

EP_A127**RARE CASE OF PINEAL GERMINOMA WITH ARGININE VASOPRESSIN DEFICIENCY**

<https://doi.org/10.15605/jafes.039.S1.138>

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INTRODUCTION/BACKGROUND

Pineal tumour is an uncommon type of brain tumour in Malaysia, with low incidence rate of 2.1% based on a demographic study done in 1 of the states in Peninsular Malaysia. On the other hand, germinoma is a common cause of intracranial tumor leading to arginine vasopressin deficiency (AVP-D). In a literature review, 82% of 95 patients were diagnosed with AVP-D.

CASE

We report a case of a 22-year-old Malay male, who was diagnosed with pineal gland tumour complicated with obstructive hydrocephalus. He underwent endoscopic biopsy of pineal region tumor and right ventriculoperitoneal shunt insertion in January 2024.

Two days after his surgical intervention, he developed an episode of polyuria with urine output more 5 ml/kg/hr for consecutive hours in the ward without any accompanying thirst. Investigations showed high serum osmolality of 294 mOsm/kg (NR: 271-286 mOsm/kg) with inappropriately low urine osmolality of 161 mOsm/kg (NR: 300-900 mOsm/kg).

He was diagnosed with AVP-D and responded well to intravenous desmopressin (DDAVP), a vasopressin synthetic analogue, with reduction in urine volume and passing more concentrated urine. Over the next few days, he started to develop more episodes of polyuria, requiring regular dosing of desmopressin.

The tissue histopathology of the pineal gland revealed germ cell tumour consistent with germinoma.

CONCLUSION

AVP-D is a known complication of intracranial germ cell tumour. Our case report shows that AVP-D may also happen as a consequence of neurosurgical intervention. Careful clinical and biochemical postoperative monitoring remains essential to correctly diagnose and manage AVP-D following neurosurgery intervention.

EP_A128**WORSENING OF EXISTING CRANIAL DIABETES INSIPIDUS SYMPTOMS AFTER COMMENCEMENT OF HYDROCORTISONE**

<https://doi.org/10.15605/jafes.039.S1.139>

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INTRODUCTION/BACKGROUND

Central diabetes insipidus (DI) is a condition that causes persistent thirst and frequent urination. It can develop when a pituitary tumour compresses on the posterior pituitary gland and impairs the gland's ability to secrete vasopressin hormone.

CASE

We report a case of a 22-year-old male who presented with a 5-month history of delayed responses, progressive visual loss and lethargy. He also complained of excessive thirst, frequent urination and increased urine volume which was diluted. Clinically, he was dehydrated. Ophthalmological assessment revealed monocular blindness. Computed tomography of the brain reported a suprasellar mass with obstructive hydrocephalus. Anterior pituitary hormone profile revealed panhypopituitarism and laboratory results confirmed the presence of DI. He was managed with hydrocortisone tablets and BD dosing of desmopressin. Unfortunately, he developed episodes of worsening polyuria, and his sodium increased from 143 to 150 mmol 24 hours following commencement of hydrocortisone. His urine output improved following an increment of desmopressin to TDS dosing and his serum sodium normalized.

Anterior pituitary hormone profiles showed panhypopituitarism with stalk effect; TSH: 1.192 m IU/L, FT4: 3.9 pmol/L, LH: 0.35 IU/L, FSH: 0.61 IU/L, testosterone: <0.35 nmol/l, ACTH: <0.22 pmol/L, morning cortisol: 108 nmol/L and serum prolactin: 2042 m IU/L. His urine sodium: 47 mmol/L, serum osmolality: 345 mosm/kg, and urine osmolality: 277 mOsm/kg. Pituitary MRI showed a well-defined lobulated suprasellar cystic lesion measuring 2.4 x 2.3 x 1.7 cm, causing compression to the floor of the 3rd ventricle which leads to obstructive hydrocephalus. There was a focally enhancing lesion at the pineal gland. He subsequently underwent a right ventriculoperitoneal shunt and supraorbital craniotomy and biopsy. HPE was reported as germinoma. He is currently stable on hydrocortisone, thyroxine and sublingual desmopressin replacement.

CONCLUSION

Glucocorticoid replacement may precipitate diabetes insipidus in the setting of adrenal insufficiency. Low cortisol levels will stimulate the release of antidiuretic hormone and increase water reabsorption in the kidney. This effect can be inhibited by exogenous steroids which may then rapidly unmask or worsen central DI.

EP_A129
**SPONTANEOUS RHINORRHOEA
SECONDARY TO NORMAL-PRESSURE
PARTIAL EMPTY SELLA SYNDROME**

<https://doi.org/10.15605/jafes.039.S1.140>

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INTRODUCTION/BACKGROUND

Empty sella syndrome is a disorder where the sella turcica is partially or completely filled with cerebrospinal fluid (CSF), resulting in compression and displacement of the pituitary gland. Empty sella is frequently coupled with increased intracranial pressure which causes spontaneous CSF leaks. Some people may experience normal-pressure CSF leaks, which can be due to idiopathic or congenital abnormalities. Both high-pressure and normal-pressure CSF leaks are commonly seen in middle-aged obese women.

CASE

A 43-year-old obese female with a BMI of 34.9 kg/m² presented with a two-day history of headache and clear nasal discharge persisting for a month. She reported no weakness or visual disturbances. Upon examination, her blood pressure measured 142/70 mmHg, her Glasgow Coma Scale was intact, and her neurological assessment revealed no abnormalities. The ophthalmological evaluation showed no signs of papilledema. Subsequently, a lumbar puncture was performed, indicating a normal opening pressure of 12 cm H₂O. Brain MRI revealed a partially empty sella, while CT imaging of the paranasal sinuses aimed to identify the cause of the CSF leak, was suggestive of cribriform plate dehiscence. Evaluation of anterior pituitary hormones yielded results within normal ranges. The case was then referred to a neurosurgical team for further evaluation and potential surgical intervention.

The occurrence of a spontaneous cCSF leak frequently coincides with the radiographic discovery of an empty sella. Its clinical manifestation can vary, encompassing symptoms such as headaches, rhinorrhoea, visual impairments, and hormonal imbalances. Assessing pituitary hormone levels

is crucial, with hormonal supplementation warranted when deficiencies are identified. Surgical intervention is essential in managing CSF leaks to mitigate potential complications like meningitis and brain abscesses.

CONCLUSION

A CSF leak is important to be recognised at the initial presentation, as surgical intervention is required to reduce the complication of infections.

EP_A130
**IMMUNOLOGICAL CROSSFIRE:
ENDOCRINOPATHIES IN THE AGE OF
IMMUNE CHECKPOINT INHIBITORS**

<https://doi.org/10.15605/jafes.039.S1.141>

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INTRODUCTION/BACKGROUND

Pembrolizumab is a PD1 receptor inhibitor which is a type of immune checkpoint inhibitor (ICI) for cancer therapy. Immune-related adverse events (IRAE) are more commonly encountered with increased use.

CASE

A 52-year-old male with recurrent renal cell carcinoma developed endocrinopathies following pembrolizumab. After failure of first-line treatment, he received the first dose of pembrolizumab in November 2022.

Six weeks later, he was symptomatic of hyperthyroidism with corresponding biochemistry of TSH <0.01 m IU/L (reference interval 0.55 – 4.8), free T4 of 28.8 pmol/L (reference interval 11.5 – 22.7) and free T3 of 9.7 pmol/L (reference interval 3.5 – 5.5). Following treatment with low dose and tapering carbimazole for eight weeks, he quickly became hypothyroid. Biochemistry showed TSH of 87.3 m IU/L, fT4 of 2.9 pmol/L and fT3 of 1.3 pmol/L. Anti-thyroid peroxidase and anti-thyroglobulin antibodies were negative. Carbimazole was discontinued and levothyroxine 50 mcg daily were commenced. Pembrolizumab was continued.

Four months later, he presented with lethargy and postural symptoms. A random serum cortisol level was undetectable at <14 nmol/L (reference interval 145 – 619) and ACTH level was inappropriately normal at 6 pg/ml (reference interval 0 – 46). He denied any polyuria, polydipsia, or