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Case Report

^{18}F-FDG PET/CT findings in a mucosa-associated lymphoid tissue lymphoma patient coexisting with primary myelofibrosis

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Abstract: A 61-year-old male presented with hematemesis and melena. Biopsy and immunohistochemistry confirmed mucosa-associated lymphoid tissue (MALT) lymphoma in the posterior wall of the gastric antrum, prompting further evaluation with ^{18}F-fluorodeoxyglucose (^{18}F-FDG) positron emission tomography/computed tomography (PET/CT). In addition to elevated uptake in the gastric antrum, ^{18}F-FDG PET/CT showed diffuse uptake in multiple bone marrow, initially suspected to indicate bone marrow involvement by lymphoma. Further examination identified it as primary myelofibrosis (PMF). Following concurrent therapies, ^{18}F-FDG PET/CT demonstrated negative uptake in gastric antrum, indicating complete remission of the lymphoma, while the elevated bone marrow uptake suggested progression of PMF. The coexistence of MALT lymphoma and PMF is very rare. This case highlights the image characteristics and potential diagnostic and therapeutic monitoring value of ^{18}F-FDG PET/CT in patients with concurrent MALT lymphoma and PMF.

Keywords: Mucosa-associated lymphoid tissue lymphoma, primary myelofibrosis, ^{18}F-FDG PET/CT, diagnosis, monitoring

Introduction

Mucosa-associated lymphoid tissue (MALT) lymphoma originates from post-germinal center B cells in the marginal zone and constitutes approximately 7-8% of non-Hodgkin's lymphomas, with a peak incidence in individuals aged 50-60 [1]. MALT lymphomas can arise in lymphoid tissue at various mucosal sites, most commonly in the stomach [2]. It is often associated with chronic inflammation or infection at the primary sites [3]. For instance, gastric MALT lymphoma is frequently linked to Helicobacter pylori (*HP*) infection. Clinical presentations of MALT lymphoma can be protean, depending on the affected organ. It is characterized by an indolent clinical course and frequent relapses. Treatment may involve antibiotics for infection-related cases (e.g., *HP* eradication therapy), chemotherapy, radiation therapy, immunotherapy, or a combination of these approaches.

Primary myelofibrosis (PMF) is a chronic myeloproliferative neoplasm that arises from the clonal proliferation of hematopoietic stem cells [4, 5]. It is characterized by bone marrow inflammation, reactive marrow fibrosis and extramedullary hematopoiesis [6, 7]. The incidence of PMF is low, with a reported incidence of 1.5 cases per 100,000 individuals per year, underscoring the significance of each case in advancing our understanding of this disease [8]. Despite its infrequency, PMF carries a grave prognosis, with survival times typically ranging from 2 to 5 years post-diagnosis and the onset of symptoms [9]. This disease may initially manifest with minimal or no symptoms, leading to a diagnosis that often follows incidental imaging findings. Although the pathogenesis of PMF is not fully understood, it is closely associated with aberrant activation of the Janus kinase/signal transducers and activators of transcription (*JAK-STAT*) signaling pathway [10]. Additionally, the recurrent mutations in *JAK2*, myeloproliferative leukemia virus (*MPL*) and calreticulin (*CALR*) are key drivers of this disease [11]. PMF is a condition of dismal prognosis with limited treatment options. Allogeneic hematopoietic stem cell transplantation is the only curative therapy for patients with myelofibrosis (MF); however, transplant-related morbidity and mortality preclude this option for the majority of patients. The *JAK2* inhibitor (ruxolitinib), an FDA approved drug, has demonstrated efficacy in alleviating myelofibrosis-related symptoms and improving the quality of life [12]. According to the WHO classification, PMF can be classified into prefibrotic primary myelofibrosis (pre-PMF) and overt-fibrotic primary myelofibrosis (overt-PMF) stages with fibrosis grade 0-1 in pre-PMF and grade 2-3 in overt-PMF [13].

The coexistence of lymphoma with PMF is rare, and managing concurrent malignancies in such patients necessitates a multidisciplinary approach. ^{18}F-fluorodeoxyglucose positron emission tomography/computed tomography (^{18}F-FDG PET/CT) is a multimodal molecular imaging technique that plays a crucial role in the diagnosis, staging and response monitoring of various diseases, including hematological malignancies [14]. This report presents the case of a patient with concurrent MALT lymphoma and PMF, emphasizing the role of ^{18}F-FDG PET/CT imaging findings in diagnosing and monitoring these conditions.

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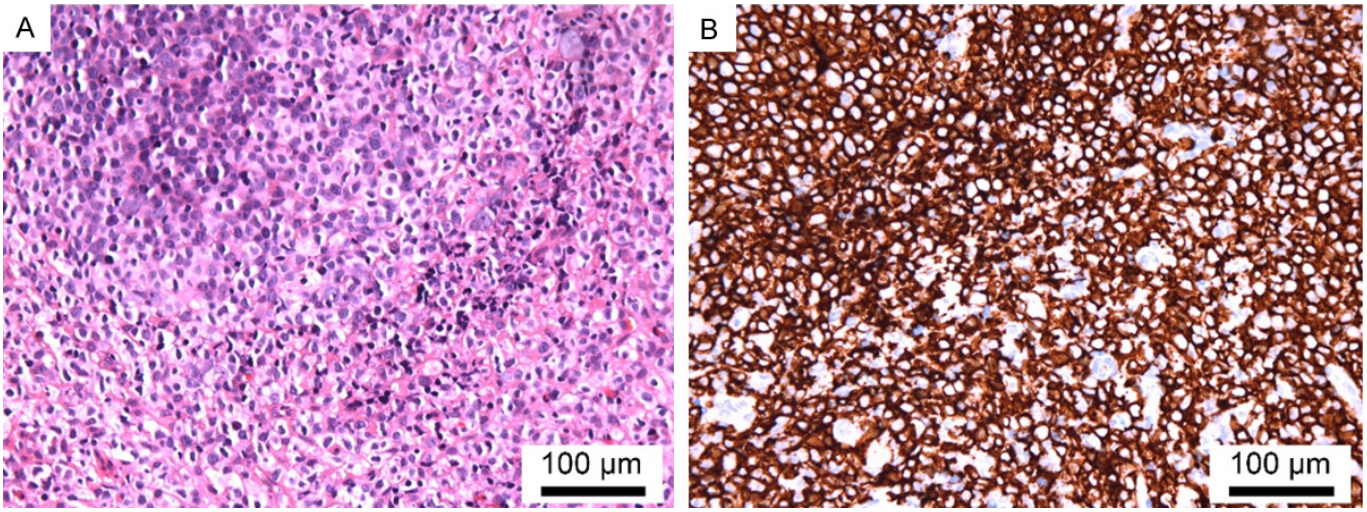

Figure 1. Biopsy specimens from the gastric antrum revealed the pathological diagnosis of MALT lymphoma. A. Hematoxylin and eosin staining; B. Immunohistochemistry of CD20.

Case presentation

The patient, a 61-year-old male, visited a local hospital in July 2020 with the chief complaints of hematemesis and melena over several days. He denied experiencing constitutional symptoms such as fever, chills, night sweats, or unintentional weight loss. His medical history included a radical resection for lung adenocarcinoma, with no known hematologic disorders and no relevant family history. Upon admission, peripheral blood analysis revealed elevated white blood cell count (15.3 G/L), thrombocytosis (617 G/L) and anemia (hemoglobin 64 g/L). A gastroscopy was promptly performed, uncovering gastric ulcers with active bleeding in the posterior wall of the gastric antrum. The patient received endoscopic hemostasis, red blood cell transfusion, acid suppression therapy, measures to reduce splanchnic blood flow, and nutritional support. A positive *HP* antibody test led to the initiation of anti-*HP* treatment.

In early August, the patient was referred to our hospital for further examination and treatment. Physical examination was unremarkable. Laboratory tests in our hospital showed haemoglobin at 77 g/L, mean corpuscular volume of 71.7 fl, mean corpuscular haemoglobin concentration of 289 g/L, and a reticulocyte count of 0.46%. His platelet count was 932 G/L, and white cell count was 9.84 G/L, with a differential comprising 79.3% neutrophils, 16.0% lymphocytes, 3.6% monocytes, 0.2% basophils and 0.9% eosinophils. Direct and indirect Coombs tests were negative. Histopathology confirmed the presence of MALT lymphoma in the posterior wall of the gastric antrum (**Figure 1**). Immunohistochemical analysis showed that tumor cells were positive for CD20, CD19, CD22, PAX5, CD21 (FDC net), P53 and Ki-67 at a high frequency, while the expression of CD3, CD138, Kappa, Lambda, IgD was negative. Chromosome analysis revealed a normal male karyotype.

To further assess the condition, ^{18}F-FDG PET/CT (**Figure 2A**) was performed. Images showed slight thickening in the gastric antrum and mildly elevated ^{18}F-FDG uptake with an SUV_{max} of 4.0 (**Figure 2B**, red circles). Notably, diffuse and inhomogeneous ^{18}F-FDG uptake was observed in the axial and distal skeleton (**Figure 2A**, blue arrowheads; **Figure 2C**, red arrows), raising suspicion for lymphomatous involvement. Subsequently, the patient underwent bone marrow aspiration biopsy. Surprisingly, bone marrow biopsy revealed active myeloproliferative activity with no evidence of lymphoma cell infiltration. Bone marrow cell morphology showed increased myelocytes and megakaryocytes in a restricted area, with reticulin fiber staining graded 0-1. Furthermore, molecular testing showed positive results for *JAK2*, *MPL*, serine/arginine-rich splicing factor 2 and ten-eleven translocation 2 mutations. Based on the findings described above, a diagnosis of pre-PMF was established.

The patient received eight cycles of R-CHOP therapy (rituximab, cyclophosphamide, doxorubicin, vincristine, and prednisone) for MALT lymphoma while also starting ruxolitinib for symptom management of PMF. After the treatment, ^{18}F-FDG PET/CT images demonstrated negative ^{18}F-FDG uptake in the gastric antrum (**Figure 2D**, red circles), but there was increased uptake in bone marrow (**Figure 2E**, red arrows; **Figure 2F**, blue arrowheads) compared to previous images. Similarly, follow-up gastroscopy revealed no lymphoma lesions. However, thrombocytosis persisted (platelet count of 450 G/L), and the proportion of JAK2-carrying clones increased from 8.03% to 74.65%, indicating progression of myelofibrosis. The patient's condition necessitates ongoing monitoring and adjustments in therapy to effectively manage PMF.

Discussion

This case of a 61-year-old male diagnosed with both MALT lymphoma and PMF is significant due to the rarity and

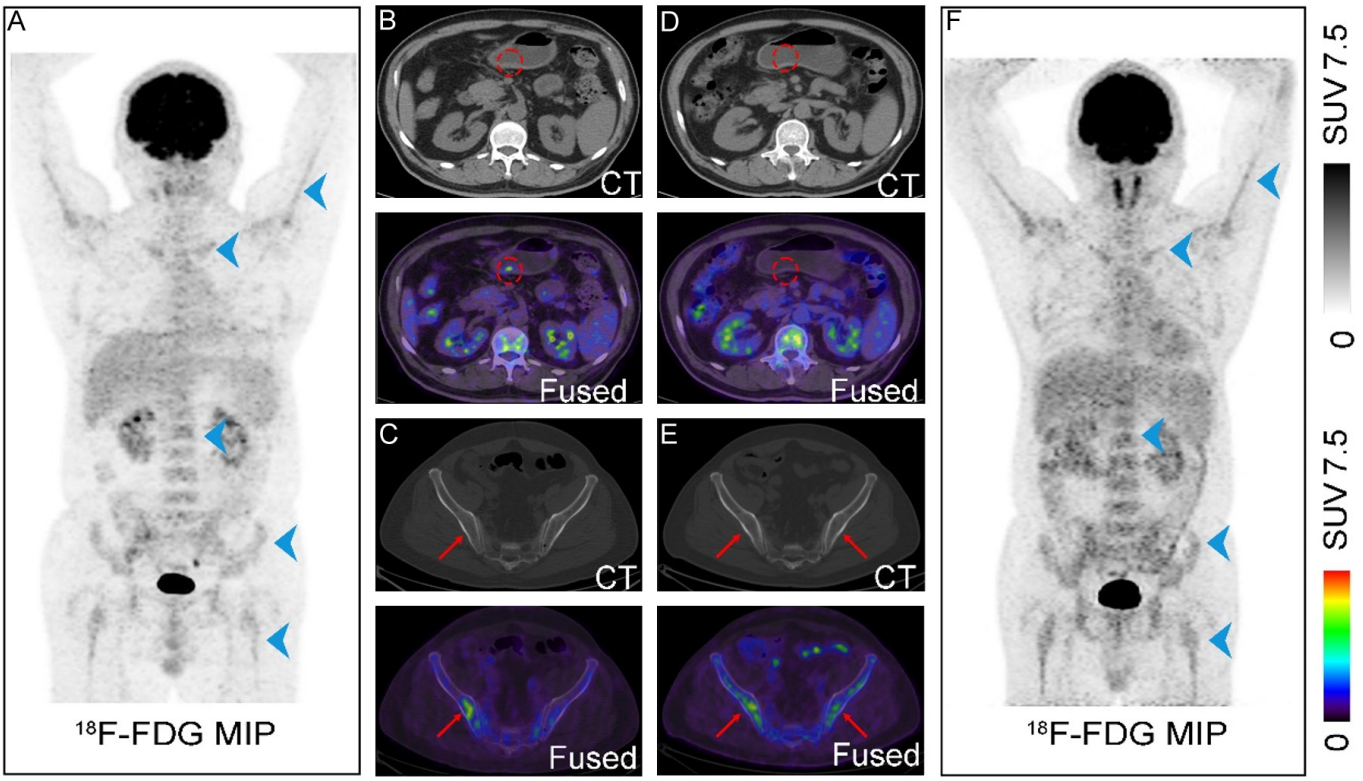

Figure 2. ^{18}F-FDG PET/CT images at baseline (A-C) and post-therapy (D-F). The ^{18}F-FDG PET maximum intensity projection (MIP) image displayed diffuse elevated ^{18}F-FDG accumulations in the axial and distal skeleton with SUV_{max} of 2.4-4.9 (A, blue arrowheads). The axial CT and fused images of the gastric antrum wall showed slightly thickened with mildly elevated ^{18}F-FDG uptake with SUV_{max} of 4.0 (B, red circles), whereas normal activities were shown in the remaining stomach. The axial CT and fused images of pelvis showed elevated ^{18}F-FDG uptake (C, red arrows). Post-therapy ^{18}F-FDG PET/CT images displayed normal ^{18}F-FDG uptake in the previous gastric antrum lesion (D, red circles). The selected axial CT and fused images of pelvis showed elevated ^{18}F-FDG uptake (E, red arrows) compared to the previous image. The ^{18}F-FDG MIP image also exhibited higher uptake in bone marrow in comparison to the previous image with SUV_{max} of 4.4-5.7 (F, blue arrowheads).

complexity of such concurrent hematologic malignancies. These two malignancies arise from distinct cell lines during the hematopoietic process. There are currently no studies definitively establishing a connection between lymphoma and myelofibrosis. A plausible hypothesis revolves around cytokines released by lymphoma cells, such as transforming growth factor-β (TGF-β), platelet-derived growth factor, basic fibroblast growth factor (β-FGF), vascular endothelial growth factor, tumor necrosis factor α, and interleukin-1 may play a significant role in the development of myelofibrosis [15]. For example, it was reported that elevated levels of TGF-β and β-FGF in patients with follicular lymphoma may contribute to the development of myelofibrosis by stimulating fibroblast activity and collagen production in the bone marrow [16]. The tumor microenvironment in MALT lymphoma may alter the bone marrow niche, contributing to the dysregulation of hematopoietic stem cells and the development of myelofibrosis [17]. The interaction between malignant lymphocytes and the bone marrow stroma can create an environment conducive to fibrosis [18]. While there have been reports suggesting that the R-CHOP regimen could be beneficial for managing MF, this has not been conclusively proven [19, 20]. Furthermore, chemotherapy for lymphoma may change the bone marrow environment, potentially worsening pre-existing myelofibrosis or triggering its progression. In this case, in this patient, PMF progressed after chemotherapy for MALT lymphoma, highlighting the complex relationship between the two conditions and the need for careful monitoring during treatment.

MF can occur after lymphoma, concurrently with it, or even precede its onset. However, when both MF and lymphoma are present at the same time, distinguishing between PMF and SMF can be challenging. Distinguished from PMF, SMF is diagnosed based on the presence of defined underlying diseases, including autoimmune diseases, hematological malignancies, metastatic carcinoma, hyperparathyroidism, grey platelet syndrome, rickets, osteopetrosis, toxic marrow injury following irradiation or chemical exposure, chronic inflammation and infection [15, 21, 22]. This patient had no cause for SMF. Genetic analysis for mutations in the *JAK2*, *MPL*, and *CALR* genes are very specific to PMF and can identify mutations in approximately 90% of PMF patients [16]. In this case, both *JAK2* and *MPL* mutations were detected, supporting the diagnosis of PMF.

Existing literature on the coexistence of MALT lymphoma and PMF is sparse, with most studies focusing on the indi-

vidual characteristics and management of each condition separately. Given the complexity and rarity of concurrent PMF and MALT lymphoma, comprehensive diagnostic evaluations and a multidisciplinary approach are essential. This case highlights the role of ^{18}F-FDG PET/CT, which provided critical insights into disease extent and treatment monitoring.

The baseline ^{18}F-FDG PET/CT scan was instrumental in clearly detecting both the gastric lymphoma and PMF lesions. Most gastric MALT lymphomas typically exhibit a slight to moderate ^{18}F-FDG uptake, often accompanied by thickening of the gastric wall [23]. These characteristics are compatible with those of this case with focal and slight accumulation. It is important to note that elevated ^{18}F-FDG uptake in the stomach can be attributed to diverse factors, including physiological uptake, benign lesions and tumors [23, 24]. Usually, the distribution pattern of ^{18}F-FDG uptake can provide valuable clues to the underlying pathology; diffuse ^{18}F-FDG uptake is more suggestive of benign gastric conditions such as gastritis, while focal ^{18}F-FDG uptake may indicate a substantial risk of malignant lesions [25, 26]. Particularly, this report highlights the potential usefulness of ^{18}F-FDG PET/CT in the detection of PMF. Diffuse increased bone marrow ^{18}F-FDG uptake can be observed in a variety of conditions, including infections, hematologic malignancies, and the administration of hematopoietic cytokines such as granulocyte colony-stimulating factor or granulocyte-macrophage colony-stimulating factor [27-29]. However, the patient had no infection, history of hematologic malignancies other than lymphoma or hematopoietic cytokines therapy history. CT scans demonstrate varying degrees of osseous sclerosis and multiple cyst-like lucencies. The osseous alterations induced by myelofibrosis typically manifest as symmetrical and diffuse distributions, particularly in red bone marrow regions like the spine, pelvis, and the long bones of the limbs [30]. The overall morphology of the affected bones typically shows no obvious changes, although the internal structure of the bone marrow may be significantly altered [31]. While PET/CT is not the primary imaging modality for PMF, it can provide information on bone marrow involvement and the extent of fibrosis in whole-body images, which is not typically visualized well with conventional CT. In this case, the PET/CT scan demonstrated diffuse ^{18}F-FDG uptake in the bone marrow, a finding consistent with previous reports [32]. These conditions should not be ignored in clinical work.

Moreover, ^{18}F-FDG PET/CT could serve as a valuable tool in monitoring treatment responses for both MALT lymphoma and PMF. In recent years, the utilization of ^{18}F-FDG PET/CT has become increasingly prevalent, serving as a valuable morphofunctional imaging technique for the initial diagnosis and ongoing therapeutic surveillance of a spectrum of hematological malignancies [33, 34]. As is well known, ^{18}F-FDG PET/CT has become the standard diagnosis and efficacy assessment method for most lymphomas, and has demonstrated significant clinical value in the therapeutic evaluation of patients with ^{18}F-FDG-avid MALT lymphoma [35, 36]. Some studies have shown that ^{18}F-FDG PET/CT has the potential for noninvasive monitoring of bone marrow metabolism in myelofibrosis and is a feasible method for noninvasively monitoring treatment response [32, 37, 38]. As our understanding of these diseases and the capabilities of imaging technology continue to evolve, we anticipate further advancements in the diagnostic and therapeutic landscape.

In conclusion, here we present a rare case of MALT lymphoma and PMF coexistence, highlighting the image characteristics and potential clinical utility of ^{18}F-FDG PET/CT in diagnosing and monitoring both conditions. PET/CT offers comprehensive, whole-body imaging, aiding clinicians in understanding disease dynamics, assessing treatment responses, and guiding patient management decisions.

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Disclosure of conflict of interest

None.

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