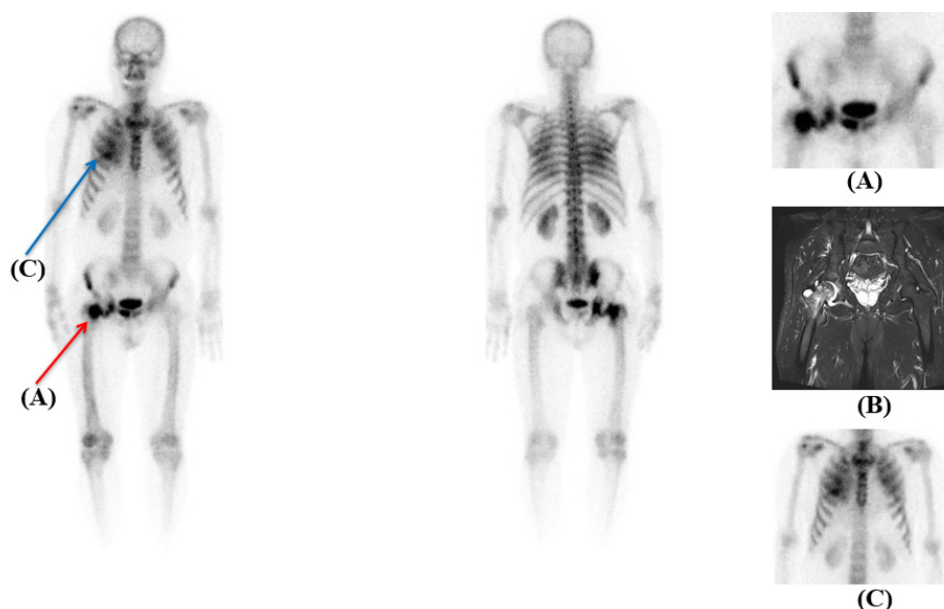


Fig. 1. (A) Bone scintigraphy performed 2 hours after intravenous injection of Tc-99m DPD shows increased uptake in the right femoral head, hip, and sacroiliac joint. (B) Pelvic MR images reveal geographic low signal areas in both femoral heads consistent with avascular necrosis, along with bone marrow edema and synovitis in the right hip joint. (C) Notably, diffuse pulmonary uptake of Tc-99m DPD is also observed, suggestive of pulmonary microcalcification.

Based on the patient's recent cardiovascular history and absence of pulmonary disease, the diffuse pulmonary uptake was interpreted as microcalcification secondary to microemboli from coronary stent thrombosis. These emboli likely caused microvascular obstruction and localized calcium deposition, leading to abnormal tracer accumulation[6-9].

Discussion

Pulmonary accumulation of bone-seeking radiopharmaceuticals is uncommon and typically indicative of underlying pathological calcification. Conditions such as renal failure, hyperparathyroidism, and widespread metastatic calcification have been linked to such findings[5]. Pulmonary accumulation of bone-seeking radiopharmaceuticals is uncommon and typically indicative of underlying pathological calcification.

Although drug-eluting stents are effective in reducing restenosis, they may paradoxically increase thrombogenic potential. Resulting microthrombi can lodge in the pulmonary microvasculature, leading to endothelial injury, macrophage recruitment, and cytokine release (e.g., TNF- α , IL-1). These processes, in turn, promote matrix remodeling and ectopic calcium deposition[4, 6, 7, 9]. Given that Tc-99m DPD exhibits high affinity for calcium salts and hydroxyapatite, it effectively highlights these microcalcifications on scintigraphic imaging[3].

Previous reports have similarly identified diffuse pulmonary tracer uptake in patients with cardiovascular or renal comorbidities, reinforcing the broader diagnostic applicability of bone scintigraphy beyond skeletal imaging. Our findings align with this literature, providing further evidence that non-osseous uptake patterns can reveal otherwise occult systemic conditions.

In cases of pulmonary microcalcification, radiotracers accumulate by binding to microscopic calcium deposits, rendering them detectable on whole-body bone scans[1,3]. Technetium-labeled agents have demonstrated utility in detecting microembolic events via SPECT imaging, further validating nuclear medicine's role in visualizing microvascular pathology[6,7]. Studies have also shown a correlation between systemic vascular calcification and abnormal radiotracer distribution, particularly among individuals with cardiovascular or renal disease[9]. Pulmonary uptake of bone-seeking agents has also been reported in acute respiratory conditions, presenting imaging patterns analogous to those observed in this case[8]. Taken together, these findings support the interpretation that the diffuse pulmonary uptake observed in this patient may result from ongoing microembolic calcification secondary to coronary stent thrombosis.

This case highlights the diagnostic utility of bone scintigraphy in detecting systemic pathology that extends beyond the skeletal system. The observation of diffuse pulmonary Tc-99m DPD uptake—likely secondary to coronary stent thrombosis—emphasizes the importance of contextualizing nuclear imaging findings within the patient's cardiovascular and interventional history. A multidisciplinary approach that integrates nuclear imaging with clinical evaluation is essential for accurate diagnosis and management of such complex cases.

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