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PET/CT Variants and Pitfalls in Liver, Biliary Tract, Gallbladder and Pancreas

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A wide variety of pathological anomalies may occur in the liver, biliary system, and pancreas. It is a necessity to use many different imaging techniques in order to distinguish such varied pathologies, especially those from malignant processes. Positron Emission Tomography/Computed Tomography (PET/CT) is an imaging method that has proven its diagnostic value in oncology and can be used for different clinical purposes. Fluoro-18 fluoro-2-deoxy-D-glucose has a wide range of uses as a dominant radiopharmaceutical in routine molecular imaging, however, molecular imaging has started to play a more important role in personalized cancer treatment in recent years with new Fluoro-18 and Gallium-68 labeled tracers. Although molecular imaging has a strong diagnostic effect, the surprises and pitfalls of molecular imaging can lead us to unexpected and misleading results. Prior to PET/CT analysis and reporting, information about possible technical and physiological pitfalls, normal histological features of tissues, inflammatory pathologies, specific clinical features of the case, treatment-related complications and past treatments should be evaluated in advance to avoid misinterpretation. In this review, the physiological and pathophysiological variants as well as pitfalls encountered in PET/CT imaging of the liver, biliary tract, gallbladder, and pancreas will be examined. Other benign and malignant pathologies that have been reported to date and that have led to incorrect evaluation will be listed. It is expected that the devices, software, and artificial intelligence applications that will be developed in the near future will enable much more effective and faster imaging that will reduce the potential causes of error. However, as a result of the dynamic and evolving structure of the information obtained by molecular imaging, the inclusion of the newly developed radiopharmaceuticals in routine practice will continue to carry new potentials as well as new troubles. Although molecular imaging will be the flagship of diagnostic oncology in the 21st century, the correct analysis and interpretation by the physician will continue to form the basis of achieving optimal performance.

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Introduction

In recent years, molecular imaging has become an indispensable part of diagnostic oncology. With widespread use of Fluorine-18 (^{18}F) and Gallium-68 (^{68}Ga) labeled radiopharmaceuticals, which have become much easier to use in recent years, high diagnostic impact has been achieved. PET imaging with PET/CT or PET/MRI is also increasingly being used to see the molecular properties of malignancies in the hepatopancreaticobiliary system. Undoubtedly, the first radiopharmaceutical that comes to mind in PET imaging is ^{18}F fluoro-2-deoxy-D-glucose (FDG), which is a glucose analog. ^{18}F-FDG PET/CT has offered fast, accurate, and practical advantages in staging, recurrence, evaluation of treatment response, and prognosis in many malignant diseases.[1] However, the risk of false positive or false negative results in ^{18}F-FDG PET imaging will never be reduced to zero, and there is still a need for different radioactive agents that are more specific, targeted, and better suited for theranostic purposes.

^{68}Ga labeled PET imaging agents have rapidly taken their place in routine clinical practice in recent years. ^{68}Ga is a highly popular radionuclide in PET applications due to its ease of use, appropriate half-life, easy binding with targeted

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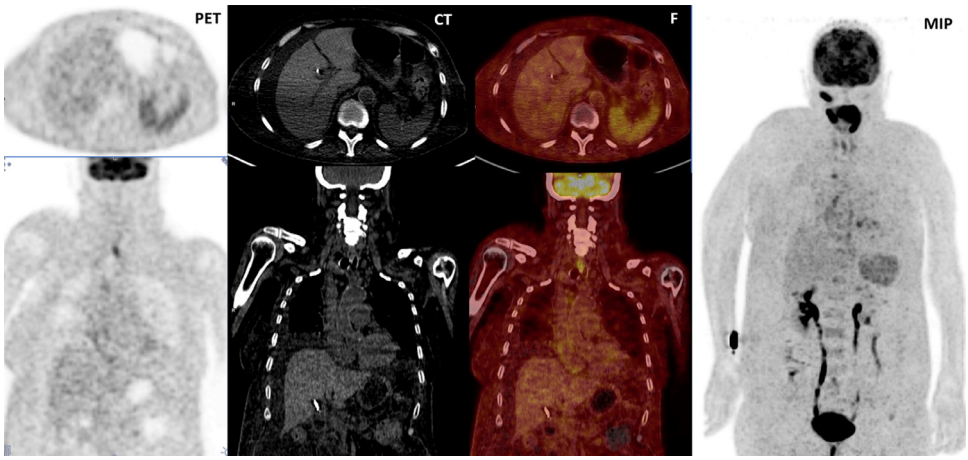

Figure 1 A 76-year-old woman with idiopathic thrombocytopenic purpura and follicular lymphoma, has hypoalbuminemia. A diffuse decrease ^{18}F-FDG uptake was observed in the liver parenchyma. (F: fusion images, MIP: maximum intensity projection).

peptides and ligands, and acceptable radiation dose for patient safety.[2] Somatostatin receptors are widely expressed on the surface of neuroendocrine cells.[3] ^{68}Ga-DOTATATE, DOTANOC, and DOTATOC are prominent somatostatin analogs in PET/CT imaging. These radiopharmaceuticals have become the preferred radioactive agents due to their high diagnostic accuracy and specific molecular target potential, especially in tumors with high somatostatin receptors expression such as pancreatic neuroendocrine tumors (NETs).[4] Another ^{68}Ga labeled radioligand that has recently been used in molecular imaging is a prostate-specific membrane antigen (PSMA), which is a transmembrane glycoprotein. Since PSMA is expressed 100-1000 times higher in prostate cancer (PC) tissue than normal tissue, it is widely used for PC diagnosis, staging, and follow-up. However, the presence of PSMA expression in non-prostate malignancies seems to have a potential in the differential diagnosis of some benign and malignant diseases due to its relationship with neovascularization.[5–7] Since various liver, gallbladder and pancreas pathologies show PSMA expression, it is also discussed in this paper.

Regardless of the radiopharmaceutical used, it is important to have information about the technical pitfalls in PET/CT imaging. Potential technical pitfalls include misregistration (anatomical alignment error-incorrect superposition, respiratory movements or movement of the patient during imaging), truncation (PET and CT visual field differences, loss of CT images), attenuation correction, and partial volume effect. In the presence of such pitfalls, small-sized tumors and lesions adjacent to high activity areas are especially affected. The bile ducts, gallbladder, pancreas, and especially the liver, which is the target organ in many malignant diseases, have a large number of nonmalignant diseases that accompany the main disease or can mimic the malignancy and may appear on PET images. In addition, surgical treatments and interventional procedures performed before PET/CT imaging, radiation therapy, effects due to chemotherapy and complications secondary to any treatment may lead to inflammatory processes, causing changes in tissue properties and thus potential entrapment.[1,8] The technical pitfalls and artifacts in PET/CT imaging are beyond the scope of this review. However, physiological uptake differences that may lead to evaluation errors, PET findings related to conditions to other diseases or treatment complications will be discussed, and clinical information that will facilitate reaching the correct diagnostic interpretation will be assessed.

Physiological/Physiopathological Factors on ^{18}F-FDG uptake in Liver and Pancreas

Physiological pitfalls include the normal physiological distribution of radiopharmaceuticals and the differences caused by the specific histological features of each tissue. Several studies have reported that hyperglycemia has no significant effect on ^{18}F-FDG uptake in the liver parenchyma on PET/CT imaging.[9] Sarıkaya et al., in a study on patients undergoing ^{18}F-FDG PET/CT imaging with different blood glucose levels, found that SUVmean obtained from hyperglycemic patients was not different from normoglycemics, while it was calculated that liver activity decreased significantly in hypoglycemic patients compared to normoglycemics.[10] Similarly, diffuse low ^{18}F-FDG uptake in the normal liver parenchyma has been reported in patients with hypoalbuminemia[11] (Fig. 1). Another relatively common condition that has an effect on ^{18}F-FDG uptake is hepatosteatosis. Changes due to fat in the liver can be in different characteristic patterns in diffuse, geographical, focal, subcapsular, and perivascular forms. It has been reported that ^{18}F-FDG uptake is significantly reduced in diffuse fatty liver compared to normal liver.[12] This pathology generally does not have a clinically meaningful result in ^{18}F-FDG PET/CT evaluation. However, focal hepatic infiltration may have intense ^{18}F-FDG uptake, and this involvement can be interpreted in favor of malignant pathology.[13,14] On the other hand, the relative increase in normal ^{18}F-FDG uptake of the parenchyma protected from fat compared to the decreased uptake of the diffuse fatty parenchyma may also lead to erroneous interpretation.[15] Another issue affecting uptake in the liver parenchyma is the distribution time between ^{18}F-FDG injection and imaging.

Table 1 Liver Pathologies with High ^{18}F-FDG Uptake in PET/CT

Non-tumoral Processes
1. Medical treatments
Filgrastim (focal uptake)[23]
Ustekinumab (diffuse uptake)[24]
2. Radiation therapy
Oesophagus Cancer (focal uptake)[25,26]
3. Surgery (Focal)
Surgical Retractor Injury[27]
Metastasectomy[28]
4. Percutaneous interventions (focal or ring-like uptake)
Radiofrequency or microwave[30]
5. Transplantation (multifocal uptake)
Inflammatory and ischemic involvement in bile ducts[29]
Infectious Diseases
1. Focal Uptake
Hydatid cyst[31]
Hepatic or peritoneal tuberculosis[32,33]
Hepatosplenic candidiasis[34]
Hepatic schistomatozis[35]
Aerococcus viridians abscess[36]
Hepatosplenic Actinomycosis[37]
2. Ring-like Uptake
Amoebic abscess[38]
Hydatid cyst[39]
3. Diffuse Uptake
Q fever[40]
Amoebiasis[41]
Tuberculosis[42]
Granulomatous Diseases
1. Focal Uptake
Sarcoidosis[43,44]
Sarcoidosis-lymphoma syndrome[46,47]
IgG related inflammatory pseudotumor[48,49]
Langerhans cell histiocytosis[50]
2. Diffuse Uptake
Sarcoidosis[45]
Rare diseases other than liver
Diffuse Uptake
Protoporphyria[51]
Benign liver tumors
Focal uptake
Hepatocellular adenoma[52,53]
Liver mycopericytoma[54]
Rare malignant diseases
1. Focal uptake
Multiple Myeloma[73]
Primary hepatic lymphoma[74]
Hepatic sarcomatous cholangiocarcinoma[75]
Primary neuroendocrine carcinoma[76]
Malignant peritoneal mesothelioma[77]
Hepatic epithelioid hemangioendothelioma[78]
Hepatic angiosarcoma[79]
2. Diffuse uptake
Hepatic epithelioid hemangioendothelioma[80]
Lymphoma[81,82]
Metastasis[83]

Cheng et al. reported that similar to the blood pool in the spleen, a long distribution time of 2-3 hours in the liver causes a decrease in both SUVmean and SUVmax.[16] For all these reasons, it should not be ignored that there may be significant changes in physiological ^{18}F-FDG uptake in the liver parenchyma, independent of the primary disease, in PET/CT evaluation. It should be noted that this background involvement may have even more important consequences for the scoring used in the evaluation of lymphoma and head and neck tumors.

Although aging does not have a significant effect on liver volume, some studies revealed that functional hepatocyte volume and total hepatic glucose synthesis decreases.[17,18] It has been reported that a large number of metabolic factors, varying from this functional loss with aging, may play a role.[19] On the other hand, Meier et al., later Lin et al., and with a much larger patient series, Cao et al. reported that ^{18}F-FDG uptake increased in the liver with aging.[19-21] According to the study data of Cao et al., an increase of up to 2 times the average ^{18}F-FDG uptake was measured in the normal liver parenchyma in older age groups compared to children. Despite decreasing hepatic glucose synthesis with aging, the most plausible hypothesis as the cause of increased parenchymal ^{18}F-FDG uptake may be the cumulative inflammatory changes secondary to the release of age-related hepatotoxins suggested by Meier et al.[19]

For the pancreas, the most important situation is the changes caused by diabetes, which is a very common problem today, as well as the treatments for diabetes. Pancreatic volume loss in patients treated with insulin was reported by Gilbeau et al.[22] Although there is a partial decrease in pancreatic exocrine and endocrine functions with aging in nondiabetics, Meier et al. reported that pancreatic ^{18}F-FDG uptake does not differ between age groups.[19] It is seen that the most important situation for the pancreas in PET/CT evaluation is the potential changes that the presence of diabetes can make on the organ.

Liver

^{18}F-FDG uptake in Benign Processes and Diseases

Detection of liver metastasis plays a very important role in the tumor staging and results in significant changes in the treatment strategy. The use of ^{18}F-FDG PET/CT is generally accepted for the detection of liver metastases in many cancers and has become an important component of diagnosis. However, for the correct interpretation of pathological processes with PET/CT imaging, the ^{18}F-FDG uptake patterns of the liver in physiological and non-oncological diseases must be well understood (Table 1). Before PET/CT evaluation, all

medical treatments the patient receives should be noted in detail, whether for oncology reasons or not. For example, the use of granulocyte colony stimulating factor "Filgrastim" can lead to extramedullary hematopoiesis in the liver, and in this case focal intense ^{18}F-FDG uptake can be observed,[23] and also diffuse high ^{18}F-FDG uptake in the liver has been reported with the use of Ustekinumab, a human monoclonal antibody.[24] These reports show that many of the medical treatment agents coming into use may have potential unexpected effects on the liver. On the other hand, radiation treatments applied to regions close to the liver may cause focal intense uptake in the parenchyma, such as in treatment of distal esophagus cancers.[25,26] Similarly, it has been reported that inflammatory focal ^{18}F-FDG involvement may occur after surgical interventions to the liver, even as a result of relatively minor trauma such as surgical retraction[27] as well as major surgeries such as metastasectomy.[28] Intense ^{18}F-FDG uptake caused by severe inflammatory and ischemic changes including bile duct necrosis, acute cholangitis, bile duct obstruction, and periportal fibrosis after liver transplantation can mimic malignancy.[29,30] In addition, in the period following percutaneous ablative approaches, focal or ring-like intense ^{18}F-FDG uptake can be seen in the intervention areas in the first months due to inflammation[29] (Fig. 2).

Although it is easy to access patient information regarding these treatments or interventions before PET/CT imaging, surprising liver involvement, which infective, inflammatory, or granulomatous diseases that may be misinterpreted as metastases, of which the presence is not known, may be encountered frequently. Focal intense involvements in many infections that affect the liver, such as hepatic echinococcosis,[31] hepatic or peritoneal tuberculosis,[32,33] hepatosplenic candidiasis,[34] hepatic schistosomiasis,[35] and hepatic aerococcus viridans abscess,[36] hepatosplenic Actinomycosis,[37] and ring-like ^{18}F-FDG involvements in amoebic abscess[38] and hydatid cyst[39] have been reported. In addition to these uptake patterns, unexpected diffuse intense increased pathological ^{18}F-FDG uptake in the liver has been reported in Q fever,[40] amoebiasis,[41] and tuberculosis.[42] Among these infections, tuberculosis stands out as a more common problem. In particular, miliary tuberculosis (MT), a disseminated form of tuberculosis, has caused many misdiagnoses and unnecessary invasive procedures. While MT is seen in 2% of the population, it is reported tens of times more in people with immunosuppression or malignancy. Di Renzo et al. observed ^{18}F-FDG uptake that did not correlate with CT in the liver of a 76-year-old male patient diagnosed with multiple myeloma. Upon this, while the malignancy was not confirmed in the biopsy material they took, acid- resistant bacilli were observed with Ziehl-Neelsen staining, and a diagnosis of tuberculosis was made. In 2 patients with MT, ^{18}F-FDG PET/CT imaging led to an incorrect diagnosis of malignancy, and one patient underwent diagnostic laparoscopy and the other hepatic resection.[33]

Focal or diffuse ^{18}F-FDG uptake due to granulomatous diseases can be observed in PET/CT imaging and may lead to evaluation errors. Although focal uptake[43,44] can often be observed in the liver in sarcoidosis, unexpectedly diffuse liver involvement has also been reported.[45] Similarly, focal intense uptake similar to malignant pathologies in sarcoidosis-lymphoma syndrome,[46,47] IgG-related inflammatory pseudotumor (IP),[48,49] and Langerhans cell histiocytosis[50] have been reported. Unexpected diffuse liver involvement has been demonstrated in X-linked protoporphyria, which is a rare subtype of protoporphyria.[51]

Increased ^{18}F-FDG uptake in primary benign tumors of the liver can lead to interpretation errors and may be evaluated as malignant. ^{18}F-FDG uptake is not generally expected in hepatocellular adenomas, but high radiopharmaceutical uptake has been presented in some case reports.[52,53] Sumiyoshi et al. reported increased ^{18}F-FDG uptake consistent with malignancy in a mass observed in the liver. Partial hepatectomy was performed, and the lesion was diagnosed as hepatocellular adenoma after histopathological evaluation.

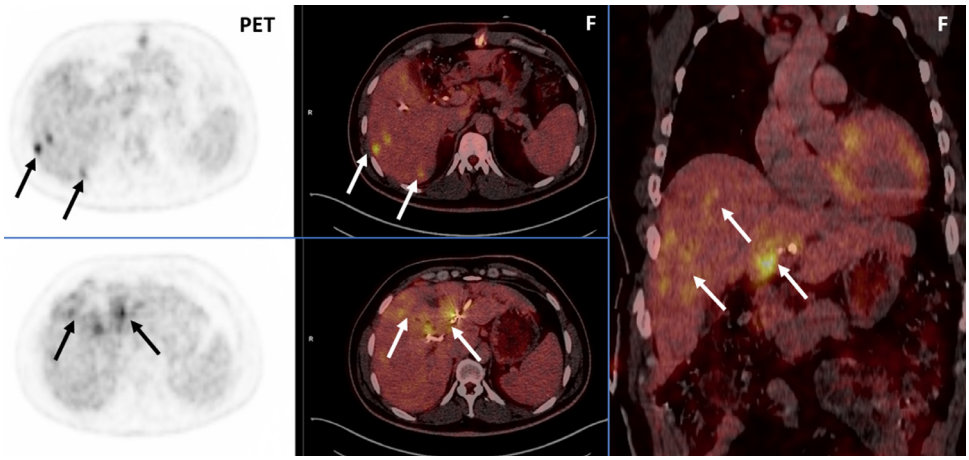

Figure 2 A 42-year-old man followed up post surgically for cholangiocarcinoma. There are multiple focal increased ^{18}F-FDG uptakes (arrows) in PET/CT that were not observed in the previous study. When these involvements mimicking metastasis were evaluated together with the patient's clinical findings and MRI results, it was thought to be compatible with cholangitis and multifocal abscess foci. (F: fusion images).

This pathology, which is often characterized by an increase in inflammatory cells or an increase in cell density, has been reported to be caused by HNF1 alpha mutation. It is thought that there is a relationship between increased ^{18}F-FDG uptake and this mutation.[52] Kang et al. reported a case of multiple ring-like ^{18}F-FDG uptake of myopericytoma, which is a benign tumor, and the patient underwent partial hepatectomy with suspicion of malignancy.[54] Cavernous hemangiomas are the most common benign hepatic tumors with an incidence of up to 20%. In PET/CT, hemangiomas almost always show ^{18}F-FDG uptake at the same level as normal liver parenchyma. However, moderate ^{18}F-FDG accumulation may make the diagnosis difficult rarely. Kaida et al. showed that a mild increase in ^{18}F-FDG activity accordant with a mural nodule in cystic tumor on PET/CT imaging. The patient was diagnosed with a typical biliary cystadenocarcinoma and received extended right hepatectomy, however, diagnosis of degenerative hemangioma was made as a result of histopathology.[55] In benign liver tumors such as focal nodular hyperplasia and angiomyolipoma, ^{18}F-FDG uptake is observed at the same level with normal liver parenchyma, and metabolic imaging findings can be confused with well-differentiated hepatocellular carcinoma (HCC) from malignant lesions. However, differential diagnosis can be made with high accuracy with MRI performed before PET/CT in these patient groups, and focal nodular hyperplasia and angiomyolipoma do not cause a major problem in daily PET/CT practice.

The bile ducts do not show ^{18}F-FDG uptake in a normal healthy individual. However, there are pitfalls in PET/CT imaging of the biliary tract as in all hepatobiliary systems. Cholangitis and cholangitic abscesses are important complications of bile drainage tubes and stents. Although it is difficult to differentiate these lesions from malignancies, the inflammatory process should be considered first due to the information obtained about the application. Causes of bile duct abscess and inflammation, such as sclerosing cholangitis, cholangitis, and cholecystitis, may cause false positive interpretation, but it should be kept in mind that they may also mask an underlying malignant lesion.[56] In differential diagnosis of false positive ^{18}F-FDG activity caused by inflammation that can be seen after tumor-induced bile retention, chemotherapy, or invasive procedures such as intraluminal bile duct stents, late imaging may contribute to the correct identification of these involvements.

On the other hand, the lack of ^{18}F-FDG uptake in benign tumors or lesions diagnosed as malignant by other radiological methods contributes to the correct diagnosis. Hepatic sclerosing hemangioma is a degenerative tumor associated with hepatic cavernous hemangioma, characterized by fibrosis and hyalinization. Atypical hemangiomas can be misdiagnosed as primary or metastatic tumors in morphological imaging methods. Yugawa et al. reported that PET/CT imaging did not show any ^{18}F-FDG affinity in multiple hepatic sclerosing hemangioma lesions confirmed histopathologically after lobectomy or partial hepatectomy. Researchers underlined that this atypical appearance of sclerosing hemangiomas can be easily confused with HCC in the cirrhotic liver.[57] Hepatic segmental atrophy and nodular elastosis is rare benign lesion in the liver. This focal liver lesion characterized by loss of hepatic parenchyma and a continuing inflammatory process ultimately leads to fibrotic changes. Gang et al. reported that pathological ^{18}F-FDG activity was not observed in these lesions, which are generally evaluated as malignant by other radiological methods.[58] Oxaliplatin-based chemotherapy is a preferred method in the adjuvant treatment of colon cancer. Focal sinusoidal obstruction syndrome is a liver injury that occurs as a complication of oxaliplatin treatment and can mimic metastatic disease on MRI. On the other hand, it has been shown that ^{18}F-FDG uptake is not observed in these lesions, and PET/CT contributes to the correct diagnosis.[59]

^{18}F-FDG Uptake in Primary Malignant Liver Tumors

^{18}F-FDG uptake is observed at a similar level to normal liver parenchyma due to low GLUT-1 and GLUT-2 expression and high glucose-6-phosphatase activity in well and moderately differentiated HCCs, while ^{18}F-FDG affinity is generally high in poorly differentiated HCC.[60] The sensitivity of ^{18}F-FDG PET/CT is limited and is between 50-65%, however high uptake shows poor histopathological differentiation and a more aggressive clinical course.[61] High glycometabolic activity is a predictor for recurrence and poor survival after both transarterial and liver transplantation.[62,63] Moreover, it is possible to detect some HCC foci, which are difficult to diagnose morphologically, with ^{18}F-FDG PET/CT. Adachi et al. diagnosed ectopic HCC, for the first time in the literature, by confirming histopathologically after observing intense ^{18}F-FDG uptake in the dorsal of the pancreatic corpus with PET/CT.[64] Again, Dong et al. contributed to the diagnosis of pedunculated HCC, which may be confused with a right adrenal gland tumor, with ^{18}F-FDG PET/CT imaging.[65]

The most common liver tumor after HCC is intrahepatic cholangiocarcinoma. Intrahepatic cholangiocarcinoma is divided into 2 subgroups as the peripheral type (originating from the small bile ducts) and the perihilar type (originating from the large bile ducts), and there are differences in ^{18}F-FDG uptake patterns between subgroups. While a nodular form and intense uptake is observed in the peripheral type, which constitutes 90% of patients, the remaining perihilar type has an infiltrating feature and low ^{18}F-FDG uptake pattern.[66,67] Apart from these 2 subtypes, another rare subtype is cholangiolocellular carcinoma originating from the small bile ducts. Low ^{18}F-FDG uptake and false negative reporting can be made in PET/CT images in cases with this subtype.[8] Takamura et al. reported that patients with cholangiocellular carcinoma achieved significantly lower SUVmax than cholangiocarcinoma, and also they concluded that the method was a good indicator for recurrence due to significantly increased ^{18}F-FDG uptake in cases with recurrence.[68] However, a case with cholangiolocellular carcinoma with high ^{18}F-FDG uptake was reported by Mori et al.[69] This shows that different uptake patterns can be observed in cholangiocellular carcinomas. Late PET imaging may contribute

to the correct interpretation of low ^{18}F-FDG uptake that may exist in cholangiocarcinoma. A tumor lesion with low uptake value in early images may show increased uptake in late images, contributing to the diagnosis of malignancy.[70] On the other hand, ^{18}F-FDG PET/CT imaging of cholangiocarcinomas is important in detecting nodal staging and metastasis, and its importance in detecting recurrence in previously resected cases has also been documented.[71,72]

^{18}F-FDG uptake in other relatively rare malignant tumors of the liver can also lead to pitfalls in evaluation. These rare malignant diseases that cause focal uptake include multiple myeloma,[73] primary hepatic lymphoma,[74] primary intrahepatic sarcomatosis cholangiocarcinoma,[75] and primary liver neuroendocrine carcinoma[76] and mesothelioma.[77] Hepatic epithelioid hemangioendothelioma (HEHE) is a very rare vascular tumor with uncertain prognosis. HEHE may also show low or slightly increased glucose metabolism compared to normal parenchyma in ^{18}F-FDG PET/CT.[78] It is rarely reported in the literature that hepatic angiosarcoma shows weak, multiple, focal, and inhomogeneous involvement on ^{18}F-FDGPET/CT.[79] Furthermore, HEHE,[80] lymphoma,[81,82] and liver metastases[83] are fewer common causes of diffuse ^{18}F-FDG uptake pattern (Fig. 3).

^{18}F-FDG PET/CT may also play an important role in rare neoplasms of the biliary tract. Tu et al. observed increased ^{18}F-FDG uptake in multiple locations in the intrahepatic bile ducts and common bile duct in the PET/CT in a patient who presented with cholangitis and obstructive jaundice. After resection, the rare diagnosis of biliary papillomatosis with high malignancy potential was confirmed.[84] Ikeno et al. reported that ^{18}F-FDG uptake was significantly higher in preoperative invasive biliary papillomatosis or intraductal papillary neoplasms compared to non-invasive lesions.[85]

^{68}Ga-DOTA-Peptides and ^{68}Ga-PSMA in Liver Neoplasm and Diseases

PET/CT with ^{68}Ga-DOTA-peptides is effectively used in daily practice in the detection of metastatic lesions, especially in

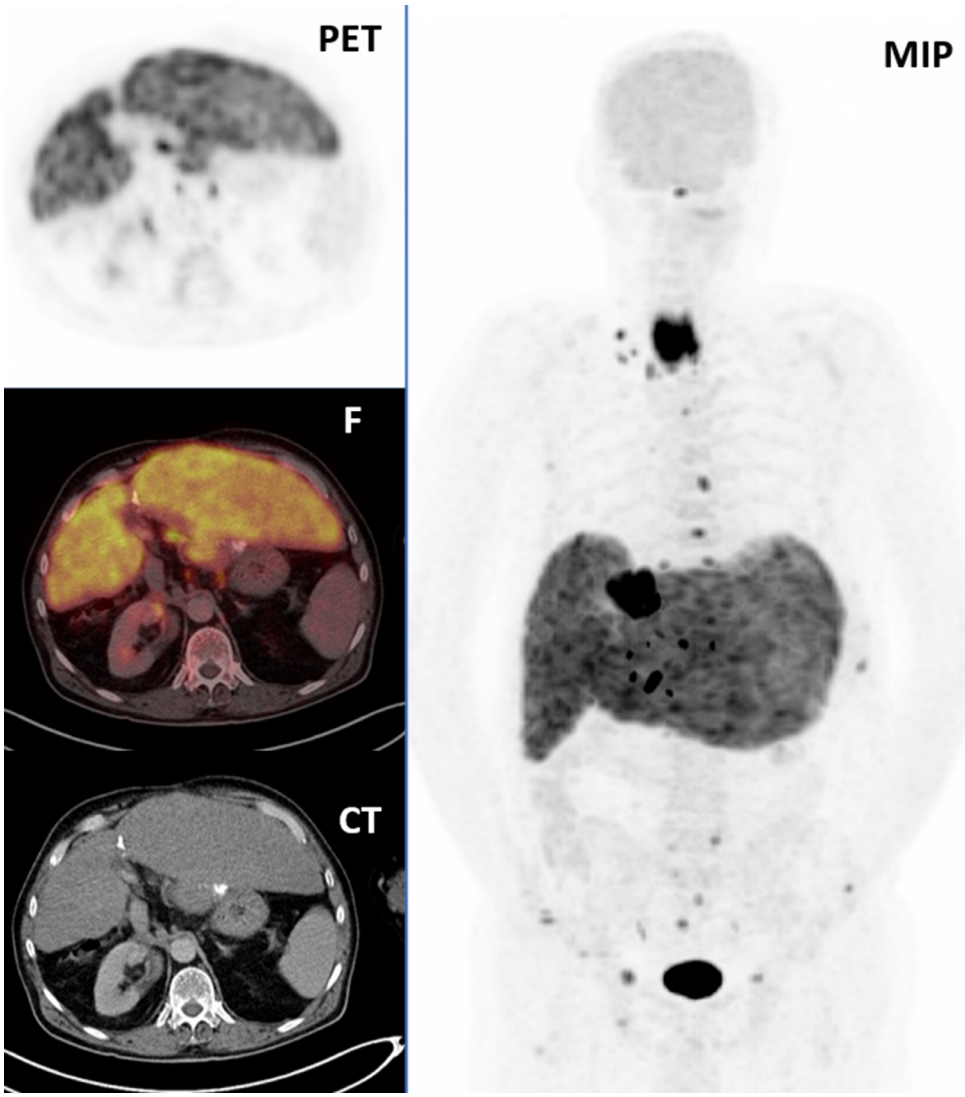

Figure 3 A 76-year-old woman with diffuse large B-cell lymphoma showed intensive diffuse ^{18}F-FDG uptake in the liver on PET/CT images for restaging study. (F: fusion images, MIP: maximum intensity projection).

well- and moderately differentiated NETs. More than half of the NETs originate from the gastrointestinal tract and pancreas, and metastases are frequently observed in the liver. Although extremely rare, cases of primary hepatic NETs have been reported using ^{68}Ga-DOTA-peptides imaging.[86] However, the activity observed in the liver with ^{68}Ga-DOTA-peptides must be accurately assessed as there are potential pitfalls to arise during NET research such as high ^{68}Ga-DOTANOC or ^{68}Ga-DOTATATE uptakes in some HCC.[87,88] Dual metabolic imaging using ^{68}Ga-DOTA-peptides and ^{18}F-FDG PET/CT together is a useful approach to evaluate the effectiveness of treatment.[89] Although ^{68}Ga-DOTA-peptides uptake by normal liver parenchyma is higher than ^{18}F-FDG, diffuse and homogeneous physiological uptake is observed. Hod et al. observed focal hepatic uptake in ^{68}Ga-DOTATATE PET/CT in the follow-up of a patient with lung carcinoid tumor, and the lesion was thought to be metastatic. However, physiological involvement of the area protected from focal fat in the liver parenchyma accompanied by widespread steatosis in the background in correlative imaging with MRI was found to lead to a false positive evaluation[90] (Table 2).

The liver is the third most common organ in which distant metastasis is observed in PC. On the other hand, the liver is among the organs in which intense physiological expression is observed in the biodistribution of PSMA, and it has also been reported that ^{68}Ga-PSMA is expressed in hepatobiliary system malignancies such as HCC and cholangiocarcinoma.[91-93] Comparative studies in HCC patients reported that ^{68}Ga- PSMA expression is superior to ^{18}F-FDG, and dual imaging with these radiotracers may be appropriate in some studies.[94,95] (Fig. 4). Kuyumcu et al. reported that both ^{18}F-FDG and ^{68}Ga-PSMA uptake were negative in 2 patients, ^{18}F-FDG uptake was higher than ^{68}Ga-PSMA in 4 patients, and ^{68}Ga-PSMA expression was higher than ^{18}F-FDG uptake in 9 of 17 patients with HCC.[95] Chen et al. evaluated vascular and peritumoral PSMA expression with immunohistochemistry, and expression was observed in primary cholangiocarcinoma (86.7%). However, they did not observe expression in pancreatobiliary adenocarcinoma and metastatic liver lesions, and also PSMA expression was not observed in any of the 5 cases diagnosed with benign hepatic adenoma in this study.[96]On the other hand, Bhardwaj et al. found intense ^{68}Ga-PSMA expression in a patient with hepatic hemangioma.[97] This involvement may lead to erroneous interpretation in favor of metastatic disease. Increased ^{68}Ga-PSMA expression can be observed in other benign liver pathologies such as sarcoidosis.[98] In a recent review by Galiza Barbosa et al., all nonprostatic diseases and normal variants with ^{68}Ga-PSMA expression are listed. Variants showing higher ^{68}Ga-PSMA expression compared to liver and biliary parenchyma are biliary tract and liver perfusion defects[5] (Fig. 5) (Table 2).

Table 2 Physiological Variants, Benign and Rare Malignant Diseases with High Uptake in ^{68}Ga-DOTA-Peptides and ^{68}Ga-PSMA

^{68}Ga-DOTA-Peptides	^{68}Ga-PSMA
Liver	
Physiologic variants	*Physiologic variants*
• Physiological uptake of the area protected fat liver[90]	• Liver perfusion defects[5]
Malignant Diseases	*Benign Diseases* / *Malignant Diseases*
• Primary hepatic NET[86]	• Hemangioma[97] / • HCC[94,95]
• HCC[87,88]	• Sarcoidosis[98] / • Cholangiocarcinoma[92,93]
Gall Bladder	
^{68}Ga-DOTA-Peptides	^{68}Ga-PSMA
Benign Disease	*Physiologic variant*
• Paraganglioma[121]	• Normal biliary excretion[122,123]
Pancreas	
^{68}Ga-DOTA-Peptides	^{68}Ga-PSMA
Physiologic variants	*Physiologic variants*
• Physiological uptakes of caput and uncinate process[138-141]	• Splenosis, accessory spleen (Fig 10)
• Splenosis and accessory spleen[142]	
• Intrapancreatic splenic tissue[143-145]	
• Heterotopic pancreas[146]	
Malignant Disease	*Malignant Diseases*
• Pancreatic lymphoma[147]	• Ductal adenocarcinoma[7,148]
	• Serous cystadenoma[149]

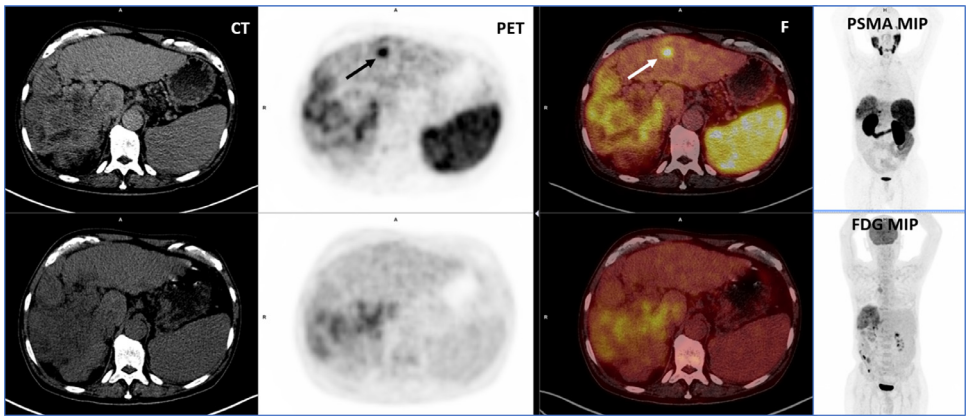

Figure 4 A 61-year-old man with a liver cirrhosis shows heterogeneous increased uptake in a large lesion with poor margins in both ^{18}F-FDG and ^{68}Ga-PSMA PET/CT in the right lobe. Additionally, second lesion in the left lobe (arrows) with no FDG uptake showed expression of ^{68}Ga-PSMA. A diagnosis of hepatocellular carcinoma was made with histopathological examination in both lesions. (F: fusion images, PSMA: ^{68}Ga-PSMA PET/CT, FDG: ^{18}F-FDG PET/CT, MIP: maximum intensity projection).

Other PET Tracers in Liver neoplasm and diseases

The low ^{18}F-FDG uptake of well-differentiated HCCs has opened up an area for the use of other radiopharmaceuticals in metabolic imaging for this patient group. For this purpose, agents that are prominent and are precursors of phosphatidylcholine membrane synthesis that find clinical use are ^{11}C-choline, ^{18}F-fluorocholine, and ^{11}C-acetatate.[99] However, not surprisingly, there are conditions and pathologies that show an unexpected uptake pattern with these radiopharmaceuticals. Lhommel et al. showed that in a case with normal serum AFP level, there was intense ^{11}C-acetate uptake in a suspected liver mass of 7 cm in MRI, while no uptake was observed in ^{18}F-FDG PET/CT. Although metabolic imaging results were compatible with well-differentiated HCC, a diagnosis of benign angiomyolipoma was revealed with histologic evaluation.[100] In a retrospective analysis of ^{18}F-fluorocholine PET/CT imaging results in 368 PC patients by Roland et al., they reported intense radiotracer uptake in a case of hepatic cavernous hemangioma.[101] Physiological radiotracer uptake that can be confused with malignant involvement in focal nodular fat-sparing hepatic parenchyma in ^{11}C-choline PET/CT imaging has been reported.[102]

Gallbladder

^{18}F-FDG Uptake in Benign Processes and Diseases

Choosing the correct imaging method is vital to the differential diagnosis between gallbladder (GB) carcinoma and cholecystitis. Gallbladder carcinoma is the most common tumor of the biliary tract that does not have any specific symptoms; it is diagnosed late due to its indolent course. An incorrect diagnostic process increases mortality and morbidity rates; on the contrary, early diagnosis contributes to good prognosis. ^{18}F-FDG PET/CT is a widely used noninvasive method for determining the metabolic characterization of GB

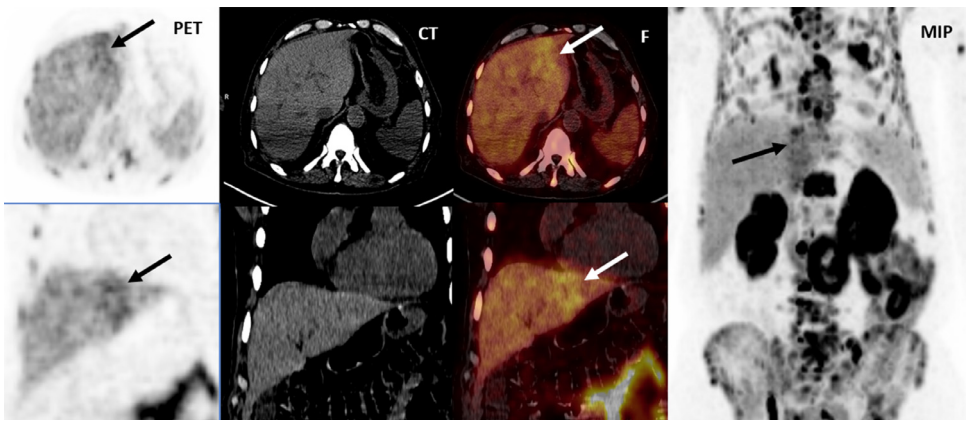

Figure 5 A 76-year-old patient with a Gleason score of 9 (5 + 4) is followed up for multiple metastatic acinar type prostate adenocarcinoma. Heterogeneous intense PSMA expression was observed in the left lobe of the liver (arrows). On MRI, no lesion was detected in the left lobe of the liver and this PSMA expression was accepted as a physiological variant. (F: fusion images, MIP: maximum intensity projection).

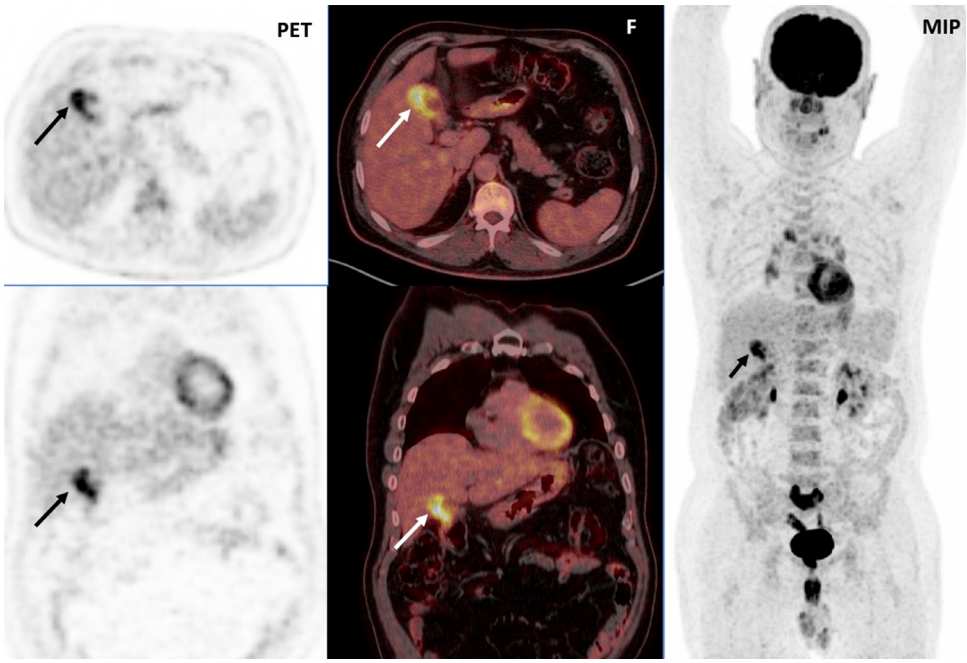

Figure 6 A 57-year-old man with rectal cancer received ^{18}F-FDG PET/CT imaging for staging. PET images showed intensive ^{18}F-FDG uptake in gallbladder walls. The patient underwent surgery for the rectum and gall bladder in the same operation session, and a diagnosis of cholecystitis with xanthogranulomatous pattern was made as a result of histopathological examination. (F: fusion images, MIP: maximum intensity projection).

tumors.[103] Although the low uptake in small or low-grade tumors lead to false negative interpretations for ^{18}F-FDG PET/CT imaging, numerous studies have been published regarding the reliability of the method. Ramos−Font et al., as a result of scanning suspicious lesions in the GB of 49 patients with ^{18}F-FDG PET/CT, 34 malignant tumors and 15 benign lesions were successfully differentially diagnosed. However, they reported a false positive result in a patient with xantogranulomatous cholecystitis and another patient with lymphadenitis caused by granulomatous sarcoidosis.[104] Increased ^{18}F-FDG uptake in cases of xanthogranulomatous cholecystitis has been reported in other case reports[105,106] (Fig. 6). Similarly, hyalinizing cholecystitis a rare cause of GB wall thickening with increased uptake of ^{18}F-FDG, should be considered when making the differential diagnosis.[107] Therefore, it should not be ignored that benign pathologies originating from the GB may cause false positive interpretations.[108] Akimoto et al. observed an improvement in laboratory results with antibiotic treatment in a case who presented with abdominal pain and fever, in which high serum CA 19-9, increased CRP, and a tumor that could not be traced anatomically but showed increased ^{18}F-FDG uptake in PET images. The researchers underlined the difficulties in distinguishing between inflammation and malignancy in ^{18}F-FDG PET/CT imaging and emphasized that high SUVmax can be observed in ^{18}F-FDG uptake similar to GB carcinoma in inflammatory conditions. The diagnosis of acute cholecystitis was confirmed by surgical results after a decrease in serum CA 19-9 levels with antibiotic treatment.[109] Gallbladder tuberculosis is another relatively uncommon disease reported as false positive malignancy in PET/CT.[110,111] Bedmutha et al. made a pre-diagnosis of malignancy due to intense ^{18}F-FDG uptake in PET/CT in a mass lesion with soft tissue density in the GB. However, they concluded that the cause of this mass was persistent ineffective inflammatory response and chronic granulomatous disorder (gallbladder malakoplakia).[112] It has been also reported that gallstones may also show an increased ^{18}F-FDG uptake pattern.[113]

^{18}F-FDG Uptake in Malignant Tumors Other than Primary Adenocarcinomas

Although tumors originating from the GB are almost always adenocarcinoma, intense involvement of tumors such as malignant melanoma,[114] primary GB NET,[115] and extramedullary plasmacytoma of the GB[116] have been reported in the literature. It has also been reported that focal or diffuse intense ^{18}F-FDG uptake can be observed in the GB in non-Hodgkin lymphoma.[117]

Physiological or Secretory Radiopharmaceutical Uptake in Gallbladder

Increased ^{18}F-FDG uptake in the GB lumen that does not indicate any pathology has also been reported.[118,119] In the retrospective analysis performed by Asmar et al. in a large cohort group of 8096 patients with ^{18}F-FDG PET/CT, they determined incidental GB uptake in 54 (0.67%) patients. In these patients, the fasting blood glucose level was found to be higher than in the control group, and the probability of

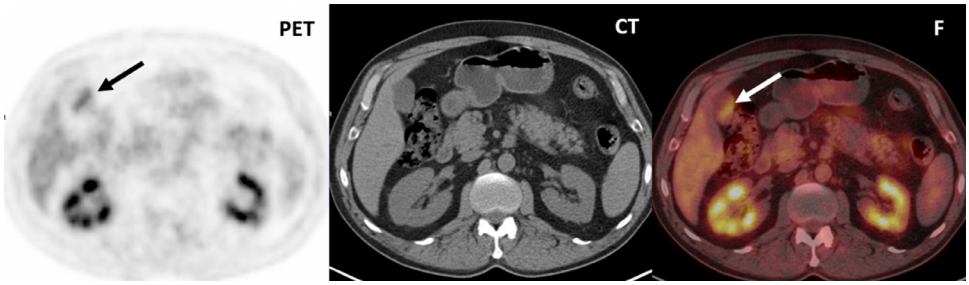

Figure 7 ^{18}F-FDG uptake (arrows) was observed in the gallbladder lumen, which was evaluated in favor of bile radiopharmaceutical excretion in ^{18}F-FDG PET/CT imaging in a 58-year-old man who was followed up for lung cancer. This finding was not observed in previous studies, and no pathology was observed in the gallbladder by ultrasonography. (F: fusion images).

GB diseases was also higher than in the control group in the 3-year follow-up.[120] In these cases, the probability that the reason for radiotracer accumulation is secretory ^{18}F-FDG may be an acceptable reason (Fig. 7).

Similar diagnostic problems for molecular imaging apply to other radiopharmaceuticals. Abdul Sater et al. reported a case with paraganglioma in which increased receptor activity was detected in the GB wall with ^{68}Ga-DOTATATE PET/CT. In the same case, a similar intense uptake was observed in ^{18}F-DOPA PET/CT, while no pathological involvement was detected with ^{18}F-FDG PET/CT.[121] Keidar et al. reported 5% unexpected non-malignant GB involvement by examining ^{68}Ga-PSMA PET/CT results in patients with PC.[122] The study by Demirci et al. reported similar involvement in 10%. The researchers highlighted that liver and GB involvement may occur in these patients due to intense PSMA-like protein expression and hepatobiliary clearance in the liver.[123] Balan et al. observed intense involvement mimicking metastasis in the GB lumen with ^{18}F-DOPA PET/CT in a 55-year-old patient with a diagnosis of extra-adrenal pheochromocytoma. However, in correlative CT images, it was decided that the involvement observed in PET imaging was related to normal biliary excretion, and it was revealed that there was no pathological liver or GB involvement[124] (Table 2).

PANCREAS

^{18}F-FDG uptake in Benign Processes and Diseases

Diagnosis of pancreatic cancers is still challenging despite today's advanced imaging methods, so the importance of molecular imaging is increasing in this area. Studies have reported that ^{18}F-FDG PET/CT imaging has 85%-100% sensitivity and 67-99% specificity in distinguishing benign and malignant lesions of the pancreas.[125-127] The pancreatic tissue shows minimal or no ^{18}F-FDG uptake, but it is not possible to interpret with the same precision for benign lesions. Adenocarcinomas often occlude the pancreatic duct and cause secondary distal pancreatitis, and chronic pancreatitis is a well-known risk factor for adenocarcinomas. However, it should be kept in mind that although pancreatitis showing high ^{18}F-FDG uptake develops secondary to tumor obstruction, different etiologies may also cause pancreatitis. Autoimmune pancreatitis may be an important diagnostic problem in distinguishing malignancy, but there are differences with the uptake pattern due to a malignant tumor. While ^{18}F-FDG uptake observed in the tumor is almost always focal, diffuse involvement is observed in most cases of autoimmune pancreatitis. However, focal and multifocal uptake patterns can also be observed in autoimmune pancreatitis.[128] Although Zang et al. reported that increased uptake in the late images taken 2 hours after ^{18}F-FDG injection compared to the early images in the pancreatic adenocarcinoma compared to autoimmune pancreatitis, the difference was not strong enough to use in differantial diagnosis the 2 groups. Another imaging finding that may lead to differential diagnosis between the 2 groups is pancreatic duct dilatation. It was reported that pancreatic duct dilatation was observed more than twice as wide in the tumor group compared to the autoimmune pancreatitis group (55% vs 23%). Other findings leading to a distinction between the 2 groups are intense ^{18}F-FDG uptake observed in the extrapancreatic regions, especially in the prostate and salivary glands, except the area where the primary lesion is observed in cases with autoimmune pancreatitis[128,129] (Fig. 8). Mass-forming pancreatitis can often be confused with pancreatic adenocarcinomas on CT imaging. ^{18}F-FDG PET/CT imaging can contribute to the differential diagnosis of mass-forming pancreatitis with the relatively low uptake observed in inflammatory lesions.[54] However, the severity of the inflammatory process is effective on the level of radiopharmaceutical uptake. Ye et al. reported a case of increased ^{18}F-FDG uptake in focal form of mass-forming chronic pancreatitis and hepatic schistosomiasis.[35] Increased ^{18}F-FDG uptake in both the pancreas and the liver, like this case, has the potential to make the diagnosis more difficult, and it may not be easy to avoid the diagnostic pitfalls. Another rare cause of pancreatitis is heterotopic pancreas, which is actually an abnormality. Heterotopic pancreas is the presence of pancreatic tissue lying outside of its normal location and lacking anatomic or vascular connections with the pancreas. Dong et al. reported inflammatory-induced high ^{18}F-FDG uptake in the heterotopic pancreas.[130]

The increased ^{18}F-FDG uptake that can be seen in the pancreas after systemic treatments may also cause

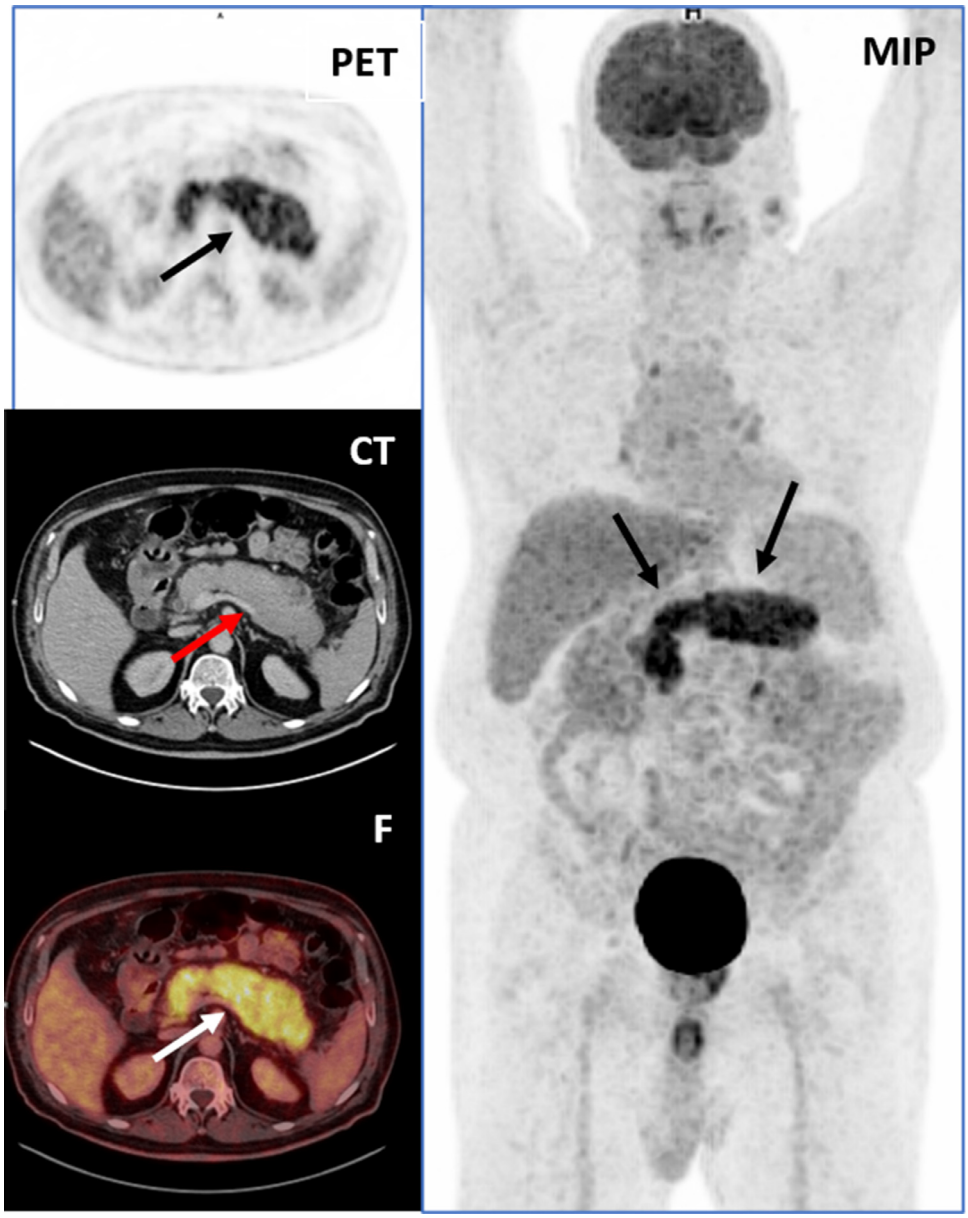

Figure 8 71-year-old man, ^{18}F-FDG PET/CT study was performed for metabolic characterization of the mass in the pancreas. Diffuse intense ^{18}F-FDG uptake was observed in the whole pancreas (arrows), and a diagnosis of autoimmune pancreatitis was made with radiological evaluation and laboratory results. (F: fusion images, MIP: maximum intensity projection).

misinterpretations. Das et al. reported focal increased ^{18}F-FDG uptake accompanied by pancreatic tail enlargement in PET/CT imaging performed after 3 cycles of pembrolizumab treatment in a patient diagnosed with lung adenocarcinoma. By drawing attention to the diagnosis of immunotherapy-induced pancreatitis in the case, misinterpretation was avoided.[131] A necrotic space-occupying lesion showing ^{18}F-FDG uptake was also observed in a patient with metastatic osteosarcoma who was treated with regorafenib, a multikinase inhibitor.[132] Considering the frequency of use of tyrosine kinase inhibitors, it should be not forgotten that acute pancreatitis is a cause that may lead to erroneous assessment.

One of the reasons for false positive result related to the surgery is the application of cyanoacrylate. Belyaev et al. sealed with cyanoacrylate to prevent postoperative pancreatic fistula in the pancreaticojejunostomy area in a case they operated on for pancreatic cancer. In the follow-up PET/CT, strong inflammation and foreign-body reaction towards cyanoacrylate were found in the second operation, which was performed with the thought of recurrence due to intense involvement in the anastomosis line.[133] Another reported a rare cause of increased ^{18}F-FDG uptake is primary pancreatic candidiasis.[134]

In addition to the adenocarcinoma of the pancreas, another pancreatic tumor showing increased ^{18}F-FDG uptake, although relatively rare, is solid pseudopapillary neoplasm (SPN). If the tumor size is small, it is almost impossible to distinguish from pancreatic ductal carcinomas. In case

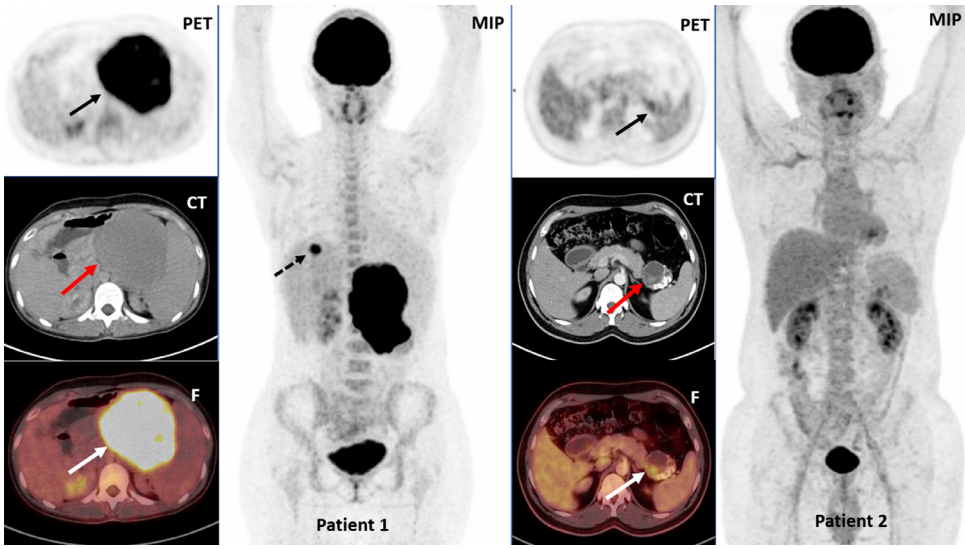

Figure 9 *Patient 1*; 22-year-old woman, ^{18}F-FDG PET/CT imaging was performed for a mass lesion in the pancreas. In addition to intense radiotracer uptake in the pancreatic mass (arrows), a metastatic lesion in the liver (dashed arrow) was revealed. As a result of pancreatectomy with splenectomy and also liver metastasectomy, solid pseudopapillary neoplasia of the pancreas and liver metastasis were diagnosed (Left side). *Patient 2*; A 46-year-old man was diagnosed with solid pseudopapillary neoplasia after distal pancreatectomy with splenectomy. Before surgery, a semisolid mass with low ^{18}F-FDG uptake has been observed in the tail of the pancreas on PET/CT (arrows) (right side). (F: fusion images, MIP: maximum intensity projection).

of an increase in the size of the lesion, the mixture monitoring of the solid and cystic structure and CT with findings of calcification and bleeding can help in the differential diagnosis of the tumor.[135] Although metastasis is not expected, it is rarely present in cases presenting with metastasis. SPN may show intense involvement or may present in the form of low ^{18}F-FDG uptake, cystic predominantly (Fig. 9). On the other hand, serous cystadenoma, mucinous cystadenoma, and intraductal pancreatic neoplasms (IPMN) are cystic pancreatic tumors, and these tumors and mucinous lesions show low ^{18}F-FDG uptake.

^{18}F-FDG Uptake in Rare Malignant Tumors

Systemic isolated metastasis of the pancreas is an extremely rare condition, but it is obvious that metastasis of an extrapancreatic malignancy must be distinguished in the presence of a hypermetabolic focus in the pancreas during the diagnostic process.[136] Similarly, pancreatic involvement is observed in non-Hodgkin lymphomas, though not frequently. However, the absence of any organ or lymphatic involvement other than the pancreas with ^{18}F-FDG PET/CT in primary pancreatic lymphoma may cause difficulties in diagnosis. Wang et al. reported a case with intensive ^{18}F-FDG uptake around the central photopenic region of the head of the pancreas, which was diagnosed as diffuse large B-cell lymphoma by histopathological evaluation.[137]

^{68}Ga-DOTA-Peptides

Nowadays ^{68}Ga-DOTA-peptides has taken its place in routine practice as a preferred method for PET/CT imaging of well and moderately differentiated NETs. On the other hand, the physiological uptake patterns of the pancreas, in ^{68}Ga-DOTA-peptides PET/CT imaging should be carefully evaluated. ^{68}Ga-DOTANOC and ^{68}Ga-DOTATATE PET/CT have high physiological uptake levels in the pancreas. The most intense physiological involvement in the pancreas is observed in the uncinate process and then in the head, and the uptake of radiotracers can reach the malignancy level.[138-141] In these regions, it is essential to support the intense involvement observed in PET with morphological imaging methods in order to be interpreted as malignant in order to avoid unnecessary surgical interventions.

Physiological or pathological radiotracer activities associated with the spleen located in the vicinity of the pancreatic tail may be the cause of pitfall. The first of these is splenosis in the form of well-defined round peritoneal soft tissue nodules, which often show intense involvement in patients who underwent splenectomy, and intense physiological involvement of the accessory spleen.[142] However, one of the most confusing uptakes in pancreas is the intrapancreatic splenic tissue due to physiological activity.[143-145] Another physiological pitfall for ^{68}Ga-DOTA-peptides imaging is the uptake monitored in heterotopic pancreas. Moreover, while pancreatitis should be present in heterotopia for ^{18}F-FDG uptake, it is sufficient to have normal pancreatic tissue for ^{68}Ga-DOTA-peptides uptake. Unexpected radiotracer involvement in the upper abdomen may well be misdiagnosed as a NET. Zilli et al. showed that ^{68}Ga-DOTATOC activity observed in the duodenum and interpreted in favor of a NET was the intestinal location of the heterotopic pancreas by histopathological evaluation of the laparoscopic enucleation of the duodenal parietal lesion.[146] Another reason for the pathological uptake

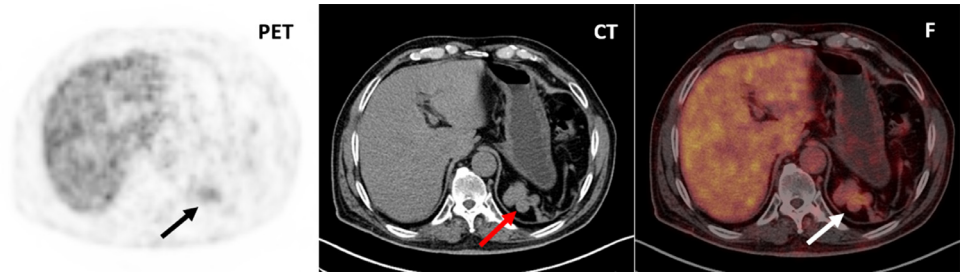

Figure 10 A 73-year-old man with a history of splenectomy was diagnosed with prostate cancer and ^{68}Ga-PSMA PET/CT was performed for staging. A solid lesion with a lobulated contour with moderate radioligand expression was observed at the operation site. The lesion was evaluated to be compatible with splenosis (arrows). (F: fusion images).

of ^{68}Ga-DOTA-peptides in PET/CT imaging may be pancreatic lymphoma. ^{68}Ga-DOTATOC activity in diffuse large B cell non-Hodgkin lymphoma is a well-known and expected finding. However, increased radiotracer uptake in primary pancreatic lymphoma may create a pitfall that may cause an interpretation error for the diagnosis of the tumor in PET/CT[147] (Table 2).

^{68}Ga-PSMA and Other PET Tracers

In a study examining the normal distribution pattern associated with PSMA, different involvements from low and heterogeneous distribution to intense focal expression in the pancreas were reported. PSMA expression was confirmed by immunohistochemical staining of islet cells of the pancreas, and the varying heterogeneous distribution or expression capacity in islet cells was interpreted as the cause of different levels of uptake.[123] PSMA expression was detected in the neovascularization of pancreatic ductal adenocarcinoma cells. Moreover, increased uptake has also been significantly associated with poor survival.[148] Vamadevan et al. observed PSMA expression in the neck of the pancreas on ^{68}Ga-PSMA PET/CT imaging in a patient with PC, and a diagnosis of NET was made by histopathological analysis of the lesion.[7] Chan et al. also reported that pancreatic serous cystadenoma was the cause of focal ^{68}Ga-PSMA incidental expression in the head of the pancreas on PET/CT imaging in a patient with PC.[149] Splenosis and accessory spleen may be confused with metastasis due to increased radioligand uptake in ^{68}Ga-PSMA PET/CT imaging similar to ^{68}Ga-DOTA-peptides (Fig. 10) (Table 2).

The most intense physiological uptake of ^{11}C-acetate, in the abdomen is observed in the pancreas, and a decrease in radiopharmaceutical uptake in the presence of pancreatitis is an expected finding.[150] In recent years, the diagnostic potential of ^{18}F-DOPA PET/CT has been reported, especially in insulinoma.[151] However, false positive ^{18}F-DOPA uptake has also been reported in cases with a diagnosis of pancreatic serous cystadenoma and solid pseudopapillary tumor.[152,153]

Conclusion

Molecular imaging is increasingly used in diagnostic oncology. However, intense physiological uptakes or involvements of the non-malignant pathologies that cause unexpected abnormal uptakes can also be observed constantly. Abnormal or unexpected involvements may be due to the systemic or regional oncological or non-oncological treatment methods applied, as well as benign or other malignant diseases accompanying the main problem. During the evaluation and reporting of PET/CT, the clinical findings, laboratory and the results or comment obtained from morphological imaging methods should be taken into consideration during metabolic evaluation. In addition, physicians working in the field of molecular imaging can turn the pitfalls in molecular images into a successful diagnostic process with experience and awareness and can completely change treatment management. Although the exciting power of molecular imaging methods contributes significantly to problem solving, it seems that the need for histological confirmation will continue in the near future.

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