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High [^{18}F] 2-fluoro-2-deoxy-D-glucose (FDG) uptake of adrenocortical adenoma showing subclinical Cushing's syndrome

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A 48-year-old woman with left adrenal tumor, which showed increased uptake of [^{18}F] 2-fluoro-2-deoxy-D-glucose (FDG) was presented. Her adrenal tumor was incidentally discovered, although she had no remarkable illness, and her blood pressure was normal. Hormonal examination including dexamethason suppression test and diurnal variation in serum cortisol level confirmed preclinical Cushing's syndrome. CT, MRI and ^{131}I-adosterol scintigraphy showed findings consistent with adenoma. FDG-PET revealed that tumor had standardized uptake value of 4.8, which was higher than usual benign tumors. Histological diagnosis of the resected adrenal tumor was adrenocortical adenoma without evidence of malignancy. Although the current literature showed that adenomas in general did not exhibit increased FDG uptake, adenoma in the present case with subclinical Cushing's syndrome showed intense uptake of FDG, suggesting FDG-PET could evaluate hormonal function of an adrenocortical adenoma in a completely asymptomatic normocortisolism patient.

Key words: FDG-PET, preclinical Cushing's syndrome, adrenocortical adenoma, adrenal incidentaloma

INTRODUCTION

RECENTLY, adrenal incidentalomas are occasionally depicted by computed tomography (CT) and ultrasonography (US), which are now widely available. Although patients with these tumors are usually asymptomatic, approximately 10% of adrenal incidentalomas produce cortisol. They are called subclinical Cushing's syndrome or preclinical Cushing's syndrome. We herein report a case of an incidentally discovered left adrenocortical adenoma, which showed a high uptake of [^{18}F] 2-fluoro-2-deoxy-D-glucose (FDG) on positron emission tomography (PET), and it was confirmed subclinical Cushing's syndrome by hormonal examinations.

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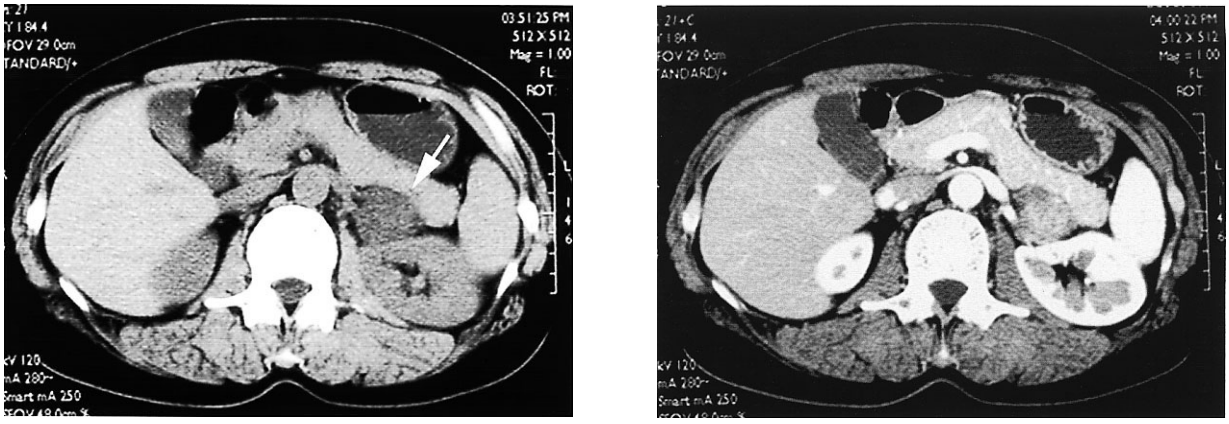
CASE REPORT

A 48-year-old woman was admitted to our hospital for the further examination of an incidentally discovered left adrenal tumor. Four months before admission, she felt a dull pain in the left side of her back, and her family physician discovered a left suprarenal mass lesion on US. She had no remarkable illness throughout her life, and there was no family history of adrenal disease or endocrinopathy. Her height was 159.8 cm and weight was 61.0 kg, and no significant change in her weight was found during the preceding year.

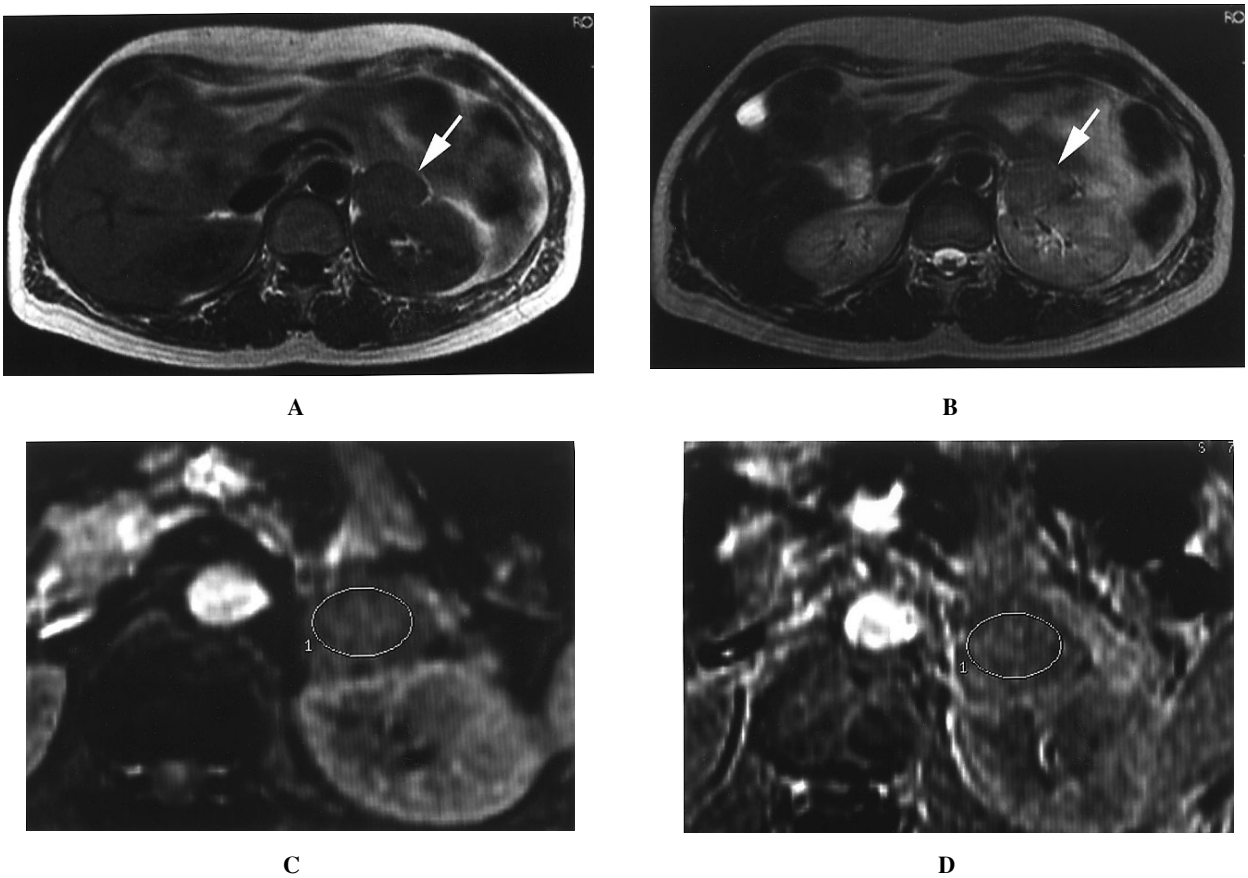
On physical examination, the patient appeared well. The pulse was 72 per minute and regular. The blood pressure was 118/78 mmHg. The chest and abdomen were normal. There was no peripheral edema. The results of a neurological examination indicated normal.

The urine was normal. The levels of transaminase, lactate dehydrogenase, alkaline phosphatase, bilirubin, creatinine, glucose, and electrolytes were normal. Hormonal examinations revealed decreased levels of plasma



A B

Fig. 1 An unenhanced CT of upper abdomen (A) shows a well-circumscribed, low-density mass (*arrow*) of the left adrenal gland, and this tumor is homogeneously enhanced (B).



A B C D

Fig. 2 On T1-weighted (A; TR = 211 msec, TE = 4.8 msec) and T2-weighted (B; TR = 1300 msec, TE = 64 msec) MR images, the left adrenal tumor (*arrow*) is slightly hyperintense as compared to normal adrenal gland. The signal intensity of the tumor on the out-of-phase GE image (C) is lower than that on the in-phase GE image (D), representing rich lipid content. This finding strongly suggests the diagnosis of adrenocortical adenoma.

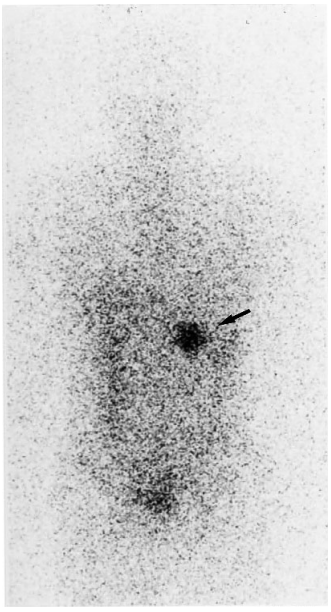


Fig. 3 ^{131}I-adosterol scintigraphy demonstrates an increased uptake of tracer in the left adrenal mass (*arrow*).

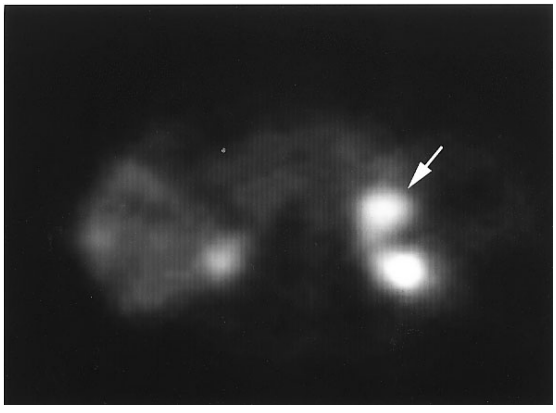


Fig. 4 Axial PET with FDG demonstrates a significant uptake in the left adrenal mass (*arrow*).

adrenocorticotropic hormone (ACTH: 6 pg/m*l*) and increased urinary level of 17[α]-hydroxycorticosteroid (17-OHCS: 10.6 mg/day). The serum levels of 13.6 μg/d*l* of cortisol, 35 pg/m*l* of aldosterone and 0.6 ng/m*l*/hour of rennin were all normal in the fasting state. The urinary levels of 17-ketosteroids (5.0 mg/day), noradrenaline (136.0 μg/day), dopamine (658.3 μg/day), and adrenaline (10.7 μg/day) were all normal. Dexamethason administration failed to suppress the serum cortisol level (16.3 μg/day at 1 mg dexamethason, 14.4 μg/day at 8 mg dexamethason) and the diurnal variation in the serum cortisol level was absent. These results confirmed the diagnosis of preclinical Cushing's syndrome. Thyroid function, parathyroid hormone level, and glucose tolerance test were normal. Examination of serum tumor markers revealed that α-fetoprotein (12.5 ng/m*l*) was abnormal.

An abdominal CT (Fig. 1) showed a well-circumscribed, low-density mass lesion (4.0 × 3.0 cm) of the left adrenal gland. After intravenous administration of contrast material, this tumor was homogeneously enhanced. On both T1-weighted and T2-weighted fast spin echo (FSE) magnetic resonance (MR) imagings (Figs. 2a, b), the left adrenal mass was slightly hyperintense compared to normal adrenal tissue. The signal loss on the out-of-phase gradient-echo (GE) images compared to the in-phase GE images suggested rich lipid content in the tumor (Figs. 2c, d), thus adrenocortical adenoma was the most likely diagnosis.

^{131}I-adosterol scintigraphy showed an increased tracer uptake at the left adrenal gland (Fig. 3), although ^{131}I-MIBG scintigraphy showed no uptake at left adrenal gland. These findings were consistent with functioning adrenocortical adenoma. PET with FDG (Fig. 4) showed a high uptake at left adrenal mass. Semi quantitative analysis of the FDG uptake revealed that standardized uptake value (SUV) of the tumor was 4.8, which was high enough to be suspicious of malignant tumor.

This tumor has been surgically removed, and the pathological diagnosis was adrenocortical adenoma without any evidence of malignancy.

DISCUSSION

Malignant adrenal tumors and pheochromocytomas occasionally show an increased FDG uptake, and benign adrenal tumor, such as adrenocortical adenomas usually show no increase in the uptake.[1,2] Although Lin et al.[3] reported a case of adrenal hyperplasia in Cushing's syndrome, in which an increased FDG uptake was observed, our case is the first report of an adrenocortical adenoma with an increased FDG uptake in a patient with subclinical Cushing's syndrome.

We speculate that the increased glucose metabolism in the adrenocortical adenoma may be responsible for the increased FDG uptake. In this case, the average daily cortisol was within the normal limit, but ACTH was suppressed and impaired cortisol suppression by dexamethasone was observed. In addition, adrenocortical scintigraphy using ^{131}I-adsterol revealed unilateral norcholesterol uptake by adenoma. These data suggested that adenoma of this patient was hormonally active, despite that the patient was asymptomatic. The signal loss in the adrenal tumor on the out-of-phase GE images represented rich lipid content, consistent with adrenocortical adenoma, although MRI cannot differentiate hyperfunctioning adenoma from nonhyperfunctioning adenoma.[4]

Rossi et al.[5] recently suggested that clinically silent hypercortisolism may not be completely asymptomatic. Even a completely asymptomatic normocortisolism patient demonstrated an increased FDG uptake as shown

in the present case. The subclinical Cushing's syndrome patient that is supposed to be not completely asymptomatic as mentioned in the paper may demonstrate an increased FDG uptake. This point should be clarified by further study.

We reported a case of subclinical Cushing's syndrome due to an adrenocortical adenoma, in which an increased FDG uptake was demonstrated on PET. This case suggested that FDG-PET may have potential to evaluate hormonal function of an adrenocortical adenoma by means of glucose metabolism, even in a completely asymptomatic normocortisolism patient. Further study with series of patients is needed to verify this hypothesis.

REFERENCES

1. Maurea S, Mainolfi C, Bazzicalupo L, Panico MR, Lyparato C, Alfano B, et al. Imaging of adrenal tumors using FDG PET: comparison of benign and malignant lesions. *AJR* 1999; 173: 25–29.
2. Yun M, Kim W, Alnafisi N, Lacorte L, Jang S, Alavi A. ^{18}F-FDG PET in characterizing adrenal lesions detected on CT or MRI. *J Nucl Med* 2001; 42: 1795–1799.
3. Lin EC, Heigans R. Adrenal hyperplasia in Cushing's syndrome demonstrated by FDG positron emission tomographic imaging. *Clin Nucl Med* 2002; 27: 516–517.
4. Tsushima Y, Ishizaka H, Matsumoto M. Adrenal masses: Differentiation with chemical shift, fast low-angle shot MR imaging. *Radiology* 1993; 186: 705–709.
5. Rossi R, Tauchmanova L, Luciano A, Di Martino M, Battista C, Del Viscovo L, et al. Subclinical Cushing's syndrome in patients with adrenal incidentaloma: Clinical and biochemical features. *J Clin Endocrinol Metab* 2000; 85: 1440–1448.