American Journal of Case Reports

e-ISSN 1941-5923
© Am J Case Rep, 2021; 22: e928875
DOI: 10.12659/AJCR.928875

Received: 2020.09.28
Accepted: 2021.02.02
Available online: 2021.02.24
Published: 2021.04.04

Giant Adrenocortical Carcinoma: A Case Report and Review of the Relevant Literature

Authors' Contribution:
Study Design A
Data Collection B
Statistical Analysis C
Data Interpretation D
Manuscript Preparation E
Literature Search F
Funds Collection G

ADE 1 Ioannis Mantzoros
ADEF 1 Stefanos Bitsianis
BCDEF 1 Lydia Loutzidou
BDEF 1 Georgios Ntampakis
CDE 1 Christos Chatzakis
BEF 1 Panagiotis Christidis
AE 2 Anna Gkiouliava
AE 2 Eleni Koraki
EFG 1 Stamatios Aggelopoulos

1 4th Academic Department of General Surgery, General Hospital of Thessaloniki "G. Papanikolaou", Aristotle University of Thessaloniki, Exochi, Thessaloniki, Greece
2 Department of Anesthesiology, General Hospital of Thessaloniki "G. Papanikolaou", Exochi, Thessaloniki, Greece

Corresponding Author: Panagiotis Christidis, e-mail: panagiotischristidis13@gmail.com
Conflict of interest: None declared

Patient: Female, 63-year-old
Final Diagnosis: Adrenal cortical carcinoma
Symptoms: Abdominal pain and discomfort
Medication: —
Clinical Procedure: Excision of the recurrent mass along with the tail of the pancreas and a small part of the left lobe of the liver • extended open surgical excision of the mass with an esophago-jejunal anastomosis and a side to side jejuno-jejunal anastomosis
Specialty: Endocrinology and Metabolic • Oncology • Surgery

Objective: Rare disease

Background: Adrenocortical carcinomas are rare and aggressive tumors often diagnosed as incidentalomas. The malignancy can present with abnormal hormone secretion or the tumor may be non-functioning and present as a palpable mass causing discomfort. Here, we present a case of an adrenal cortical carcinoma originally identified as an incidentaloma.

Case Report: A 63-year-old woman presented with abdominal pain and discomfort. A large abdominal mass, occupying the left upper and lower quadrant, was palpated. Imaging revealed a mass occupying the left abdomen between the stomach and the spleen, applying pressure on the pylorus, duodenum, splenic vessels, and pancreas. The mass size was 21.2×13×14.6 cm. Hormonal investigations were normal. Surgical exploration was performed, and the tumor was excised. Pathological analysis revealed an adrenocortical carcinoma and the patient underwent adjuvant chemotherapy. Twelve months later, the carcinoma recurred. The patient underwent a second operation in which the recurrent mass was excised along with the tail of the pancreas and a small part of the left lobe of the liver. The postoperative period was uneventful, and the patient was discharged home on the 7th postoperative day. No further adjuvant therapy was applied. The patient remains disease-free 18 months after the reoperation.

Conclusions: Giant adrenocortical carcinomas, although rare, pose a challenge to the surgical team both diagnostically and therapeutically. Surgical excision with the appropriate oncologic support can guarantee excellent outcomes.

Keywords: Adrenal Gland Neoplasms • Adrenocortical Carcinoma • Case Reports • Incidental Findings • Mitotane

Full-text PDF: https://www.amjcaserep.com/abstract/index/idArt/928875

1613 1 6 23

Indexed in: [PMC] [PubMed] [Emerging Sources Citation Index (ESCI)] [Web of Science by Clarivate]

Background

Adrenocortical carcinomas (ACC) are rare and frequently aggressive tumors. Their worldwide incidence is estimated at 2 per million cases annually in the USA, affecting women more frequently than men [1]. The 5-year survival rate varies from 15% to 84% and is directly correlated with the stage of the cancer at presentation [2]. ACCs can be divided in 2 groups: functioning (hormone-producing) and non-functioning tumors [3].

The clinical presentation depends on the size, location, and hormonal production status. A large tumor can be the cause of abdominal pain and discomfort to the patient due to a mass effect. The patient might also present with generalized symptoms such as weight loss, malaise, and dyspnea [4].

The cornerstone therapy has always been complete surgical removal of the tumor and adjacent organs. Adjuvant chemotherapy with mitotane and cytotoxic factors can be used for functional adrenal carcinomas according to the stage of the disease and the status of the patient [5].

Here, we present the case of a patient with a large abdominal mass that turned out to be ACC and the patient underwent a radical surgical resection.

Case Report

A 63-year-old woman was referred from the gastroenterology clinic of our hospital to our unit with a giant solid mass in the upper abdomen of unknown origin. Her initial symptoms were abdominal pain and discomfort. Her past medical history included type II diabetes mellitus treated with metformin and a history of hysterectomy for uterine fibroids. On examination, a large abdominal mass was palpated occupying mostly the left upper and lower abdominal quadrant. Upper gastrointestinal endoscopy was normal. After investigation with CT and MRI abdominal scans (**Figures 1, 2**, respectively), a mass was found occupying the left abdomen, located between the stomach and the spleen. The mass was shown to originate from the left pararenal space extending to the greater curvature of the stomach, applying pressure to the pylorus, duodenum and splenic vessels, as well as being in direct contact with the pancreas (**Figure 1**). Due to the size (21.2×13×14.6 cm) and location of the mass, an adrenocortical carcinoma was suspected. After biochemical studies, no hormonal abnormalities were discovered (serum cortisol level: 188 ng/ml, normal reference range: 50-230 ng/mL), dehydroepiandrosterone level: 67 μg/dl (normal reference range: 45-270 μg/mL), plasma

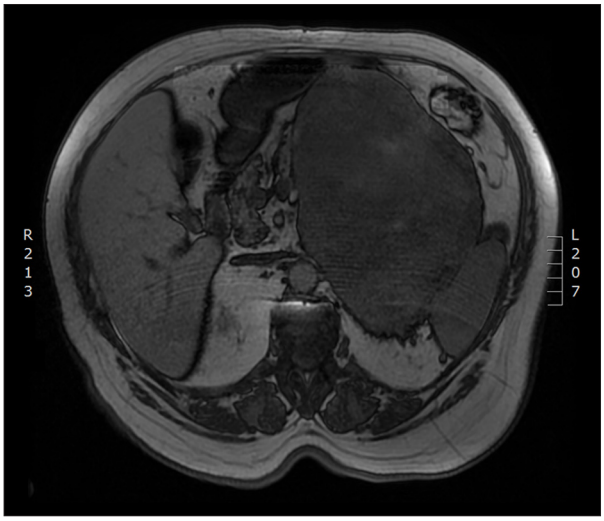

Figure 2. MRI scan with abdominal mass next to the left kidney.

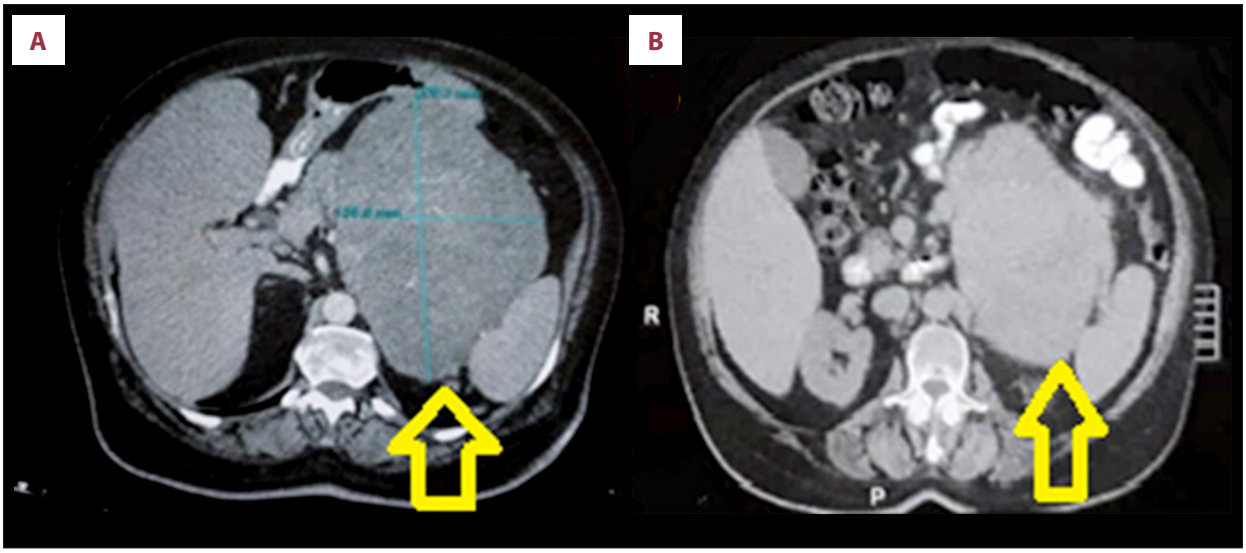

Figure 1. (**A, B**) CT scan with abdominal mass next to the left kidney.

Indexed in: [PMC] [PubMed] [Emerging Sources Citation Index (ESCI)] [Web of Science by Clarivate]

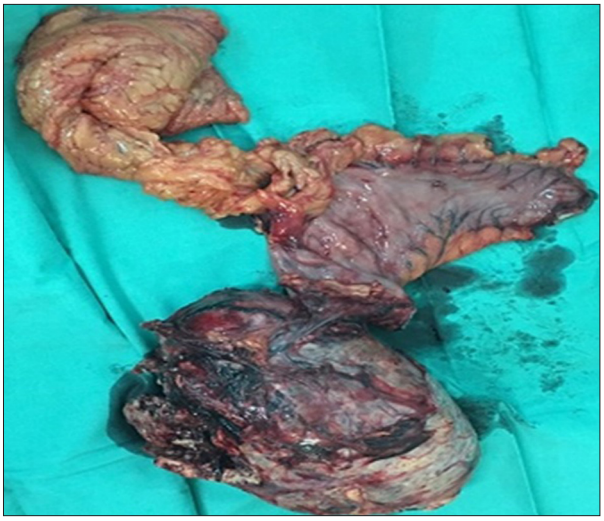

Figure 3. Histopathologic specimen from the first operation. Adrenal mass with the stomach and greater omentum attached.

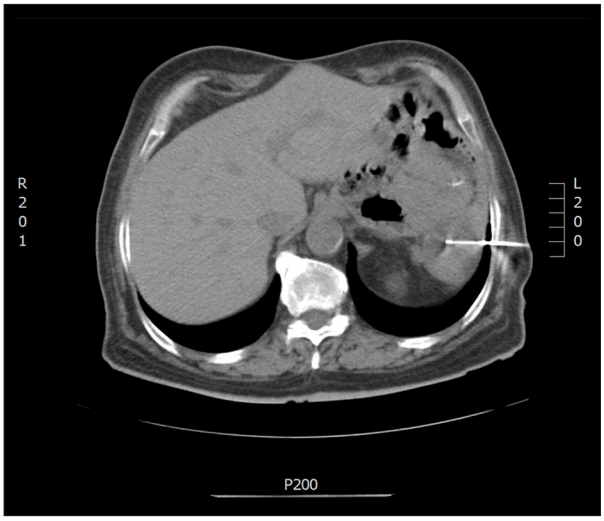

Figure 4. CT-guided biopsy of the recurred tumor, albeit the patient underwent radical surgical excision and adjuvant chemotherapy.

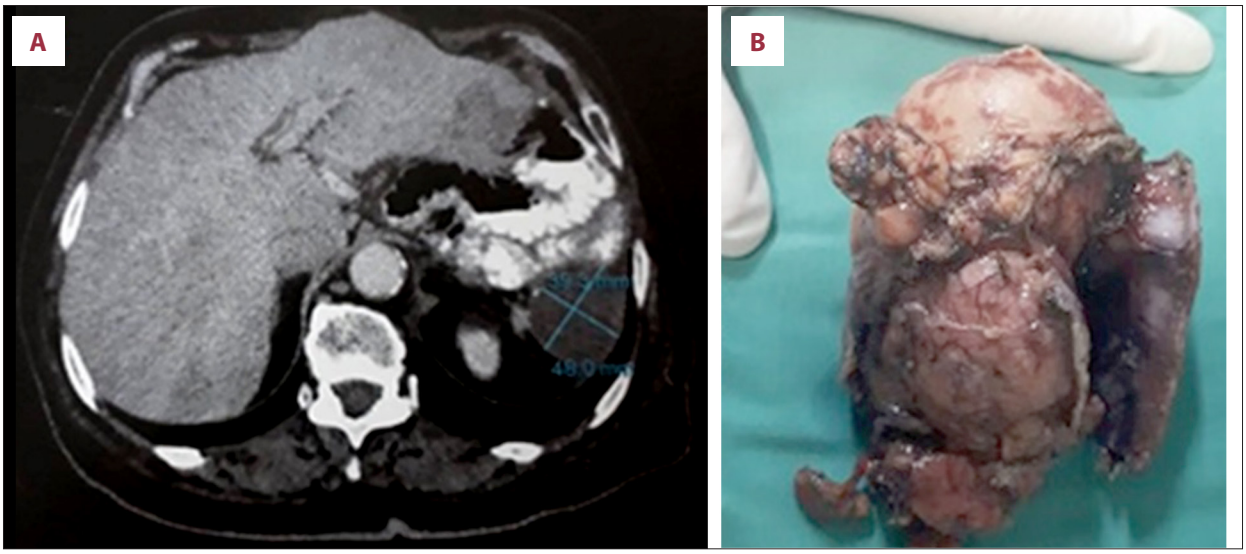

Figure 5. (**A**) CT scan revealing the recurrent tumor superior to left kidney (**B**): Histopathologic specimen from the second operation. Recurrent tumor excised. The tail of the pancreas and a small part of the left lobe of the liver were also removed during the second operation.

fractioned metanephrines 31 pg/mL (normal reference range: 12-60 pg/mL), plasma fractioned normetanephrines 75 pg/mL (normal reference range: 18-111 pg/mL), and aldosterone-to-renin ratio of <20 (normal reference range: 20-40).

The patient underwent an extended open surgical excision of the mass along with the stomach that was attached to it (**Figure 3**). An esophago-jejunal anastomosis and a side-to-side jejuno-jejunal anastomosis were performed. Resection margins were clear of residual mass, confirmed by frozen section study. Due to the duration of the operation (3.5 hours) and the co-morbidities of the patient (diabetes mellitus, obesity) she was transferred to the Intensive Care Unit (ICU) for postoperative resuscitation and remained there for the first 24 hours. In the immediate postoperative period, she developed a lower respiratory tract infection that was treated successfully with intravenous antibiotics. Otherwise, her postoperative period was uneventful and she was discharged home on the 22nd day.

The histology exam revealed that the mass was an adrenal cortical neoplasm (WHO 2004 Classification-tumors of endocrine organs) with the following immunochemistry profile: vimentin (+), CD56 (+), synaptophysin (+), melan-A (+/–), calretinin (+/–), CD138 (+). The modified Weiss score system incorporates the

Indexed in: [PMC] [PubMed] [Emerging Sources Citation Index (ESCI)] [Web of Science by Clarivate]

Table 1. Giant ACCs reported in the literature [3-5,7,10-19].

Author	Age	Sex	Presentation	Laboratory studies	Tumor size (cm)	Site	Histological findings	Immunohistochemical studies
Alastrue Vidal et al	45	F	Virilization	Elevated: T, DHEA-S	16.2	R	ACC	N/R
Alastrue Vidal et al	50	F	Virilization	Elevated: T, DHEA-S	20.3	R	ACC	N/R
Almarzouq et al	30	F	Abdominal pain, weight loss	Normal	20	L	ACC	Vimentin(+), Synaptophysin(+)
Bacalbasa et al	65	M	Caval compression syndrome, abdominal pain	N/R	35	R	ACC	Vimentin(+), Synaptophysin(+), Melan-A(+), Calretinin(+), Ki-67(+) (14%)
Bagchi et al	35	F	Altered menstrual symptoms	Elevated: serum cortisol, norepinephrine, dopamine	21	L	ACC	N/R
Benassai et al	53	M	Palpable mass on the L flank	Normal	24	L	ACC	N/R
Brown and Bacal	64	M	Abdominal distention	Normal	19	R	ACC	N/R
Chentli et al	34	F	Cushing	Elevated: Serum GLC, E2, T, 17OH P, CA125, Decreased: ACTH, K+, Hb	14.5	R ovary	ACC	Inhibin-A(+), Melan-A(+), SF1(+)
Chung et al	36	F	Incidental finding	Elevated: DHEA-S, Urinary Free Cortisol, 17-KS Decreased: ACTH	12	L	ACC	Synaptophysin(+), CD56(+), Inhibin-A(+), Melan-A(+)
Coli et al	75	F	Abdominal pain	N/R	15	L	Sarcomatoid ACC	MNF-116(+), Vimentin(+), Desmin(+), Actin(+), H-Caldesmon(+), Myogenin(+), HMB-45(+)
Fancellu et al	41	M	Feminization	Elevated: E2, Cortisol, ACTH Decreased: T, Gonadotropins	27	L	ACC	Melan-A(+), Synaptophysin(+)
Fernandez et al	64	F	Abdominal pain	N/R	12	R	ACC	N/R
Fimmano et al	61	M	N/R	N/R	24	R	ACC	N/R
Fulawka et al	27	M	Non-specific	N/R	22	L	ACC	Vimentin(+), Inhibin(+), Synaptophysin(+), BCL-2(+), Calretinin(+)
Ghorayeb et al	50	M	Palpable mass on the L flank	Elevated: DHEA	18	L	ACC	Ki-67(+) (12%), IGF-2(+), β-Catenin(+)
Habibi et al	38	F	Abdominal pain, palpable mass	Normal	22	L	ACC	Ki-67(+) (15-20%)
Hatano et al	60	M	Feminization	Elevated: E2, Preg, P, DOC, 17OH-P, DHEA-S Decreased: T, LH, FSH	13	R	ACC	Ki-67(+) (18%), SF1(+)

Indexed in: [PMC] [PubMed] [Emerging Sources Citation Index (ESCI)] [Web of Science by Clarivate]

Table 1 continued. Giant ACCs reported in the literature [3-5,7,10-19].

Author	Age	Sex	Presentation	Laboratory studies	Tumor size (cm)	Site	Histological findings	Immunohistochemical studies
Hoang et al	39	M	Ascites, abdominal mass	N/R	14	R	ACC	Cytokeratin(+), Vimentin(+), Synaptophysin(+)
Hoang et al	53	F	Abdominal pain	N/R	17	L	ACC	Cytokeratin(+), Vimentin(+), Synaptophysin(+)
Hoang et al	58	M	1 year History of rapidly enlarging adrenal mass	N/R	13	R	ACC	Cytokeratin(+), Vimentin(+), Synaptophysin(+)
Hsieh et al	82	F	Primary hyperaldo-steronism	Elevated: ALDO, Decreased: Plasma renin activity	13	L	Myxoid ACC	Synaptophysin(+), Melan-A(+), Vimentin(+)
Kalra et al	34	M	Incidental finding	Normal	16	L	ACC	Inhibin-A(+), Melan-A(+)
Kashiwagi et al	47	F	Lower back pain	Decreased: Hb	13.5	L	ACC	N/R
Khan et al	40	M	Incidental finding	Normal	30	R	ACC	N/R
Kovecsi et al	71	M	Weight loss, epigastric pain	Normal	13	R	ACC	Vimentin(+), Inhibin(+), Synaptophysin(+), NSE(+), Ki-67(+) (30%)
Kunieda et al	52	M	Weight loss, palpable mass	Elevated: Cortisol, S, DHEA-S, DHEA, 17-KS Decreased: ACTH	29	R	ACC	N/R
Lee et al	61	M	Right flank pain	VMA	12	R	Sarcomatoid ACC	Cytokeratin(+), Vimentin(+), NSE(+)
Lee et al	21	M	R flank pain, palpable mass	N/R	21	R	ACC	N/R
Meshikhes et al	20	M	R flank pain, palpable mass	Normal	24	L	ACC	Vimentin(+), Inhibin(+), Cytokeratin(+)
Ohwada et al	47	F	Incidental finding	Normal	18	R	ACC	N/R
Ohwada et al	68	M	Incidental finding	Normal	16	R	ACC	N/R
Ohwada et al	62	M	Weight loss, bilateral Lower extremities edema	Normal	20	R	ACC	N/R
Ohwada et al	43	F	Cushing	Elevated: 17-OHCS, 17-KS, DHEA-S	15	R	ACC	N/R
Onkar and Shilpi	47	M	Non-specific	Normal	22	L	ACC	N/R
Permana et al	21	F	Virilization	Elevated: T, DHEA-S, E2, Morning Cortisol Decreased: LH, FSH,	15.6	R	ACC	NSE(+), HEP1(+), CD56(+)

Indexed in: [PMC] [PubMed] [Emerging Sources Citation Index (ESCI)] [Web of Science by Clarivate]

Table 1 continued. Giant ACCs reported in the literature [3-5,7,10-19].

Author	Age	Sex	Presentation	Laboratory studies	Tumor size (cm)	Site	Histological findings	Immunohistochemical studies
Reyes et al	42	F	Right flank pain	Normal	12	R	ACC	N/R
Saeger et al	53	F	Incidental finding	N/R	13	R	Sarcomatoid ACC	β-Catenin(+), Vimentin(+), Synaptophysin(+), Desmin(+), SF1(+), Melan-A(+), Ki-67(+) (60%)
Sasaki et al	45	M	Epigastric pain, weight loss	Normal	17	L	Sarcomatoid ACC	Synaptophysin(+), Melan-A(+), Vimentin(+), Calretinin(+), Desmin(+), Myogenin(+), Myoglobin(+)
Souto et al	54	F	Cushing	Elevated: DHEA-S, AE, 17OH-P, T, Urinary free cortisol decreased: LH	21	L	ACC	Ki-67(+) (20%)
Straka et al	40	M	PE	NSE	26	R	ACC	Ki-67(+) (12%)
Sung et al	48	F	Palpable mass	N/R	19	R	Myxoid ACC	Ki-67(+) (4%)
Sung et al	59	F	Incidental finding	N/R	12.5	L	Myxoid ACC	Ki-67(+) (5%)
Sung et al	48	F	Non-specific	N/R	16	R	Myxoid ACC	Ki-67(+) (18%)
Sung et al	51	M	Non-specific	N/R	15	R	Sarcomatoid ACC	Ki-67(+) (12%)
Tseng et al	56	M	AKI, PE	Normal	24	R	ACC	Melan-A(+)
Uruc et al	48	F	Abdominal pain	Elevated: T, DHEA-S	23	L	ACC	Vimentin(+), Synaptophysin(+), Cytokeratin(+), Ki-67(+) (13%)
Veron Esquivel et al	39	F	HTN, HypoK, metabolic alkalosis	Elevated: ALDO, renin, cortisol, T, AE	13	R	ACC	N/R
Wei et al	53	F	Palpable mass	Elevated: T, P	12	L	ACC	N/R
Wilkinson et al	64	F	Abdominal pain	Normal	12	L	ACC	N/R
Wolf et al	46	M	Feminization, varicocele L	Elevated: P2, E, 17-OHCS, 17-KS	17	L	ACC	N/R
Yavascaoglu et al	51	M	L flank pain, weight loss, bilateral leg edema	Normal	18	L	ACC	N/R
Yeh et al	53	F	Virilization	Elevated: T, DHEA-S, AE	12	R	ACC	N/R

ACC – adrenocortical carcinoma; VMA – vanilmandelic acid; NSE – neuron specific enolase; N/R – not reported; ALDO – aldosterone; AE – androstenedione; T – testosterone; E – estrogens; P2 – pregnadiol; 17-OHCS – 17-hydroxycorticosteroids; 17-KS – 17-Ketosteroids; AKI – acute kidney injury; PE – pulmonary edema; SF1 – steroidogenic factor 1; DHEA-S – dehydroepiandrostenedione-sulfate; IGF-2 – insulin-like growth factor 2; GLC – glucose; P – progesterone; ACTH – adrenocorticotropic hormone; Hb – hemoglobin; Preg – pregnenolone; DOC – deoxycorticosterone; 17OH-P – 17-hydroxyprogesterone; LH – luteinizing hormone; FSH – follicle stimulating hormone, S – 11-Deoxycortisol.

Indexed in: [PMC] [PubMed] [Emerging Sources Citation Index (ESCI)] [Web of Science by Clarivate]

following criteria: 1) mitotic rate >5 per 50 high-power fields, 2) cytoplasm (clear cells comprising 25% or less of the tumor), 3) abnormal mitoses, 4) necrosis, and 5) capsular invasion. According to the system, each of these criteria is differently weighted and a score of 3 or more suggests malignancy [6]. In our patient, the modified Weiss score system of 6, size of 20 cm in diameter, and the ki-67 proliferation index of 5-7% were all suggestive of carcinoma. The patient underwent adjuvant chemotherapy postoperatively with mitotane treatment.

Despite adjuvant therapy, she presented with a cystic lesion 1 year after the operation. A CT-guided core biopsy was performed, which revealed recurrent adrenocortical carcinoma (**Figure 4**). The recurrence was located posterior lateral to the esophagojejunal anastomosis (**Figure 5A**). She underwent a second operation in which the recurrent mass was excised along with the tail of the pancreas and a small part of the left lobe of the liver (**Figure 5B**). The postoperative period was uneventful, and the patient discharged home on the 7th postoperative day. No further adjuvant therapy was applied. The patient undergoes follow-up with CT scan every 6 months, which revealed no recurrence 1.5 year after the reoperation, and no metastasis was observed.

Discussion

Adrenal cortical carcinoma (ACC) is an uncommon malignant endocrine neoplasm. ACC accounts for 0.02% of all cancers reported annually in the United States. It has an estimated incidence of 0.02 to 0.2 cases per million population per year [7]. The clinical presentation of adrenal cortical tumors depends on their size and hormonal status. Many non-functioning adrenal tumors are incidental findings and not always related to the patient's clinical presentation, and are therefore called incidentalomas [8].

Less than 2% of incidentalomas under 4 cm in size are primary adrenal carcinomas, while the risk for adrenal carcinoma increases to 25% in adrenal masses greater than 6 cm. The sensitivity and specificity for cutoffs of 4 cm are 98% and 59%, respectively [9,10]. Our patient presented with an abdominal mass of 21.2×13×14.6 cm, which rarely has an adrenal origin.

After thorough review of the literature, cases of adrenal carcinomas larger than 12 cm were documented (**Table 1**) [3-5,7,11-20]. The mean size of these tumors was 18 cm. The largest tumor, reported by Bacalbasa et al, was 35 cm [21]. The size of the tumor we resected was at the 74th percentile of the normal distribution curve. The tumor appears to be larger than the mean tumor size reported in the literature (**Figure 6**). The clinical manifestation of ACCs depends on their hormone-producing status. Non-functioning tumors can present with unspecific symptoms like abdominal pain, malaise, hematuria, and weight loss or no symptoms at all (incidental findings). Functioning tumors have a varied presentation, including Cushing's syndrome, virilization, and feminization [8]. Our patient had non-specific symptoms, none of which were associated with hormone production (abdominal pain, discomfort, palpable abdominal mass). Blood hormonal levels where within normal levels. The imaging studies that are most commonly and effectively used for the differential diagnosis between the types of adrenal masses are non-contrast CT, MRI, and adrenal scintigraphy [5].

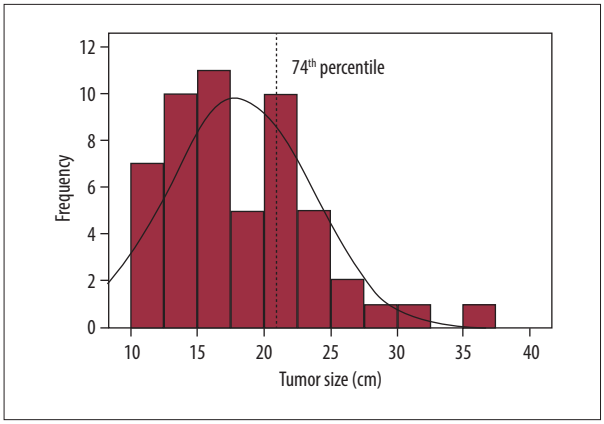

Figure 6. The size of the tumor we resected was at the 74th percentile of the normal distribution curve. The tumor appears to be larger than the mean tumor size reported in the literature.

The laboratory tests used for the differential diagnosis for adrenal masses depends on the clinical signs of hormonal production. In cases of obvious endocrine symptomatology there are a number of measurements that indicate the functionality of the mass. Dexamethasone suppression test and 24-hour urine cortisol sample are used in the presence of Cushing's syndrome, while plasma estradiol or estrone is measured when feminization is present [5,8]. When an adrenal lesion is incidentally found, appropriate laboratory tests to be performed are electrolyte levels measurement in the presence of hypertension to rule out hyperaldosteronism, urinary metanephrines and catecholamines levels to exclude pheochromocytoma, and estrogen or androgen levels where relevant symptoms exist [8]. Our patient underwent an abdominal CT and MRI scan, in which the mass was found, and a full blood biochemical work-up including cortisol, aldosterone, renin, metanephrines, normetaphrines, and dehydroepiandrosterone level measurement.

Radical surgical resection is suggested for all patients presenting with adrenal tumor (stage I, II, or III disease), while achieving an R0 excision remains the most important prognostic factor for survival. Laparoscopic adrenalectomy is an option for experienced surgeons and masses <6 cm, whereas open adrenalectomy remains the suggested option for larger masses, signs of local invasion, or if carcinoma/malignancy is suspected [22].

Indexed in: [PMC] [PubMed] [Emerging Sources Citation Index (ESCI)] [Web of Science by Clarivate]

We performed an extended surgical excision of the tumor along with the stomach that was attached to it and completed the operation with the appropriate anastomosis (esophago-jejunal anastomosis and a side-to-side jejuno-jejunal anastomosis). Radical resection is also the criterion standard strategy for recurrence. Similarly, the attached organs such as the left liver lobe and the tail of the pancreas were excised along with the recurrent mass. Our patient needed a second operation 1 year later due to recurrence, but she remains cancer free to date.

Mitotane, as a single agent or in combination with other cytotoxic drugs, is the current standard treatment for advanced ACCs [23]. Treatment regimens in ACC are mitotane monotherapy, EDP-M (etoposide, doxorubicin, cisplatin plus mitotane), or streptozotocin plus mitotane. However, time to progression was observed to be significantly better in patients treated with mitotane [20]. In our case, despite open radical excision and adjuvant chemotherapy with mitotane, adrenocortical carcinoma reoccurred after 1 year.

Recently, gene expression profiling has improved our understanding of the oncogenesis of ACC and helped identify potential new targets for treatment [20]. New avenues for ACC therapy have been opened by studies investigating the biological and molecular bases of this disease [23]. Several pathways have been identified in the tumorigenesis of ACC. IGF-2, mTOR, EGFR, and VEGF are overexpressed in ACC [23]. In vitro and in vivo studies have been performed to identify potential targeted therapies for ACC [20]. Those include IGF-1 receptor antagonists, β-catenin antagonists, SF-1 (steroidogenic factor 1) inverse agonists, and mTOR antagonists. PPARγ and estrogen receptors have also been identified as potential markers for ACC tumor-genesis [23].

Conclusions

Non-functioning adrenal incidentalomas are rare and present with vague symptoms such as dull abdominal pain, nausea, and discomfort. Their size is a strong indicator of their malignant potential. Masses larger than 6 cm have increased risk being carcinomas. The criterion standard treatment for adrenocortical carcinomas is open radical excision with adjuvant chemotherapy, although the recurrence rate and disease-fee survival remain unsatisfactory.

Conflict of Interest

None.

References:

1. Bilimoria KY, Shen WT, Elaraj D, et al. Adrenocortical carcinoma in the United States: Treatment utilization and prognostic factors. Cancer. 2008;113(11):3130-36
2. Kim Y, Margonis GA, Prescott JD, et al. Nomograms to predict recurrence-free and overall survival after curative resection of adrenocortical carcinoma. JAMA Surg. 2016;151(4):365-73
3. Hatano M, Takenaka Y, Inoue I, et al. Feminizing adrenocortical carcinoma with distinct histopathological findings. Intern Med. 2016;55(22):3301-7
4. Benassai G, Desiato V, Benassai G, et al. Adrenocortical carcinoma: What the surgeon needs to know. Case report and literature review. Vol. 12, International Journal of Surgery. Elsevier Ltd.; 2014;S22-28
5. Uruc F, Urkmez A, Yuksel OH, et al. Androgen secreting giant adrenocortical carcinoma with no metastases: A case report and review of the literature. Can Urol Assoc J. 2015;9(9-10):E644-47
6. Aubert S, Wacrenier A, Leroy X, et al. Weiss system revisited: A clinicopathologic and immunohistochemical study of 49 adrenocortical tumors. Am J Surg Pathol. 2002;26(12):1612-19
7. Phan AT. Adrenal cortical carcinoma-review of current knowledge and treatment practices. Vol. 21, Hematology/Oncology Clinics of North America. Hematol Oncol Clin North Am; 2007;489-507
8. Alastrué Vidal A, Navinés López J, Julián Ibáñez JF, et al. Adrenohepatic fusion: Adhesion or invasion in primary virilizant giant adrenal carcinoma? Implications for surgical resection. Two case report and review of the literature. Int J Surg Case Rep. 2016;18:24-29
9. Ye Y, Yuan X, Chen M, et al. Management of adrenal incidentaloma: the role of adrenalectomy may be underestimated. BMC Surg. 2016;16(1):41
10. Grumbach MM, Biller BMK, Braunstein GD, et al. Management of the clinically inapparent adrenal mass ("incidentaloma"). Ann Intern Med. 2003;138(5):424-29
11. Permana H, Darmawan G, Ritonga E, et al. An interesting case of hepatic adrenocortical carcinoma. Acta Med Indones. 2018;50(3):257-59
12. Meshikhes AWN, Abdel Gawad WM, Al-Saeed JY. Young male with left adrenal mass. BMJ Case Rep. 2016;2016:bcr2016215669
13. Chentli F, Terki N, Azzoug S. Ectopic adrenocortical carcinoma located in the ovary. Eur J Endocrinol. 2016;175(4):K17-23
14. Habibi M, Karakoyun R, Demirci E, Alikanoglu AS. Chylous ascites after resection of giant adrenocortical carcinoma. Gland Surg. 2016;5(6):639-43
15. Sung TY, Choi YM, Kim WG, et al. Myxoid and sarcomatoid variants of adrenocortical carcinoma: Analysis of rare variants in single tertiary care center. J Korean Med Sci. 2017;32(5):764-71
16. Ghorayeb N El, Grunenwald S, Nolet S, et al. First case report of an adrenocortical carcinoma caused by a BRCA2 mutation. Medicine (Baltimore). 2016;95(36):e4756
17. Saeger W, Mohren W, Behrend M, et al. Sarcomatoid adrenal carcinoma: Case report with contribution to pathogenesis. Endocr Pathol. 2017;28(2):139-45
18. Esquivel DV, Batiz F, Vega AF, Carrillo Gonzalez PA. Adrenocortical carcinoma, an unusual cause of secondary hypertension. BMJ Case Rep. 2016;2016:bcr2016217918
19. Estévez Fernández S, Artime Rial M, Domínguez Comesaña E, Sánchez Santos R. Giant adrenal cortical carcinoma. Cir Esp. 2017;95(9):542
20. Maluf DF, de Oliveira BH, Lalli E. Therapy of adrenocortical cancer: Present and future. Am J Cancer Res. 2011;1(2):222-32
21. Bacalbasa N, Terzea D, Jianu V, et al. Multiple visceral resection for giant non-secretory adrenocortical carcinoma in an elderly patient: A case report. Anticancer Res. 2015;35(4):2169-74
22. Fassnacht M, Arlt W, Bancos I, Dralle H, et al. Management of adrenal incidentalomas: European Society of Endocrinology Clinical Practice Guideline in collaboration with the European Network for the Study of Adrenal Tumors. Eur J Endocrinol. 2016;175(2):G1-34
23. Aufforth RD, Nilubol N. Emerging therapy for adrenocortical carcinoma. Int J Endocr Oncol. 2014;1(2):173-82

Indexed in: [PMC] [PubMed] [Emerging Sources Citation Index (ESCI)] [Web of Science by Clarivate]