

Case Report



Application of Simultaneous 18F-FDG PET/MRI for Evaluating Residual Lesion in Pyogenic Spine Infection: a Case Report

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Conflict of Interest

No conflicts of interest.

Compliance with ethical standards

The patient has consented to the submission of the paper to the journal.

ABSTRACT

Magnetic resonance imaging (MRI) is the preferred imaging method for evaluating treatment response in spine infection. However, there are still no definite correlation between follow-up MRI findings and clinical status. Recently, Fluorine-18 fluorodeoxyglucose positron emission tomography (18F-FDG PET) shows great potential as diagnostic and monitoring options. Simultaneous 18F-FDG PET/MRI makes us to expect a huge synergic effect on diagnosis and evaluation of treatment response with metabolic and anatomical advantages in spine infection. We introduce an application of 18F-FDG PET/MRI for evaluating residual lesion in the patient with pyogenic spine infection.

Keywords: 18F-FDG PET; MRI; Residual lesion; Spine infection

INTRODUCTION

Magnetic resonance imaging (MRI) is the preferred imaging method for diagnosing vertebral osteomyelitis, discitis, and epidural abscess [1, 2]. After a sufficient pathogen-specific antibiotics therapy with or without surgical debridement, follow-up MRI is sometimes used to evaluate treatment response and residual lesions. According to prior studies [3-5], some MRI findings may persist or worsen over time despite clinical improvement. Kowalski et al. [6] reported that soft tissues rather than bony findings should be the focus of interpreting follow-up MRI results. Nevertheless, there are still no specific correlations between follow-up MRI findings and clinical status, and the routine use of follow-up MRI is not always conclusive.

Recently, the use of Fluorine-18 fluorodeoxyglucose positron emission tomography (18F-FDG PET) for evaluating treatment response was also considered in spine infection. Kim et al. [7] introduced the usefulness of 18F-FDG PET with computed tomography (CT) for differentiating residual infectious lesions and post-infectious granulations after treatment. MRI has merits over CT scan for showing the changes of bone marrow and soft tissues in spinal pathologies, simultaneous 18F-FDG PET/MRI makes us to expect a huge synergistic effect on diagnosis and evaluation of treatment response with metabolic and anatomical advantages in spine infection. In this case, we describe the application of 18F-FDG PET/MRI

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for evaluating residual lesions in the patient with pyogenic spine infection showing a sharp re-elevation of C-reactive protein (CRP) level after discontinuation of antibiotics therapy.

CASE REPORT

A 59-year-old-man presented with a history of progressive bilateral leg weakness and back pain for 10 days. On neurological examination, he demonstrated grade 1 (trace) motor weakness and sensory impairment below the T9 dermatome with increased bilateral deep tendon reflexes. He underwent posterior fusion with pedicle screw fixation on the T10-L2 due to acute burst fracture of T12 seven months ago at another medical center after a fall. MRI demonstrated a compressed thoracic cord by ventral and dorsal epidural abscess with spondylodiscitis on T9-10. There was no aggravation of T12 bony structure (compression ratio and canal compromise) on the CT scan (Fig. 1). The blood inflammatory indexes including CRP and erythrocyte sedimentation rate (ESR) were increased by 3.83 mg/dL and 75 mm/h, respectively (normal ranges are defined as <0.5 mg/dL for CRP and <20 mm/h for ESR).

The patient underwent a decompressive laminectomy from lower T8 to 10. Thick granulation tissues on the dorsal epidural space compressed thoracic cord ventrally, and a small yellowish-colored abscess was noted. After a wide-range decompressive laminectomy was performed and granulation tissues were removed from the dorsal space, the cord compression was relieved. We then removed the inserted pedicle screws to facilitate healing of spine infection. In a culture study, methicillin-resistant *Staphylococcus aureus* was identified, for which parenteral vancomycin (1g/24 hours, trough level of 15 - 20mg/L), as susceptible antimicrobial was started. The patient experienced no drug-related side effects and was tolerable. CRP was normalized after 18 days of antibiotics therapy, and the ESR showed a decreasing trend. We discontinued the vancomycin on the 42nd day based on CRP and ESR with the improvement of back pain. Serial blood tests were conducted to detect recurrence of the infectious lesion. Unfortunately, sharp re-elevation of the inflammatory indexes was detected, especially in CRP, which reached to 8.49 mg/dL at seven days after the discontinuation of vancomycin. During this period, there were no signs of infection from another cause (no fever, diarrhea, coldness, or phlebitis) (Fig. 2).

The patient underwent 18F-FDG PET/MRI to identify the presence of residual infectious lesions at seven days after the discontinuation of vancomycin (Fig. 3), 18F-FDG PET/MRI revealed gadolinium enhancement on the overall bone marrow of T9 and 10 bodies, T9-10 disc, previous compression fracture site of T12, destroyed endplates of T9-10, and ventral epidural space. Additionally, increased FDG uptake with maximum standardized uptake value (SUVmax) 5.78 was also seen in these enhanced areas. We concluded that 18F-FDG PET/ MRI imply the presence of a persistent residual infectious lesion and treatment failure. An additional 29 days of vancomycin was administered and CRP and ESR were monitored. On the follow-up 18F-FDG PET/MRI (Fig. 4), sustained gadolinium enhancement on the destroyed endplate of T9 and T10, ventral epidural space, and old fracture site at T12; even though there are decrease of FDG uptake with SUVmax 3.34 compared to the prior examination. On the other hand, the edema is distinctly disappeared than gadolinium enhancement at the bone marrow and around endplates. These lesions also presented decreased FDG uptake. Antibiotic therapy was terminated based on the 18F-FDG PET/MRI results and normalized CRP. ESR was finally normalized at day 116, and normalized CRP and ESR lasted until day 222. The neurological function was recovered and the patient became ambulatory.





Figure 1. Radiography shows the posterior fusion state with pedicle screw fixation at T10-L2. MRI and CT scan reveal a compressed thoracic cord by ventral and dorsal epidural abscess with spondylodiscitis at T9-10 (red arrow). Destroyed endplates and bone erosion of T9 and T10 are evident on the CT scan (white arrow). MRI, magnetic resonance imaging; CT, computed tomography.

DISCUSSION

Pathophysiologically, the earliest response to infection of vertebral body is the accumulation of extracellular fluid within the bone marrow [8]. Infected bone marrow also enhances diffusely after contrast material is administered [9]. Post-inflammatory phase is histologically characterized by the presence of vascularized fibrous tissue, fatty bone marrow transformation, subchondral fibrosis, and osteosclerosis, which are clearly demonstrated



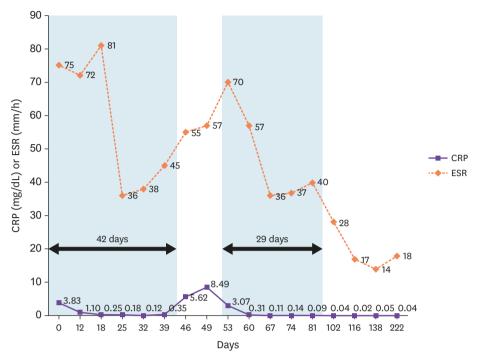


Figure 2. The initial blood inflammatory indexes including CRP and ESR were increased by 3.83 mg/dL and 75 mm/h, respectively. CRP was normalized after 18 days of vancomycin administration for MRSA, and ESR also showed a decreasing trend. The vancomycin was discontinued on the 42nd day based on improved CRP and ESR levels as well as clinical improvement. Sharp re-elevation of CRP and ESR level were detected, especially in CRP, which reached 8.49 mg/dL at seven days after the discontinuation of vancomycin. Additional 29 days of vancomycin was administered due to the persistent residual infectious lesions on F-18 FDG PET/MRI. Vancomycin administration was terminated based on the F-18 FDG PET/MRI results and normalized CRP level. The ESR finally normalized at day 116, and the normalized CRP and ESR lasted until day 222.
CRP, C-reactive protein; ESR, erythrocyte sedimentation rate; F-18 FDG PET/MRI, Fluorine-18 fluorodeoxyglucose positron emission tomography/ magnetic resonance imaging.

by MRI [8]. According to the already established MRI findings in the literatures, the disappearance of edema and enhancement of the bone marrow, resolution of paravertebral soft-tissue changes, and decreased enhancement of the phlegmon of epidural space or intervertebral disc were the reliable findings implying improvement of the spine infection. Some articles [4, 9] showed that resolution of the paravertebral soft-tissue changes and fat deposition in the bone marrow are reliable signs of the healing process and are relatively well correlated with the resolving clinical signs and symptoms despite the persistent gadolinium enhancement. Unfortunately, none of these findings are clear answers.

The response to antibiotics therapy is usually assessed in terms of the clinical status and the trend of inflammatory indexes. CRP and ESR are sensitive indexes that indicate a state of active inflammation [10]. However, their variations are influenced by an even mild undercurrent of another inflammatory process, which may cause misinterpretation of the results [11]. Repeated biopsy and culture studies for detecting residual infectious lesion are neither reasonable nor perfect. In these situations, 18F-FDG PET can play a big role in monitoring infectious lesion. 18F-FDG PET can quantify inflammatory change and is able to discriminate residual infectious lesions after antibiotics treatment [7, 12]. 18F-FDG PET measures the metabolic activity of the tissues in a non-invasive and semi-quantitative way and provides very accurate localization of the hypermetabolic activity. MRI has anatomical advantages over CT scan; it can show subtle changes of spinal structure such as bone marrow

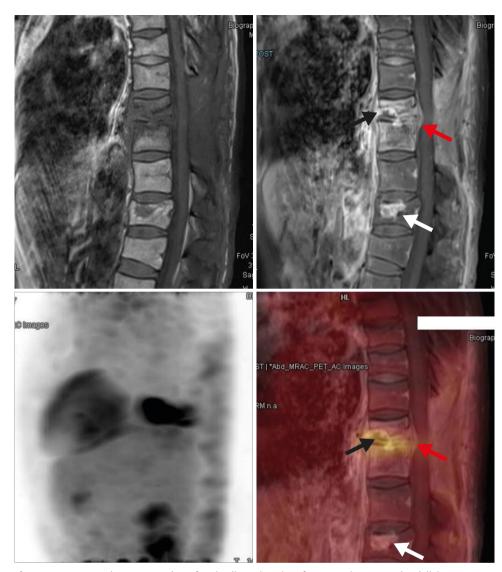


Figure 3. F-18 FDG PET/MRI at seven days after the discontinuation of vancomycin presented gadolinium enhancement on the overall bone marrow of T9-10, T9-10 disc, prior compression fracture site at T12 (white arrow), and the phlegmon of destroyed endplates of T9-T10 (black arrow) with ventral epidural space (red arrow). Increased FDG uptake (SUVmax 5.78) was also detected in these enhanced areas. F-18 FDG PET/MRI result implies the presence of a persistent residual infectious lesion and treatment failure.

F-18 FDG PET/MRI, Fluorine-18 fluorodeoxyglucose positron emission tomography/ magnetic resonance imaging; SUVmax, maximum standardized uptake value.

and neural components. The combination of the advantages from 18F-FDG PET and MRI offers a new type of hybrid imaging that provides highly sensitive metabolic and high-resolution anatomic imaging [13]. In addition, simultaneous 18F-FDG PET/MRI can minimize the errors arising from metabolic and spatial differences between two separated examinations.

Here we describe our experience of applying 18F-FDG PET/MRI in the patient with equivocal MRI findings under a sharp re-elevation of CRP level after discontinuation of antibiotics therapy. 18F-FDG PET/MRI identified the presence of residual infectious lesions by detecting persistently increased FDG uptake, and it provides specific MRI findings including edema and gadolinium enhancement compatible with metabolic activity. On the MRI performed after additional 29 days antibiotics therapy, the edema at the bone marrow had disappeared.

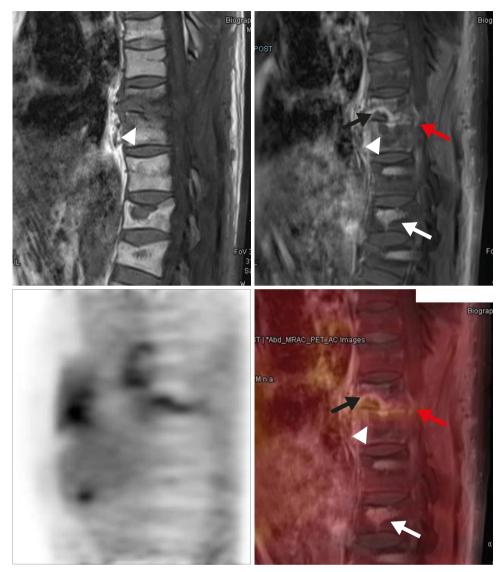


Figure 4. F-18 FDG PET/MRI performed after an additional 29 days of vancomycin treatment showing sustained gadolinium enhancement on the destroyed endplate of T9 and T10 (black arrow), ventral epidural space (red arrow), and old fracture site at T12 (white arrow); even though there are decrease of FDG uptake (SUVmax 3.34) compared to the prior examination. On the other hand, the edema is distinctly disappeared than gadolinium enhancement at the bone marrow and around endplates. These lesions also presented decreased FDG uptake. F-18 FDG PET/MRI, Fluorine-18 fluorodeoxyglucose positron emission tomography/ magnetic resonance imaging; SUVmax, maximum standardized uptake value.

There was a tendency of persistent gadolinium enhancement on the destroyed endplates of T9-10, and old compression fracture site of T12 continues even though some sort of decline compared with prior MRI. However, FDG uptake declined at the overall bone marrow and paravertebral soft tissues. Gadolinium enhancement and increased FDG uptake were more focused at the endplates than other sites after completion of treatment.

The increased FDG uptake on 18F-FDG PET/MRI at seven days after the discontinuation of vancomycin was a compatible finding that implied the presence of residual infectious lesions, which led to re-elevation of the CRP level after discontinuation of the antibiotics therapy. In contrast, based on the clinical progress and considering the finding of the old compression



fracture site with persistent increased FDG uptake and gadolinium enhancement, equivalent findings at the destroyed endplates, bony structure, and ventral epidural space on 18F-FDG PET/MRI after additional 29 days antibiotic therapy can be interpreted as a healing process such as vascularized fibrosis. In this case, we could differentiate healing process from residual infectious lesions by measuring metabolic activity with 18F-FDG PET/MRI; it was not accessible by the enhancement pattern of MRI alone. Bone marrow healing as shown by disappearance of edema is a more reliable MRI finding to decide to stop antibiotics therapy based on the decrease of FDG uptake. This case gives the possibility of 18F-FDG PET/MRI for evaluating treatment response in spine infection. Further studies with more patients are required to demonstrate our results.

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