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Schmorl's Nodes can cause increased ^{68}Ga -DOTA-TATE activity on PET/CT, mimicking metastasis in patients with neuroendocrine malignancy

Georgios Z. Papadakis, MD^a, Corina Millo, MD^b, Ulas Bagci, PhD^c, Samira M. Sadowski, MD^d, and Constatntine A. Stratakis, MD, D(med)Sci^e

^aRadiology and Imaging Sciences, Warren Grant Magnuson Clinical Center (CC), National Institutes of Health (NIH), Bethesda, MD 20892, USA

^bDivision of Nuclear Medicine, RAD&IS; Clinical Center, National Institutes of Health (NIH), Bethesda, MD20892,USA

^cCenter for Research in Computer Vision (CRCV), Electrical and Computer Science Department, University of Central Florida (UCF), Orlando, FL 32816, USA

^dEndocrine Oncology Branch, National Cancer Institute, National Institutes of Health, Bethesda, MD 20892, USA

^eSection on Endocrinology and Genetics, Program on Developmental Endocrinology & Genetics, Eunice Kennedy Shriver National Institute of Child Health and Human Development, National Institutes of Health (NIH), Bethesda, MD 20892,USA

Abstract

Schmorl's node (SN) is the herniation of the nucleus pulposus (NP) through the cartilaginous and bony endplate into the adjacent vertebral body. It is documented that SNs produce areas of moderately increased ^{18}F -FDG uptake. We present a case of a patient with history of neuroendocrine tumor (NET), who underwent ^{68}Ga -DOTA-TATE- PET/CT for follow-up, showing increased focal vertebral uptake suggestive of bone metastasis. Computed tomography (CT) revealed typical findings of a SN. The presented case indicates that SNs should be considered when encountering focally increased skeletal uptake in ^{68}Ga -DOTA-TATE-PET/CT studies, which can mimic metastasis in patients with history of NETs.

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Address for correspondence: Constatntine A. Stratakis, MD, D(med)Sci, Scientific Director, Eunice Kennedy Shriver National Institute of Child Health and Human Development (NICHD), Chief Section on Endocrinology & Genetics (SEGEN), PDEGEN, NICHD, NIH, Building 10, CRC, Room 1-3330, 10 Center Dr., MSC1103, Bethesda, Maryland 20892, tel 301-496-4686/496-6683, fax 301-402-0574/480-0378, stratakc@mail.nih.gov.

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A 73 year-old man with history of recurrent pancreatic NET underwent ^{68}Ga -DOTA-TATE PET/CT study for follow-up. PET images showed focal increased ^{68}Ga -DOTA-TATE uptake (SUVmax: 6.39) at the level of T7 vertebral body (**A** sagittal, **B** axial ^{68}Ga -DOTA-TATE fused PET/CT images), raising the suspicion of skeletal metastasis. CT bone window images revealed a radiolucent lesion (**C** sagittal, **D** axial CT images) in the body of T7 vertebra surrounded by osteosclerotic bone, in an otherwise intact vertebra. The adjacent endplate was disrupted with protrusion of disc material into the vertebral body, characteristic of a SN. Consecutive spine CT scans were evaluated over a long period, showing stability of the lesion and no evidence of any other pathological process in the T7 vertebra. SNs represent herniations of the NP of the intervertebral disc into the adjacent cartilaginous

endplate of the vertebral body [1,2]. There is no consensus regarding the pathogenesis of SNs. They are either considered as developmental defects due the abnormal persistence of vascular channels within the intervertebral disc which result to weakening of the vertebral endplate, or SNs are perceived as the secondary result of subchondral osteonecrosis related to metabolic (osteoporosis, hyperparathyroidism), neoplastic, and degenerative disc disease or traumatic injury. Histological examination of *en-bloc* slices through Schmorl's nodes has shown ischemic necrosis beneath the cartilaginous endplate, supporting the theory that SNs are secondary to osteonecrosis with disc components migrating into vertebral necrotic areas [1-3]. The reported in the literature prevalence of SNs in the general population ranges widely from 2 to 76%, with multiple lesions commonly found in the same individual [4]. Like in the presented case, it has been demonstrated that SNs appear more frequently in the T7-L1 region, and most often (62.3%) affect the inferior surface of the vertebral body [5]. The majority of SNs are asymptomatic, however in certain cases can be responsible for low back pain especially in the presence of inflammatory changes and edema in the bone marrow [6]. SNs most commonly don't show increased 18F-FDG activity. However, it is reported that SNs can occasionally produce areas of moderately increased 18F-FDG uptake, which is attributed to the inflammatory response induced by the protrusion of NP into the well vascularized vertebral body and the contact of intraspongious disc material with the bone marrow [7,8]. 68Ga-DOTA-TATE is a radiolabelled analogue of somatostatin (SST) suitable for PET imaging with wide application in the diagnostic work-up and follow-up of patients with NETs, since somatostatin receptors (SSTRs) are over-expressed by the majority of these tumors [9-11]. Furthermore, it has been documented that SSTRs are expressed and upregulated on white blood cells including leukocytes and macrophages, suggesting the potential role of PET/CT using 68Ga-DOTA-conjugated peptides (SST analogues) for imaging of inflammatory processes [12-15]. The presented case indicates that inflammatory changes induced by the formation of a SN, can result to increased 68DOTA-TATE activity which has a predominant affinity for SSTR subtype 2, and should not be erroneously interpreted as sites of bone metastasis.