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Case report

Unveiling a malignant oncocytic adrenocortical carcinoma: A clinico-radiological challenge resolved by immunohistology and a brief review of literature

Ashim Roy[1], Pranjal Kalita[1], Evarisalin Marbaniang[1], Vandana Raphael[1], Caleb Harris[2], Sumit Kumar[3], Donboklang Lynser[4]

[1]*Department of Pathology, Post Graduate Trainee, North Eastern Indira Gandhi Regional Institute of Health & Medical Sciences, Shillong, India*
[2]*Department of Surgical Oncology, North Eastern Indira Gandhi Regional Institute of Health & Medical Sciences, Shillong, India*
[3]*Department of Radiotherapy & Oncology, North Eastern Indira Gandhi Regional Institute of Health & Medical Sciences, Shillong, India*
[4]*Department of Radiology & Imaging, North Eastern Indira Gandhi Regional Institute of Health & Medical Sciences, Shillong, India*

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ABSTRACT

Oncocytic adrenocortical carcinoma is a rare histopathological variant of adrenocortical carcinoma, a malignant tumor of adrenal cortical cells. The exact incidence of this unusual variant of adrenocortical carcinoma is unknown. It mainly occurs in the 5th decade of life with no gender predilection, without any established risk factor, and is characterised by the majority (>90%) of the tumor cells displaying oncocytic morphology. The patients mostly present with an abdominal mass with associated mass effects, or as an incidental finding detected during radiological evaluation for other reasons, and or occasionally with a functional status, usually hypercortisolism. It is an indolent variant with a lower stage, rare local invasion, delayed recurrence, and improved survivability, by virtue of which it differs from conventional adrenocortical carcinomas both clinically and prognostically.

We present a case of non-functional left-sided oncocytic adrenocortical carcinoma in a 40-year-old female who was diagnosed radiologically as a high-grade malignancy, whereas histomorphology, along with immunohistochemistry, played a key role in diagnosis as oncocytic adrenocortical carcinoma.

Correspondence:
Dr. Pranjal Kalita, MD
Department of Pathology,
North Eastern Indira Gandhi Regional Institute of Health & Medical Sciences, Shillong, India
ORCID ID: 0000-0001-5721-6142
Contact: kalita.pranjal4@gmail.com

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INTRODUCTION

Oncocytic Adrenocortical Carcinoma (OACC), a comparatively rare variant of oncocytic adrenal neoplasm, was first recognized and classified as a distinctive subtype by the World Health Organization (WHO) in 2017.[1] The exact prevalence of OACC is highly debatable as only case reports and small case studies have been published to date.[1] OACC incidence has been reported to be 0.72 per million population.[2] It is characterized by a majority (>90%) of the tumor cells having oncocytic morphology due to abundant mitochondria identified by electron microscopy. It mainly occurs in the 5th decade of life (mean age 44 years) with no

sex predilection, mostly as a sporadic tumor without any established risk factors. Patients usually present with solitary incidentalomas (non-functional~60%), detected during radiological evaluation for other reasons, or an abdominal mass with associated mass effects, occurring slightly more on the left side. Functional forms with hormone production (mimicking Pheochromocytoma) and presenting with hypercortisolism or Conn's syndrome are rarely described.[3] It differs from conventional adrenocortical carcinomas (ACC) clinically and prognostically, being an indolent variant with lower stage, rare local invasion, delayed recurrence, and improved survivability.

Herein, we report a rare case of OACC, presenting with a clinico-radiological dilemma where immunohistochemistry played a pivotal role in the diagnosis, ultimately bearing therapeutic implications.

CASE REPORT:

A 40-year female patient presented with complaints of on-and-off episodes of left sided abdominal pain and constipation for one month. Multiple evaluations were done in different peripheral hospitals in view of abdominal pain, where radiological examinations showed possibilities of Solid pseudopapillary neoplasm of the pancreas (SPEN), retroperitoneal gastrointestinal stromal tumor (GIST), and liposarcoma (LPS), but none suggested any possibility of a renal or suprarenal neoplasm. The patient was normotensive, and on per abdominal examination, there was a large lump of approximately 36 weeks of gestational age felt in the epigastrium and umbilical quadrants with tenderness. Tumor markers (CEA, CA-125, and CA19.9) were normal. There was no feature of any endocrine abnormality (Blood counts, Liver and Renal Function Tests, Serum cortisol, Urinary metanephrines, and sex hormones were within normal limits for age). Contrast-Enhanced Computed Tomography (CECT) of the thorax, abdomen, and pelvis was suggestive of a Retroperitoneal Sarcoma (fig. 1 a,b).

Ultrasound-guided Fine Needle Aspiration Cytology (FNAC) attempted from the mass yielded only necrotic debris.

The patient underwent exploratory laparotomy, and a 30x15 cm mass was identified. Intra-operatively, the mass was felt involving the body and tail of the pancreas, which was sent for histopathological examination. The gross appearance of the specimen showed a mass with attached spleen and part of normal pancreas weighing 6.5 kilograms and measuring (24 x 22 x 15) cm with tense, congested outer capsule and solid cystic with necrotic areas on cut surface. (fig. 2 a,b) Hematoxylin and Eosin-stained slides showed an encapsulated tumor with oncocytic cells arranged in sheets having bizarre hyperchromatic nuclei with interspersed multinucleated giant cells, areas of haemorrhage and necrosis. High mitotic count (>20/10 mm²) was observed. No capsular or lymphovascular invasion or infiltration into pancreatic or splenic tissue was seen. (fig. 3 a,b,c,d) In view of absence of organ specific findings, a series of differential diagnoses were considered. To confirm the diagnosis and rule out other differentials, a series of immunohistochemical (IHC) stains was performed (Table 1). (fig. 4 a-f). Lin Weiss Bisceglia criteria were fulfilled (Table 2). A diagnosis of Oncocytic Adrenocortical Carcinoma was favoured primarily based on IHC findings. TNM grade was given as pT2N0Mx. Post-surgery period was uneventful, and the patient was followed up with mitotane.

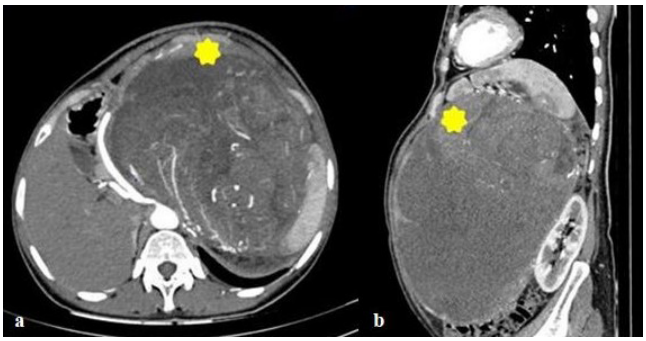

Figure 1: *CECT scan showing a large (19x18x25) well defined retroperitoneal mass on left side with solid cystic areas displacing pancreas, bowel and large vessels and compression of kidney but with a maintained fat plane.*

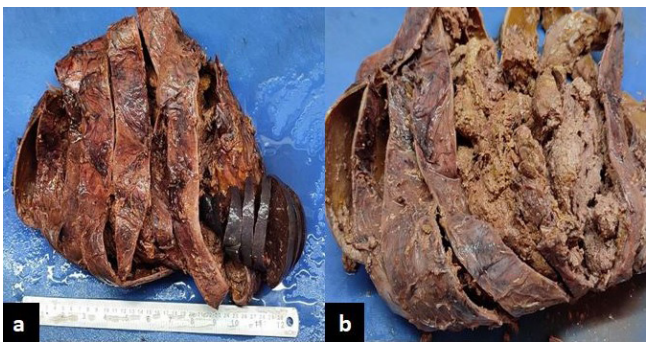

Figure 2: *Gross appearance of the tumor with attached spleen, (A) Tense, congested encapsulated surface; (B) Cut open surface with solid-cystic-necrotic area.*

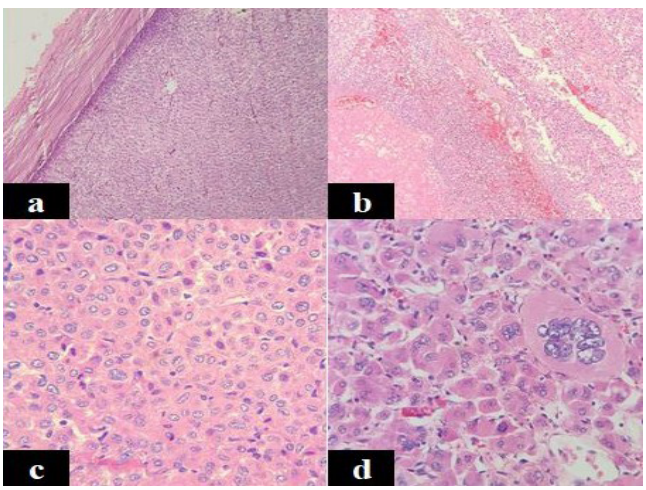

Figure 3: *Histomorphology showing (A) an encapsulated tumor (HE stain,40x); (B) Areas of necrosis and haemorrhage (HE*

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stain,40x); (C) Sheets of Oncocytic cells with increased mitosis (HE stain,200x); (D) Oncocytic cells with interspersed multinucleated cell (HE stain,200x).

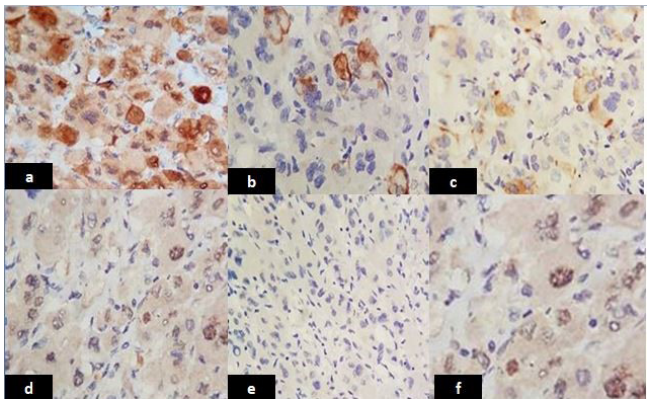

Figure 4: *Immunohistochemistry showing (A) Positivity for Calretinin; (B) Focal positivity for Pan CK; (C) Focal positivity for Synaptophysin; (D) Ki-67 labelling index of 40%; (E) Negative for HMB 45; (F) Positivity for SF-1 (IHC,200x).*

Table 1: Immunohistochemical panel to substantiate diagnosis of Oncocytic adrenocortical carcinoma

Immunohistochemistry panel	Inference	Differential Diagnosis
CD 56, Chromogranin	Negative	Rules out Phaeochromocytoma
a. β-catenin b. E-cadherin	Negative Retained	Rules out Solid pseudopapillary neoplasm
HMB45, SMA, Desmin	Negative	Rules out perivascular epithelioid cell tumor
HepPar1, Glypican 3	Negative	Rules out Hepatocellular Carcimoma
PAX8, CD10	Negative	Rules out Renal Cell Carcinoma
Ki67 proliferation index	40%	Rules out Adrenal cortical oncocytoma
a. SF-1, Calretinin, Inhibin, Melan A b. Pan CK and Synaptophysin c. Ki67 proliferation index	Positive Focally positive 40%	Favors Oncocytic Adrenocortical Carcinoma

* CD: Cluster of Differentiation, β-catenin: Beta- catenin, E-cadherin: Epithelial- cadherin, HMB45: Human Melanoma Black 45, SMA: Smooth Muscle Actin, HepPar1: Hepatocyte Paraffin 1, PAX8: Paired Box 8, SF1: Steroidogenic Factor 1, Pan CK: Pan Cytokeratin

Table 2: Lin Weiss Bisceglia Criteria fulfilling the diagnosis of malignant oncocytic adrenal cortical carcinoma.

Lin Weiss Bisceglia		Finding observed in present case
Major Criteria	A. >5 mitosis/50 high power fields	(>20/10 mm²)
	B. Atypical mitosis	Present
Minor Criteria	A. Size >10 cm and/ or weight >200 g	Size=(24 x 22 x 15)cm
		Weight=6.5 kilograms

DISCUSSION:

OACC, although rarer in occurrence than the conventional ACC, is usually a straightforward diagnosis, and histomorphology alone serves the diagnostic purpose. Fine needle aspiration cytology appears to be of use to suggest a preoperative diagnosis, but due to the huge size of the tumor, as in our reported case, and potential presence of heterogeneous areas, this technique may not fully characterize the tumor.[3] Macroscopically, oncocytic ACCs are large, round, well-circumscribed, encapsulated, yellow-brown tumors ranging in size from 2 to 20 cm and surrounded by a rim of normal adrenal tissue component. Considering the massive tumor size and dimensions, the origin of the tumor could not be ascertained by clinico-radiological modalities. Microscopically, the tumor shows diffuse sheets, nested and trabecular patterns of round to polygonal cells with abundant eosinophilic granular cytoplasm, large nuclei with prominent nucleoli, occasional binucleated and multinucleated forms, which are non-specific and may be associated with tumors originating from different sites.[4] The main differential diagnoses to consider are adrenal cortical oncocytoma, conventional ACC, phaeochromocytoma (oncocytic variant), renal cell carcinoma, and metastasis.

Steroidogenic Factor-1 (SF-1) is the most specific biomarker to confirm Adrenocortical origin with a sensitivity of 98% and specificity of 100%.[5] Also, positivity for calretinin is a novel finding in OACC, as it is absent in conventional ACCs. Other markers, namely alpha-inhibin and Melan-A, and a high Ki-67 labelling index, favour a malignant neoplasm originating from the adrenal organ. The key highlight of the case includes the pivotal role played by IHC, which not only pointed to the organ of origin but also helped in differentiating it from its aggressive mimickers and thereby bearing therapeutic and prognostic implications for the patient. In 2004, criteria were established under the Lin-Weiss-Bisceglia system, where the presence of even one major criterion—such as a high mitotic rate (>5/50 HPF), atypical mitosis, or venous invasion—classifies the tumor as malignant, which was applicable in our case.[6] OACC has a high risk of local relapse (5%), distant metastasis (3.7%), and mortality (7.4%) (but much lower than conventional ACCs). The most common reported metastatic sites to develop are the liver and lungs.[2] Wong et al. found that the overall median survival for patients with these tumors is 58 months, thereby proving a more favorable prognosis than conventional ACCs.[6] The 5-year survival rate is 20-35%, which increases to 50-60% after adequate surgery.[7] Multimodal approach with wide surgical excision as the mainstay treatments along with palliative/adjuvant radiofrequency ablation and Mitotane or other chemotherapeutic agents have shown to delay and prevent local recurrence.[8] European Society of Endocrinology Clinical Practice Guidelines on the management of ACCs in adults, in collaboration with the European Network for the study of Adrenal Tumors, suggest the use of mitotane as adjuvant therapy in tumors with a high risk of recurrence, with Ki-67>10%, microscopic positive

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resection margins, and stage III tumors.[2] In the case reported, Mitotane was used primarily considering the massive size, high Ki- 67 labeling index to prevent local recurrence. Two months post-therapy, the patient was planned for CECT and positron emission tomography/computed tomography (PET-CT) to rule out metastasis. Considering the financial constraints, the patient was unable to undergo CECT and PET-CT on 2-month follow-up, but was asymptomatic clinically.

Mills JK et al., in their study, highlight the indolent nature, delayed presentation, and lower stage of OACC compared to conventional ACC.[9] A thorough PubMed search for "Oncocytic Adrenocortical Carcinoma" revealed 48 results. Eighteen case reports, which had clinico-biochemical, radiological, and pathological findings, were detailed (Table 3). Two non-English texts were not included due to a language barrier. Based on the clinical findings of the cases included, OACC is primarily a disease of the elderly. However, a case of OACC in an 18-month-old child has also been noted. OACC usually presents as a non-functional left-sided swelling primarily affecting females. As most of the swellings were non-functional, they presented as large masses with pressure symptoms on the adjoining organs rather than direct infiltration into them. Although in most of the cases the histomorphological diagnosis was straightforward, immunohistochemistry was used to substantiate the diagnosis and rule out various differentials.

The authors of the present case report suggest a limited panel of immunohistochemistry markers, namely SF-1 (specifying adrenal origin), Inhibin, Melan-A, and Calretinin (positive in adrenal cortical tumor), Ki-67 labelling index: High (> 5%), which helps in the diagnosis of OACC. Various other markers, Synaptophysin, Chromogranin-A, CD56, Vimentin, AE1/AE3, show variable positivity. Electron microscopy (EM) was done in a few cases, showing abundant mitochondria. However, the authors opine that ancillary techniques like EM are not a necessity for the diagnosis of OACC. No uniformity was noted on the use of adjuvant therapy with Mitotane and its correlation with the absence of recurrence, metastasis, and overall survival.

Table 3: Summary of cases reported as Oncocytic Adrenal Cortical Carcinoma.

Author	Year	n	Age, Sex	Functional/ Non-functional	Size at first presentation	Site	Recurrence/ Metastasis	Treatment received
Picut B. et al.[11]	2025	01	60y, F	F	(8×5) cm and (1.8× 2.2) cm	L	Recurrence (2 months follow-up)	Surgery Mitotane Radiotherapy Glucocorticoid Replacement Therapy
Santos CD et al.[12]	2024	01	64y, F	NF	(14.2 × 11.8 × 12.5) cm	L	Not available (Patient on follow-up)	Surgery Chemotherapy
Li Y et al.[10]	2024	01	35y, F	NF	(8.5x6.5x5) cm	R	No Recurrence/ Metastasis	Surgery alone
Liu H et al.[13]	2023	01	72y, M	F [Urinary VMA level: Mildly increased]	(7.8× 6.8) cm	R	No Recurrence/ Metastasis (12 months follow-up)	Surgery alone
Al-Rashdan R et al.[14]	2023	01	47y, M	F [Increased Cortisol]	(17×12×12)cm	L	No Recurrence/ Metastasis (1 months follow-up)	Surgery Mitotane
Coppola Bottazzi E et al.[3]	2023	01	88y, F	NF	< 5cm	L	No Recurrence/ Metastasis (24 months follow-up)	Surgery alone
Huang CP et al.[15]	2022	01	42y, F	NF	(3.9 × 2.9 × 2.5) cm	L	Metastasis (Detected after 1 year)	Surgery alone
Singh Y et al.[7]	2022	01	21y, M	F [Increased Cortisol]	(9.6x8.9x7.6) cm	L	No Recurrence/ Metastasis (On 10 months follow-up)	Surgery alone
Sinai Khandeparkar SG et al.[8]	2022	01	45y, F	NF	(19 × 18 × 7.5) cm	L	Patient succumbed post-operatively	Surgery alone
Cardona Attard CD et al.[2]	2022	01	40y, F	NF	(3.5×3.4×5.6)cm	L	No Recurrence/ Metastasis	Surgery alone

Author	Year	n	Age, Sex	Functional/ Non-functional	Size at first presentation	Site	Recurrence/ Metastasis	Treatment received
Babaya N et al.[16]	2021	01	21y, F	F [Serum (DHEA), (DHEA-S), testosterone, and urine 17-ketosteroid levels were raised]	(8.2 × 5.0 × 5.0)cm	L	No Recurrence/ Metastasis (12 months follow-up)	Surgery alone
Akın O et al.[17]	2021	01	1y 6m, M	F [Serum total testosterone, androstenedione, and DHEA-SO4 were raised]	(2.2x1.7) cm	R	No Recurrence/ Metastasis. Had a second tumor in different location diagnosed as RMS	Surgery alone
Lehr I et al.[18]	2020	01	55y, F	Not available	(13.3 x 7.7 x 10.8)cm	L	Not available	Not available
Harada K et al.[4]	2020	01	70y, M	NF	6.2 cm (Greatest dimension)	L	No Recurrence/ Metastasis (05 months follow-up)	Surgery Mitotane Glucocorticoid Replacement Therapy
Kaur RJ et al.[19]	2019	01	65y, F	F [Androgen ,serum steroid precursors, and estrogen excess,(ACTH) independent cortisol excess	(9.2 × 5.9 × 4.8)cm	R	No Recurrence/ Metastasis (26 months follow-up)	Surgery Glucocorticoid-replacement therapy Mitotane
Panizzo V et al.[20]	2018	01	48y, M	NF	(12.3×9.8×11.3)cm	R	No Recurrence/ Metastasis (24 months follow-up)	Surgery alone
Al Balooshi B et al.[21]	2018	01	37y, M	NF	19 cm (Greatest dimension)	L	Not available	Surgery alone
Kalra S et al.[1]	2015	01	34y, M	NF	(16×11×8) cm	L	No Recurrence/ Metastasis (3 months follow-up)	Surgery alone

* M: Male, F: Female, F: Functional, NF: Non- Functional, L: Left, R: Right, VMA: Vanillylmandelic Acid, DHEA: Dehydroepiandrosterone, ACTH: Adrenocorticotropic Hormone

CONCLUSIONS:

This case highlights the rarity, the clinico-radiological dilemma it posed, and the utility of histomorphology and IHC analysis to solve it. The possibility of OACC should be kept in mind while evaluating retroperitoneal masses, especially those with minimal clinico-biochemical features. Effective management relies on clinical, biochemical, radiological, and pathological findings. The need of the hour is a multimodal therapeutic approach with a careful follow-up to prevent recurrence, distant metastasis, and mortality. The underlying need to differentiate this histotype from Conventional ACCs due to better clinical and prognostic behaviour makes this case unique.

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