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A case of hepatic inflammatory pseudotumor identified by FDG-PET

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A 53-year-old man with a history of nausea and elevated liver functions presented to our clinic. A CT scan showed a small tumor in the right lobe of the liver. Fluorine-18-fluorodeoxyglucose (^{18}F-FDG) positron emission tomography confirm abnormal metabolic activity with a high standardized uptake value of 7.3 in the lesion. These findings could indicate a malignancy such as well-differentiated hepatocellular carcinoma or cholangiocarcinoma, or a benign lesion such as hepatic abscess. He was diagnosed by histopathological examination as having an epithelioid granuloma with many inflammatory cells. This is the rare report of hepatic inflammatory pseudotumor featuring markedly increased ^{18}F-FDG uptake.

Key words: positron-emission tomography, inflammatory pseudotumor, liver, false positive reactions

INTRODUCTION

THERE HAVE BEEN FEW REPORTS of hepatic inflammatory pseudotumor (IPT) diagnosed with fluorine-18-fluorodeoxyglucose positron emission tomography (^{18}F-FDG-PET).[1,2] This lesion is usually diagnosed clinically or with conventional imaging modalities.[3,4] The clinical diagnosis is difficult in patients with nonspecific symptoms and no serological evidence of inflammation. Laparoscopy and/or open hepatectomy are often performed unnecessarily after misdiagnosis of IPT as a hepatocellular carcinoma or cholangiocarcinoma.[5]

We review herein the PET findings and clinical course in an asymptomatic patient with IPT featuring markedly increased FDG uptake.

CASE REPORT

A 53-year-old man with a history of nausea and elevated liver functions presented to our clinic. Past history was significant only for cholecystectomy one year earlier. There was no palpable abdominal mass. The gamma-glutamyl transferase was 192 IU/*l* (normal, 10–50 IU/*l*) and the alkaline phosphatase was 429 IU/*l* (normal, 80–260 IU/*l*). The WBC count and C-reactive protein were both within normal limits. The clinical impression was of acute cholangitis. A CT scan of the abdomen was performed.

Contrast-enhanced CT (Fig. 1) revealed a 2.0 × 1.5 cm lesion in the right anterior inferior segment of the liver. It displayed heterogeneous early-phase enhancement and delayed-phase isodensity. There was no associated biliary dilatation.

These findings were felt to be nonspecific, and could indicate a malignancy such as well-differentiated hepatocellular carcinoma or cholangiocarcinoma, or a benign lesion such as hepatic abscess. A PET scan (HEADTOME IV SET-1400W-10, Shimadzu, Kyoto, Japan) was performed to evaluate for metastatic disease. Transmission data was acquired prior to injection with rotating germanium-68 rod sources. Then the fasting patient was injected intravenously with 240 MBq ^{18}F-FDG and emission data acquired 50 minutes later. PET images were visually compared with the corresponding CT images, using anatomic landmarks for localization. For quantitative analysis, a 3-pixel ROI was placed over the area

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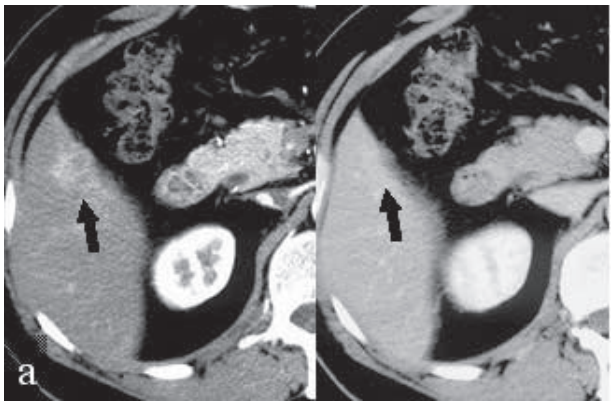

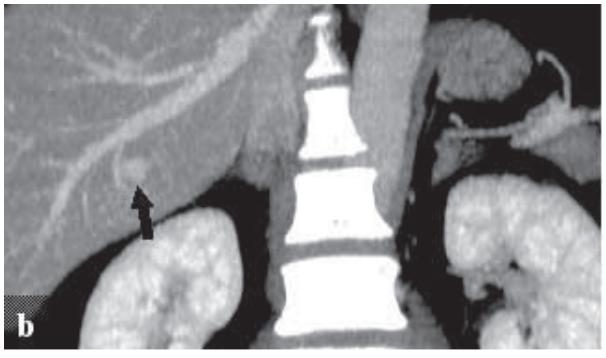

Fig. 1 (a) An early-phase axial contrast-enhanced CT scan shows an enhancing nodule (*arrow*) in the right anterior inferior segment of the liver (*left image*). The nodule exhibits no contrast (*arrow*) with normal liver parenchyma in the delayed phase (*right image*). (b) An early-phase coronal contrast-enhanced CT scan shows the same nodule (*arrow*).

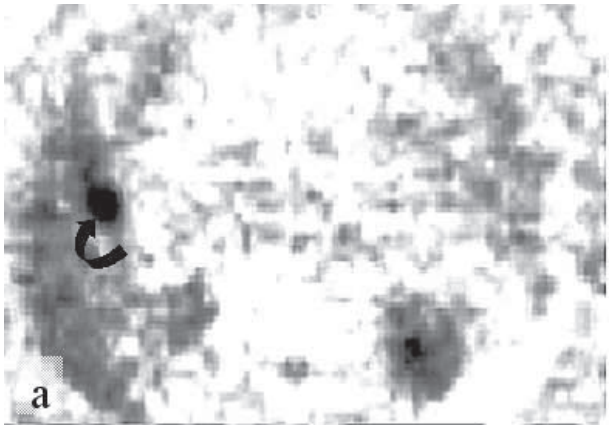

Fig. 2 Axial (a) and coronal (b) FDG-PET images confirm abnormal metabolic activity with a very high standardized uptake value of 7.3 in the nodule (*curved arrows*).

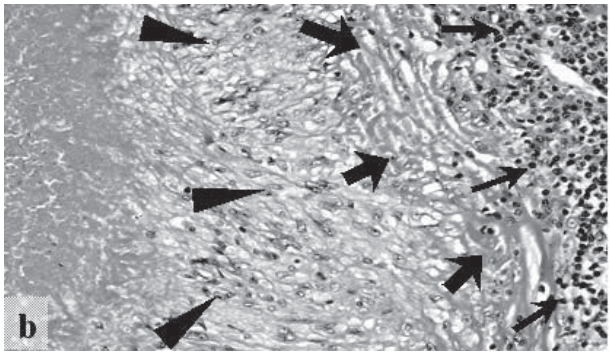

Fig. 3 Photomicrographs of a needle biopsy specimen obtained from the hepatic tumor. Hematoxylin and eosin (HE) staining shows many lymphocytes, fibroblasts, and lymphatic follicles (a, × 40). Higher magnification (b, × 200) reveals abundant lymphocytes (*small arrows*), indistinct fibroblasts (*large arrows*), and epithelioid cells (*arrowheads*), leading to the diagnosis of epithelioid granuloma as inflammatory pseudotumor.

of maximum activity in the lesion to generate an SUV. The lesion exhibited a high SUV of 7.3 (Fig. 2). No other abnormalities were identified on PET.

Because the normal C-reactive protein and high SUV suggested an intrahepatic cholangiocarcinoma rather than abscess or hepatocellular carcinoma, we initially planned a partial hepatic resection. Given the slow growth of the lesion over the ensuing three months, we decided to

perform an ultrasound-guided percutaneous biopsy.

The biopsy specimens revealed an epithelioid granuloma with many lymphocytes, fibroblasts, and lymphatic follicles (Fig. 3), suggesting chronic inflammation. Following a course of symptomatic treatment, the patient's liver function returned to normal eight weeks after the biopsy.

DISCUSSION

IPT has been reported in every site in the body, but is extremely rare in the liver.[3,4] Hepatic IPT is an important consideration in the differential diagnosis of cholangiocarcinoma and other hepatic malignancies.

Our patient was afebrile. There was a discrepancy between his lack of serologic inflammation and his high FDG uptake on PET. Other authors have reported that IPT with serologic inflammation can be diagnosed with conventional modalities such as contrast-enhanced CT.[6] Fukuya et al. reported that patients with smaller hepatic IPT's had no serological evidence of inflammation, and did not exhibit a hepatic lesion on delayed-phase CT scan.[7] It may be difficult to detect IPT in patients with nonspecific symptoms, although CT and FDG-PET clearly revealed a small lesion in our case.

Conventional imaging modalities such as CT, MRI, and ultrasonography cannot differentiate hepatic IPT from neoplasm. While hepatic neoplasms may have relatively low FDG uptake compared to other solid malignancies, they generally have higher uptake than inflammatory processes.[8,9] Recent quantitative studies of glucose utilization in liver tumors have shown that FDG-PET is useful for tumor characterization. The SUV of 7.3 found in our case is much higher than the median values for hepatic neoplasms such as cholangiocarcinoma, metastatic liver tumors, and multifocal hepatocellular carcinomas.[10] As is well known, FDG uptake is not specific to malignant neoplasms, and may be observed in a variety of tissues with increased glucose consumption.[11,12] Changes in FDG uptake correlate with the number of inflammatory cells in various inflammatory and infectious disorders.[13] The high FDG uptake in our patient may have reflected the activity of lymphocytes invading the lesion.

Several theories as to the etiology of IPT have been proposed, but its origins are not fully understood.[14] In our patient, IPT may have had an infectious etiology secondary to cholecystectomy, though no viral or bacterial infection was proven pathologically.

Although the finding of increased FDG uptake is highly sensitive in screening, its low specificity hampers its utility as a confirmatory test. Knowledge of the physiologic and benign pathologic causes of increased FDG uptake is necessary for the accurate interpretation of findings. We have described a case of intrahepatic IPT that was false-positive for malignancy on FDG-PET scan. This case should contribute to the gamut of false-positive FDG PET findings in clinical oncology. It is another example of why special attention is required in the differential diagnosis of hepatic lesions in FDG-PET.

REFERENCES

1. Babar-Craig H, Gill H, Almeyda R, Wong WL, Farrell R. Inflammatory pseudotumour of the neck with multifocal sites on positron emission tomography scan imaging. *J Laryngol Otol* 2005; 119: 219–221.
2. Hsu CH, Lee CM, Lin SY. Inflammatory pseudotumor resulting from foreign body in abdominal cavity detected by FDG PET. *Clin Nucl Med* 2003; 28: 842–844.
3. Horiuchi R, Uchida T, Kojima T, Shikata T. Inflammatory pseudotumor of the liver. Clinicopathologic study and review of the literature. *Cancer* 1990; 65: 1583–1590.
4. Soudack M, Shechter A, Malkin L, Hayek T, Gaitini D. Inflammatory pseudotumor of the liver: sonographic and computed tomographic features with complete regression. *J Ultrasound Med* 2000; 19: 501–504.
5. Nakama T, Hayashi K, Komada N, Ochiai T, Hori T, Shioiri S, et al. Inflammatory pseudotumor of the liver diagnosed by needle liver biopsy under ultrasonographic tomography guidance. *J Gastroenterol* 2000; 35: 641–645.
6. Yoon KH, Ha HK, Lee JS, Suh JH, Kim MH, Kim PN, et al. Inflammatory pseudotumor of the liver in patients with recurrent pyogenic cholangitis: CT-histopathologic correlation. *Radiology* 1999; 211: 373–379.
7. Fukuya T, Honda H, Matsumata T, Kawanami T, Shimoda Y, Muranaka T, et al. Diagnosis of inflammatory pseudotumor of the liver: value of CT. *Am J Roentgenol* 1994; 163: 1087–1091.
8. Delbeke D, Martin WH, Sandler MP, Chapman WC, Wright JK Jr, Pinson CW. Evaluation of benign vs. malignant hepatic lesions with positron emission tomography. *Arch Surg* 1998; 133: 510–516.
9. Shreve PD, Anzai Y, Wahl RL. Pitfalls in oncologic diagnosis with FDG PET imaging: physiologic and benign variants. *Radiographics* 1999; 19: 61–77; quiz 150–151.
10. Iwata Y, Shiomi S, Sasaki N, Jomura H, Nishiguchi S, Seki S, et al. Clinical usefulness of positron emission tomography with fluorine-18-fluorodeoxyglucose in the diagnosis of liver tumors. *Ann Nucl Med* 2000; 14: 121–126.
11. Kubota K. From tumor biology to clinical PET: a review of positron emission tomography (PET) in oncology. *Ann Nucl Med* 2001; 15: 471–486.
12. Yamada S, Kubota K, Kubota R, Ido T, Tamahashi N. High accumulation of fluorine-18-fluorodeoxyglucose in turpentine-induced inflammatory tissue. *J Nucl Med* 1995; 36: 1301–1306.
13. Kawabe J, Okamura T, Shakudo M, Koyama K, Wanibuchi H, Sakamoto H, et al. Two cases of chronic tonsillitis studied by FDG-PET. *Ann Nucl Med* 1999; 13: 277–279.
14. Arber DA, Kamel OW, van de Rijn M, Davis RE, Medeiros LJ, Jaffe ES, et al. Frequent presence of the Epstein-Barr virus in inflammatory pseudotumor. *Hum Pathol* 1995; 26: 1093–1098.