

Andersson's lesion in ankylosing spondylitis diagnosed by ^{18}F -FDG PET/CT

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Abstract

Ankylosing spondylitis (AS) is a form of arthritis that affects the spine, the sacroiliac joints and sometimes the hips and shoulders. Andersson lesion (AL) was first described by Andersson in 1937 as a destructive vertebral body and discovertebral portion of the spine in AS. In this case, we report a 50 years old man with medical history of AS diagnosed AL by fluorine-18-fluorodeoxyglucose positron emission tomography/computed tomography (^{18}F -FDG PET/CT).

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Introduction

The prevalence of ankylosing spondylitis (AS) is generally believed to be between 0.1% and 1.4% globally [1]. The diverse inflammatory changes of the spine in patients with AS include spondylitis, spondylarthritis and Andersson lesions (AL) [2, 3]. Because of the lack of proper diagnostic criteria and the differences in the extent of spinal survey undertaken, the reported prevalence of AL varied dramatically from 1.5% to 28% [4, 5]. Both early and late technetium-99m methylene diphosphonate ($^{99\text{m}}\text{Tc}$ -MDP) bone scintigraphy can be used to identify the AL complicating AS and to differ the lesion from the infection. Focal areas of increased isotope retention in late state of AS may identify the AL [6-8]. Single photon emission tomography/computed tomography (SPET/CT) showed an extraordinary large vacuum on intervertebral disc space with end plate sclerosis and erosion, but no scintigraphic abnormality. Instead, increased tracer uptake was shown at the facet joint, the lamina and the heterotopic facet ossification [9]. In this case, we also imaged AL by fluorine-18-fluorodeoxyglucose positron emission tomography/computed tomography (^{18}F -FDG PET/CT).

Case Report and Discussion

A 50 years old man was presented with numbness in both legs for one month. His medical history was AS. He didn't have any history of malignant tumor, tuberculosis and bone or vertebral trauma or surgery. Tumor marker tests for α -fetoprotein, carcinoembryonic antigen, carbohydrate antigen 199, carbohydrate antigen 125, carbohydrate antigen 153, neuron-specific enolase, cytokeratin 19 fragment and squamous cell carcinoma-related antigen were within normal range. Antinuclear antibody and tuberculosis screening test (T-SPOT.TB) were negative. Fluorine-18-FDG PET/CT images (Figure 1) showed "bamboo-like changes" in the spine, narrowing of the intervertebral space, vertebral facet and sacroiliac joints well shown and partial vertebral fusion with abnormal ^{18}F -FDG uptake [maximum standardized uptake value (SUVmax):5.6]. There was an extraordinary large soft tissue in the T10-T11 intervertebral disc space with end plate sclerosis and erosion, showing increased ^{18}F -FDG uptake (SUVmax: 3.8). The patient underwent CT-guided percutaneous transpedicular biopsy of T10 and T11. Based on medical

history, imaging and pathological findings, the diagnose of AL was made.

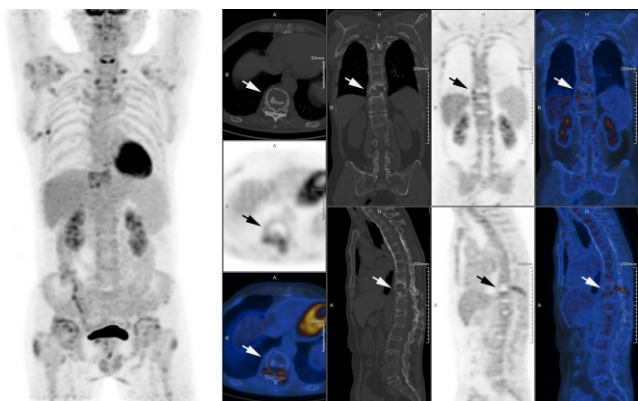


Figure 1. Fluorine-18-FDG PET/CT images show "bamboo-like changes" in the spine, narrowing of the intervertebral space, vertebral facet and sacroiliac joints and partial fusion with abnormal ^{18}F -FDG uptake (SUVmax:5.6). The lesion in the T10-T11 (arrow) was diagnosed as AL.

Usually AL involves only one single spinal level and most common affected site is thoracolumbar junction. Diagnosis of AL is some times difficult because of the preexisting spinal changes, osteoporosis, its often radiographic resemblance to infective spondylodiscitis and is often misdiagnosed as tuberculosis [10]. The role of imaging in the diagnosis, management and follow-up of patients with AS is important. The main imaging modalities available are conventional radiography, CT, ultrasound, nuclear medicine tests including PET and magnetic resonance imaging [11]. Andersson's lesion is morphologically different from Schmorl's nodes [12]. The AS activity is reflected by bone formation rather than inflammation, since the bone tracer ^{18}F -fluoride was seen in these patients with high correlation to structural bony changes in the uptake areas. We know that during bone remodeling or tissue repair, fluoride is incorporated into the skeleton at sites

of active osteoblastic bone synthesis, thus ^{18}F -fluoride uptake represents direct in-vivo molecular imaging of bone synthetic activity [13, 14].

The authors declare that they have no conflicts of interest.

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