Various inflammatory lesions indicated as malignancy by PET in the abdomen

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Learning objectives

1. In this exhibit, we illustrate the CT and MR imaging findings of inflammatory lesions indicated as malignancy by PET in the abdomen.

2. We correlate these findings with the pathologic result.

3. We discuss and illustrate imaging findings to help to differentiate these false-positive lesions from true malignancies.

Background

18F-FDG Positron emission tomography (PET) is very useful checkup tool in the staging and follow-up evaluation for malignancy, such as head and neck cancer, esophageal cancer, lung cancer, breast cancer, colorectal cancer, lymphoma, and melanoma. The extent of FDG uptake is various for each malignancy.

Overall sensitivity and specificity of PET was estimated to be 84% and 86%, respectively, and the plan for treatment can be changed approximately one third of the patient by PET.

However, 18F-FDG uptake is not a specific probe for malignancy. The range of 18F-FDG uptake in malignancy, which is measured as standardized uptake value (SUV), is overlapped with physiologic uptake or benign nonphysiologic condition.

Physiologic 18F-FDG uptake can be seen in brain, orbital muscle, Waldeyer's tonsillar ring, myocardium, digestive tract, urinary tract, gonads, and thymus. Benign nonphysiologic lesions with increased 18F-FDG uptake include inflammatory process such as acute or chronic infection, granulomatous diseases such as sarcoidosis, autoimmune disease, postoperative healing scars and postradiation therapy, post-traumatic bone and soft tissue abnormalities, and benign tumors.

Knowledge and perception for these conditions is important to decrease the rate of false-positive results in oncologic patients.

Imaging findings OR Procedure details
1. Introduction

18F-FDG is transported into cells by glucose transporter proteins (GLUTs), similar to that for unlabeled D-glucose. Many malignant cells express higher numbers of specific membrane transport proteins, with greater affinity for deoxyglucose than normal cells, which permits increased, glucose flow into the cancerous cells. The SUV of malignant neoplasms ranges from slightly greater than 2 to as high as 20.

Malignant cells are, however, not the only cells that exhibit an increased uptake of 18F-FDG. Among benign nonphysiologic condition with increased FDG uptake, more than 73% of benign lesions were inflammatory nature, with post-traumatic change in addition to benign tumors (Table 1).

Multiple reports have shown that lesions with a high concentration of inflammatory cells, such as granulocytes, lymphocytes, and macrophages, also show increased 18F-FDG uptake, which can be mistaken for malignancy in patients with proven or suspected cancer. These cells have been found to have enhanced levels of GLUT 3 or GLUT 1.

Tumor necrosis factor # is a cytokine produced primarily by monocytes and activates macrophages. Activated monocytes directly activate reduced nicotinamide adenine dinucleotide phosphate (NADPH) oxidase, which imposes an acute metabolic demand met by an increased use of both internal energy stores and exogenous metabolites such as glucose. This can explain increased 18F-FDG uptake in inflammatory condition.

2. Infectious diseases

Gastrointestinal tract

In the digestive tract, normal stomach often reveals FDG uptake. SUV is usually less than 3.8, but SUVs as high as 5.6 can also occur. Normal colon and small intestine commonly demonstrate increased FDG uptake in patients who have fasted. Isolated 18F-FDG uptake shows SUV of less than 4, but intense uptake (SUV as high as 10) can occur, particularly in the right colon. These focal uptakes can be confused with inflammatory conditions, so the correlation with CT findings is required.

The origin of the 18F-FDG uptake in the digestive tract is unknown; active smooth muscle, metabolically active mucosa, swallowed secretions, or colonic microbial uptake were suggested.
Active inflammation of bowel

- Normal physiological uptake of 18F-FDG along the gastrointestinal tract often occurs at gastro-esophageal junction, ileocecal valve region, right colon, or all along the small and large intestine.
- Segmental uptake may suggest focal inflammation when it is associated with bowel wall thickening with or without infiltration of adjacent mesenteric fat or reactive lymph node enlargement on CT.
- §Acute diverticulitis, appendicitis, or active inflammatory bowel disease (figure 1) can lead to increased uptake of 18F-FDG, but sometimes only endoscopy and biopsy might exclude malignancy.

Liver

Abscess

- Pyogenic, fungal, or parasitic
- Histologically, liver abscess is composed of a liquefied cavity filled with debris, lined by chronic inflammatory infiltrate consisting of macrophages, lymphocytes, eosinophils, and neutrophils.
- Therefore, any abscess in the liver could be associated with increased 18F-FDG uptake (figure 2).

Hydatid cyst

- Echinococcus is parasitic infection from ingestion of eggs of the tapeworm Echinococcus granulosus, either by eating contaminated food or through contact with dogs.
- A hydatid cyst is composed of 3 layers: Inner germinal layer (endocyst), Translucent thin interleaved membrane (ectocyst), Outer layer (pericyst)
- Inflammatory infiltrates of outer layer may explain increased uptake of 18F-FDG, which may be found in the periphery of these cysts on PET

Gallbladder, Biliary tree, and Pancreas

Acute cholecystitis and ascending cholangitis

- PET/CT images often can differentiate biliary inflammation from tumor, especially if a stone is seen on CT.

Acute pancreatitis
• Pancreatitis is often presented with diffuse or segmental enlargement of the pancreas, with infiltration of peripancreatic fat, necrosis, or pseudocyst formation on CT. Those CT findings are associated with increased 18F-FDG uptake.

Spleen

Abscess and active granulomatous disease such as tuberculosis or brucellosis can lead to increased uptake of 18F-FDG.

Lymph Node

Active granulomatous disease such as tuberculosis lymphadenitis can show increased uptake of 18F-FDG in lymph nodes (figure 3). But reactive change also can be associated with increased uptake.

Adrenal gland

Benign non-physiologic lesion in adrenal gland usually show increased 18F-FDG uptake in case of adrenal cortical adenoma or pheochromocytoma. But infectious condition such as tuberculosis can also show high 18F-FDG uptake (figure 4).

3. Other inflammatory lesions

Gastrointestinal tract

Typhlitis

• Neutropenic condition or mucosal injury from cytotoxic drugs can attribute the development of typhlitis.
• Circumferential edematous wall thickening, cecal distention, stranding of the adjacent mesenteric fat are common accompanying findings. Pneumatosis or pneumoperitoneum indicates bowel ischemia/infarction and bowel perforation due to severe inflammatory change.
• These can be visualized as increased 18F-FDG uptake.

Graft versus host disease (GVHD)

• Denuded gastrointestinal mucosa in small and large bowel might be replaced by granulation after allogeneic stem cell transplantation.
• Hyperemic bowel mucosa surrounded by lower-attenuation outer bowel wall layers ("target sign"), bowel loop separation, and mesenteric fat stranding might be detected on CT, which also can be seen in infectious condition.
• Low-intensity uptake of 18F-FDG may be helpful for differentiation.

**Pancreas**

Autoimmune pancreatitis

• Autoimmune pancreatitis shows similar findings on CT with pancreatitis resulted from alcohol or stone. Diffuse or segmental edematous change of pancreas, with infiltration of peripancreatic fat, necrosis can be seen.
• Histopathologically it shows aggregatuib of CD4- or CD8-positive lymphocytes and IgG4-positive plasma cells.
• These findings are associated with increased 18F-FDG uptake (figure 5).

**Spleen**

Sarcoidosis

• Sarcoidosis involving the spleen can lead to increased uptake of 18F-FDG.
• Non-caseating granulomas are associated with increased 18F-FDG uptake, and lymph node involvement can be accompanied.

**Mesentery**

Sclerosing mesenteritis

• Sclerosing mesenteritis is a benign disease entity of unknown etiology affecting the small bowel mesentery.
• Various neoplastic conditions such as lymphoma, gastrointestinal tumors, and urogenital tumors, has been reported.
• Histologically, sclerosing mesenteritis show fat necrosis.
• High, false-positive 18F-FDG uptake can be seen.

4. Imaging pitfalls

**Iatrogenic change**

Wound repair
• Medical procedure can cause focal 18F-FDG uptake, such cases of ostomy (ex, colonostomy, ileostomy) or various indwelling stents. Healing process involves an inflammatory reaction even in the absence of infection.
• In the granulation tissue, leukocytic infiltration can be present, associated with wound repair and resorption of necrotic debris and hematoma.
• The granulation tissue associated with resorption of a hematoma or thrombus also results increased 18F-FDG uptake (figure 6).

Immediate postoperative change

• PET taken on immediate postoperative period can show increased uptake of 18F-FDG and may be indistinguishable from uptake in peritoneal malignancy: it can be avoided if PET is performed at least 4 to 6 weeks after surgery.

Radiation therapy

• Radiation induces inflammatory changes and increases glucose metabolism: so it could be better that PET would not be not performed until 2 to 3 months after radiation or chemoradiation.

**Epiploic appendage**

Primary epiploic appendagitis

• Primary epiploic appendagitis results from torsion or thrombosis of one of the fatty epiploic appendages projecting from the colonic serosa.
• Primary epiploic appendagitis may mimick malignancy, associated with increased 18F-FDG uptake and on PET. However, thickening of the visceral peritoneum and hazy infiltration of the fat within the appendage make diagnosis possible.

**Images for this section:**
**Fig. 1:** Table 1. Benign nonphysiologic conditions with increased 18F-FDG in the abdomen

<table>
<thead>
<tr>
<th>Organ</th>
<th>Lesion type</th>
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<th>Lesion type</th>
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<tbody>
<tr>
<td>Esophagus</td>
<td>Esophagitis</td>
<td>Liver, gall bladder, biliary tree</td>
<td>Cyst, hydatid cyst, Bacterial, fungal, amebic, Echinococcal abscess</td>
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<td>Stomach</td>
<td>Gastritis, gastric ulcer</td>
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<td>Acute cholangitis, Acute cholecystitis</td>
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<td>Bowel</td>
<td>Enterocolitis, diverticulitis, Inflammatory bowel disease (Crohn’s disease, ulcerative colitis), Perianal fistula, typhilitis, Acute radiation enterocolitis, Colostomy</td>
<td>Spleen</td>
<td>Granulomatous infection (Tuberculosis, Brucella melitensis,) Sarcoidosis</td>
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<td>Appendix</td>
<td>Appendicitis</td>
<td>Peritoneum and mesentery</td>
<td>Peritonitis, abscess, fistula, Sclerosing mesenteritis, Primary epiploic appendicitis</td>
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</table>
Fig. 2: Figure 1. A 75 year-old female with chronic colitis. (a) Focal wall thickening with pericolic fat infiltration in hepatic flexure (arrow).
**Fig. 3:** Figure 1. (b) Increased 18F-FDG uptake in hepatoc flexure with SUV = 4.4 (arrow). Pathologic diagnosis revealed chronic colitis.
Fig. 4: Figure 2. A 41 year-old female with non-neoplastic biliary stricture. (a) Ill-defined focal low attenuation in S1 (arrow) associated with intrahepatic duct dilatation (arrowhead).
Fig. 5: Figure 2. (b) Increased 18F-FDG uptake in S1 with SUV = 3.8 (arrow). Pathologic diagnosis was periductal lymphocytic infiltration, revealing focal inflammatory change of liver.
Fig. 6: Figure 3. A 68 year old female with tuberculous Lymphadenitis. (a) Multiple enlarged lymph nodes in left gastric, porta hepatis (arrow), perisplenic (arrowhead), portocaval space, peripancreatic area, paraaortic area, right cardiophrenic angle, and around distal esophagus.
**Fig. 7**: Figure 3. (b) Hypermetabolic lymph nodes in left gastric, porta hepatis (arrow), perisplenic area (arrowhead), portocaval space, peripancreatic area, paraaortic area, right cardiophrenic angle, and around distal esophagus. (SUV = 5.4 - 17.5) Pathologic diagnosis revealed chronic granulomatous inflammation with caseation necrosis, consistent with Tbc lymphadenitis. Some lymph nodes show internal low attenuation foci suggesting necrosis.
**Fig. 8:** Figure 4. A 71 year old male with adrenal tuberculosis. (a) Diffuse nodular thickening of left adrenal gland (arrow).
**Fig. 9:** Figure 4. (b) Increased 18F-FDG uptake in left adrenal gland with SUV = 6.2 (arrow). Pathologic diagnosis revealed chronic granulomatous inflammation with caseous necrosis, consistent with tuberculosis.
Fig. 10: Figure 5. A 74 year old male with autoimmune pancreatitis. (a)Swelling of pancreas (arrow) and peripancreatic fluid collection (arrowhead).
**Fig. 11:** Figure 5. (b) Marked uptake of 18F-FDG pancreas head (SUV = 6.4) (arrowhead) and heterogeneous uptake in pancreas body and tail (arrow).
**Fig. 12:** Figure 6. A 66 year-old male with history of hemicolecction for colon cancer. (a) Abnormal wall thickening at the colonic anastomotic site and enhancing soft-tissue mass (arrow) along the SMA.
Fig. 13: Figure 6. (b) Increased 18F-FDG uptake in the soft tissue mass with SUV = 9.6 (arrow). Pathologic diagnosis revealed inflammatory fibrosis.
Conclusion

We sometimes encountered inflammatory lesions indicated as malignancy by PET, and their final diagnoses were made only after operation. This exhibit can be familiarized the radiologists with relevant CT imaging and PET appearances of inflammatory lesions mimicking malignancies in the abdomen.

Radiologists should understand the normal physiologic distribution and different benign nonphysiologic conditions to optimize appropriate interpretation.

Personal Information

References