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Case Report

Adrenal cyst presenting with clinical features of a pheochromocytoma *,**

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ARTICLE INFO

Article history: Received 28 March 2024 Revised 29 May 2024 Accepted 3 June 2024

Keywords: Adrenal cyst Urology Adrenal incidentaloma Pheochromocytoma

ABSTRACT

Benign adrenal cysts are relatively uncommon variants of all adrenal incidentalomas. When identified, most benign adrenal cysts are asymptomatic, which differentiates them from other functional adrenal lesions. There are various types of adrenal cysts, although the most common being an endothelial cyst. Ultimately, evaluation and management approaches are aimed at ruling out a functional adrenal mass and management of symptoms if present. We present a unique presentation of an otherwise healthy male with a large incidental adrenal cyst, later identified as a benign endothelial cyst, who presented with classic symptoms of catecholamine excess. The patient had a negative hormonal evaluation, and his episodic symptoms were resolved with surgical removal of the adrenal mass. This case report and brief review provides valuable insight into the evaluation and management of a unique clinical scenario, where a benign cystic mass led to compression-related symptoms of catecholamine excess that were resolved after removal of the nonfunctional, cystic, mass.

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^{*} Competing Interests: The authors declare that they have no known competing financial interests or personal relationships that could have appeared to influence the work reported in this paper. DoD Disclaimer: The opinions and assertions contained herein are the private opinions of the authors and are not to be construed as reflecting the views of the Uniformed Services University of the Health Sciences, the U.S. Department of Defense, the U.S. Department of the Army, or the U.S. Department of the Navy.

^{☆☆} Acknowledgments: None.

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Introduction

Encompassing only about 1%-2% of all adrenal incidentalomas, benign adrenal cysts are relatively uncommon [1–3]. Typically, benign adrenal cysts are nonfunctioning lesions; however, a metabolic evaluation is often undertaken to rule out a functional adrenal mass, most concerning of which includes cystic pheochromocytoma or cystic adrenocortical carcinoma [3–8]. An even smaller proportion of benign adrenal cysts are noted to be derived from endothelium [8]. Large adrenal cysts presenting with signs of gastric outlet obstruction and hypertension have been previously reported [9,10]. We aim to present a unique case of a patient who presented with symptoms characteristic of a pheochromocytoma which ultimately proved to be related to mass effect of a large, benign, endothelial adrenal cyst.

Case report

A previously healthy 30-year-old male presented to a community emergency department (ED) with acute onset tachycardia, lightheadedness, chest tightness, and nausea following a large meal. The patient's symptoms were brief in nature. Interestingly, nine months earlier, the patient had experienced a similar episode of tachycardia and headache during a febrile illness attributed to rhinovirus. During the present visit, the ED evaluation was notable only for mild tachycardia to 108 beats per minute and nonspecific T wave inversions on electrocardiogram, a finding which was similarly observed during his prior episode. Complete metabolic panel (CMP), complete blood count (CBC), and high sensitivity troponin (HS-TP) were all normal. The patient was discharged home that evening but returned the next morning to the ED for similar symptoms but with the addition of fever to 38°C, pounding headache, and vertigo. On this occasion, the ED evaluation indicated tachycardia to 110 beats per minute, normal repeat CMP, CBC, and normal HS-TP; however, cross-sectional imaging, obtained to evaluate the patient for pulmonary emboli, revealed a right-sided suprarenal mass measuring 7 cm with enhancement to 25 Hounsfield units (HU). Of note, the imaging study was negative for pulmonary emboli. The patient was admitted to the internal medicine service for expedited work up of his adrenal mass. A triple phase CT scan confirmed the adrenal mass, which measured 7 x 5.3 x 6.5 cm and had a fluid density of 22 HU (Fig. 1). The patient was discharged home and referred for outpatient endocrinology evaluation. He subsequently underwent an unremarkable metabolic evaluation notable for plasma metanephrines 33.7 pg/mL (normal), normetanephrine 83.3 pg/mL (normal), plasma aldosterone 1.4 ng/dL (normal), plasma renin 0.650 ng/mL/hr (normal), DHEA 96.2 μg/dL (low), urine metanephrines 101 µg/L (normal), 1 mg dexamethasone suppression test 4.160 µg/dL (normal), salivary cortisol 0.010 μ g/dL (normal), TSH 2.73 μ IU/mL (normal). The patient was evaluated by urologic oncology and the decision was made to proceed with robot-assisted laparoscopic right adrenalectomy based on the size of the lesion and the presumption that compression of this large cystic lesion led to functional pheochromocytoma-like symptoms. The patient underwent uncomplicated right adrenalectomy and was discharged home on post operative day 2. Pathologic examination of the tissue revealed an adrenal gland with the large cyst measuring 5.5 x 5.0 x 2.7 cm arising from within the adrenal gland, adjacent to the medulla, and distorting the architecture of the adrenal cortex. Most of the adrenal gland was involved by the mass. Microscopically, the adrenal gland showed a cyst lined by vascular endothelium, including valves. The final pathology was read as right adrenal gland with benign endothelial (vascular) cyst (Figs. 2 and 3). The patient continues to do well without any significant sequalae as a result of his benign adrenal cyst. He has experienced no further episodes of tachycardia, hypertension, headaches, or hyperhidrosis postoperatively.



Fig. 1 – Contrast enhanced axial and coronal CT demonstrates a unilocular, non-enhancing water-density right adrenal cyst, measuring 5.8 cm (white arrow).

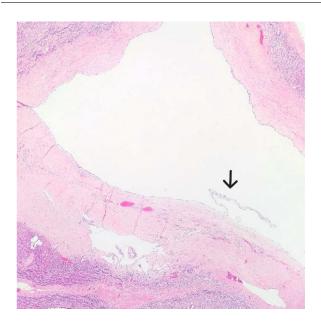


Fig. 2 – Photomicrograph of an endothelial-lined cyst with valve (black arrow), within the adrenal gland (dark purple tissue on outer edges). (H&E, original magnification 20x).

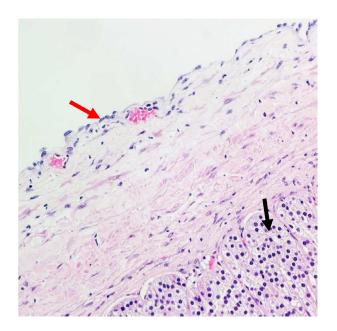


Fig. 3 – Photomicrograph of endothelial-type cyst lining (single layer of flattened cells with plump nuclei at left; red arrow) and adjacent normal adrenal cortex (pale pink foamy cells at bottom right; black arrow). (H&E, original magnification 200x).

Discussion

Adrenal cysts, although rare, have a growing incidence due to incidental detection related to advancements in imaging techniques [1,2,11]. There are few reports of adrenal cysts in the literature [9,10,12–16]. The lack of data compounded with the

various histological etiologies and heterogenous clinical manifestations of these adrenal lesions present diagnostic and management challenges for clinicians.

Adrenal cysts encompass a diverse spectrum of radiographic, hormonal, and histological subtypes of adrenal incidentalomas. Clinicopathological and imaging studies have demonstrated that these cysts are most prevalent in patients aged 30-50 years, with females being three times more likely to develop them than males [3,6,17]. Adrenal cysts have been documented as just a few millimeters up to 50 cm with a unilateral adrenal preference, although bilateral cysts have been reported in < 10% of cases [3]. Both computed tomography (CT) and magnetic resonance imaging (MRI) are key tools to initially characterize adrenal lesions, yet differentiating benign from malignant cysts remains challenging. Though most nonfunctional cysts are benign, an inactive hormone profile does not rule out malignancy [11]. Depending on size, location, hormone profile, and origin, these cysts can present with various symptoms such as mass effect, hypertension, palpable flank mass, infection, rupture, gastrointestinal symptoms, or vague abdominal pain. In the presented case, our patient presented with numerous sympathetically- mediated symptoms.

The current adrenal cyst histological classification is based on the 1966 Foster autopsy series which separates these entities into endothelial cysts (45%), pseudocysts (39%), epithelial cysts (9%), and parasitic cysts (7%) [18]. Not listed in this classification, but equally valuable in differential diagnoses for adrenal cysts, are solid adrenal masses with cystic changes, such as cystic adenomas, cystic pheochromocytomas, adrenal cortical carcinomas (ACC), cystic metastases, neurogenic tumors, hemangiomas, adrenal abscesses, teratomas, lymphomas, and granulomatous disease [19,20]. Table 1 encompasses typical imaging findings associated with these various adrenal lesions.

Endothelial cysts, thought to originate from vascular or lymphatic channels, are generally simple, benign cysts and are rarely associated with malignancy [16,18]. On CT, most endothelial cysts are homogenous, low density (< 20 HU), smooth lesions without intralesional enhancement [20]. MRI typically shows homogenous low T1/high T2 intensity with a few reports of intralesional hemorrhage, resulting in high intrinsic T1 signal content [20]. A few outliers have shown internal septations noted on lymphangiomatous subtypes and possible rim-enhancement due to surrounding compressed adrenal tissue [23]. These subtle variations on imaging may raise false malignancy concerns. More complex pseudocysts represent the second most common variant. These cysts are thought to result from prior adrenal hemorrhage secondary to infections, trauma, burns, or shock [18]. Generally, pseudocysts are high T2 signal, encapsulated, unilocular masses with variable T1 signal depending on the hemorrhagic or proteinaceous content. Compared to endothelial cysts, they may have calcifications in the capsule, lack an inner wall lining, and septations are more common. Unfortunately, these features are difficult to differentiate between metastases, necrosis, and abscesses on CT and MRI. Furthermore, these lesions have a low association with other neoplastic tissues, such as pheochromocytoma or other malignancies [15]. Epithelial and parasitic cysts are less common, non-vascular cysts. Epithelial cysts are true benign lesions with imaging findings suggestive of sim-

Table 1 – Review of typical computed tomography (CT), magnetic resonance imaging (MRI), and nuclear medicine imaging findings for various adrenal lesions [20–22].

		Imaging Modality		
		CT	MRI	Nuclear Medicine
Lesion type	Endothelial cyst	<20HU, homogenous, no intralesional enhancement	Low T1/high T2, homogeneous, +/- high intrinsic T1	*Paucity of data
	Pseudocysts	Well-defined round masses with fluid attenuation, no enhancement, may contain complexity, +/- calcifications	High T2, encapsulated, unilocular, +/- T1 intensity	*Paucity of data
	Pheochromocytoma (Cystic)	Enhancing rim, can be large, complex, cystic components common	T2 hyperintensity ("light-bulb" sign), avidly enhancing, intratumoral clefts possible	MIBG avidity common, 18F-FDG-PET can identify suspicious lesions that are MIBG negative
	Cystic metastases	Commonly bilateral, ill-defined margins, heterogenous, thick enhancing rim	Rim enhancement with low T1/ high T2	18F-FDG-PET avidity for metabolic activity
	Adrenocortical	Enhancing, heterogenous, large	T1 isointense with	18F-FDG-PET avidity,
	carcinoma	(>4cm), cystic components common for lesions >6cm, calcifications common, invasive behavior common	scattered hyperintensity	helpful for metastasis
	Adrenal adenomas	<3cm, =10HU, Homogenous, lipid rich</td <td>Loss of signal intensity on out-of-phase T1</td> <td>No significant FDG uptake on 18F-FDG-PET</td>	Loss of signal intensity on out-of-phase T1	No significant FDG uptake on 18F-FDG-PET

^{*} HU, Hounsfield unit; MIBG, metaiodobenzylguanidine; T1, longitudinal relaxation time; T2, transverse relaxation time; 18F-FDG-PET, ¹⁸F-fluorodeoxyglucose positron emission tomography.

ple cysts. Parasitic cysts are a rare subtype of adrenal cysts that are usually due to an *Echinococcus granulos*is or leishmaniasis infection [24,25]. Those patients typically have associated hepatic or splenic cysts and other constitutional symptoms [26]. A cystic mass with infolding and collapse of the parasitic membranes, producing the "water lily" sign, is the classic imaging appearance. Extra-adrenal involvement of either liver, spleen, or lungs would be expected [20].

Clinicians should consider solid adrenal masses with cystic changes on their differential when initial imaging reveals cystic lesions. Cystic pheochromocytomas are usually large, more complex-appearing lesions, that may outgrow their blood supply and result in interval cystic degenerative changes [27]. Most have an enhancing rim with an internal adrenomedullary tumor derived from chromaffin cells, hidden within septae, that may have positive biochemical findings [27]. Traditionally, pheochromocytomas tend to by hyperintense on T2 weighted MRI and avidly enhance with contrast [28,29]. MRI may reveal intra-tumoral clefts of high T2 signal approaching that of CSF, historically referred to as the "light bulb sign" [30]. Iodine meta-iodobenzylguadnidine (MIBG) scintigraphy can be used in the setting of suspected pheochromocytoma to localize primary lesions as well as identify distant metastases [28]. Additionally, MIBG scintigraphy is preferred over positron emission tomography (PET) CT for the detection of benign pheochromocytomas [28]. Clinicians need to be aware that spontaneous rupture of these cysts could lead to a life-threatening adrenergic crisis [27,31,32]. Cystic metastases to the adrenal gland appear similar in imaging presentation to pseudocysts - rim enhancement with low T1/high

T2 signal [22]. Common primary tumors with hematogenous adrenal spread patterns include lung, kidney, and lymphoma [22]. Some argue that ¹⁸F-fluorodeoxyglucose positron emission tomography (18F-FDG-PET) may be a useful modality in these lesions to delineate benign versus metabolically active lesions [22]. Adrenocortical carcinomas (ACC) are an aggressive malignant adrenal lesion that originate from the cortex [33]. Approximately 60% of these lesions are functional and present with other hormonal conditions, such as Cushing syndrome and hyperaldosteronism. There is thought to be a biological loading component since these have been reported in familial disorders such as multiple endocrine neoplasia (MEN) syndrome type 1, Beckwith-Wiedmann syndrome, and Li-Fraumeni [33]. ACCs are enhancing, heterogenous, and large (> 4 cm) masses on CT. In comparison, adrenal adenomas are benign, homogeneous, lipid-rich, cortex tumors that can secrete glucocorticoids, cortisol, and/or aldosterone [20]. These 2 adrenal lesions are distinguished histologically using the Weiss Scoring system, a standardized system consisting of 9 pathological criteria to differentiate adenoma from ACC [34,35].

The management of adrenal cysts remains ambiguous due to the lack of definitive preoperative diagnosis. The initial work-up for an adrenal incidentaloma involves both unenhanced CT adrenal imaging and hormonal profiling in parallel [11,36]. If the non-contrast imaging reveals a lesion > 4 cm or density of > 10 HU, then proceeding with an enhanced CT is recommended. Conservative management is generally recommended for cysts that are non-functional, < 4 cm, homogenous, < 10 HU, and/or asymptomatic. Cysts that grow > 1 cm

per year, develop hormonal autonomy, > 10 HU, large size, are symptomatic, heterogeneous or have other suspicious imaging findings may require intervention.

Our patient presented with a mix of benign and suspicious clinical and imaging features that ultimately led to surgery after shared decision-making. His adrenergic clinical symptoms, particularly after larger meals, in the context of a large, cystic lesion, elevated concern for a cystic pheochromocytoma with pseudo-gastric outlet obstruction due to mass effect, which also contributed to compression-mediated catecholamine release [9,37,38]. Despite the negative hormonal profile, this diagnosis was still a consideration as similar reports have been made for non-functioning lesions [39,40]. However, upon histological analysis, the patients adrenal incidentaloma was confirmed to be a benign, endothelial cyst. The patient's symptoms resolved following surgery, suggesting that even in the setting of a benign endothelial cyst, the sympathomimetic response to large meals was likely a result of mass effect from the large adrenal cyst. We hypothesize that due to the mass effect in the setting of larger meals, there was a temporary increase in circulating catecholamines due to medullary compression, which resolved between meals with gastric decompression and was unrecognizable on hormonal evaluation. This case underscores the potential for a large benign adrenal lesion to mimic the presentation of a hormonally active entity, most likely due to mass-effect. In this case, we catalog this rare clinical entity and demonstrate that surgical removal was curative of the patient's symptoms.

Patient consent

Written consent for publication was obtained from the patient who is described within this case report.

Data availability

The data within this case report is presented herein. Access to data outside of what is presented within this manuscript is protected for patient confidentiality.

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