

A Commentary on Neuropathology of Pituitary Adenomas and Sellar Lesions

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Description

The pituitary gland can give rise to varied pathological processes, both neoplastic and inflammatory [1]. Among these, the vast majority of tumors are pituitary neuroendocrine neoplasms (Pit NETs), originating from the anterior pituitary lobe [2]. Proper classification and evaluation of these neoplasms is essential to provide proper patient management. Historically, classification relied on rather arbitrary stratification based on differences in staining with H&E [3]. Immunohistochemistry assay staining for pituitary hormones is now fundamental in elucidating the nature of these neoplasms, as well as newer transcription factor markers [2,4-9]. Transcription factors illuminate cell lineage into acidophilic lineage (somatotrophs, lactotrophs, thyrotrophs), corticotroph lineage, and gonadotroph lineage [4], in cases where no hormone staining/hormone excess is present (i.e., hitherto "null cell adenomas"). The clinical imperativeness of these stratifications is illustrated by certain adenoma subtypes behaving more aggressively by their very nature, including lactotroph adenomas (in men) and plurihormonal PIT-1 positive adenomas (previously called silent subtype 3 adenoma) [4]. Tumor invasion and elevated Ki-67 proliferation indices are also predictive of worse clinical outcomes, regardless of adenoma subtype [4,10,11]. Aggressive adenomas over time may progress to the rare but deadly pituitary carcinoma, resistant to conventional treatment modalities, defined by distant systemic metastases or noncontiguous spread of tumor cells within the Central Nervous System (CNS) [2,3,12,13].

Other pathologic processes (both neoplastic and inflammatory) may mimic pituitary adenomas on imaging, or sometimes even clinically (i.e. compression of the optic chiasm). The most frequent tumor among these are craniopharyngiomas, originating from Rathke pouch epithelium. The pituitary gland is also the second most common CNS site for germ cell neoplasms, after the pineal gland [14,15]. Less commonly, tumors of the posterior lobe occur, including pituicytoma, granular cell tumor, and spindle cell oncocytoma, which, unlike Pit NETS, lack staining for synaptophysin or pituitary hormones [16]. Additional possibilities include pituitary blastoma (an embryonal, undifferentiated malignancy), or tumors from the overlying meninges (sellar diaphragm), most commonly, meningiomas [1,17].

Inflammatory and related processes also occur in the pituitary region. These include lymphocytic hypophysitis (an autoimmune disorder often occurring in pregnancy), IgG4 disease (an autoimmune disorder rich in IgG4 plasma cells with resulting organ fibrosis), and Langerhans cell histiocytosis (a neoplastic, clonal proliferation of Langerhans Cells) [4,18].

The pictures are excellent, and illuminating. Normal pituitary microanatomy is juxtaposed and contrasted with the pathology of Pit NETs. Pituitary adenoma classification, with adenoma subtypes, anterior pituitary cell differentiation based on transcription factors, and more aggressive adenomas, is accurately listed. Diagnostic immunohistochemistry of the various adenomas and lesions

of the pituitary region are included, with H&Es of related lesions and tumors grouped appropriately for easy differential diagnostic reference.

Conclusion

This manuscript is very interesting and by furnishing a comprehensive review of pituitary adenomas and sellar lesions, adds to the medical literature. The most up to date methods to properly diagnose and classify these entities are provided, fundamental to patient care and treatment decisions.

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Conflict of Interest

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