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【 CASE REPORT 】

Adrenocortical Carcinoma Diagnosed by Endoscopic Ultrasound-guided Fine-needle Aspiration

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Abstract:
Adrenocortical carcinoma (ACC) is a rare malignancy with a very poor prognosis. A 77-year-old man underwent imaging studies due to poorly controlled hypertension, which revealed a mass measuring 43 mm in diameter near the left adrenal gland. There were no findings indicative of pheochromocytoma. Endoscopic ultrasound-guided fine-needle aspiration (EUS-FNA) was performed for the preoperative pathological evaluation, and the findings indicated a possibility of ACC. Based on these results, curative surgery was performed. If the diagnosis of pheochromocytoma is excluded, then EUS-FNA for adrenal lesions is relatively safe. It can also be used for the preoperative diagnosis of ACC.

Key words: adrenocortical carcinoma, EUS-FNA

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Introduction

Adrenocortical carcinoma (ACC) is a rare malignant tumor with a reported frequency of 0.5-2 cases per 1 million individuals annually (1). It usually occurs in adults in the fourth to fifth decade of life (2). Additionally, a smaller peak is observed in children aged <5 years (3). It occurs more frequently in women than in men; the female to male ratio is between 2.5 and 3 to 1 (4). Approximately 55% of the affected patients present with symptoms indicative of hypersecretion of adrenocortical hormones; 30-40% have symptoms related to the tumor volume, and 10% are asymptomatic (5). The mortality rate associated with ACC is >70% (6). Stages I, II, III and IV accounted for 3%, 29%, 19%, and 50% of the cases, respectively (7). Approximately 40% of the cases have distant metastases (8). The median duration of survival is 101 months for stages I and II, whereas it is 15 months for stages III and IV (2). Advanced cases have a poor prognosis.

ACC is a rare tumor that is sometimes difficult to differentiate when only imaging findings are used. Moreover, owing to its rapid progression, few cases are diagnosed at an operable stage. In this report, we present a case of ACC diagnosed by endoscopic ultrasound-guided fine-needle aspiration (EUS-FNA), which was surgically resectable.

Case Report

The patient was a 77-year-old man who had been treated by another doctor for hypertension for the past 3 years. His hypertension was poorly controlled by oral therapy; therefore, he was referred to a hospital to rule out secondary hypertension. Abdominal computed tomography (CT) revealed a heterogeneous mass, measuring 43 mm in diameter, and abutting the left adrenal gland. The margins of the mass were enhanced on contrast imaging; however, the central part was not enhanced, thus indicating either degeneration or necrosis of the cyst (Fig. 1). T1-weighted magnetic resonance imaging (MRI) showed that the mass had low signal

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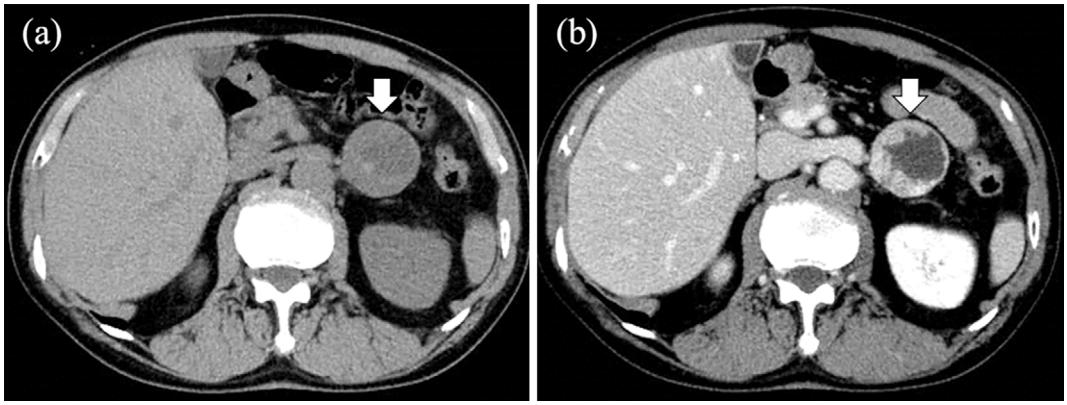

Figure 1. **Computed tomography findings. Abdominal plain computed tomography showing a mass measuring 43 mm in diameter in contact with the left adrenal gland (arrow) (a). The margins of the mass were enhanced on contrast; however, the central part was not enhanced, indicating cystic degeneration or necrosis (arrow) (b).**

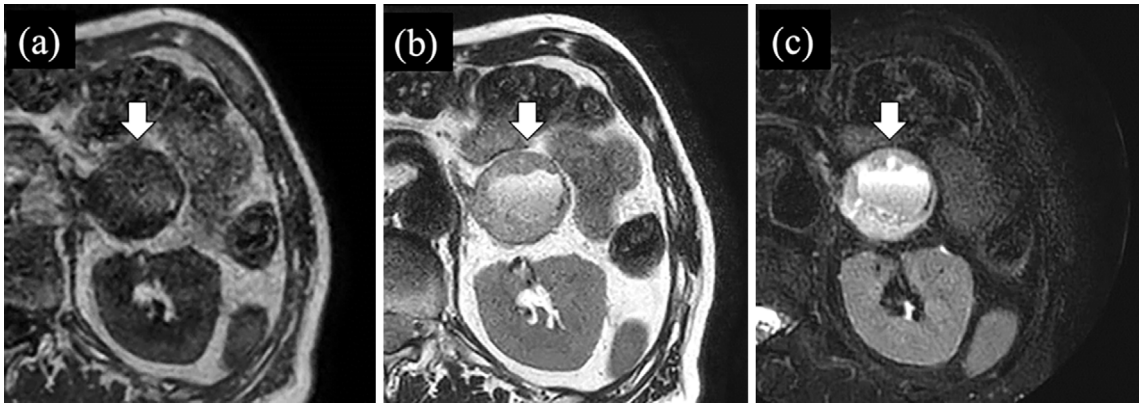

Figure 2. **Magnetic resonance imaging (MRI) findings. T1-weighted MRI showed that the mass had low signal intensity. It also contained a faint high-signal area within the mass (arrow) (a). T2-weighted MRI showed that the mass had a relatively high-signal intensity. Inside the mass, there was a region of high-signal intensity with liquid surface formation (arrow) (b). The fat-suppressed T2-weighted MRI showed no change in the internal properties of the tumor compared to the normal T2-weighted MRI (arrow) (c).**

intensity. It also showed a faint high-signal area within the mass. T2-weighted MRI showed that the mass had a relatively high signal intensity. There was a high-signal region with liquid surface formation inside the mass. Fat-suppressed T2-weighted MRI showed no change in the internal properties of the tumor compared with the normal T2-weighted MRI. These results indicated that the lesion was an adrenal mass with cystic degeneration containing blood components (Fig. 2). The results of endocrinological examinations are shown in Table 1. Specifically, under the administration of candesartan cilexeil 12 mg/day, the plasma aldosterone level was 126 pg/mL, the serum potassium level was 3.6 mEq/L, and the plasma renin activity was 0.4 ng/mL/h. The aldosterone-renin ratio (ARR) was relatively high. However, iodine-131 adsterol scintigraphy showed a normal uptake in both adrenal glands (right, 2.1%; left, 1.4%) (Fig. 3). This suggested that it could be an aldosterone-secreting tumor. The level of serum adrenocorticotropic hormone (ACTH) was 10.7 pg/mL and that of serum cortisol was 12.0 μg/dL. A low-dose dexamethasone suppression test revealed subclinical Cushing's syndrome based on the serum ACTH level of 9.8 pg/mL and the unsuppressed serum cortisol level of 7.7 μg/dL. By contrast, the serum dehydroepiandrosterone sulfate (DHEA-S) level was 72 μg/dL, which was within the normal range.

He was referred to our hospital for further diagnosis and treatment. The levels of plasma adrenaline, noradrenaline, and dopamine were 136 pg/mL, 1,100 pg/mL, and 27 pg/mL, respectively. The level of plasma noradrenaline was relatively high. However, the 24-hour urinary metanephrine was 0.15 mg/day, and normetanephrine was 0.23 mg/day, both of which were not elevated. Iodine-123 adrenal metaiodobenzylguanidine (MIBG) scintigraphy showed no increase in the uptake of this tumor (Fig. 4). The possibility of pheochromocytoma was considered to be low. The lesion was relatively large with hormone secretion, and there was a

Table 1. Endocrinological Examinations

	Pre-operation	Post-operation	Normal range
Hormonal profile			
Serum ACTH (pg/mL)	10.7	16.2	7.2-63.3
Serum cortisol (μg/dL)	12.0	11.9	3.7-19.4
Plasma renin activity (ng/mL/h)	0.4	0.8	0.3-2.9
Plasma aldosterone concentration (pg/mL)	126	77.0	35.7-240
Serum DHEA-S (μg/dL)	72	54	5-253
Plasma adrenaline (pg/mL)	136	N/A	0-100
Plasma noradrenaline (pg/mL)	1,100	N/A	100-450
Plasma dopamine (pg/mL)	27	N/A	0-20
24-hour urinary metanephrine (mg/day)	0.15	0.22	0.05-0.20
24-hour urinary normetanephrine (mg/day)	0.23	0.44	0.10-0.28
Low dose dexamethasone suppression test			
Serum ACTH (pg/mL)	9.8		
Serum cortisol (μg/dL)	7.7		

ACTH: adrenocorticotropic hormone, DHEA-S: dehydroepiandrosterone sulfate

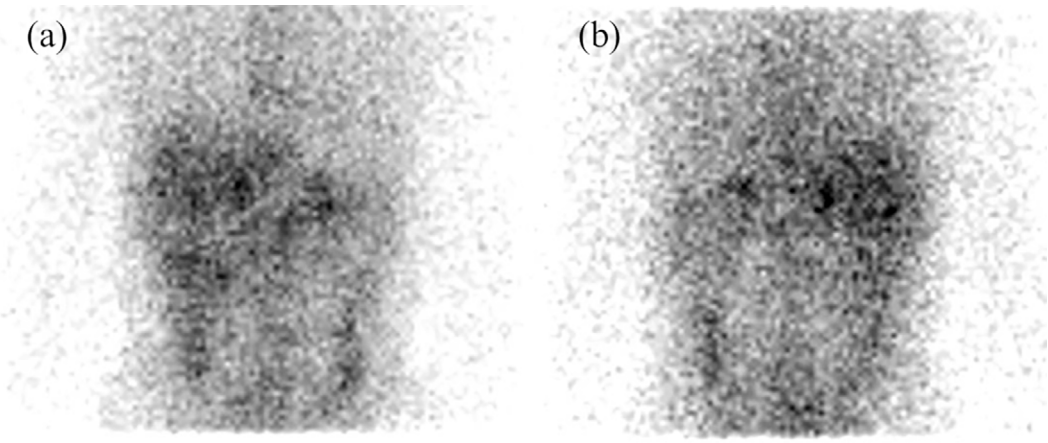

Figure 3. Iodine-131 adsterol scintigraphy findings. Iodine-131 adsterol scintigraphy showed normal uptake in both adrenal glands (right, 2.1%; left, 1.4%). a: Anterior, b: Posterior.

possibility of malignancy; therefore, surgery was considered, and a biopsy was performed for the preoperative evaluation.

EUS (GF-UCT260; Olympus Optical Tokyo, Japan) detected a 38-mm diameter mass adjacent to the left adrenal gland via a gastric approach. It was not continuous with the tail of the pancreas or the left kidney. The surface was smooth, with cystic degeneration inside; however, there was no obvious calcification. EUS-FNA was performed via the gastric approach, and two passes were made with a 22-gauge needle (Acquire; Boston Scientific, Marlborough, USA). Solid lesions of the tumor edges were punctured (Fig. 5). No fluctuations in blood pressure occurred either during or after the procedure. The histopathological findings were as follows: Atypical cells with round nuclei and mostly eosinophilic cytoplasm were seen in alveolar-like patterns. There were some swollen nuclei; however, the overall nuclear atypia was mild to moderate, and the mitotic image was unclear. Immunohistologically, the tumor cells were positive for steroidogenic factor-1 (SF-1) and negative for S 100 protein, cytokeratin AE1/AE3, and chromogranin A, and the Ki-67 index was approximately 30% (Fig. 6). Based on the above, the possibility of a primary adrenal tumor, especially ACC, was suspected.

Left adrenal resection was performed, and the left adrenal gland and adrenal tumor were excised as a single mass. The excised material was a 9.5-6.5 cm mass containing a cyst with internal hemorrhaging and normal adrenal glands. Histopathologically, there was a diffuse proliferation of atypical cells with eosinophilic cytoplasm in most regions of the tumor. Partially, there were myxoid areas of mucus deposition in the stroma. Overall, the tumor cells had nuclear atypia, and some mitotic images were observed [>5/50 high power field (HPF)], but no atypical mitotic images were observed. Coagulation necrosis was observed in the tumor. The tumor invaded the capsule; however, extracapsular invasion was unclear. Elastica van Gieson (EVG) staining showed venous and sinusoidal capillary invasion; however, no lymphatic invasion was observed. Immunohistologically, SF-1 was positive, and the Ki-67 index was approximately 15% (Fig. 7). Seven of the Weiss criteria (9, 10) (Table 1)

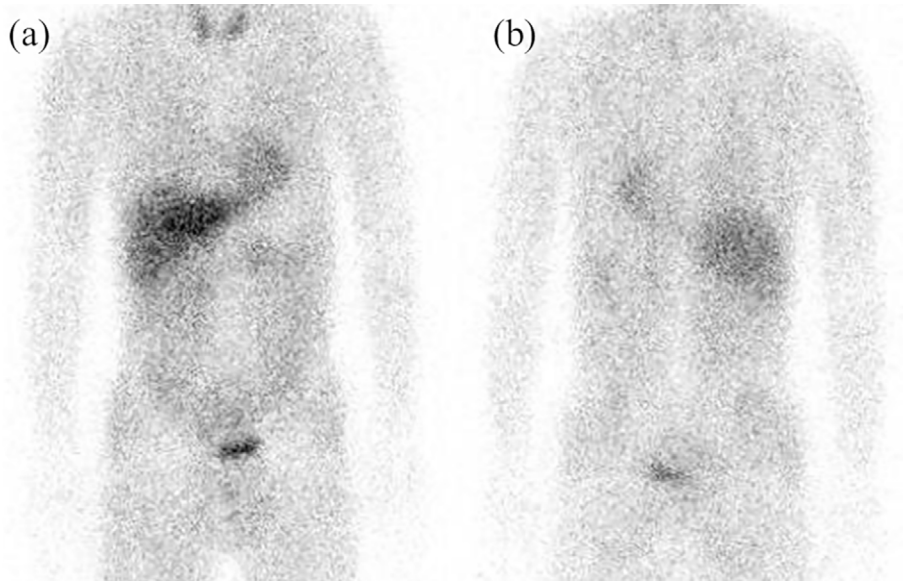

Figure 4. **Iodine-123 adrenal MIBG scintigraphy findings. Iodine-123 adrenal MIBG scintigraphy showed no increase in the uptake of this tumor. a: Anterior, b: Posterior.**

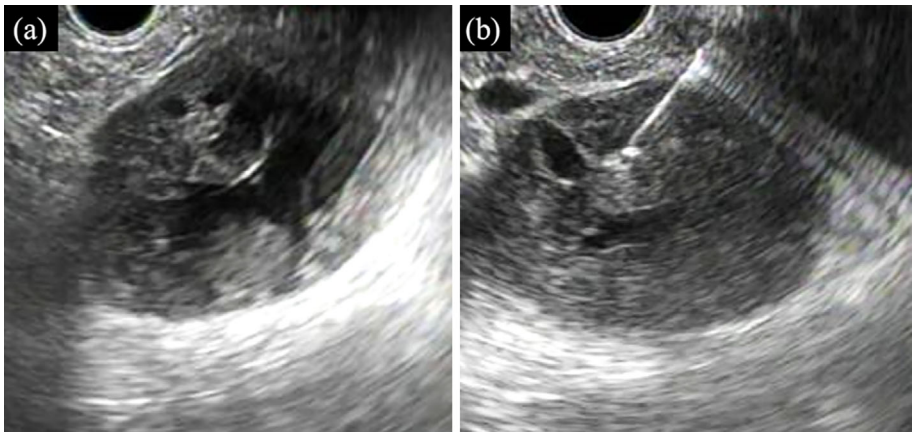

Figure 5. **Endoscopic ultrasound (EUS) findings. EUS via a gastric approach detected a 38 mm diameter mass adjacent to the left adrenal gland. The surface was smooth with cystic degeneration; however, there was no obvious calcification (a). EUS-FNA was performed via the gastric approach, and two passes were made using a 22-gauge needle (b).**

were met: nuclear atypia, an increased mitotic rate, the percentage of clear cytoplasm, coagulation necrosis, invasion of venous and sinusoidal structures, and capsular invasion. Considering the presence of mucus in the tumor stroma, it was diagnosed as an ACC of myxoid variant type. The final pathological stage was T2N0M0 stage II. After the surgery, his hormone levels, including aldosterone and renin, normalized (Table 1), and the antihypertensive medication could also be reduced. We recommended adjuvant therapy with mitotane, but he declined, so he has thereafter been carefully monitored on an outpatient basis.

Discussion

ACC most commonly presents with features of excessive hormonal secretion or symptoms of compression due to the enlarging mass. An increasing percentage of patients with ACC are diagnosed with incidentaloma during abdominal imaging (12). In the case of adrenal incidentalomas, it is especially important to differentiate primary benign lesions from malignant lesions and to rule out metastasis for the proper management and staging of each case. Most adrenal incidentalomas are benign, and the determination of the malignant potential of an adrenal mass depends on the size of the lesion, imaging features, and hormonal status (13). Tumors measuring greater than 40 mm and heterogeneous enhancement on CT are important discriminators of malignancy in adrenal incidentalomas (14). In this case, the tumor diameter was 43 mm, which was large, and the contrast enhancement on CT was heterogeneous, thus leading to a suspected malignancy. ACC often shows a low-attenuation central region on CT that represents tumor necrosis, irregular

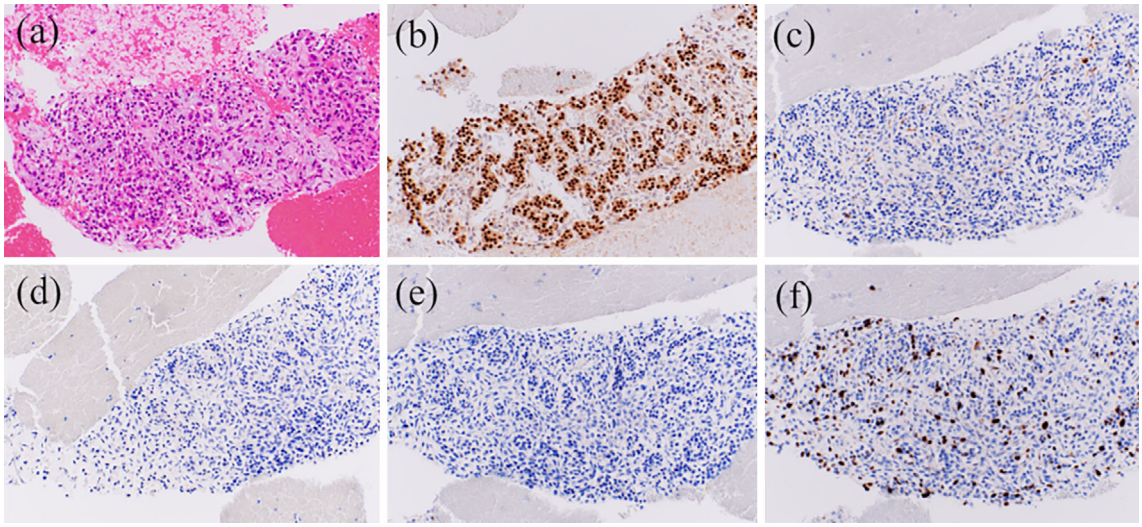

Figure 6. **Histological findings of EUS-FNA. Atypical cells with round nuclei and mostly eosinophilic cytoplasm were seen in alveolar-like patterns (Hematoxylin and Eosin staining, ×200) (a). Immunohistologically, the tumor cells were positive for steroidogenic factor-1 (SF-1) (b), negative for S100 protein (c), cytokeratin AE1/AE3 (d), and chromogranin A (e), and a Ki-67 index of approximately 30% (×200) (f).**

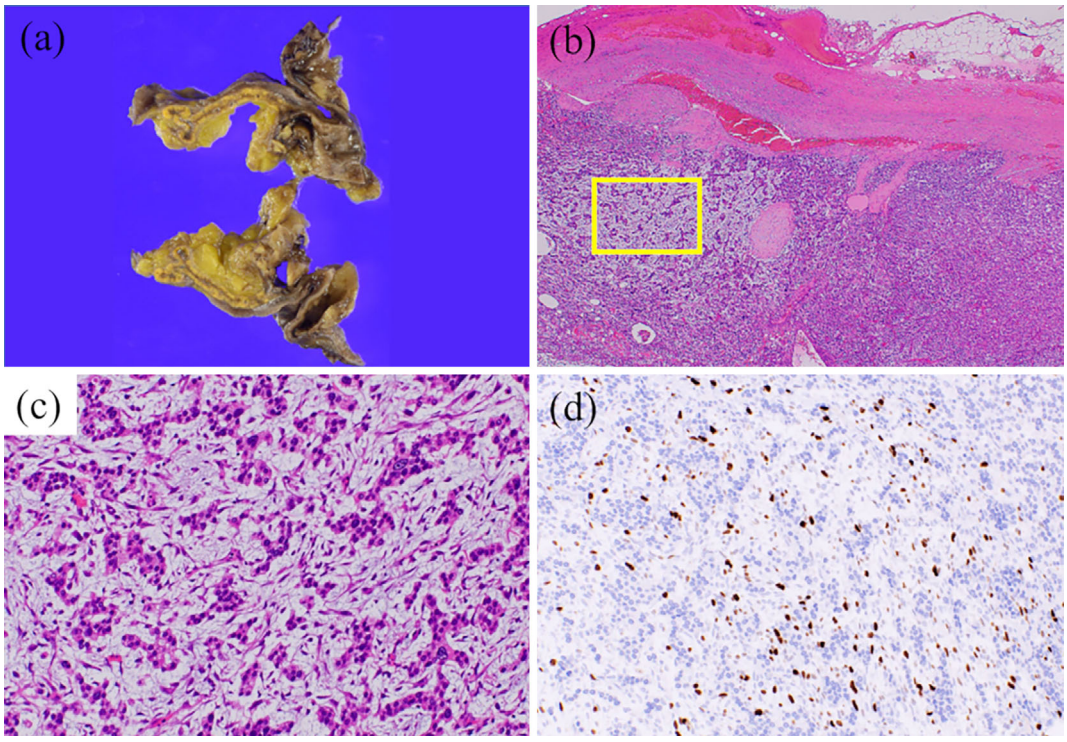

Figure 7. **The excised material and histological findings of surgery. The excised material consisted of a 9.5×6.5 cm mass containing a cyst with internal hemorrhage and normal adrenal glands (a). Histopathologically, there was a diffuse proliferation of atypical cells with eosinophilic cytoplasm in most areas of the tumor (Hematoxylin and Eosin staining, ×40) (b). Partially, there were myxoid areas of mucus deposition in the stroma [enlarged image of the yellow rectangle in (b). ×200] (c). Immunohistologically, the Ki-67 index was approximately 15% (×200) (d).**

contrast enhancement, calcification, and a thin capsule-like margin surrounding the tumor (15). On ultrasound imaging, ACC usually appears as a rounded or oval well-defined hypoechoic mass, with a few displaying a thick partial or complete echogenic rim (16). Although conventional imaging modalities, as mentioned above, are used to rule out adrenal involvement in different malignancies, false-positive and negative-results have been observed in approximately 10%

Table 2. Adrenal Lesions That Must Be Differentiated from Adrenocortical Carcinoma (18-23).

	Location	US	CT	MRI	Endocrinology	Histology
Adrenocortical carcinoma	Cortex	Large (>6 cm), oval, hypoechoic, heterogeneous, hypervascular, hemorrhage, necrosis, echogenic rim, calcifications	High attenuation (>10 HU), heterogeneous contrast enhancement, invasions, metastases	Hetero-intensity on both T1WI and T2WI, small signal loss on out-of-phase images (<30%)	Cushing syndrome or virilization, less commonly Conn's syndrome or feminization (if functional)	SF-1 (+), high Ki-67 index
Pheochromocytoma	Cortex	Large (>5 cm), well-defined, hypoechoic, hypervascular, cystic necrosis, calcifications	High attenuation (>10 HU), homogenous contrast enhancement	Hyperintensity on T2WI referred to as "light bulb sign"	Secreting catecholamine, sometimes Cushing syndrome, high uptake on MIBG scintigraphy	Chromogranin A (+)
Adrenal adenoma	Medulla	Small (<3 cm), oval, well-defined, hypoechoic, homogenous, hypovascular	Low attenuation (<10 HU), homogeneous, mild contrast enhancement	Signal loss on out-of-phase images (>20%)	Cushing syndrome or Conn's syndrome (if functional)	SF-1 (+), low Ki-67 index
Adrenal metastasis	Cortex and medulla	Usually bilateral, <3 cm, solid homogeneous; large, heterogeneous by necrosis or hemorrhage	Other metastases lesions, high attenuation (>10 HU), less contrast washout	Hypointensity on T1WI, hyperintensity on T2WI, lack of signal loss on out-of-phase images	-	Depending on a primary
Adrenal lymphoma	Interstitial	Large (average 8 cm), well-defined, hypoechoic, homogeneous, mimicking cysts, no calcifications	Low attenuation, homogeneous, mild to moderate contrast enhancement	Hypointensity on T1WI, hetero-hyperintensity on T2WI	-	Atypical lymphocyte
Ganglioneuroma	Medulla	Large (>5 cm), irregular, hyperechoic, homogeneous, calcifications	Low attenuation, homogeneous, mild and progressive contrast enhancement	Low homointensity on T1WI, hetero-hyperintensity on T2WI	-	Mature ganglion cells, Schwann cells, mucous matrix and nerve fibers
Adrenal hemorrhage	Cortex and medulla	Oval, mild echoic in the center of adrenal glands	High attenuation (50-90 HU), surrounded by fat stranding	Depending on the age of the hematoma	-	No solid components
Adrenal pseudocyst	Cortex and medulla	Well-defined, cystic fluid, wall calcifications	Cystic structure with thin wall	Depending on the age of the hematoma	-	No solid components
Adrenal hemangioma	Interstitial	Large (2.5-25 cm), oval, well-defined, heterogeneous, hemorrhage, calcifications	Peripheral patchy enhancement with central filling	Hyperintensity on T2WI and focal hyperintensity on T1WI	-	Angioblastic cells

US: ultrasound, CT: computed tomography, MRI: magnetic resonance imaging, HU: Hounsfield unit, T1WI: T1-weighted image, T2WI: T2-weighted image, SF-1: steroidogenic factor 1, MIBG: metaiodobenzylguanidine

of the cases (17). In addition, ACC has imaging findings similar to those of pheochromocytoma, adrenal adenoma, adrenal metastasis, adrenal lymphoma, ganglioneuroma, adrenal hemorrhaging, adrenal pseudocyst, and adrenal hemangioma (Table 2) (18-23). Although malignancy was suspected in this case, it was difficult to differentiate ACC from other diseases such as pheochromocytoma, adrenal lymphoma, and adrenal metastasis based on the imaging findings alone. Therefore, endocrinological examinations were necessary. When evaluating some primary adrenal neoplasms, the differential diagnoses can be refined by laboratory biochemical testing, including serum cortisol, aldosterone, and metanephrine measurements (24). ACC secretes various hormones, including androgens, cortisol, estrogen, and aldosterone (25). Among adult patients with ACC, 30% present with Cushing's syndrome and 20% with virilization. Feminization and hyperaldosteronism are much rarer, accounting for approximately 2% of the cases (26). In the present case, subclinical Cushing's syndrome was revealed by the low-dose dexamethasone suppression test. Although the accumulation of lesions was not clear on iodine-131 adsterol scintigraphy, the plasma aldosterone level and plasma renin

Table 3. Weiss System for Differentiating Benign from Malignant Adrenocortical Neoplasms (9, 10).

Criteria
1. High nuclear grade [grade 3 or 4 according to the criteria of Fuhrman et al. (11)]
2. Mitotic rate greater than 5 per 50 high-power fields
3. Atypical mitoses
4. Clear cells comprising ≤25% of the tumor
5. Diffuse architecture (greater than one-third of the tumor)
6. Necrosis
7. Invasion of venous structures
8. Invasion of sinusoidal structures
9. Invasion of capsule of tumor

* The presence of three or more criteria correlates with subsequent malignant behavior.

activity revealed the aldosterone production capacity of the tumor. However, the DHEA-S level was within the normal limits. Endocrinological examinations were somewhat atypical, but ACC was suspected first, considering the imaging findings. Resection was considered, and a pathological examination was performed to confirm the diagnosis before surgery.

Adrenal FNA for the diagnosis of predominantly solid masses gained attention in the 1970s and has been increasingly used in clinical practice following improvements in imaging technologies and deep-seated FNA techniques (27). For decades, adrenal FNA has been primarily performed by interventional radiologists using percutaneous approaches guided by CT. In recent years, EUS-FNA advancements have provided an alternative approach for the biopsy of adrenal mass lesions. EUS-FNA sampling of the adrenal gland started in the 1990s and has become more common with improvements in technology and equipment (28). EUS is useful as a diagnostic imaging modality because of its superior resolution, and it also provides direct access to the left and sometimes to the right adrenal glands. Complications of FNA in the adrenal glands, such as adrenal hematoma, abdominal pain, adrenal abscess formation, and tumor recurrence along the needle track, have been reported but are infrequent (29). In a review of 416 patients who underwent EUS-FNA of the adrenal glands, no major complications were reported, except for one case of adrenal hemorrhaging (30). EUS-FNA of the adrenal gland can be performed safely. However, if the targets of the puncture are pheochromocytomas, extreme caution is required because it might cause hemorrhaging and hypertensive crisis (31). When performing EUS-FNA for adrenal tumors, it is important to rule out pheochromocytoma. In addition, we always have phentolamine, an alpha-blocker, on standby, to deal with a hypertensive crisis if it occurs. In this case, the iodine-123 adrenal MIBG scintigraphy showed no accumulation in the lesion, and 24-hour urine metanephrine and normetanephrine levels were within the normal limits, suggesting a low probability of pheochromocytoma. However, we took all possible preparations for the examination and were able to perform the procedure safely.

Pathologically, ACC is one of the most difficult tumors to differentiate benign disease from malignancy, and it is often impossible to distinguish a benign tumor from amalignant tumor using only commonly used indices such as nuclear atypia and vascular invasion. A scoring system that combines multiple indices is recommended for the diagnosis of ACC, and the Weiss criteria are the most commonly used (9, 10) (Table 3). It is useful in that it is simple and can diagnose ACC based on the pathological findings alone. ACC can be diagnosed using EUS-FNA; however, it is important to understand that there are certain limitations to this diagnosis. To diagnose ACC accurately, an evaluation of the entire lesion after surgery is necessary. However, EUS is a useful tool to confirm the location of the tumor and adjacent organs, and EUS-FNA allows for pathological evaluations including immunostaining. ACC is often difficult to differentiate from other diseases based on imaging, as it sometimes shows endocrinologically atypical characteristics. Preoperative pathological evaluations are important because it can identify benign and malignant tumors and exclude other malignant tumors such as pheochromocytoma, adrenal metastasis, and adrenal lymphoma. Therefore, it is worthwhile to consider tissue sampling by EUS-FNA before performing surgical resection. ACC is a rare tumor, and there are few reports of cases diagnosed by EUS-FNA. To the best of our knowledge, only three cases of ACC diagnosed using EUS-FNA have so far been reported (32-34). This case is valuable because the ACC was diagnosed using EUS-FNA and then surgically resected.

We encountered a rare case of ACC diagnosed using EUS-FNA. When performing EUS-FNA for adrenal tumors, it is important to rule out pheochromocytoma in advance. There were no major complications at the time of examination, and complete resection was possible based on the diagnosis. An early diagnosis of ACC and surgery is important for improving the prognosis. In conclusion, EUS-FNA is therefore considered to be useful for the preoperative diagnosis of ACC.

The authors state that they have no Conflict of Interest (COI).

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