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Adrenal Cortical Adenoma: The Fourth Component Of Carney Triad and an Association With Subclinical Cushing Syndrome

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Abstract

Carney triad is the combination of gastric stromal sarcoma, pulmonary chondroma, and extra-adrenal paraganglioma. Herein, we describe the clinical, imaging, pathologic, and follow-up findings from 14 patients for a fourth component of the syndrome, adrenal adenoma, and clinical and imaging findings consistent with the tumor from 14 others. The adrenal neoplasm was asymptomatic and usually a late finding. Results of adrenocortical function tests were normal. Computed tomography revealed low-density adrenal masses that were consistent with adenomas. Bilateral lesions were present in 4 patients. In 13 of the 14 patients who underwent surgery, resected adrenal glands and biopsy specimens featured 1 or more circumscribed, yellow tumors, up to 3.5 cm in diameter, composed of well-differentiated polygonal cells with clear vacuolated cytoplasm and a smaller component of eosinophilic cells. The extratumoral cortex had combinations normal histologic features, discrete clear cell micronodules, zonal clear cell hypertrophy, or marked atrophy. The lesion in the 14th patient was different, grossly and microscopically resembling the usual sporadic cortisol-secreting adenoma. After the tumor was excised, the patient required glucocorticoid support. None of the tumors recurred or metastasized. Fourteen additional patients had unilateral or bilateral adrenal tumors consistent with adenomas detected by imaging studies.

Keywords

adrenal; Carney triad; adrenocortical adenoma; Cushing syndrome

Introduction

The Carney triad (CTr) was described in 1977 as “the triad of gastric leiomyosarcoma, functioning extra-adrenal paraganglioma, and pulmonary chondroma” (1). The syndrome was found in 7 unrelated young women. One, aged 16 years, had a 5-mm adrenocortical adenoma. Later, when adenomas were found in several other patients with the syndrome, it was evident that adrenocortical adenoma was a fourth component of CTr (2). The 3 tumors in the initial description of the syndrome were multicentric and multifocal; and they affected young patients, suggesting a genetic cause. The gene for CTr remains elusive but genomic changes have been found (3).

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The gastric tumors, the most common component of the disorder, originate in the interstitial cells of Cajal (4) and have been extensively studied (5, 6). They usually occur in the gastric antrum and metastasize to the liver and lymph nodes. They differ clinically, pathologically, and behaviorally from sporadic, nonsyndromic gastric gastrointestinal stromal tumors (GISTs) (6.

The pulmonary chondromas are benign lesions composed solely of cartilage (7). The neoplasm may be single or multiple and unilateral or bilateral. It is often misinterpreted pathologically as pulmonary hamartoma, but it lacks bronchial epithelium, an essential component of pulmonary hamartoma (8).

The paraganglionic component, the least frequent of the 3 major components, and may occur anywhere in the paraganglionic system, including unusual locations such as the aortopulmonary body and the heart, and may be malignant (2).

This article presents the clinical, imaging, pathologic, behavioral, and follow-up findings from 28 patients who had CTr: 14 had 1 or more proven adrenocortical adenomas and 14 had an adrenal tumor, consistent with cortical adenoma, that was detected by imaging. In 13 of the 14 who underwent adrenal surgery, the resected adrenal neoplasms were unilateral or bilateral, multifocal, and composed of clear vacuolated cells with a variable population of eosinophilic cells. The extratumoral cortex had clear cell micronodules and other abnormalities. The pathologic findings in the 14th patient (who, in retrospect, had subclinical Cushing syndrome) resembled those of a sporadic cortisol-secreting adenoma. None of the tumors in the 14 operated patients recurred or metastasized.

Patients and Methods

In coauthor J.A.C.'s database of 149 patients with CTr, 28 (19%) had 1 or more proven or suspected adrenocortical tumors. The presence of 2 of the 3 major component tumors multifocally was accepted as sufficient for diagnosis of the syndrome. Of the 28 patients (27 female, 1 male), 11 were evaluated at Mayo Clinic, Rochester, Minnesota; 1 was evaluated at the National Institutes of Health, Bethesda, Maryland; 10 were described in publications (9–17); and 6 were communicated to J.A.C. Patient records and publications were reviewed for adrenal symptomatology; results of abdominal imaging, iodine-131 metaiodobenzylguanidine (^{131}I-MIBG) scintigraphy, and adrenocortical function testing; treatment of the adrenal neoplasm; and follow-up. Two patients (patients 2 and 8) had paraganglioma (pheochromocytoma) in addition to a cortical tumor in the resected gland. In 24 patients the lesions were discovered by imaging; in 4 patients, the lesions were found at surgery.

The resected adrenal glands and adrenal excisional biopsy specimens were fixed in formalin and embedded in paraffin. Hematoxylin-eosin (H&E)-stained slides were available for 13 patients, and 1 or more formalin-fixed, paraffin-embedded blocks were obtained for 9 of the 13. Sections 4μm thick were used for H&E staining, histochemistry (periodic acid—Schiff, reticulin, and Masson trichrome stains), and immunocytochemistry. The thickness of the extra-tumoral cortex was measured in H&E-stained sections at ×100 magnification with a 1-cm intraocular scale divided into 100 units.

For immunostaining, antibodies were directed against the following: vimentin (Dual Env; PT link: 1/500 BRD [background reduction diluent]; Dako; V9), synaptophysin (Ventana; CC1 [cell conditoner 1] mild; 1/50 BRD; Leica [Novocastro]; 27G12), inhibin-A (Advance; PT link; 1/60 BRD; AbD Serotec; R1), melan A (Advance; PT link; 1/500 BRD; Dako; A103), and CD56 (Ventana; CC1 mild; 1/100 BRD; Dako; 123C3).

Results

Table 1 presents the CTr components in the 28 patients, the age at diagnosis of the first lesion (mean, 22 years), and the age at detection of proven (mean, 32 years) or imaged (mean, 37 years) adrenal cortical tumors. No patient had adrenal symptoms preoperatively, and results of limited laboratory testing of adrenocortical function were normal (patient 28 had complete testing). Four patients had normal levels of plasma cortisol, and 2 others had normal results from dexamethasone suppression testing. One patient had elevated levels of plasma cortisol before and after adrenalectomy; they were interpreted as resulting from estrogen therapy (11). Patient 1 had normal 24-hour urinary free cortisol levels 4 years before adrenalectomy, but adrenal insufficiency (confirmed by laboratory testing) developed after adrenalectomy; cortical function normalized in 3 months. Four patients had bilateral lesions that were pathologically proven (1 patient), pathologically proven and contralaterally imaged (1 patient), or imaged bilaterally (2 patients).

Imaging results, tumor treatment, and follow-up information for the 28 patients are shown in Table 2. Computed tomographic (CT) images were compatible with a low-density or lipid-rich lesion, suggestive of a benign adenoma. Results of ^{131}I-MIBG scintigraphy were negative for 11 patients.

The rate of growth of the adrenal tumors was unpredictable. Patient 6 had normal adrenal imaging findings at age 23; a 7-mm left adrenal mass was present at age 24; 5 years later, it measured 2 × 1.7 cm with signal characteristics consistent with a lipid-rich adenoma. Bilateral 1-cm nodules found in patient 10 at age 24 were unchanged 23 years later. The longest interval separating detection of a contralateral adrenal tumor was 26 years.

Table 3 presents selected pathologic findings and initial diagnoses for the 14 patients who underwent surgery.

Pathology

Gross—The resected adrenal glands and excisional biopsy specimens from 13 patients featured 1 to 6 circumscribed, solid, yellow tumors, 4 mm to 3.5 cm in diameter (Figure 1A). Close to 20 1- to 2- mm yellow lesions were attached to the adrenal capsule of 1 tumor (Figure 1A). The extratumoral cortex usually appeared normal with an outer yellow layer and an inner brown layer (Figure 1A). The findings in the remaining patient were different (Figure 1C). There were 2 juxtaposed tumors: one was tan with red, brown, black, and yellow; the other was dark tan with radiating dark red zones. The attached cortex was thin and yellow.

Light Microscopy—The tumors were limited by a thin adrenal capsule, or an incomplete fibrous pseudocapsule, or they were circumscribed and in direct contact with stretched or compressed cortex. There were areas of scarring in some of the neoplasms.

With H&E staining, the tumors had 2 types of cells. Most were polygonal, medium-sized, and well-outlined with clear vacuolated cytoplasm (Figure 2). They were arranged in tightly packed clusters that formed broad sheets, some of which appeared vaguely lobulated. Occasional cells were ballooned, and some smaller ones had a tinge of cytoplasmic eosinophilia. A minority of the cells were small and poorly outlined, and they had eosinophilic cytoplasm (Figure 2A). These cells were randomly disposed in zones of various sizes and shapes and in narrow ramifying bands among the clear cells. The degree of eosinophilia ranged from weak to very strong; the cytoplasm varied from fine granular to homogenous. The 2 cell types were separate with minimal transition between them. In patient 1, the eosinophilic cells were arranged in large, irregularly shaped zones or round

nodules (Figure 3A). Some cells were large or huge (Figure 3B); a few had lipochrome (Figure 3C).

The nuclei were small, round, and centrally located, with dense darkly staining chromatin and 1 or 2 very small nucleoli. They were uniformly distributed in the cells and occasionally tightly stacked in some of the eosinophilic cells. Scattered larger cells had correspondingly enlarged nuclei (Figure 2B). Multinucleation was rare. There was nuclear atypia (nucleomegaly, hyperchromatism, and intranuclear vacuoles) in the eosinophilic cells in patient 1 (Figure 3D). Mitoses were not seen.

The stroma was inconspicuous. Some tumors had fine arborizing or stellate scarring, and there were some dilated, occasionally hyalinized, blood vessels.

The extratumoral cortex adjacent to but not surrounding the tumor showed combinations of normal histology features (Figure 1B), segmental atrophy (Figure 1D), discrete clear cell micronodules (Figure 4A), zonal clear-cell hypertrophy (Figure 4A), and cortical extrusions (Figs. 1A and 4B). Micronodules, present in 5 of 8 patients, were defined as roughly spherical, unencapsulated areas of hypertrophied cortical clear cells, usually less than 1 mm but up to 2 mm in diameter. There was sufficient extratumoral cortex available in 7 specimens for thickness measurements: 0.43 mm (28 measurements) to 0.91 mm (16 measurements) in 4 glands; 0.96 mm (33 measurements) in the specimen in Figure 1B; and 1.46 mm in 1 section (15 measurements) and 0.55 mm in a second section (4 measurements) in the remaining cortex. The thickness of the normal adult cortex is 0.7 to 1.3 mm (21).

The extratumoral capsule was intact except where it was penetrated by cortical extrusions in 2 patients (Figures 1A and 4B). A focus of ovarian thecal metaplasia extended from the capsule into the cortex in 3 of the female patients.

Immunostaining—The immunostaining results showed variability in 1) the degree of staining (strong, weak, or equivocal); the site of cell staining (cytoplasm or cell membrane), the type of staining (single large cytoplasmic body, coarse granularity, or fine granularity); and inconsistency (adjacent histologically similar cells stained or did not stain). Differences in tissue processing and age of sections may have been contributing factors. Also, the functional state and the particular presumptive steroid products of the tumor cells may have differed among the tumors. Only consistent results are presented (Figure 5). Results for the tumors were as follows—vimentin, negative (7 of 7); synaptophysin—positive, focal, clear cells, membrane (6 of 7); inhibin-A—positive, weak, patchy, membrane and cytoplasm (7 of 7); melan A—strong, diffuse, coarsely granular including cytoplasmic body (7 of 7); and CD56—positive, patchy, weak (7 of 7). Results for the extra-tumoral cortex were as follows: vimentin—positive (4 of 6); synaptophysin—positive, weak (3 of 6); inhibin-A—positive, strong (4 of 6); melan A —positive, diffuse or patchy, strong (5 of 6); and CD56—positive, patchy, moderately strong (6 of 6). The clear cell micronodules in 2 cases stained as follows: melan A—positive and other stains—negative.

Discussion

This study describes the clinical, imaging, pathologic, and behavioral features of 28 proven or suspected adrenocortical adenomas from 28 patients with CTr. The tumors were asymptomatic and were discovered incidentally during imaging studies or at surgery for gastric stromal sarcoma. They occurred almost exclusively in women (96%) and were detected through a wide age range (16 – 68 years); sometimes a long interval (up to 58 years) separated detection of the first CTr tumor and discovery of the adrenal lesion, which was generally the last component of the syndrome to be detected.

The adrenal neoplasm was single or multiple, unilateral or bilateral, circumscribed, small to intermediate in size, and almost always yellow. Microscopically, it was well delineated and composed of moderately sized cells with clear vacuolated cytoplasm (resembling cells of the normal adrenal zona fasciculata) and a variably sized component of eosinophilic cells (resembling those of the normal zona reticularis). Clear cell micronodules (microscopically similar to the tumors) were present in the extratumoral cortex, which elsewhere was normal, segmentally hyperplastic, or atrophic.

Clear-cell micronodules are a common finding at autopsy in older individuals and are not considered neoplastic or preneoplastic (22). However, since they occurred in 5 of 8 patients in the present study, they were most likely part of the adrenal pathologic features of CTr, particularly because they were multiple and morphologically similar to the clear cells in the cortical adenomas. Clear-cell micronodules probably remain in patients who had partial adrenalectomy and in the contralateral gland of the patients who had unilateral adrenalectomy; new adenomas might develop from these micronodules.

The findings from patient 1 deserve comment because they were different clinically and pathologically from the others. The patient's 2 adenomas were similar to the usual sporadic cortisol-secreting adrenal adenoma. Intraoperative pathologic examination of the resected gland revealed marked atrophy of the extratumoral cortex, as is found with a cortisol-producing adenoma. This suggested that the patient's neoplasm had been producing excessive amounts of cortisol that had affected corticotropin secretion. If this was true, the cortex in the patient's remaining adrenal was likely atrophic also and a postoperative Addisonian crisis could occur. As a precautionary measure, corticosteroid was administered intraoperatively. After adrenalectomy, there was a 3-month period of gradually improving adrenocortical function (confirmed by laboratory assessment) during which the patient needed glucocorticoid replacement.

The pathologic and clinical evolutionary features noted in this patient are consistent with the patient having had subclinical Cushing syndrome preoperatively. The probability seems remote that a sporadic cortisol-secreting adenoma, itself an uncommon tumor, occurred by chance in a patient with CTr, a very rare disorder. Therefore, it must be anticipated that glucocorticoid secretory autonomy may develop in exceptional patients who have CTr. Interestingly, the histology of this patient's atrophic cortex (small eosinophilic cells) was different from that found with sporadic cortisol-secreting adrenal adenoma (ballooned clear lipid-filled cells). The severe focal cortical atrophy in 2 other patients may have been a normal phenomenon (19)

The long-term follow-up data after resection of the adrenal tumors (mean follow-up, 13 years) provided good insight into their natural history. None recurred locally or metastasized, providing convincing evidence that the tumors were benign and giving reassurance that the unresected adrenal tumors (thought to be adenomas by imaging) for the 14 patients who did not undergo surgery were also benign. Average follow-up in these patients was 9 years. These findings suggest that the adrenal adenomas in CTr should be managed conservatively. The adrenal glands may be examined during follow-up CT monitoring of the gastric tumor. If resection of an apparently nonfunctioning adrenal adenoma is planned, subclinical Cushing syndrome should be ruled out and ^{131}I-MIBG scintigraphy (to rule out pheochromocytoma) should be obtained.

Our adrenal findings in CTr patients are different clinically and pathologically from those in several genetic syndromes: Carney complex features primary pigmented nodular adrenocortical disease (20); McCune-Albright syndrome displays bimorphic adrenocortical disease (21); and Beckwith-Wiedemann syndrome manifests with cytomegaly and fetal

cortical abnormality (22). The findings described herein add another entity to this group of disorders. Multiple bilateral cortical tumors also occur in other conditions, including corticotropin-independent macronodular hyperplasia which features multiple yellow clear and compact cell adenomas in a hyperplastic cortex (23), and multiple endocrine neoplasia1 (MEN1) which features bilateral, hyperplastic, and usually nonfunctional lesions (24).

In summary, we have described the clinical, pathologic, and behavioral features of the adrenocortical tumor that is the fourth component of CTr. Clinically, the tumor was asymptomatic and behaviorly it was benign. The pathologic features were included multifocal and multicentric clear cell adenomas and extratumoral clear cell micronodules. One patient had cortisol-producing tumors and subclinical Cushing syndrome. The pathologic findings are an addition to the group of genetically mediated bilateral adrenocortical tumors.

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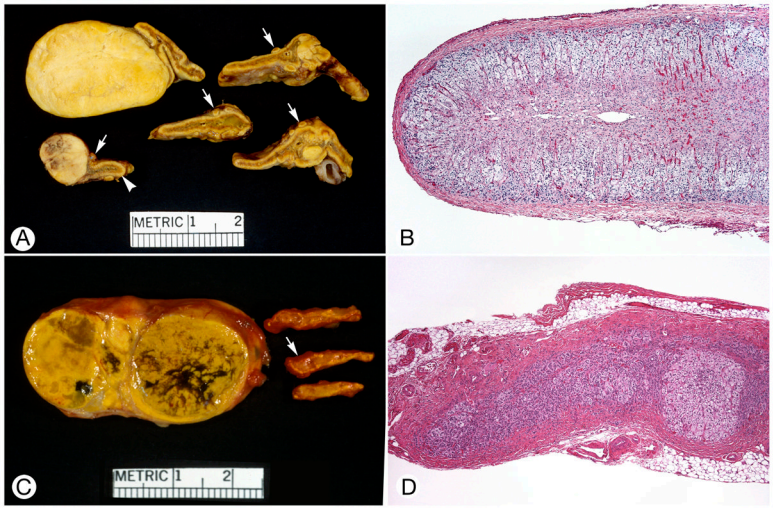

Figure 1.

Adrenocortical Adenomas and Extra-tumoral Cortex. A and B, patient 5, C and D, patient 1. A, Slices of the adrenal gland showed 4 predominantly yellow, circumscribed tumors, ranging in diameter from 3 mm to 3 cm. Several small paracortical nodules were evident (arrows). The extratumoral cortex (arrowhead) had an outer yellow (zona fasciculata) and an inner brown layer (zona reticularis). B, The extratumoral cortex was composed of a thin outer cellular layer (zona glomerulosa), a central clear cell layer (zona fasciculata), and a deep layer of eosinophilic cells (zona reticularis). It was 0.96 mm thick. C, Abutting circumscribed adenomas, each approximately 2 cm in diameter. One tumor (left) was lobulated and tan with brown and black areas. The other tumor was marbled with tan and dark red and brown areas. Three slices of the extratumoral adrenal were occupied mostly by pearly gray medulla. The visible cortex was thin and yellow (arrow). D, The cortex was atrophic and lacked normal zonation. It was composed of small eosinophilic cells and had a capsule-to-capsule thickness of 0.34 mm. A clear cell micronodule was present.

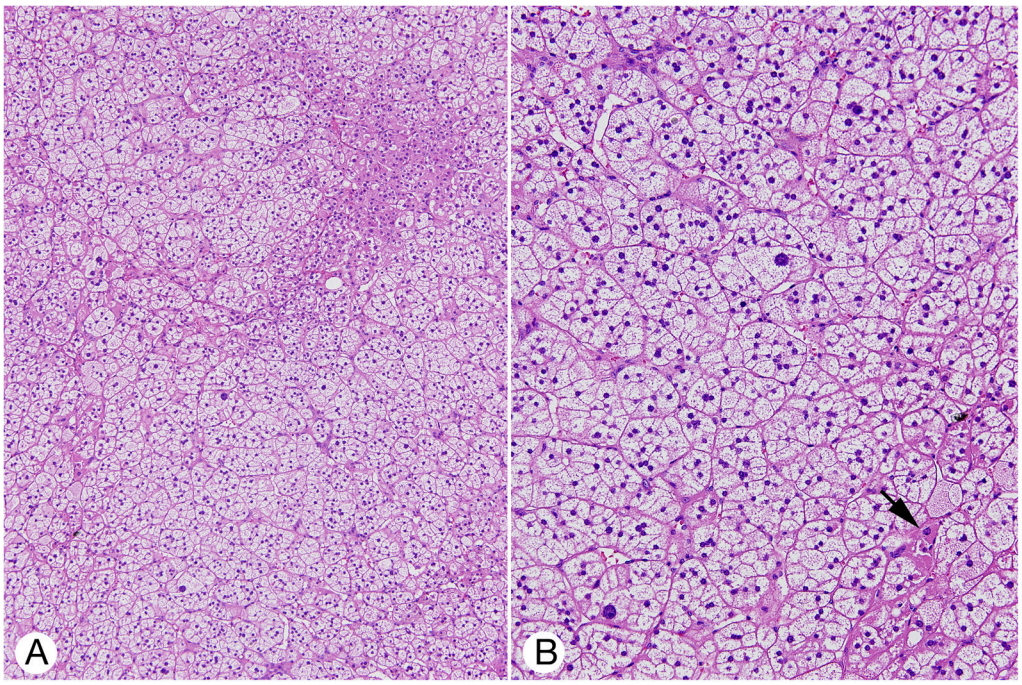

Figure 2.
Adrenocortical Adenoma. A, Clear cells and an irregularly shaped zone of eosinophilic cells. B, Tightly packed clusters of sharply outlined clear cells with small hyperchromatic nuclei, several of which were enlarged. A single cell had eosinophilic cytoplasm (arrow).

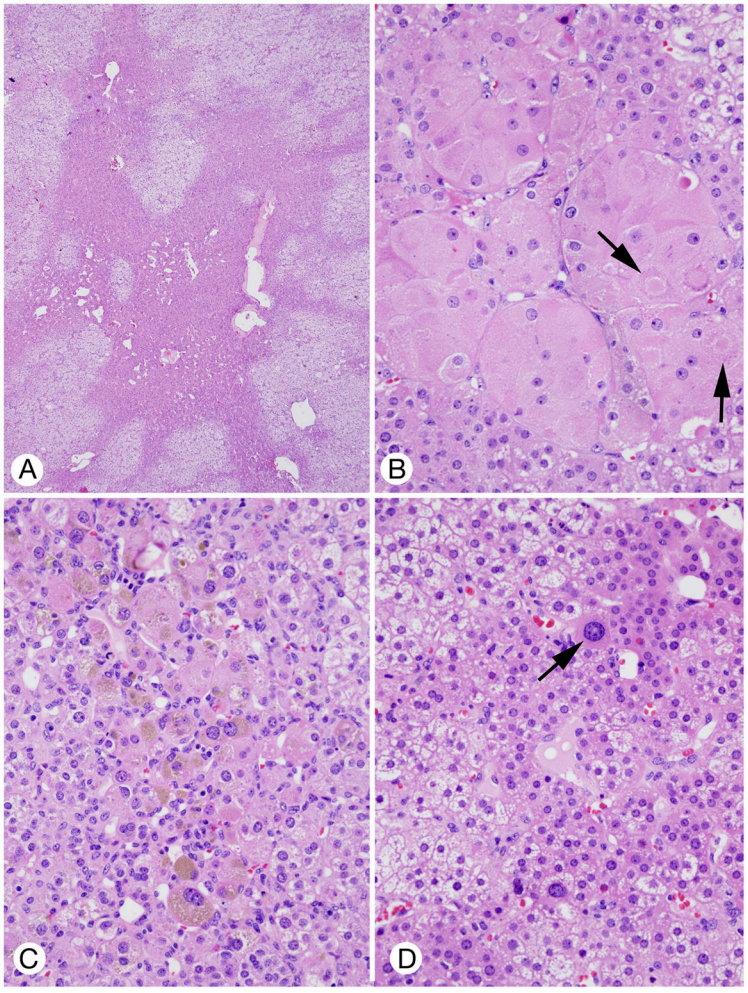

Figure 3.
Adrenocortical Adenomas in Patient 1 who had Postoperative Hypocortisolism. A, Tumor had separate eosinophilic and clear cell components. B, Focus of huge cells with homogeneous eosinophilic cytoplasm with spherical rings (arrows). The nuclei were larger and more vesicular than those of the surrounding typical tumor cells (top right and bottom left). C, Eosinophilic cells varied in size; the larger ones had lipochrome. D, Two greatly enlarged nuclei, 1 with an intranuclear vacuole (arrow).

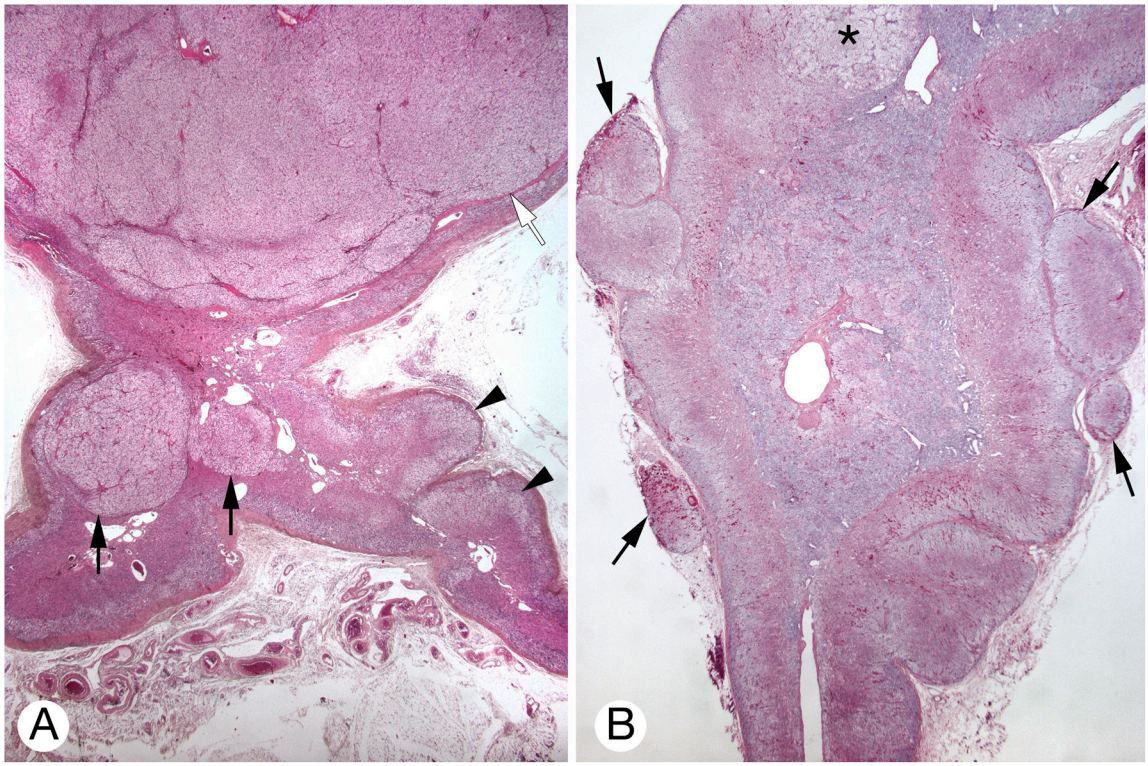

Figure 4.
Cortical Adenoma, Micronodules, and Cortical Excresences. A, The circumscribed clear cell tumor was partially surrounded by a thin pseudocapsule (white arrow). There were 2 clear cell micronodules (black arrows) and zonal clear cell hypertrophy (arrowheads) in the extratumoral cortex. B, The cortex varied in thickness and featured several excrescences (arrows) and a clear cell micronodule (asterisk).

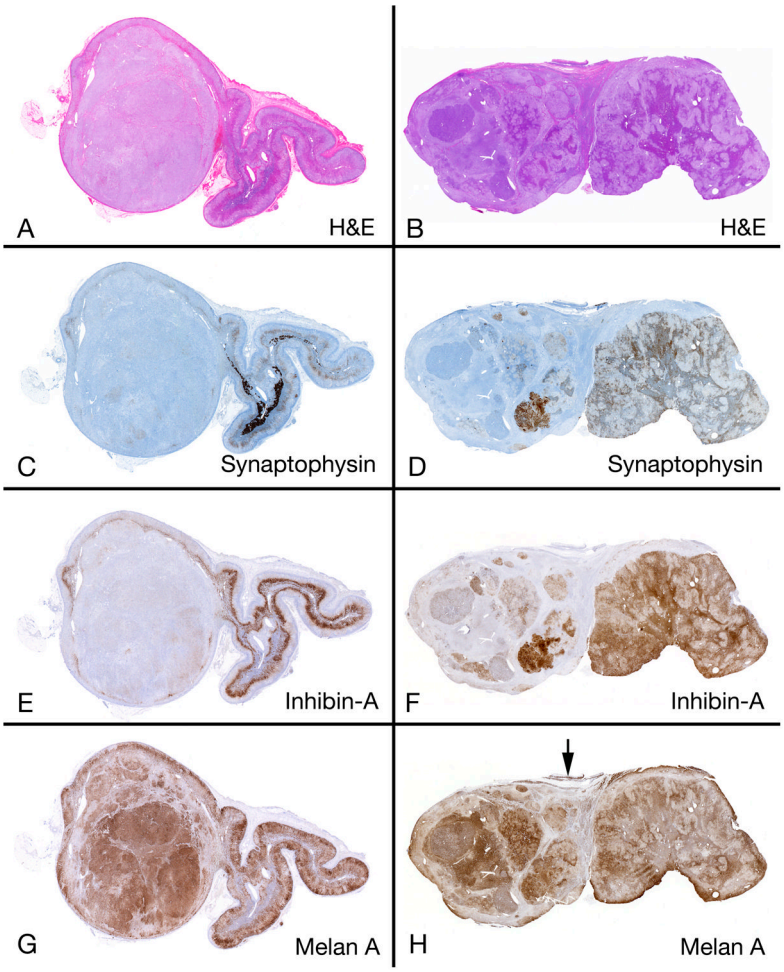

Figure 5.

Hematoxylin-eosin Staining and Immunostaining of Cortical Adenomas. Images in panels A, C, E and G are from the specimen shown in Figure 1 A (patient 5); images in panels B, D, F and H are from Figure 1C. (patient 1). The relative uniformity of nonstaining and staining of the clear cells the specimen shown in A contrasted with the variable staining of eosinophilic and clear cells in B. The darker areas in B were zones of eosinophilic cells. Immunostaining with synaptophysin and inhibin-A showed little or weak staining in C and E and strong focal staining in D and F. The medulla in C was strongly positive for synaptophysin (normal result). Melan A stained both tumors (G and H) and a thin rim of cortex (arrow in H).

Table 1

Neoplasms in 28 Patients With Carney Triad (CTr) and 1 or More Proven or Imaged Adrenal Cortical Adenomas

	Component[a]				Age at detection of tumor, y	
Patient	**Gastric Stromal Sarcoma**	**Pulmonary Chondroma**	**Para- ganglioma**	**Adrenal Adenoma**	**First CTr Tumor**	**Adrenal Adenoma**
1	+	+	+	+	11	47
2	+	+	+	+	17	17 and 29
3	+	+		+	26	47
4	+	+	+	+	41	41
5	+	+	+	+	22	22
6	+	+		+	18	35 and 61
7	+	+		+	15	16
8	+	+		+	20	21
9	+	+	+	+	18	37
10	+	+		+	27	37
11	+	+	+	+	48	50
12	+	+		+	20	27
13					20	30
14	+	+	+	+	22	22
15	+	+	+	(+)	25	25
16	+	+		(+)	23	24
17	+		+	(+)	13	57
18	+	+		(+)	49	37
19	+	+		(+)	24	26(bilateral)

	Component[a]				Age at detection of tumor, y	
Patient	Gastric Stromal Sarcoma	Pulmonary Chondroma	Para- ganglioma	Adrenal Adenoma	First CTr Tumor	Adrenal Adenoma
20	+	+	+	(+)	10	36
21	+	+		(+)	10	68
22	+	+		(+)	11	36
23	+	+	+	(+)	21	27
24	+	+		(+)	49	54
25	+	+	+	(+)	17	37
26	+	+		(+)	29	29
27	+	+		(+)	30	30 and 40
28	+	+		(+)	27	27

+ = histologically proven tumor; (+) = tumor detected by imaging.

Table 2

Adrenal Imaging, Treatment, and Follow-up Data for 28 Patients With Proven or Imaged adrenocortical Adenoma and Carney Triad*

Patient	Lesions Detected With Imaging[a]	Treatment	Follow-up, y
1	Lt: 2.5-cm mass with fatty characteristics	Adrenalectomy	12
2	Lt: Not detected. Rt: Small low density lesion	Lt: Local excision at age 9 y. Rt: adrenal-ectomy at age 29 y	17
3	Lt: 1 × 1 × 2-cm mass	Adrenalectomy	5
4	Lt: Enlargement consistent with adenoma	Adrenalectomy	17
5	Lt: 2.3-cm low-density mass	Adrenalectomy	15
6	Lt: Not detected. Rt; 1.4 × 1.2 × 0.9 cm and 1.2 × 1 ×1 cm masses	Adrenalectomy	11
7	Not detected	Local excision	18
8	PET: Rt: Hypodense lesions compatible with nonsecretory adenomas	Adrenalectomy	18
9	Lt: Well-delineated nodule with little enhancement	Adrenalectomy	22
10	Rt: 3 lesions, each 2 cm; 1 consistent with adenoma	Adrenalectomy	6
11	Not detected	Local excision	33
12	Lt: 3-cm hypodense mass	Local excision	11
13	Rt: Adrenal tumor	Adrenalectomy	4
14	Lt: Not detected	Local excision	7
15	Lt: 2.5-cm mass compatible with lipid- poor adenoma	None	4
16	Lt: 2 ×1.7-cm mass with characteristics of benign adenoma	None	8
17	Rt: 2-cm mass, likely adenoma	None	8
18	Rt: 2-cm mass	None	15
19	Rt and lt: 1-cm mass	None	12
20	MRI—Lt: 3 × 2.5-cm mass	None	16
21	Rt: 1.2 × 2.2-cm mass	None	5
22	Rt and lt: Enlarged adrenal glands	None	No information
23	MRI—Lt: 1.7 × 1.3-cm mass	None	4
24	Rt: 1.3 × 0.9-cm nodule	None	16
25	MRI—Rt: 2 1.5 × 1.9- cm and 1.6 × 1.7-cm masses	None	7
26	PET—Lt: 1.7-cm and 1.1- cm masses. Rt: Thickened	None	None yet (recent case)
27	Rt: 1 × 1.5-cm mass. Lt: 0.8	None	9
28	Lt: 1.5-cm mass	None	None yet (recent case)

Abbreviations: CT, computed tomography; Lt, left: MRI, magnetic resonance imaging; PET, positron emission tomography; Rt, right.

[a]Imaging was by CT unless otherwise indicated

Table 3

Selected Adrenal Pathologic Findings and Initial Diagnosis for 14 Patients With Carney Triad.

	Adrenal Gland[a]		Tumor		
Patient	Rt	Lt	Size, cm	Cut Surface	Initial Pathologic Diagnosis
1		+	4 (2 tumors, each 2)	Variegated tan, brown, black and yellow	Adenoma, atrophic cortex with clear cell micronodules
2	+	+	Rt: 0.5 Lt: 1	Yellow	Adenomas
3		+	2.2 and 1.5	Yellow	Adenomas
4		+	3.5 ×1.6	Yellow	Adenoma
5		+	6 lesions (0.3–3)	Yellow. Cortical excrescences	Adenomas, cortical excrescences
6		+	2	Yellow	Adenoma
7		+	0.8	Not mentioned	Adenoma
8	+		3 × 2.5 × 1	Yellow	Adenoma
9		+	2 by 1.5 by 1	Yellow-orange	Adenoma
10		+	0.4, 1.1 and 1.8	Bright orange to yellow	Adenomas
11		+	0.5	Yellow-red	Adenoma
12		+	3	Not mentioned	Adenoma
13		+	3	Not mentioned	Adenoma
14		+	3	Yellow	Adenoma

Abbreviations: Lt, left: Rt, right

[a]Plus sign indicates presence of adrenocortical adenoma