Journal of the ASEAN Federation of Endocrine Societies

CASE REPORT

Adrenocortical Carcinoma with Suspected Dual Cortisol and Aldosterone Secretion: A Case Report

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Abstract

Adrenocortical carcinoma (ACC) is a rare malignancy, accounting for 0.05-2% of all malignant tumors. Most cases are sporadic and asymptomatic, often discovered incidentally via imaging. However, 50-60% present with autonomous hormonal secretion, with 45% producing excess cortisol, and only 1% exhibiting mineralocorticoid excess.

We report a 43-year-old Filipino female with a 6-month history of abdominal bloating, proximal muscle weakness, edema, and hypertension. Laboratory tests showed hypokalemia (serum potassium 2.5-3.05 mmol/L; normal 3.3-5.0 mmol/L). Imaging revealed a neoplastic right suprarenal mass (adrenal origin vs confluent lymphadenopathy) described as an incidental, lipid–poor right adrenal mass measuring 4.7 x 3.6 x 6.1 cm, with contrast-enhanced Hounsfield units of 40, along with periportal and paraaortic lymphadenopathies.

Biochemical evaluation indicated an intermediate probability of primary aldosteronism: suppressed plasma renin at 0.97 ug/L/hr (0.97 ng/mL/hr), normal plasma aldosterone 599.46 pmol/L (21.61 ng/dl), and aldosterone-renin ratio of 618 (pmol/L)/ (ng/L/hr) (22.27 ng/dl/ng/mL/hr). Ideally, confirmatory testing with an aldosterone suppression test prior to imaging and adrenal vein sampling (AVS) should be performed to lateralize the lesion before surgery; however, the patient opted to proceed with surgery. The patient also had overt hypercortisolism, with non-suppressed serum cortisol of 31.88 µg/dL (reference <1.8 µg/dL) on a low-dose dexamethasone suppression test, suppressed ACTH at 2.81 pg/mL (reference 7.7–63.6 pg/mL), and clinical features of Cushing syndrome, including facial plethora, and proximal muscle weakness. While these findings support the diagnosis of an ACTH-independent hypercortisolism, current guidelines recommend confirmatory testing, including 24-hour urinary free cortisol or late-night salivary cortisol, before definitive evaluation to distinguish ACTH-dependent from ACTH-independent disease. Concomitant catecholamine and androgen excess were excluded with normal 24-hour urine metanephrines and serum DHEAS.

Surgical removal via open adrenalectomy was performed. Histopathology confirmed ACC, supported by immunohistochemistry. Postoperative treatment included radiotherapy and planned adjuvant mitotane therapy. During follow-up, the patient's blood glucose and blood pressure worsened, requiring medication adjustments. Unfortunately, the patient succumbed to the disease seven months post-surgery.

This case highlights the difficulties in assessing adrenal tumors that concurrently secrete cortisol and aldosterone, especially when biochemical results are inconclusive, emphasizing the need for careful assessment and prompt intervention in this rare coexistence.

Key words: adrenocortical carcinoma, hyperaldosteronism, hypercortisolism

INTRODUCTION

Adrenocortical carcinomas (ACC) are rare and highly malignant tumors, accounting for less than 2% of all malignant cancers. Most adult cases are sporadic, with many patients remaining asymptomatic and diagnosed incidentally during imaging.[1] However, some patients present with symptoms caused by autonomous hormone secretion or local effects from an abdominal or adrenal mass. Typically, 50 to 60% of ACCs manifest as functioning adrenal tumors. Symptoms of excess cortisol are observed in 45% of cases, while signs of mineralocorticoid excess are much less common, affecting only about 1% of patients.

Dual hormone-secreting ACCs, producing both cortisol and aldosterone, are rare and pose diagnostic challenges, as biochemical abnormalities may overlap and must be interpreted in the context of clinical features. Current

eISSN 2308-118x (Online)
Printed in the Philippines

Received: January 16, 2026. Accepted: March 5, 2026.
Published online first: August 7, 2026.
https://doi.org/10.15605/jafes.041.02.5875

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guideline-based evaluation of primary aldosteronism involves screening with measurement of the aldosterone–renin ratio followed by confirmatory suppression testing, and localization with imaging or adrenal vein sampling.[2] On the other hand, assessment of cortisol excess requires two concordantly abnormal findings on dexamethasone suppression testing, 24-hour urinary free cortisol, or late-night salivary cortisol, followed by further evaluation for its cause via ACTH measurement.[3]

The diagnosis of ACCs involves biochemical testing, followed by surgical resection and histopathologic confirmation. Adjuvant therapies, such as radiotherapy and mitotane, and regular monitoring for disease progression or recurrence are recommended. This report describes a rare case of adrenocortical carcinoma that initially presented with clinical and biochemical findings suggestive of cortisol excess and possible aldosterone co-secretion, although confirmatory testing for aldosterone was not performed, highlighting the challenges in evaluating dual hormone-secreting tumors.

CASE

A 43-year-old female with no known comorbidities presented with a six-month history of facial swelling, bilateral lower extremity weakness, and bipedal edema, along with generalized bloating that was relieved with rest. The patient had no history of medication use and denied smoking or alcohol consumption. A cardiology consultation was performed six months before admission, which revealed hypertriglyceridemia; she was started on fenofibrate 200 mg once daily and was highly compliant. Follow-up history showed persistent bipedal edema, bilateral lower extremity weakness, and abdominal bloating; therefore, a gastroenterology consult was arranged, and an EGD showed signs of gastritis from *Helicobacter pylori* infection. The patient was sent home with 40 mg pantoprazole once daily, clarithromycin twice daily, and amoxicillin three times daily for 10 days.

During follow-up, the patient reported persistent bipedal edema, bilateral lower extremity weakness, and abdominal bloating, now accompanied by elevated blood pressure and blood glucose levels. Blood pressure was controlled with valsartan 80 mg once daily. Workup showed hypokalemia (serum potassium 3.05 mmol/L) and hepatomegaly with severe fatty changes on whole abdomen ultrasound. The patient was subsequently started on furosemide 20 mg once daily and spironolactone 25 mg once daily, with a recommendation for an endocrinology consult to evaluate the elevated blood glucose.

During follow-up with the cardiology service, the patient continued to have persistent bilateral lower extremity weakness, bilateral lower extremity edema, and abdominal bloating. Additionally, the patient's persistently elevated blood pressure levels necessitated an adjustment of medication to valsartan/amlodipine 160/10 mg once daily, while spironolactone was maintained. A whole abdominal CT scan with triple contrast was subsequently performed to evaluate the persistent abdominal bloating. The results showed an enhancing right suprarenal mass measuring 4.7 x 3.6 x 6.1 cm, likely neoplastic (adrenal origin versus confluent lymphadenopathy), and periportal and para-aortic lymphadenopathies likely indicating metastasis. It was identified as a lipid-poor soft-tissue mass with contrast-enhanced Hounsfield units of 40. Heterogeneous enhancement, non-contrast Hounsfield units, contrast washout values, and neovascularization were not evaluated because an adrenal protocol was not conducted during the initial imaging. The patient was referred to the endocrinology service for further evaluation.

From an endocrinology perspective, the patient denied any episodes of diplopia, increased shoe or ring size, or headache. The patient had no complaints of increased perspiration, heat or cold intolerance, tremors, or changes in BM. The patient had no history of steroid use and denies any episodes of easy bruising but reports episodes of proximal muscle weakness and weight gain (56 kg to 67 kg within one month). Physical examination revealed moon facies, a dorsocervical fat pad, and no violaceous striae over the abdomen.

In the case of an incidentally discovered adrenal mass, hypertension, and hypokalemia (serum potassium levels between 2.5 and 3.05 mmol/L), workup for primary aldosteronism was performed after correcting serum potassium levels and discontinuing spironolactone. Plasma renin activity was at the lower end of the reference range at 0.97 ug/L/hr (0.97 ng/mL/hr), while plasma aldosterone was 599.46 pmol/L (21.61 ng/dL). The aldosterone-renin ratio was elevated to 618 (pmol/L)/(ng/L/hr) (22.27 ng/dL/ng/mL/hr); therefore, it was considered a positive screening test for primary aldosteronism. Given these findings, the patient has an intermediate likelihood of primary aldosteronism. Ideally, confirmatory aldosterone suppression testing should be performed before adrenal computed tomography and adrenal venous sampling (AVS) to determine lateralization prior to surgical intervention in patients over 35 years old.[2] In this case, aldosterone suppression testing was recommended but not performed due to financial constraints, as the patient chose to proceed with surgery instead. Nevertheless, although AVS was discussed as part of the ideal guideline-directed workup for primary aldosteronism, proceeding with definitive surgical management was ultimately deemed more clinically appropriate than pursuing AVS for localization, given the clinical findings and radiographic features suggestive of adrenocortical carcinoma with possible nodal metastases.

In line with this, an important consideration in the diagnostic workup was to first rule out potentially life-threatening conditions, such as pheochromocytoma, prior to surgery, followed by optimizing blood pressure and metabolic control, and correcting hypokalemia before definitive intervention. Additional biochemical workup

Table 1. Biochemical testing results

Laboratory test	Result	Reference range
Plasma aldosterone	599.46 pmol/L (21.61 ng/dl)	111 – 860 pmol/L (4 – 31 ng/dl)
Plasma renin activity	0.97 ug/L/hr (0.97 ng/ml/hr)	0.39-3.94 ng/L/hr (0.39-3.94 ng/ml/hr)
Aldosterone – Renin ratio	618 pmol/L per ng/L/hr (22.27 ng/dl/ng/mL/hr)	<554 pmol/L per ng/L/hr (<20 ng/dl/ng/mL/hr)
Serum cortisol (1 mg dexamethasone suppression test)	31.88 mcg/dl	<1.8 mcg/dl
Serum ACTH	2.81 pg/ml	7.2-63.6 pg/ml
Dehydroepiandrosterone sulfate (DHEAS)	0.44 mg/L	0.27 – 2.4 mg/l
TSH	0.196 uIU/ml	0.27-4.20 uIU/ml
FT4	13.10	12-22 pmol/L
HbA1c	9.3%	
24-hour urine metanephrines	0.23 mg/ 24 hrs	0 – 1 mg/ 24 hours

was therefore performed, including evaluation for cortisol excess and pheochromocytoma. Catecholamine and androgen excess were ruled out with normal 24-hour urine metanephrines and DHEAS levels — 0.23 mg/24 hours and 0.44 mg/L, respectively. Serum cortisol after a low-dose dexamethasone suppression test was not suppressed (31.88 mcg/dL), and ACTH levels were 2.81 pg/mL, suggesting ACTH-independent Cushing syndrome secondary to an adrenal mass. Similarly, guideline recommendations for additional workup, including 24-hour urinary free cortisol or late-night salivary cortisol, were not performed before definitive evaluation to distinguish ACTH-dependent from ACTH-independent disease, as the patient opted to proceed with surgical management instead.[3]

The patient was subsequently referred to the urology service for eventual adrenalectomy. Clearances from cardiology and endocrinology were obtained, showing normal levels of creatinine, serum sodium, serum ionized calcium, and complete blood count. The 12-lead ECG indicated cardiomegaly. However, based on Simpson's criteria, 2D echocardiography results were within normal limits, with an ejection fraction of 71%.

The patient then underwent an open adrenalectomy, revealing the following intraoperative findings: a right adrenal mass measuring 6 cm adherent to the inferior vena cava, the superior pole of the kidney, the inferior surface of the liver, and the portal vein. The right adrenal mass was dissected from these structures, and 19 lymph nodes attached to the inferior vena cava were also removed. Histopathologic evaluation of the adrenal tumor under light microscopy demonstrated features consistent with a Weiss score of 3, indicating malignant behavior. Immunohistochemical studies showed a Ki-67 index of 30%, suggesting high proliferative activity. Additionally, the tumor was confirmed as adrenal cortical carcinoma through additional stains (calretinin and inhibin) (Figure 1). Given the malignant histopathologic features and elevated proliferative index, adjuvant therapy was recommended to lower the risk of recurrence and enhance disease management. The patient underwent 25 radiotherapy sessions, which were well tolerated. An oncology consultation was performed, and the patient was advised to undergo chemotherapy with mitotane. Postoperatively, there was early improvement in blood pressure and glucose control, along with reduced signs and symptoms of hypercortisolism. The patient was instructed to follow up regularly to monitor for tumor recurrence but was lost to follow-up. During subsequent visits, the patient's blood glucose and blood pressure increased, requiring medication adjustments. Unfortunately, the patient succumbed to the disease seven months after surgery.

DISCUSSION

Adrenocortical carcinoma (ACC) is a rare cancer occurring at a rate of 0.5-2 cases per million people per year, with the highest incidence between ages 40 and 60, primarily affecting women.[4] ACC typically presents as a functioning adrenal tumor in 50-60% of cases. Most patients exhibit symptoms of hypercortisolism, accounting for 45% of cases, while only 1% of the tumors produce aldosterone. The patient denied any episodes of easy bruising but reported proximal muscle weakness, weight gain (from 56 kg to 67 kg in 1 month), along with physical signs like moon facies, dorsocervical fat pad, and bipedal edema, which indicate hypercortisolism from Cushing Syndrome. Additionally, the combination of hypertension and hypokalemia in the presence of a unilateral adrenal mass should prompt evaluation for mineralocorticoid excess.

The diagnosis of ACC involves a combination of clinical, biochemical, and radiological assessments. A comprehensive hormonal workup is essential, as it can serve as a marker for tumor recurrence and help evaluate the patient's clinical features. Additionally, this workup aids in preoperative, intraoperative, and postoperative assessments.[5] The absence of autonomous steroid secretion decreases the likelihood that an adrenal mass is an adrenocortical cancer.[5]

Autonomous hormone co-secretion has emerged as a recurring feature in published case reports of ACC, highlighting the importance of thorough biochemical assessment in all patients with adrenal masses. Previous reports also suggest that cortisol-producing ACCs are associated with poorer clinical outcomes than nonfunctioning tumors, possibly due to the metabolic derangements and immunosuppressive effects of hypercortisolism.[6,7] A multivariate analysis from a study evaluating the prognostic

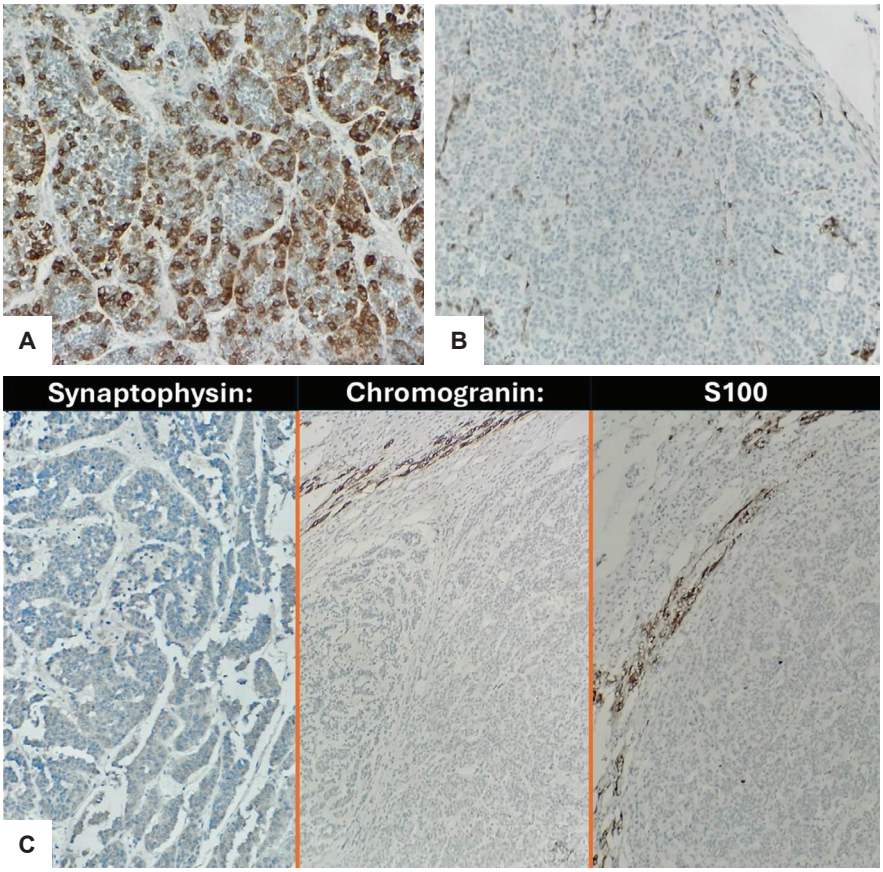

Figure 1. Immunohistochemical staining on low power magnification with inhibin, calretinin, synaptophysin, chromogranin and S100 to support the diagnosis. **(A)** Staining with inhibin shows positive membranous staining in the tumor cells. **(B)** Immunohistochemical staining with calretinin shows focal positive cytoplasmic staining in the tumor cells. **(C)** Immunohistochemical staining for synaptophysin, chromogranin and s100 all show negative findings.

role of overt hypercortisolism in completely operated ACC demonstrated that patients with hypercortisolism had decreased recurrence-free survival and overall survival, even after adjustment for established prognostic factors, including age, sex, tumor stage, and tumor size.[1,7] These findings further support that hypercortisolism is associated with a more aggressive behavior. As previously discussed, hormonal workup also aids in preoperative assessment. In this case, identifying hypercortisolism was crucial for anticipating and managing postoperative adrenal insufficiency, a potentially life-threatening complication.[5,6]

In contrast, the prognostic impact of concomitant aldosterone secretion remains uncertain because of the limited number of reported cases.[6]

Reported cases of aldosterone and cortisol co-secretion demonstrate a consistent biochemical profile characterized by hypokalemia, suppressed renin, elevated aldosterone levels, and non-suppressed cortisol.[5,8] Clinically, patients often initially present with abdominal pain or symptoms attributable to mass effect or hormone excess.[9] Additionally, most of the reported cases involved adrenal lesions measuring >5 cm, with a range of 6-22 cm, similar to the case of our patient.[8,9]

In this patient's case, plasma renin activity was suppressed below 1 ng/ml/hour, with a normal plasma aldosterone concentration of 599.46 pmol/L (21.61 ng/dl), and an elevated aldosterone-renin ratio of 618 (pmol/L)/ (ng/L/hr) (22.27 ng/dl/ng/mL/hr) indicating a positive screening for primary aldosteronism.[2] Following the 2025 clinical practice guideline of the Endocrine Society on primary aldosteronism, confirmatory aldosterone suppression testing is recommended in patients with intermediate probability prior to subtype classification. If confirmatory testing is positive, adrenal computed tomography followed by adrenal venous sampling (AVS) is advised to establish lateralization before surgical intervention, particularly in patients older than 35 years, given the limited reliability of imaging alone in distinguishing unilateral adenoma from bilateral adrenal hyperplasia.[2] However, testing was not done due to financial constraints; also, the patient was willing and was able to undergo surgery instead. Conversely, cortisol hypersecretion was evident, with non-suppressed cortisol levels at 31.88 mg/dl after a 1 mg dexamethasone suppression test. A low ACTH level of 2.81 pg/ml suggests an ACTH-independent hypercortisolism. Although the patient had a non-suppressed cortisol level after the 1 mg dexamethasone suppression test and an adrenal mass, she did not meet criteria for mild autonomous cortisol secretion (MACS), as she exhibited overt clinical features consistent with Cushing syndrome, indicating clinically significant hypercortisolism rather than subclinical disease.[8,10] Exclusion of pheochromocytoma before surgery is warranted, as preoperative preparation is necessary among patients with catecholamine excess.[5] The patient had normal 24-hour urine metanephrines; therefore, pheochromocytoma was not considered. Testing for adrenal androgen excess,

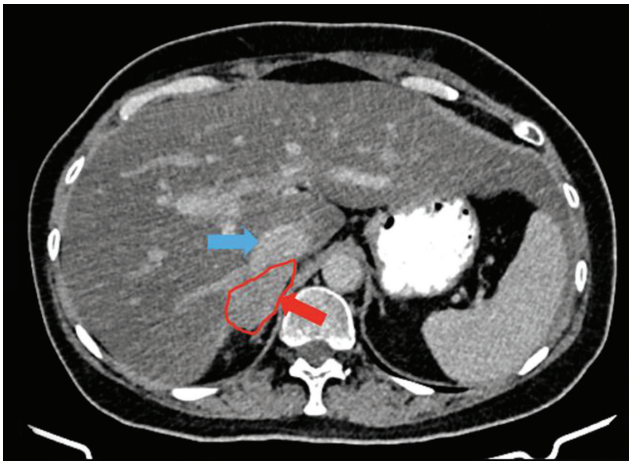

Figure 2. Axial contrast-enhanced CT image of the abdomen demonstrating a 4.7 × 3.6 × 6.1 cm enhancing right adrenal mass *(red arrow)* with post-contrast attenuation of 40 HU, consistent with a lipid-poor lesion. The mass abuts hepatic segments VI and VII and compresses the inferior vena cava *(blue arrow)*. The size (>4 cm) and imaging characteristics are suspicious for malignancy; the patient was subsequently diagnosed with adrenocortical carcinoma with suspected aldosterone and cortisol co-secretion.

for which the patient showed normal findings, was also performed since mixed cortisol and androgen secretion is highly suggestive of malignancy.[11]

The biochemical diagnosis is supported by imaging to differentiate between adrenal adenoma, adrenocortical carcinoma, pheochromocytoma, and metastatic lesions.[8] The patient was evaluated with an enhancing right suprarenal soft tissue and lipid-poor mass, measuring 4.7 x 3.6 x 6.1 cm, with Hounsfield units of 40, along with periportal and paraaortic lymphadenopathies that are likely indicative of metastasis (Figure 2). In this context, features suggestive of adrenal malignancy included a size of 4-6 cm and contrast-enhanced Hounsfield units greater than 10.[12] Heterogeneous enhancement, non-contrast Hounsfield units, contrast washout values, and neovascularization were not assessed, as an adrenal protocol was not performed during the initial imaging.

Given the high suspicion of adrenocortical carcinoma, based on a florid presentation of endocrine-related symptoms developing over six months and the onset of new metabolic comorbidities, including hypertension and Type 2 Diabetes Mellitus, along with imaging characteristics and tumor size suggestive of malignancy, an open adrenalectomy was performed to achieve complete tumor removal despite an indeterminate biochemical workup. Postoperative TNM staging is crucial for prognosis and evaluating treatment options.[1,8] In this case, the right adrenal mass was only partially resected due to its size; however, 19 locoregional lymph nodes were retrieved, categorizing it as TNM Stage III, with complete resection as the ideal treatment. Patients with advanced adrenal carcinomas may also show vascular invasion, primarily into the renal vein and inferior vena cava.[8] Grossly, ACC lesions are typically large and heterogeneous, often appearing brown, orange, or yellow due to their lipid content.

Histopathologic examination of the specimen after surgery confirms the diagnosis of ACC. The Weiss scoring system is also utilized, based on the identification of morphologic parameters under light microscopy. It classifies tumors based on morphologic and cytologic features and invasive properties.[13] The presence of three or more criteria from the Weiss score, including the following, strongly indicates malignant behavior: atypical mitoses (>33% of the tumor), capsular invasion, necrosis, and venous or sinusoidal invasion.[5,14] Histopathologic evaluation of the patient's tumor fulfilled three of the following criteria from the Weiss score, namely: atypical mitotic figures, suspicious capsule infiltration, and a diffuse architecture of more than 33% of the tumor characterized by atypical cuboidal to rounded cells arranged in nests visible at low magnification – corresponding to a Weiss score of 3 (Figure 3).

Furthermore, immunohistochemical stains play a role in differentiating primary adrenocortical tumors from non-adrenocortical tumors.[1,8] Immunohistochemical staining for the confirmation of dual secretion of aldosterone and cortisol was not conducted, as available resources were allocated towards investigations aimed at establishing the diagnosis of adrenocortical carcinoma. The immunohistochemical staining supportive of adrenocortical carcinoma diagnosis was positive, indicated by calretinin and inhibin. According to a 2022 study by Jorda et al., the mere detection of inhibin and calretinin alone does not provide sufficient sensitivity for diagnosing adrenocortical tumors, with sensitivities of 70% and 73%, respectively. However, the combined use of these two antigens enhances sensitivity to 94%.[15] Additional stains, including S100, Synaptophysin, and Chromogranin, which are markers for neuroendocrine tumors, were negative, ruling out pheochromocytoma (Figure 1).[5]

Postoperatively, prognosis is classified according to TNM ENSAT staging or GRAS criteria.[16,17] The TNM staging is evaluated both before and after surgery, with stage 1 defined as a localized adrenal tumor less than 5 cm in size, stage 2 as a localized tumor larger than 5 cm, stage 3 as a tumor invading the surrounding adipose tissue, adjacent organs, renal vein, or vena cava regardless of lymph node involvement, and finally, stage 4 as the presence of distant metastases regardless of tumor size and lymph node status.[1,15] The patient was classified as stage 3, which correlates with a 5-year survival rate of 19-54%.[16,18]

The GRAS criteria include four prognostic factors that independently predict relapse and overall survival: grade (Weiss scoring or Ki-67), primary resection status, patient's age, and presence of secretory syndrome. Resection status is classified as R0 (microscopically complete), R1 (microscopically incomplete), R2 (macroscopically incomplete), or Rx when unknown.[16] The adrenal lesion

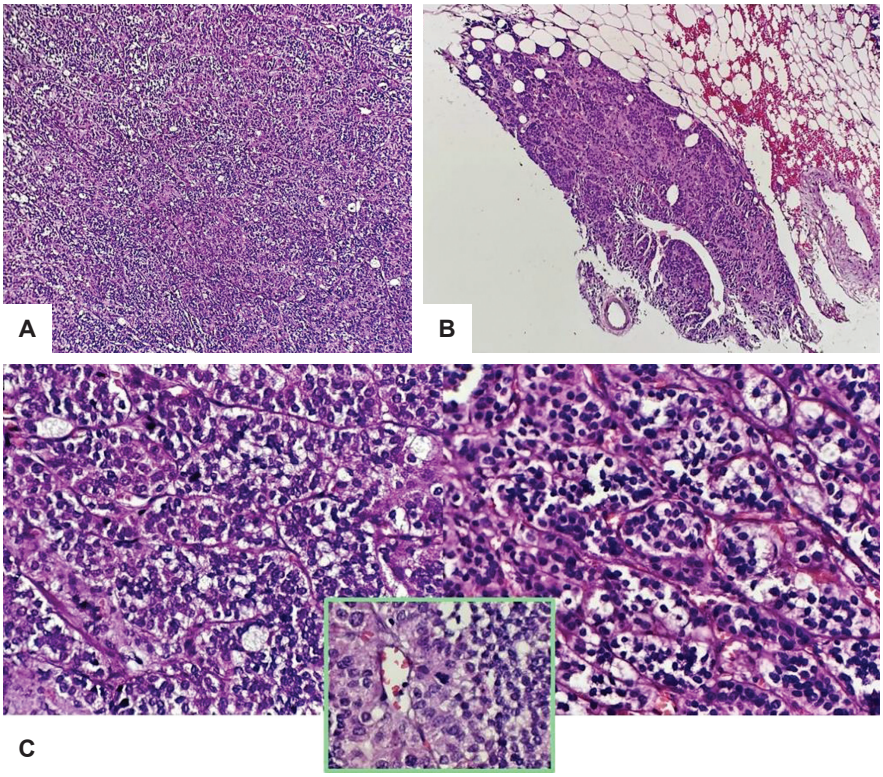

Figure 3. Histopathologic evaluation of the right adrenal gland. **(A)** Low power magnification of the adrenal mass shows atypical cuboidal to round cells disposed in nests (H&E, 10x). **(B)** Atypical cells are seen infiltrating into the peri-adrenal fat in other microsections (H&E, 10x). **(C)** High power magnification of the adrenal mass shows that the mildly pleomorphic, atypical cells have vesicular, hyperchromatic nuclei, some with prominent nucleoli. These atypical cells have moderate eosinophilic, granular to clear cytoplasm. Also noted in the picture insert is an atypical mitosis (H&E, 40x).

in our patient was incompletely resected, classified as R2, with an overall 5-year survival rate of 13%. The grade is determined by Ki-67 immunohistochemistry, which assesses cell proliferation; scores exceeding 10% indicate a poor prognosis, higher recurrence risk, and rapid disease progression.[8,16] The patient's Ki-67 was 30%, showing high proliferation and locally advanced ACC, which suggests a decreased overall survival. Due to the variable 5-year survival rates after resection, adjuvant therapy, radiotherapy, and mitotane were recommended, along with follow-up using cross-sectional imaging of the chest, abdomen, and pelvis to detect any new lesions, recurrence, or disease progression.

The patient completed 25 radiotherapy sessions without complications and was advised to start mitotane therapy; however, she was lost to follow-up. Her blood glucose and blood pressure levels rose during treatment, requiring medication adjustments. Seven months postoperatively, the patient died from the disease.

CONCLUSION

Adrenocortical carcinoma is a rare malignancy, with 50-60% of cases initially presenting as hypercortisolism and 1% exhibiting mineralocorticoid excess. To confirm the diagnosis, a detailed history, physical exam, and biochemical and imaging tests are essential in managing adrenocortical carcinoma. Symptoms can result from excess hormone production. Uncontrolled hypertension and hypokalemia should prompt evaluation for primary aldosteronism. Conversely, signs of excess cortisol, such as moon facies, dorsocervical fat pad, weight gain, and proximal muscle weakness, warrant assessment for Cushing syndrome. Features suggestive of malignancy in adrenal masses include tumor growth over 4-6 cm, neovascularization, heterogeneous enhancement with >20 HU, and invasion into nearby structures.

In this case, there was diagnostic uncertainty, as biochemical findings were indeterminate and limited resources precluded further confirmatory testing or additional pathologic evaluation to establish hormonal co-secretion definitively. This represents a limitation in fully characterizing the tumor's functional status. Nevertheless, given the florid clinical presentation, clear signs of hormone excess, and radiologic features worrisome for malignancy, the decision was made to proceed with complete surgical excision of the adrenal mass. Postoperative histopathologic examination remains essential for definitive diagnosis and prognostication. Subsequent management may include adjuvant therapies, such as mitotane or radiotherapy, along with regular monitoring for recurrence as clinically indicated.

Acknowledgments
The authors gratefully acknowledge the support and guidance of the consultants and co-fellows, whose contributions were instrumental in the completion of this work.

Ethical Consideration
Patient consent forms were obtained before manuscript submission.

Statement of Authorship
All authors certified fulfillment of ICMJE authorship criteria.

CRediT Author Statement

CFIJ: Conceptualization, Methodology, Investigation, Resources, Writing – original draft preparation, Writing – review and editing, Visualization, Project administration; **FRCG:** Conceptualization, Methodology, Investigation, Resources, Writing – review and editing, Visualization, Supervision, Project administration.

Data Availability Statement

Datasets generated and analyzed are included in the published article.

Author Disclosure

The authors declared no conflict of interest.

Funding Source

None.

References

1. Fassnacht M, Dekkers OM, Else T, et al. European Society of Endocrinology clinical practice guidelines on the management of adrenocortical carcinoma in adults, in collaboration with the European Network for the Study of Adrenal Tumors. Eur J Endocrinol. 2018;179(4):G1-46. PMID: 30299884 DOI: 10.1530/EJE-18-0608
2. Adler GK, Stowasser M, Correa RR, et al. Primary aldosteronism: An Endocrine Society clinical practice guideline. J Clin Endocrinol Metab. 2025;110(9):2453-95. PMID: 40658480 DOI: 10.1210/clinem/dgaf284
3. Nieman LK, Biller BM, Findling JW, et al. The diagnosis of Cushing's syndrome: An Endocrine Society clinical practice guideline. J Clin Endocrinol Metab. 2008;93(5):1526-40. PMID: 18334580 PMCID: PMC2386281 DOI: 10.1210/jc.2008-0125
4. Abeynayake DR, Sopan V, Perera KJC, Paramanantham A, Munasinghe TMJ. Adrenal carcinoma: A case report. J Med Case Rep. 2022;16(1): 229. PMID: 35637536 PMCID: PMC9153213 DOI: 10.1186/s13256-022-03398-4
5. Lazaro K, Adorable-Wagan P. Aldosterone-producing adrenocortical carcinoma with co-secretion of cortisol and estradiol: A case report. J ASEAN Fed Endocr Soc. 2018;33(1):57-62. PMID: 33442112 PMCID: PMC7784201 DOI: 10.15605/jafes.033.01.10
6. Eisa N. Adrenocortical carcinoma presenting with concurrent Cushing syndrome and primary hyperaldosteronism. AACE Endocrinology and Diabetes. 2026. DOI: 10.1016/j.aed.2026.02.003
7. Berruti A, Fassnacht M, Haak H, et al. Prognostic role of overt hypercortisolism in completely operated patients with adrenocortical cancer. European Urology, 2014;65(4), 832–8. PMID: 24268504 DOI: 10.1016/j.eururo.2013.11.006
8. Aliño MM, Tam-Go LM. Diagnosis and management of adrenocortical carcinoma with co-secretion of cortisol and aldosterone: a case report. J ASEAN Fed Endocr Soc. 2024;39(2):103-7. PMID: 39620173 PMCID: PMC11604353 DOI: 10.15605/jafes.039.02.13
9. Kurtulmuş N, Yarman S, Azizlerli H, Kapran Y. Co-secretion of aldosterone and cortisol by an adrenocortical carcinoma. Hormone Research. 2004;62(2), 67–70. PMID: 15218335 DOI: 10.1159/000079322
10. Prete A, Bancos I. Mild autonomous cortisol secretion: Pathophysiology, comorbidities and management approaches. Nat Rev Endocrinol. 2024;20(8):460-73. PMID: 38649778 DOI: 10.1038/s41574-024-00984-y
11. Moreno S, Montoya G, Armstrong J, et al. Profile and outcome of pure androgen-secreting adrenal tumors in women: Experience of 21 cases. Surgery. 2004;136(6):1192-8. PMID: 15657575 DOI: 10.1016/j.surg.2004.06.046
12. Villa M, Quimpo J. Endocrine protocols: A handbook on the use and interpretation of tests, 2nd ed; 2020.
13. Papotti M, Libè R, Duregon E, Volante M, Bertherat J, Tissier F. The Weiss score and beyond--Histopathology for adrenocortical carcinoma. Horm Cancer. 2011;2(6):333-40. PMID: 21997290 PMCID: PMC10358021 DOI: 10.1007/s12672-011-0088-0
14. Weiss LM, Medeiros LJ, Vickery AL Jr. Pathologic features of prognostic significance in adrenocortical carcinoma. Am J Surg Pathol. 1989;13(3):202-6. PMID: 2919718 DOI: 10.1097/00000478-198903000-00004
15. Jorda M, De MB, Nadji M. Calretinin and inhibin are useful in separating adrenocortical neoplasms from pheochromocytomas. Appl Immunohistochem Mol Morphol. 2002;10(1):67-70. PMID: 11893039 DOI: 10.1097/00129039-200203000-00012
16. Libé R, Huillard O. Adrenocortical carcinoma: Diagnosis, prognostic classification and treatment of localized and advanced disease. Cancer Treat Res Commun. 2023;37:100759. PMID: 37690343 DOI: 10.1016/j.ctarc.2023.100759
17. Libé R, Borget I, Ronchi CL, et al. Prognostic factors in stage III-IV adrenocortical carcinomas (ACC): An European Network for the Study of Adrenal Tumor (ENSAT) study. Ann Oncol. 2015;26(10): 2119-25. PMID: 26392430 DOI: 10.1093/annonc/mdv329
18. Elhassan Y, Altieri B, Berhane S, et al. Modified GRAS score for prognostic classification of adrenocortical carcinoma: An ENSAT multicentre study. Endocrine Abstracts. 2020; 70 OC1.6. DOI: 10.1530/endoabs.70.OC1.6