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PATHOMORPHOLOGY OF ADRENAL GLAND TUMORS AND HYPERPLASIAS

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Abstract: Adrenal gland tumors and hyperplasias represent a diverse group of lesions with distinct morphological, functional, and clinical characteristics. Their pathomorphology ranges from benign cortical adenomas to highly aggressive adrenocortical carcinomas, as well as medullary neoplasms such as pheochromocytoma. Hyperplastic conditions, both nodular and diffuse, constitute an important category associated with endocrine disorders including Cushing's syndrome, primary aldosteronism, and congenital adrenal hyperplasia. This study provides a comprehensive description of the structural and microscopic features of adrenal cortical and medullary tumors and hyperplasias, emphasizing their diagnostic criteria and clinicopathological significance.

Keywords: adrenal gland, adrenal tumors, adrenal hyperplasia, adrenocortical carcinoma, pheochromocytoma, pathomorphology.

Introduction

The adrenal glands play a central role in endocrine regulation through the secretion of glucocorticoids, mineralocorticoids, and catecholamines. Pathological processes involving the adrenal cortex or medulla may result in significant clinical syndromes due to hormone excess, deficiency, or mass effects. Adrenal tumors and hyperplasias encompass a wide spectrum of lesions, including benign and malignant neoplasms, functional and non-functional nodules, diffuse cortical hyperplasia, micronodular and macronodular hyperplasia, as well as medullary tumors such as pheochromocytoma and paraganglioma.

Understanding the pathomorphology of these lesions is vital for accurate diagnosis, prognostication, and treatment planning. Adrenocortical adenomas remain common findings, often discovered incidentally, while adrenocortical carcinomas are rare but highly aggressive. Hyperplastic lesions are frequently associated with endocrine disorders such as Cushing's disease, primary aldosteronism, and congenital enzymatic defects. This article aims to present an in-depth overview of the structural and microscopic characteristics of adrenal gland tumors and hyperplasias.

Materials and Methods

This study employed a descriptive and comparative pathomorphological approach to characterize structural features of adrenal gland tumors and hyperplasias. Surgical specimens were obtained from adrenalectomies performed for various clinical indications, including hormonally active tumors, incidentally discovered masses, and hyperplastic lesions associated with endocrine disorders. All samples were processed using standard histopathological techniques.

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Resected adrenal tissues were fixed in 10% neutral buffered formalin for adequate preservation of cellular and stromal architecture. Following fixation, tissues were embedded in paraffin blocks, and serial sections of 4–5 micrometers thickness were prepared using a rotary microtome. Sections were routinely stained with hematoxylin and eosin to evaluate overall morphology, cytological details, and tissue organization.

Additional histochemical and structural assessments were performed depending on the lesion type. Lipid content in cortical cells was evaluated macroscopically and microscopically by examining cytoplasmic vacuolization and cell clarity. For selected neoplasms, reticulin staining was used to assess stromal framework integrity and identify architectural disruption typical of malignant processes. Tumors with suspected neuroendocrine differentiation underwent chromaffin-specific evaluation based on their cytological features and vascular pattern.

Morphological analysis focused on key diagnostic parameters including tumor circumscription, presence or absence of a capsule, cellular uniformity, nuclear atypia, mitotic activity, and identification of necrosis or hemorrhage. For adrenocortical carcinomas, classical diagnostic systems such as evaluation of mitotic rate, cellular pleomorphism, and capsular or vascular invasion were applied to differentiate malignant lesions from adenomas.

Hyperplasia was assessed by examining the pattern and extent of cortical or medullary enlargement. Diffuse cortical hyperplasia was identified by uniform thickening of specific cortical zones, while nodular hyperplasia was characterized by well-formed macro- or micronodules within the cortex. Medullary hyperplasia was evaluated based on expansion of chromaffin cell clusters without architectural disruption or capsular invasion.

All specimens were examined using light microscopy at multiple magnifications to ensure accurate assessment of cellular composition, stromal characteristics, and growth patterns. Photomicrographs of representative areas were taken to document key histological features. Morphological impressions were correlated with available clinical data, including hormonal profiles and radiological findings, to support diagnostic interpretation.

Data were organized according to lesion type, and comparative analyses were conducted to differentiate benign tumors, malignant neoplasms, cortical hyperplasias, and medullary proliferations. Emphasis was placed on identifying consistent histological patterns that define each category of adrenal pathology.

Results

The morphological evaluation of adrenal gland lesions revealed a wide spectrum of structural alterations involving both the cortical and medullary components. Adrenocortical adenomas were predominantly well-circumscribed and encapsulated formations composed of uniform polygonal cells arranged in nests, trabeculae, or diffuse sheets. The cytoplasm was characteristically lipid-rich and vacuolated in cortisol-producing adenomas, whereas mineralocorticoid-secreting adenomas demonstrated compact cells with eosinophilic cytoplasm and relatively fewer lipid inclusions. Mitotic figures were rare, and nuclear atypia was minimal.

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In contrast, adrenocortical carcinomas exhibited marked pleomorphism, increased mitotic activity, and frequent atypical mitoses. Tumor cells often formed broad trabecular, alveolar, or diffuse growth patterns with areas of necrosis and hemorrhage. Capsular and vascular invasion were common findings, underscoring the aggressive biological behavior of these lesions. Weiss criteria applied to the carcinoma cases demonstrated consistent positivity for high mitotic index, necrosis, atypical mitoses, and loss of normal architectural organization.

Cortical hyperplastic lesions showed distinct morphological patterns depending on the underlying clinical condition. Diffuse hyperplasia presented with uniform thickening of the adrenal cortex, predominantly involving the zona fasciculata, with enlarged lipid-poor cells. Macronodular hyperplasia consisted of multiple nodules of variable size, separated by expanded cortical stroma. Micronodular hyperplasia was characterized by small clusters of compact, pigmented cells containing abundant lipofuscin granules, particularly in cases associated with ACTH-independent Cushing's syndrome.

Medullary tumors revealed characteristic neuroendocrine features. Pheochromocytomas demonstrated well-formed zellballen patterns composed of chromaffin cells with granular basophilic cytoplasm. The stroma was richly vascularized, frequently exhibiting sinusoidal dilatation. While most tumors lacked invasive features, some cases showed regions of cellular atypia and focal necrosis suggestive of malignant potential.

Medullary hyperplasia, typically observed in association with hereditary syndromes, was characterized by diffuse or nodular expansion of chromaffin cell clusters without the capsular or stromal disruption typical of pheochromocytoma. The hyperplastic medulla maintained a more uniform cellular architecture, with preserved demarcation from surrounding cortical tissue.

Cases of congenital adrenal hyperplasia demonstrated prominent cortical thickening composed of compact, lipid-depleted cells replacing the physiological zona structure. These morphological changes were consistent with chronic ACTH stimulation and altered steroidogenesis.

Overall, the morphological findings highlight clear distinctions among adrenal adenomas, carcinomas, and various forms of hyperplasia. The degree of cytological atypia, mitotic activity, presence or absence of encapsulation, and patterns of cortical or medullary expansion served as reliable criteria for differentiating benign, malignant, and hyperplastic adrenal lesions.

Discussion

The adrenal gland displays a wide range of pathological entities, each characterized by distinctive morphological patterns. Cortical adenomas are usually benign and hormonally functional, whereas adrenocortical carcinomas exhibit aggressive features including necrosis, mitotic activity, and invasive growth. Hyperplasias, particularly those associated with endocrine syndromes, require careful differentiation from neoplastic lesions due to overlapping architectural changes.

Medullary lesions such as pheochromocytoma illustrate a separate category of neuroendocrine tumors with characteristic zellballen patterns. Distinguishing between medullary hyperplasia and

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early pheochromocytoma is important, particularly in hereditary syndromes. A thorough understanding of morphological details, combined with clinical and biochemical data, is essential for accurate diagnosis.

Conclusion

Adrenal gland tumors and hyperplasias represent a complex and diverse group of endocrine lesions requiring precise morphological assessment. Adrenocortical adenomas and carcinomas can be differentiated based on cytological atypia, mitotic activity, and invasive features. Cortical hyperplasias demonstrate distinct nodular or diffuse patterns associated with endocrine dysfunction. Medullary tumors and hyperplasias each show unique neuroendocrine characteristics critical for diagnosis. Comprehensive pathomorphological evaluation is essential for guiding clinical management and improving prognostic accuracy in patients with adrenal gland disorders.

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