

pituitary stalk, but AVP-D persisted, and he was diagnosed with LINH.

The evaluation of a patient with thickening of the pituitary stalk involves assessing the function of both the anterior and posterior pituitary glands and identifying the underlying cause. LINH is characterized by lymphocytic infiltration, leading to the eventual destruction of the pituitary tissue accompanied by varying degrees of pituitary dysfunction. Histopathology remains the gold standard for diagnosis, and definite diagnosis can only be established via pituitary stalk biopsy. Due to the wide range of possible aetiologies, caution and close monitoring are strongly recommended for the treatment of presumed cases lacking histopathological confirmation.

CONCLUSION

There are no evidence-based guidelines on the management of LINH due to its rarity. The glucocorticoid response rate has been variable. An individual approach is warranted. A conservative medical approach is often used as LINH is often self-limiting, especially when symptoms of mass effect are absent.

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SECONDARY HYPERTHYROIDISM PRESENTING WITH MASSIVE STROKE

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INTRODUCTION/BACKGROUND

Hyperthyroidism secondary to pituitary adenoma is rare (TSHoma). It comprises 1-3% of all pituitary adenomas. TSHoma itself does not directly cause strokes; however, the associated hyperthyroidism predisposes patients to cardiovascular complications, including atrial fibrillation and hypertension, thereby increasing the risk of stroke.

CASE

A 54-year-old Malay female complained of sudden-onset right-sided body weakness, aphasia, and confusion. Clinical examination revealed rapid atrial fibrillation (AF) and neurological deficits consistent with a left middle cerebral artery (MCA) territory infarct. CT scan of the brain showed a sellar mass. Initial investigation revealed elevated FT4 levels, with nonsuppressed TSH, prompting further diagnostic workup to confirm secondary hyperthyroidism. Her remaining pituitary functions were normal. Transthoracic echocardiography showed normal ventricular size, mild MR, AR, and moderate TR; however,

there was no left ventricular thrombus. Pituitary MRI later showed a 1.9 x 1.9 x 3.2 cm pituitary macroadenoma with suprasellar extension. AF was subsequently controlled with a beta blocker and direct oral anticoagulants (DOACs) were initiated. Hyperthyroidism was managed with octreotide LAR and biochemical euthyroidism was achieved, and the patient showed neurological improvement.

CONCLUSION

TSHoma-associated with stroke is rarely reported. Hyperthyroidism-induced cardiovascular complications are well-documented, including the risk of stroke. Treatment strategies for TSHoma aim to control hyperthyroidism and alleviate associated complications. While surgical resection is the definitive treatment, medical therapy with somatostatin analogues may be considered, as demonstrated in this case.

The management of TSHoma is challenging due to its association with hyperthyroidism-induced cardiovascular complications, and the risk of stroke. Recognizing TSHoma and early intervention may prevent its cardiovascular complications.

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CONFRONTING THE GIANT: A CASE REPORT ON THE MANAGEMENT OF RESISTANT PROLACTINOMA

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INTRODUCTION/BACKGROUND

Giant prolactinoma (tumor size more than 4 cm) is extremely rare, even more so in adolescents. Although generally benign, giant prolactinomas are locally aggressive, extending out of the pituitary fossa causing compression to the surrounding structures. While the mainstay of treatment for prolactinoma is medical therapy, lesions of such a large calibre may exhibit resistance to dopamine agonists.

We report a case of a 14-year-old female with a giant prolactinoma complicated with obstructive hydrocephalus which was resistant to cabergoline.

CASE

A 14-year-old female presented with a 4-month history of worsening peripheral vision loss. Neuroimaging revealed a large lobulated solid cystic sellar mass with suprasellar extension measuring 4.3 x 3.7 x 4.1 cm with optic chiasm