DOI: 10.7860/JCDR/2015/15775.7005

An Uncommon Cause for Polyuria

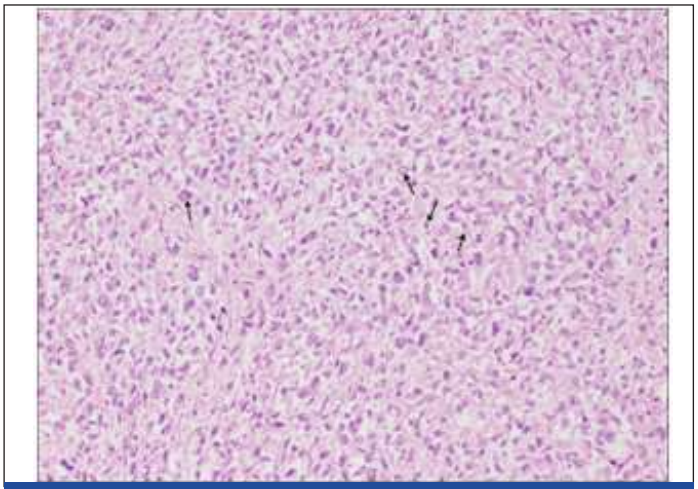
FELIX K JEBASINGH[1], DM MAHESH[2], SAMANTHA SATHYAKUMAR[3], ELANTHENRAL SIGAMANI[4], THOMAS V PAUL[5]

Keywords: Cytokeratin-7, Diabetes insipidus, Lung carcinoma, Signet Ring cell

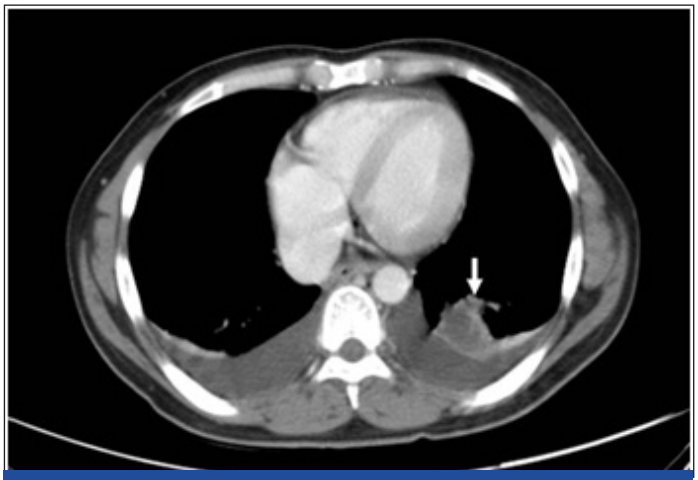
Diabetes Insipidus (DI) is characterized by passing of large volume of urine secondary to deficient secretion of antidiuretic hormone (Central DI) or renal resistance to its action (nephrogenic DI). Central DI is secondary to the destruction of neurons arising in the supraoptic and paraventricular nuclei [1]. The known causes include inflammatory or autoimmune diseases (e.g. sarcoidosis and Wegener's granulomatosis), Langerhans cell histiocytosis, craniopharyngiomas, cranial injury resulting from surgery or an accident, metastases and rarely genetic defects in antidiuretic hormone synthesis. Nephrogenic DI may result from a genetic cause (e.g.: X-linked NDI is secondary to Arginine Vasopressin (AVP) receptor 2 mutations leading to a loss of function resulting in renal resistance) or an acquired cause (e.g: hypercalcaemia and medications like lithium and cisplatin) [2]. Herewith we report on a patient who presented to us with the features of diabetes insipidus. which had an abrupt onset secondary to a metastatic disease.

A 31-year-old gentleman presented to Emergency Department with altered sensorium of 4 days duration. He was feeling unwell over 4 weeks prior to this presentation with an abrupt onset of polydipsia and polyuria (about 5-6 liters of urine/24 hours) along with a weight loss of 6 kg. There was no history of trauma, headache, fever, addictions, chronic illness or any long term medication use. On examination, his Glasgow Coma Scale (GCS) was 11 over 15. There was a group of matted lymph nodes (4X6 cm) in the right lower posterior cervical region. There were no signs of meningeal irritation and optic fundi examination and systemic examination were unremarkable. His haemogram and blood glucose levels were normal. The serum sodium level was 165mmol/l (135-145) with urine osmolarity of 105 mmol/kg (126-502). Other biochemical results are as follows: TSH-0.6µIU/ml (0.3 - 4.5); T4 (Total Thyroxine) -9.3ug/dL (4.5 - 12.5); FT4C (Free thyroxine)-1.3 ng/dL (0.8 - 2.0); FSH -0.7mIU/ml (0.7 – 11.1); LH -0.1 mIU/ml (0.8 – 7.6); Testosterone-20.0ng/dL (270-1030); Cortisol (8 AM) -11.6 ug/dL (7-25). The Computed Tomography [Table/Fig-1a] and MRI [Table/Fig-1b] of the brain are shown below. Histopathology from cervical lymph node biopsy and CT of the chest are shown in [Table/Fig-2,3] respectively. Both CT and the MRI brain showed a sellar-suprasellar mass lesion. The biopsy of the lymph node showed signet ring cells which were positive for CK7(Cytokeratin-7) and focally for TTF1(Transthyretin Factor-1) suggestive of an adenocarcinoma probably of the pulmonary origin (Signet Ring Cell Type). A subsequent CT of the chest showed a mass in the left lung. Thus the patient was diagnosed to have Diabetes Insipidus (DI) due to metastasis of the adenocarcinoma of the lung (signet ring cell type) with partial hypopituitarism. He was initiated on desomopressin with which his sensorium improved along with normalization of sodium and urine output. He was referred to medical oncology for further management.

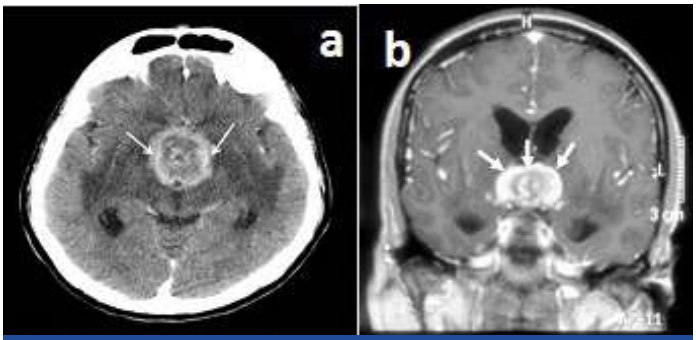
Malignancies from breast, lung, colon and prostate are the most commonest to have metastases in the pituitary gland. While most of pituitary metastases remain asymptomatic, common clinical presentation in symptomatic subjects is DI indicating preference of metastases to the posterior lobe. A CT scan usually shows a hyperdense or isodense mass which may enhance homogeneously



[Table/Fig-2]: Histopathology (H&E) of lymph node –Signet ring cells along with increased mitotic figures suggestive of an adenocarcinoma (Signet Ring Cell Type)



[Table/Fig-3]: CT Thorax–A Hypodense lobulated mass lesion in the posterior basal segment of the left lower lobe of the lung measuring 3.4 x 3.1 cm



[Table/Fig-1]: CT and MRI Brain showing a well-defined 3.4x3.2 sized heterogeneously enhancing lesion in sellar suprasellar region

or non-homogeneously with contrast, whereas MRI scan commonly demonstrates a high-intensity signal on T2 weighted images and the definitive diagnosis of metastatic involvement is always based on histological evaluation [2]. Management is usually palliative which includes medical treatment of diabetes insipidus and surgical debulking with or without radiotherapy especially when there is compromise of vision secondary to compression [3].

REFERENCES

[1] Di Iorgi N, Napoli F, Allegri AE, Olivieri I, Bertelli E, Gallizia A, et al. Diabetes insipidus--diagnosis and management. *Horm Res Paediatr*. 2012;77(2):69-84.
[2] Ratti M, Passalacqua R, Poli R, Betri E, Crispino M, Poli R, et al. Pituitary gland metastasis from rectal cancer: report of a case and literature review. *Springer Plus*. 2013;2:467.
[3] Hermet M, Delévaux I, Trouillier S, André M, Chazal J, Aumaître O. Pituitary metastasis presenting as diabetes insipidus: a report of four cases and literature review. *Rev Med Interne*. 2009;30(5):425-29.

PARTICULARS OF CONTRIBUTORS:
1. Senior Registrar, Department of Endocrinology, Diabetes & Metabolism, Christian Medical College, Vellore, India.
2. Assistant Professor, Department of Endocrinology, Diabetes & Metabolism, Christian Medical College, Vellore, India.
3. Senior Registrar, Department of Endocrinology, Diabetes & Metabolism, Christian Medical College, Vellore, India.
4. Assistant Professor, Department of Pathology, Christian Medical College, Vellore, India.
5. Professor, Department of Endocrinology, Diabetes & Metabolism, Christian Medical College, Vellore, India.

NAME, ADDRESS, E-MAIL ID OF THE CORRESPONDING AUTHOR:
Dr. Thomas V Paul,
Professor, Department of Endocrinology, Diabetes & Metabolism,
Christian Medical College, Vellore – 632 004, India.
E-mail : thomasvpaul@yahoo.com

FINANCIAL OR OTHER COMPETING INTERESTS: None.

Date of Submission: **Jul 18, 2015**
Date of Peer Review: **Sep 18, 2015**
Date of Acceptance: **Oct 15, 2015**
Date of Publishing: **Dec 01, 2015**