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Case Report

Positron emission tomography-magnetic resonance liver parenchyma attenuation correction artifact in secondary hemochromatosis

ABSTRACT

Positron emission tomography-magnetic resonance (PET-MR) hybrid imaging is a relatively new imaging modality combining the superb MR contrast capabilities among different soft-tissue structures with the high sensitivity of PET functional imaging. With the development of any new technology, a variety of limitations will be encountered including the introduction of new types of artifacts. In this case report, we present a restaging PET-MR scan for multiple myeloma that showed severely decreased fluorodeoxyglucose activity in the liver on the PET attenuated corrected images. Careful analysis showed the cause of the decreased activity to be the improper density assignment on the mu map caused by iron deposition within the liver. Follow-up imaging showed reversal of the phenomena following improvement of liver disease.

Keywords: Attenuation correction, fluorodeoxyglucose, mu map, multiple myeloma, positron emission tomography-magnetic resonance

INTRODUCTION

Positron emission tomography-magnetic resonance (PET-MR) hybrid imaging is a relatively new imaging modality combining the superb MR contrast capabilities among different soft-tissue structures with the high sensitivity of PET functional imaging. With the development of any new technology, a variety of limitations will be encountered including the introduction of new types of artifacts. In this case report, we present a restaging PET-MR scan for multiple myeloma that showed severely decreased fluorodeoxyglucose (FDG) activity in the liver on the PET attenuated corrected images. Careful analysis showed the cause of the decreased activity to be the improper density assignment on the mu map caused by iron deposition within the liver. Follow-up imaging showed a reversal of the phenomena following improvement of liver disease.

CASE REPORT

A 74-year-old female with multiple myeloma initially diagnosed 10 years ago presented for a restaging FDG PET-MR scan. The patient had relapsed immunoglobulin G (IgG) kappa multiple myeloma with 17-p deletion. For treatment, the patient had received multiple chemotherapy regimens including two autologous stem cell transplants. She suffered from generalized bone pain with worsening lower back pain. PET-MR was performed on a hybrid scanner to evaluate the extent of metastatic disease including bone involvement.

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PET-MR fusion images showed unusual severely decreased radiotracer activity throughout the liver. The corresponding T1 images revealed generalized decreased MR signal in the liver parenchyma in relation to the paraspinal musculature signal intensity which is commonly seen with iron overload. When looking at the PET imaging, the attenuation-corrected images showed severely decreased uptake which was not present on the nonattenuation corrected images indicating an attenuation correction artifact. The mean standard uptake value (SUV) of the liver was 0.6 [Figure 1].

On review of the Dixon T1 MR sequence used for attenuation correction, the out-of-phase sequence revealed normal moderate signal intensity in the liver parenchyma, but on the corresponding in-phase image, the liver demonstrated signal loss due to susceptibility artifact from excessive iron accumulation in the hepatic parenchyma. As a consequence, the generated mu maps for this patient expanded the boundary of the right lung instead of correctly assigning soft-tissue density to the liver parenchyma [Figure 2]. To confirm this, the coronal attenuation mu map was manually segmented by filling in the faulty liver reconstruction with soft-tissue density instead of lung tissue. The resulting attenuated corrected PET image showed the properly corrected liver parenchyma tracer activity with mean SUV 2.4 [Figure 3].

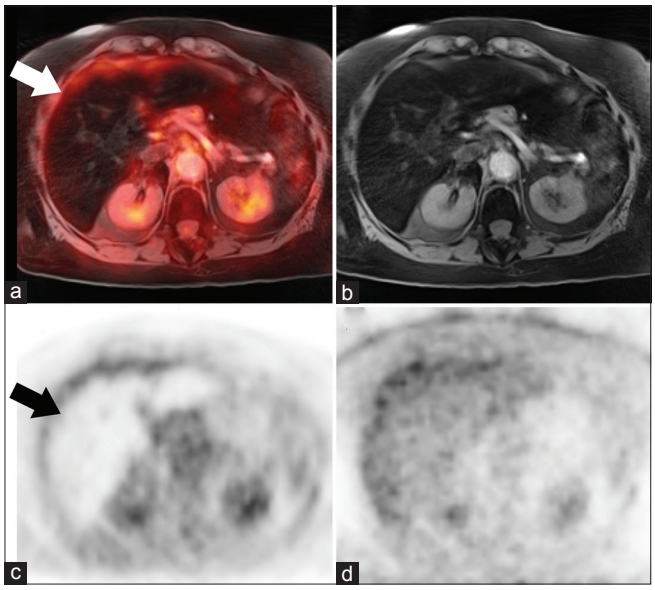

Figure 1: Positron emission tomography-magnetic resonance imaging fusion with T1 radial volumetric interpolated breath-hold examination with fat suppression acquired in the axial plane showed severely decreased radiotracer activity throughout the liver parenchyma (white arrow) (a). Corresponding axial T1 radial volumetric interpolated breath-hold examination with fat suppression image (b) revealed generalized decreased magnetic resonance signal in the liver. Axial positron emission tomography attenuation corrected image (c) showed severe decreased liver uptake (black arrow) which was not present on the nonattenuation corrected images (d)

Five months after the initial FDG PET-MR scan, the patient returned for follow-up imaging. During this period, the patient underwent oral chelator therapy for the secondary hemochromatosis. Attenuation corrected PET axial images demonstrated homogeneous, expected moderate FDG activity of the liver parenchyma. Likewise, the corresponding T1 image showed improved liver signal intensity reflecting decreased iron deposition [Figure 4].

DISCUSSION

The patient had relapsed IgG kappa multiple myeloma with 17p deletion which is associated with a poor clinical outcome, aggressive initial disease presentation, and short response duration to therapy.[1] The treatment of multiple hematopoietic and immune system disorders frequently involve hematopoietic stem cell transplantation (HSCT). Although there is a greater overall increase in survival rates, complications resulting from HSCT include infections, graft versus host disease, and hepatic sinusoidal obstruction syndrome. Patients undergoing HSCT frequently experience iron overload or secondary hemochromatosis as a result of chronic red blood cell transfusion therapy for anemia.[2] Because the body has no active mechanism to clear iron, the excess iron from transfusion accumulates in the blood, bone marrow, liver, and other organs. Since the liver is the largest organ to accumulate iron, it is readily detectable on MR imaging (MRI).[3]

The attenuation correction on the hybrid PET-MR scanner is performed using a two-point Dixon three dimensional volumetric interpolated breath-hold examination T1-weighted

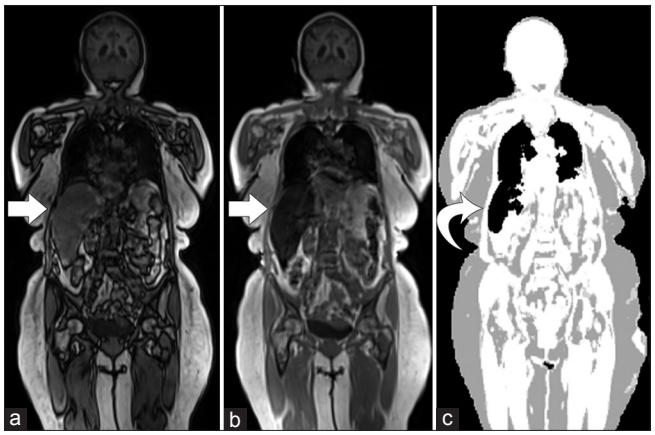

Figure 2: Dixon three-dimensional volumetric interpolated breath-hold examination T1-weighted magnetic resonance sequence in the coronal plane demonstrates normal signal intensity in the liver parenchyma (arrow) on the out-of-phase image (a) with drop in signal on the in-phase image indicating excessive iron accumulation (b). The mu map showed the expanded boundary of the right lung instead of the correctly assigned soft-tissue density of the liver parenchyma (curved arrow) (c)

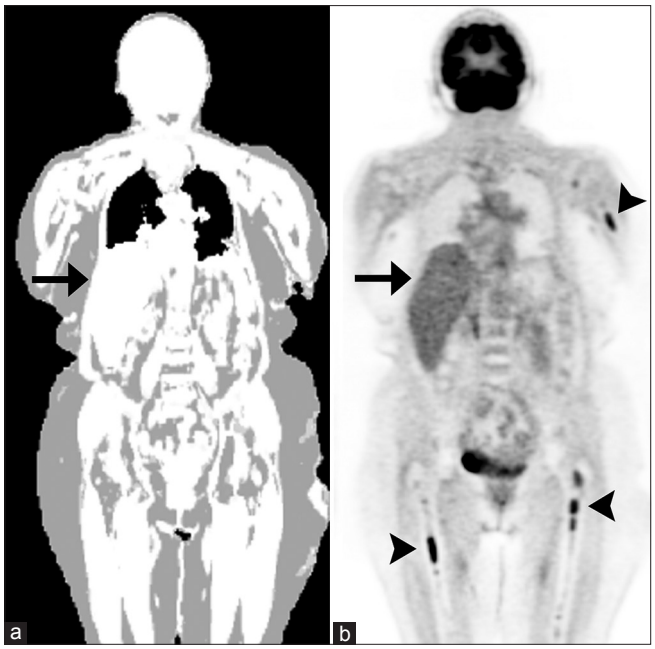

Figure 3: The coronal attenuation mu map was manually segmented by filling in the faulty liver reconstruction (thin arrow) with soft-tissue density instead of lung tissue (a). The resulting attenuation corrected positron emission tomography image in the coronal plane showed the properly corrected liver parenchyma tracer activity (thin arrow). Unrelated to the liver, multiple osseous metastases are noted on the study (arrowheads) (b)

MR sequence. The in-phase and out-of-phase MR images are acquired simultaneously with the T1 Dixon sequence using a single pulse with two specific echo times referred to as fat-water chemical shift imaging. With metallic compounds such as surgical hardware and iron deposition, the long echo time in-phase sequence manifests a more pronounced susceptibility artifact with signal loss compared to the short echo time out-of-phase sequence due to proportionately increased relaxation of proton spins with increasing echo time.[4]

When imaging with a PET-computed tomography (CT) scanner, the CT images provide accurate photon attenuation coefficients and mu values for the CT energies typically used clinically when correcting the PET images. In contrast, since MRI uses different principles, the photon attenuation coefficient cannot be computed in a direct way.[5] The most common way for MR attenuation correction (MRAC) using MRI data only is to use a Dixon MR sequence to segment the tissue into different classes of density (fat, soft tissue, lungs, and background) producing a mu map of the patient. The mu map is then used for PET attenuation correction. Problems do arise with the Dixon segmentation algorithm reversing the tissue class in up 10% of the time in early attenuation correction models. To ameliorate this problem, more recent approaches use an atlas or database of the patient for improved MRAC maps.[6]

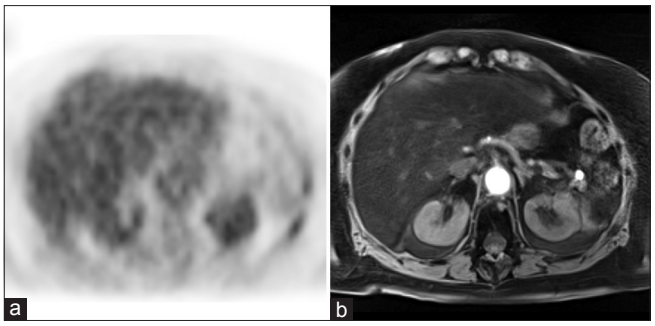

Figure 4: Fluorodeoxyglucose positron emission tomography-magnetic resonance imaging scan after oral chelator therapy with attenuation corrected positron emission tomography axial image demonstrating normal fluorodeoxyglucose uptake within the liver (a). Corresponding T1 radial volumetric interpolated breath-hold examination with fat suppression image showed improved liver signal intensity reflecting decreased iron deposition (b)

In secondary hemochromatosis, iron deposition occurs within the reticuloendothelial system including the Kupffer cells of the liver, spleen, bone marrow, and lymph nodes. On MRI imaging, T2-gradient echo sequence would show decreased signal intensity within the liver, spleen and bone marrow. In other forms of hemochromatosis, the spleen, and bone marrow are not affected. In primary hemochromatosis excess, iron deposition with decreased T2-gradient echo intensity would be seen initially in the liver, then spreading to the pancreas and thyroid, and finally progressing to the myocardium and hypophysis. Iron deposition limited to the kidney can be seen in patients with intravascular hemolysis such as mechanical stress with heart valves and hemolytic crisis of sickle cell anemia resulting in decrease T1-weighted signal of the renal cortex with accentuation of the decreased T2 signal.[7]

The excess iron accumulation in the body with secondary hemochromatosis can lead to liver toxicity, chronic liver disease, and cirrhosis. The easiest and cost-effective way to remove excess body iron is through simple phlebotomy. This is often performed alone or in combination with erythropoietin therapy. An alternative therapy is with an iron chelating medication such as deferoxamine that has been shown to be effective in the management of iron overload with patients of HSCT resulting in less transplant complications and improved patient survival.[8]

Declaration of patient consent

The authors certify that they have obtained all appropriate patient consent forms. In the form, the patient has given her consent for her images and other clinical information to be reported in the journal. The patient understands that their names and initials will not be published and due efforts will be made to conceal their identity, but anonymity cannot be guaranteed.

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Nil.

Conflicts of interest

There are no conflicts of interest.

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