**CONCLUSION**

Studies have shown that CSI has a positive predictive value up to 88.9%. Use of CSI in this patient was helpful in demonstrating absolute suppression of the contralateral zona glomerulosa of a normal adrenal gland, thus lateralising the culprit adenoma and curing a patient of hypertension.

# PP-50

## HYPOPHYSITIS COMPLICATED BY PANHYPOPITUITARISM AND CRANIAL DIABETES INSIPIDUS: A CASE SERIES

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**INTRODUCTION**

Hypophysitis is gaining greater clinical recognition over the years while continuing to be a diagnostic and therapeutic challenge. Hypophysitis is the collective term for conditions presenting with inflammation of the pituitary gland and infundibulum. It can occur as a primary entity or secondary to systemic conditions. Pituitary inflammation usually results in pituitary hormone deficiency and enlargement of the gland. Inflammatory expansion of the gland can result in compression of the optic apparatus with resulting neuro-ophthalmic consequences. We described two cases of hypophysitis complicated by panhypopituitarism and cranial diabetes insipidus (DI).

**RESULTS**

Case 1 is a 37-year-old man with severe intractable headache complicated by bilateral 3$^{rd}$, 4$^{th}$, 6$^{th}$ nerve palsies and left partial 5$^{th}$ nerve palsy. Pituitary MRI demonstrated diffusely thickened pituitary stalk with absence of posterior pituitary bright spot on T1WI. Hormonal profiles revealed panhypopituitarism with morning cortisol <11 nmol/L (185-624), ACTH <1.1 pmol/L, TSH 0.267 mIU/L (0.38-5.33), fT4 5.38 pmol/L (7.86-14.41), fasting testosterone <0.35 nmol/L (5.72-26.14), FSH 1.23 mIU/mL (1.27-19.26) and LH 0.32 mIU/mL(1.24-8.62). Connective tissue screening and tumour markers were unremarkable. Multiple analgesia failed to alleviate his headache and he was subsequently given IV methylprednisolone followed by tapering dose of prednisolone. Subsequently, his biochemical profiles demonstrated evidence of cranial DI. Case 2 is a 35-year-old woman with forgetfulness and profound lethargy. Pituitary MRI demonstrated empty sella with hypothalamic retrochiasmatic lesion with mammillary body involvement. Hormonal profiles revealed panhypopituitarism with morning cortisol 45 nmol/L, FSH 1.4 mIU/mL, LH 0.3 mIU/mL, oestradiol <18.4 nmol/L, TSH 1.03 mIU/L, fT4 below detection. Lumbar puncture cerebrospinal fluid analysis was normal. Serum angiotensin converting enzyme (ACE) was 44 U/L (16-85) and IgG-4 was 44.9 mg/dL (2.4-121). Connective tissue screening and tumour markers were unremarkable. She was replaced with hydrocortisone and thyroxine. Following glucocorticoid replacement, she demonstrated polyuria and biochemically confirmed cranial DI.

**CONCLUSION**

Evaluation of patients with suspected hypophysitis involves a thorough clinical and laboratory armamentaria to decide on the optimal management strategies.