Original Article

Characteristics of Bilateral Adrenal Lesions: Experience from an Indian Tertiary Care Centre

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Abstract

Introduction: The aetiologies in unilateral and bilateral adrenal lesions can be different with different clinical implications and management guidelines, the latter having aetiologies like hyperplasia, infections, infiltrative lesions and neoplasia. Bilateral tumours are more likely to have hereditary/syndromic associations. There is limited data on the clinical and pathological profile of bilateral adrenal lesions. **Methods:** This was a retrospective study where patients with bilateral adrenal lesions were selected from a total of 266 patients with adrenal lesions who presented to our institute between January 2016 and August 2022. The demographic, laboratory and imaging data were retrieved from the Hospital Information System and patient case files. **Results:** The study included 51 patients; the mean age at presentation was 51.15 years (range 14 to 82 years). Forty-eight patients (94.1%) were symptomatic at presentation with an average duration of symptoms being 10.68 months (range 10 days to 1 year). The most common presentation was adrenal insufficiency in 18 cases (38%), followed by fever in 17 cases (36%). The commonest aetiology, as revealed on histopathology, was histoplasmosis ($n = 22$, 43%), followed by pheochromocytoma ($n = 11$, 21.5%), metastases ($n = 6$, 11.7%), adrenal hyperplasia ($n = 5$, 9.8%), adrenocortical adenoma ($n = 1$, 1.9%), lymphoma ($n = 3$, 5.8%), neuroblastoma ($n = 1$, 1.9%), myelolipoma ($n = 1$, 1.9%) and tuberculosis ($n = 1$, 1.9%). Histoplasmosis and metastatic lesions were commonly seen in older people, and pheochromocytoma was associated with young age. 6/11 patients with a diagnosis of bilateral pheochromocytoma were associated with family history, genetic mutation and extra-adrenal involvement. **Conclusion:** The approach to bilateral adrenal lesions differs from that of unilateral lesions due to differences in aetiologies and the more significant role of genetics in some bilateral tumours. The age at presentation, presenting symptoms, lesion size and biochemical features help delineate varied underlying aetiologies.

Keywords: Adrenal, bilateral, histoplasmosis, hyperplasia, hypocortisolism, infection, pheochromocytoma

Introduction

Bilateral adrenal lesions include a varying spectrum of aetiologies like neoplasms, adrenal hyperplasia and infections.[1,2] Hypocortisolism is an important feature that should be considered when evaluating bilateral masses.[3] Hereditary or syndromic associations are more likely to be present in cases with bilateral adrenal involvement.[3] Most of the adrenal tumours present as unilateral masses and are benign non-functioning adenomas.[4] In contrast to this, bilateral adrenal masses are relatively uncommon, and exhibit varied clinical manifestations, while some cases may be asymptomatic, and others may demonstrate severe systemic features.[1,2,5] The initial diagnostic approach during evaluation remains similar to that for the unilateral masses; however, additional testing and genetic studies should be performed in such cases.[2] Imaging characteristics that assist in the differential diagnosis of adrenal lesions include size, attenuation, washout values, presence of calcification, fat, or haemorrhage and laterality. Imaging also helps to narrow the differential diagnosis of adrenal masses as there are a few lesions that present bilaterally.[5]

A large body of literature is available on unilateral adrenal lesions but there is limited data on the clinical and pathological profile of bilateral adrenal lesions. The present study aims

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to analyse the spectrum, incidence, clinical characteristics, biochemical and radiological features in patients presenting with bilateral adrenal lesions at a tertiary care institute.

Materials and Methods

Case-selection

This was a retrospective study where patients with histopathology from bilateral adrenal lesions were selected from 266 patients with histopathology from adrenal lesions who presented to the endocrine surgery and endocrinology department of our institute between January 2016 and August 2022. The demographic, laboratory and imaging data were retrieved from the Hospital Information System and patient case files. The biochemical and hormonal workup included serum cortisol, adrenocorticotropic hormone (ACTH), overnight dexamethasone suppression test (ONDST), urinary metanephrines and nor-metanephrines. Cases with unilateral adrenal lesions/masses, confirmed by radiology, were excluded from the study. Imaging parameters like the size of bilateral adrenals were recorded.

Histopathology

Tissue samples were received in the department of pathology as bilateral adrenalectomy and bilateral adrenal biopsy specimens. The tissue samples were collected in 10% buffered formalin and processed for routine histopathological examination. 4 mm thick sections from formalin-fixed paraffin-embedded blocks were cut and stained with haematoxylin and eosin stain. Special stains for fungal organisms (periodic acid Schiff and chromic acid silver methenamine), acid-fast bacilli (zeihl neelson) and immunostains (panel of immunohistochemical markers) were performed as and when required. None of the patients underwent fine needle aspiration cytology.

Statistical analysis

Data were categorized as quantitative and qualitative based on the nature of the data. Continuous data were represented as mean±SD or median (q1-q3), while categorical data was as represented as frequency (%). The normality of continuous variables was examined by Shapiro–Wilk test. For continuous variables, comparison among three groups was performed by one-way analysis of variance or Kruskal–Wallis H-test, while for categorical data, association was tested with a chi-square test. A *P* value less than 0.05 was considered significant.

Ethical aspects

The study was approved by the Institutional Ethics Committee (IEC), IEC code: 2023-200-IMP-EXP-53, 05.08.23. All the procedures followed were in accordance with the ethical standards of the IEC and the revised Helsinki Declaration of 2013. All steps were taken to ensure confidentiality of subjects was maintained and no data had any obvious personal identifiers.

Result

In our series of 51 patients (42 males and 9 females), the mean age was 50.53 ± 15.24 years (range 14 to 82 years). The most common aetiology as diagnosed on histopathology was infection (histoplasmosis *n* = 22, 43% and tuberculosis *n* = 1, 1.9%), followed by neoplasia (pheochromocytoma *n* = 11, 21.5%, metastases *n* = 6, 11.7%, adrenocortical adenoma n = 1, 1.9%, lymphoma *n* = 3, 5.8%, neuroblastoma *n* = 1, 1.9% and myelolipoma *n* = 1, 1.9%) and adrenal hyperplasia *n* = 5, 9.8%. Table 1 summarizes the clinical features and biochemical values of the study cohort [Figures 1-3].

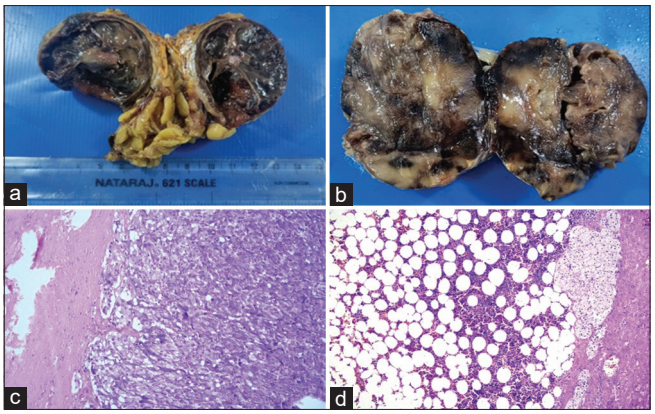

Figure 1: (a) Gross specimen of bilateral pheochromocytoma (b) Gross specimen showing bilateral myelolipoma (c) Histological section from the gross in (a) showing nests of polygonal tumour cells with abundant granular eosinophilic cytoplasm (H and E, x200) (d) Section from the (b) specimen showing an admixture of fat and marrow elements (H and E, x100)

Out of the 51 cases, 3 (5.8%) were asymptomatic and detected incidentally on ultrasonography. However, the other 48 cases (94.1%) presented with at least one symptom, with an average duration of symptoms being 51.83 ± 18.73 days. The most common presentation was adrenal insufficiency in 18 cases (38%), followed by fever in 17 (36%). Histoplasmosis and metastatic lesions were more common in the elderly, with a mean age of 59 (range 37-71) years and 67.6 (range 54-71) years, respectively. Adrenal hyperplasia was encountered in five cases (9.8%), and the most common symptoms were abdominal pain, fever and features of hypocortisolism. All five cases also had histologically proven ACTH-secreting pituitary adenoma. Serum cortisol level was high, with a mean value of 722.25 nmol/l. Bilateral pheochromocytoma was more common at a young age (29.64 ± 12.52). Six of the 11 (54.5%) bilateral pheochromocytomas were familial, having genetic mutations and extra-adrenal involvement.

As can be seen from Table 1, the clinical features of the three groups of infections, hyperplasia and neoplasia, were statistically significant. The infection group presented more commonly with complaints of fever (65.2%), the hyperplasia group presented with abdominal pain (80%) and the neoplasia group presented with hyperadrenergic spells (47.8%). No difference was found in age, gender, duration of symptoms, weight and size of adrenals, serum adrenocorticotropic hormone (ACTH), serum cortisol, overnight dexamethasone suppression test (ONDST), urinary metanephrine (MN) and normetanephrine (NMN).

Table 1: Clinical features and biochemical values of the study cohort

Variables	Infection (*n*=23)	Hyperplasia (*n*=5)	Neoplasia (*n*=23)	*P*
Mean age±SD (in years)	50.53±15.24	46±29.69	51.83±18.73	0.813*
Gender				
Male	22 (95.7%)	3 (60.0%)	17 (73.9%)	0.059
Female	1 (4.3%)	2 (40%)	6 (26.2%)	
Duration of symptoms (in days)	182 (91-365)	30 (30-365)	61 (21-243)	0.061$
Clinical presentation				
Fever	15 (65.2%)	1 (20%)	6 (26.1%)	<0.001
Pain abdomen	2 (8.7%)	4 (80%)	9 (39.1%)	<0.001
Hypocortisolism	14 (60.9%)	0 (0%)	14 (60.9%)	0.034
Hypercortisolism	2 (8.7%)	5 (100%)	0 (0%)	<0.001
Hyperadrenergic spells	1 (4.3%)	1 (20%)	11 (47.8%)	<0.001
Asymptomatic	2 (8.7%)	0 (0%)	1 (4.3%)	0.166
Serum cortisol (nmol/l)	132.4 (26-403)	770 (481.5-963)	303 (144.9-769.8)	0.027$
ACTH (pmol/l)	92.9 (16.6-125)	103.6 (57.6-157.5)	85.7 (77.6-129)	0.669$
ONDST (nmol/l)	91 (91-640)	-	97.7 (66.5-162)	0.653@
Urinary MN (mcg/24 h)	25 (25-95)	130 (130-130)	134 (78.6-397)	0.189$
Urinary NMN (mcg/24 h)	397.5 (335-572)	1398 (1398-1398)	1171 (492-3008)	0.098$
Size of right adrenal (in cm)	38.5 (15.5-111)	38.5 (21.6-73.5)	50.3 (15-125)	0.985$
Size of left adrenal (in cm)	23.7 (18-103.2)	24 (22-42)	32.1 (12-42)	0.979$
Weight of right adrenal (in gm)	-	20.5 (11-22)	39 (30-122.8)	0.02@
Weight of left adrenal (in gm)	72 (72-72)	20 (17-25.6)	40 (20-78)	0.238$

SD=Standard deviation, ACTH=Adrenocorticotropic hormone, ONDST=Overnight dexamethasone suppression test, MN=Metanephrine, NMN=Nor metanephrine. Categorical data is represented as frequency (%), and comparison is conducted using chi-square/Fisher's exact test. Continuous data are represented as mean±SD and median (Q1-Q3), *One-way analysis of variance, $Kruskal–Wallis H-test and @Mann–Whitney *U*-test were used for comparison. *P*<0.005 is considered significant

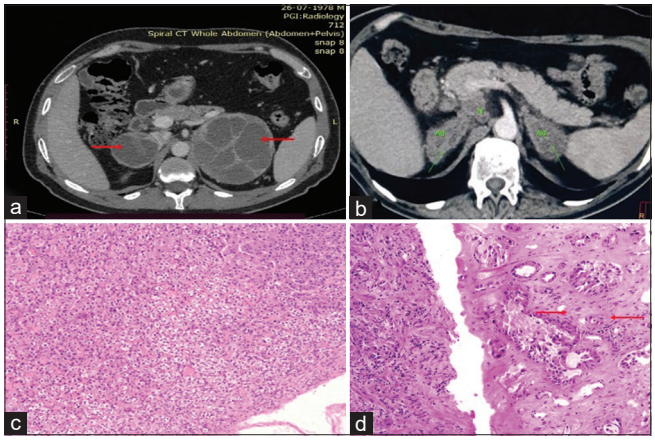

Figure 2: (a) CT scan showing bilateral adrenal masses (red arrows) in case of pheochromocytoma (b) CT scan showing bilaterally enlarged adrenals (c) Section from a case of adrenal hyperplasia with overgrowth of layers of the adrenal cortex (H and E, x200) (d) Adrenal biopsy of the case from B displaying infiltration by a tumour disposed of as irregular glands (H and E, x200)

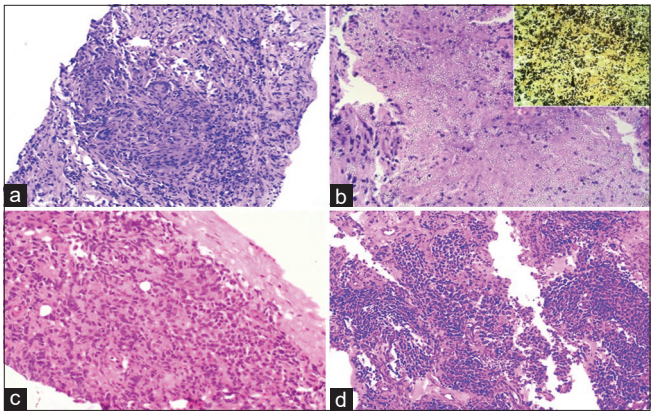

Figure 3: (a) Photomicrograph from a case of granulomatous inflammation in adrenal gland (H and E, x200). (b) Case of histoplasmosis with numerous small uniform oval, narrow-based budding yeasts (H and E, x200), highlighted on silver stain (inset). (c) Adrenal biopsy from a case of lymphoma displaying infiltration by atypical lymphoid cells (H and E, x200). (d) Case of neuroblastoma showing proliferation of small, round, blue cells in sheets (H and E, x200)

Discussion

There is limited literature on the clinical and pathological characteristics of bilateral adrenal lesions. Bilateral adrenal lesions have different sets of aetiologies as compared to diseases with unilateral lesions. We have described our case series in terms of aetiology, age, sex, bilateral versus unilateral lesions, and clinical features and compared our results with two case series, published by Zhou *et al.* and Lomte *et al.,* respectively[6,3] [Table 2]. The largest study to date was conducted by Lomte *et al.*, which studied the clinical, hormonal and radiological profile of 70 cases.

The most common aetiology in our study was histoplasmosis (43%), unlike the case series of Zohu (0%) and Lomte (2.8%), respectively.[6,3] This difference could be attributed to the endemicity of histoplasmosis in the Gangetic planes of India (Uttar Pradesh, Delhi, Haryana

Table 2: Comparison of the three study cohorts with bilateral adrenal lesions

Study (year)	Zhou *et al*. (2009)	Lomte *et al*. (2016)	Present study Niranjan *et al*. (2023)
Number of patients studied	18	70	51
Proportion of bilateral masses among all adrenal tumours	3.2%	15%	19.1%
Age (years) ±SD	43.0±17.1	39.7±16.1	50.53±15.24
Sex (male:female)	11:7	3:2	42:9
Aetiology			
Histoplasmosis	0	2 (2.8%)	22 (43%)
Pheochromocytoma	6 (33.3%)	28 (40%)	11 (21.56%)
Metastases	2 (11.1%)	4 (5.7%)	6 (11.76%)
Hyperplasia	0	3 (4.2%)	5 (9.80%)
Lymphoma	4 (22.2%)	7 (10%)	3 (5.88%)
Adenoma	4 (22.2%)	3 (4.2%)	1 (1.96%)
Tuberculosis	0	19 (27.1%)	1 (1.96%)
Others	2 (11.1%)	4 (5.71%)	3 (5.8%)

and West Bengal).[7] Histoplasmosis presents with features of adrenal insufficiency (anorexia, weight loss, low-grade fever and recurrent vomiting); however, most patients are asymptomatic and manifest features clinically when they are immunocompromised.[8] This disease is caused by a dimorphic fungus (Histoplasma capsulatum).[9] The most common route of infection is by inhalation of spores from soil contaminated with bird and bat droppings.[10] Higher engagement of males in farming and outdoor activities can be attributed to increasing male preponderance.[11] It is noteworthy that adrenal histoplasmosis contributes to a major proportion of bilateral lesions when compared to adrenal tuberculosis.

Adrenal pheochromocytoma was the second leading diagnosis in our cohort and constituted 21.5% of cases (versus 33% in Zhou and 40% in Lomte, where it was the most common aetiology).[6,3] More than 50% (6/11) of our patients with bilateral pheochromocytomas had familial/syndromic associations. Inabnet *et al.* reported 32 patients with bilateral hereditary pheochromocytomas, of which 15 cases had multiple endocrine neoplasia type 2A (MEN 2A), 12 cases had Von Hippel–Lindau disease, 3 cases of von Recklinghausen's disease, and 2 cases were of familial pheochromocytoma.[12] Similar findings were also seen in the studies by Lomte *et al.* and Amar *et al*.[3,13]

Metastatic lesions are another rare yet important cause of bilateral adrenal lesions and can be differentiated from adrenal adenoma by poor contrast washout at delayed imaging on a computed tomography scan. Adrenal metastases may occur in ~20-45% of cancer patients, depending on the localization of the primary site.[14] Because of its rich vascular supply, the adrenal gland is a common site of metastasis. Adrenal metastasis can be found synchronously or many years after nephrectomy for renal cell carcinoma.[15] Adrenal insufficiency, at times, may be the sole manifestation of the disease, as was seen in one of our cases of lung adenocarcinoma with metastasis to bilateral adrenals [Figure 2b and d].

Adrenal cortical hyperplasia is another condition that can cause bilateral enlargement of adrenals, and it can be divided into ACTH-dependent, ACTH-independent and congenital adrenal hyperplasia.[16] A detailed workup is needed to diagnose the type, which is important for the proper management of the patient and explaining the prognosis of the disease. In our case series, 5.8% of patients were of adrenal lymphoma—patients presented with pain in abdomen and features of adrenal insufficiency. Primary adrenal lymphoma, though rare, is highly aggressive and metabolically hyperactive, high-grade lymphoma and is common in elderly males.[17,18]

Bilateral non-secreting adenomas may be incidentally detected.[6] In our study group, we found one case of bilateral adrenal adenoma in a female patient who was symptomatic. Adrenal myelolipoma is a rare, benign and nonfunctional tumour, first described in 1905.[19] It comprises mature adipose tissue associated with haematopoietic elements comprising erythroid cells, myeloid cells and megakaryocytes, surrounded by a thin capsule. The majority of these lesions are small lesions and asymptomatic.[20] In rare cases, however, it can be symptomatic and bilateral. Diagnosis is based on CT findings and confirmation by histological examination.[21] In our group, we encountered one case of bilateral adrenal myelolipoma in an asymptomatic patient (incidentaloma) on initial presentation.

Adrenal insufficiency was noted in 18 (38%) of our cases. The adrenal insufficiency usually manifests when more than 90% of the adrenal gland has been destroyed.[22] Imaging modalities like computed tomography have contributed to our understanding of the patterns of adrenal involvement in various lesions, like tuberculosis. Most patients with an active or recently acquired disease (<2 years) have bilateral adrenal enlargement, whereas calcification and atrophy are encountered in cases with more distant infection or inactive disease.[23,24] Two previous studies performed at our institute reported the incidence of adrenal tuberculosis. Of 48 adrenal biopsies conducted between 2009 and 2019, 8 cases (16.7%) of adrenal tuberculosis (TB) were diagnosed.[25] In another study by Gunna *et al.*, they studied 89 patients diagnosed with primary adrenal insufficiency between 2006 and 2019 and found 13 patients (14.6%) with adrenal TB. History of extra-adrenal TB was elucidated in 24 of the total 89 patients (26.9%).[26]

Our study also included one case of adrenal neuroblastoma (NBL). NBL is composed of immature, undifferentiated, small, primitive-appearing cells called neuroblasts, which contain small, round blue nuclei, inconspicuous nucleoli and scant cytoplasm. These cells are usually arranged in the form of rosettes, known as Horner-Wright rosettes, which are a characteristic histological feature of NBL.[24,27] NBL is diagnosed by characteristic histopathological findings plus high levels of one of the catecholamines in urine.[28]

The present study is the first to include the histological spectrum of bilateral adrenal lesions, in addition to enumerating the clinical and biochemical findings. However, it also has a few limitations. Firstly, this was a retrospective study with limited sample size, and more number of cases would definitely have added to the strength of the study. Secondly, the adrenal weight was not available in cases where a biopsy was performed. Also, since this is a histopathology-based series, the cases without any biopsy or surgical intervention would have been missed. This would be because of potential referral bias as our institute is a tertiary referral research centre, many adrenal tuberculosis patients are treated based on clinical and radiological features without adrenal biopsy.

Conclusion

Clinical features, including age, size of the lesion, symptoms and biochemical parameters, help us navigate towards the diagnosis of the patient. Histoplasmosis presents with features of adrenal insufficiency and prolonged low-grade fever. Histoplasmosis is the most common aetiology in our series and can be attributed to demographic distribution. Most of the pheochromocytomas are bilateral, have familial/syndromic associations and are present at a younger age. Adrenal cortical hyperplasia warrants detailed workup for its further classification. More studies with a larger number of cases are needed to understand the nature of bilateral adrenal lesions.

Acknowledgment

Nil.

Authors' contribution

VA, PP, GN contributed to the study conception and design. Material preparation, data collection and analysis were performed by GN, PP and NG. The first draft of the manuscript was written by GN and PP; VK performed the statistical analysis. VA, AM, NJ supervised and edited the first draft of the manuscript. All authors read and approved the final manuscript. PP will act as the guarantor.

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Conflicts of interest

There are no conflicts of interest.

Data Availability statement

The raw data supporting the conclusions of this article will be made available by the authors, without undue reservation.

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