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Central Diabetes Insipidus Following Hemorrhagic Stroke- A Case Report

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Abstract: *Central Diabetes Insipidus (CDI) following intraparenchymal hemorrhage of the brain has been described as a rare presentation. Posterior pituitary ischemia has also been postulated as a possible cause of idiopathic CDI. We encountered a young male with right sided capsuloganglionic bleed who had polyuria due to central Diabetes Insipidus, requiring Desmopressin therapy.*

Key Words**:** *Diabetes Insipidus, Stroke, Polyuria*

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I. Introduction

Central Diabetes Insipidus (CDI) following hemorrhagic stroke has been described as a rare presentation.[1] Diabetes Insipidus occurs commonly in the acute phase of neurosurgical insults such as pituitary surgery, subarachnoid hemorrhage and traumatic brain injury. The onset is usually 1 to 3 days after such an insult and manifests principally as hypotonic polyuria [2]. Many neurosurgical patients have a diminished consciousness level because of brain injury, postoperative cerebral irritation, cerebral edema, sedation for airway management or a combination of these factors. Hence, their awareness of thirst or their ability to respond to it by ingesting fluids may be diminished or absent, and they are vulnerable to the development of severe hypernatremia. It is therefore particularly important to monitor the urine output and daily plasma sodium concentrations in these patients.

II. Case Report

A 47 years old male presented on the 13th day following right sided hemorrhagic stroke with progressive obtundation of sensorium. There was no history of headache, fever, trauma, hypertension or diabetes mellitus. On examination, he was normotensive, stuporous and pupils were equal in size and reaction. He had left sided hemiparesis and extensor plantar reflex bilaterally. Computerised Tomography (CT) scan of the brain revealed right sided resolving capsuloganglionic bleed with ventricular compression.[Figure1,2] Serum sodium was 163mmol/L at presentation. He developed increasing urine output (Volume 1760ml In 12 Hours) with corresponding fluid intake of 900 Ml over 12 hours. Urinary spot sodium was 243 Mmol/L. Plasma osmolality (Calculated) was 294 Mosm/Kg (285-295 Mosm/Kg). In view of his neurological status, he was subjected to a limited water deprivation test. Over three hours of absolute water deprivation, all IV and oral fluids were withheld with a close watch over his BP and urine output. During the test, he passed 760 ml of urine and developed hypotension (BP 90/60 mmHg), due to which the test was aborted and serum and urine samples were taken for osmolality. At the termination of test, he had serum sodium 145 Mmol/L, calculated plasma osmolality of 305 Mosm/Kg with corresponding urine osmolality of 357 Mosm/Kg (Expected >600 Mosm/Kg) which confirmed DI. He was started on Desmopressin Nasal Spray at A dose of 10mcg twice a day. Over the next two days, his daily urine output had reduced to 2 to 3 litres, commensurate with fluid intake. Urine osmolality increased to 685 Mosm/Kg with normalization of sodium levels and plasma osmolality. After 5 days of starting Desmopressin, his urine output decreased to 1020 ml and serum sodium levels decreased to 147 Mmol/L, when dose of Desmopressin was reduced to 10 mcg once a day. Urine osmolality was 762 Mosm/Kg. He regained full consciousness, but had persistent aphasia, facial paresis and left limb weakness with left extensor plantar. Other hormone evaluation including thyroid, cortisol, gonadotropins and testosterone were normal. MRI brain did not show any pituitary haemorrhage or necrosis. Posterior pituitary hyperintense (Bright Spot) was normally seen [Figure 3].

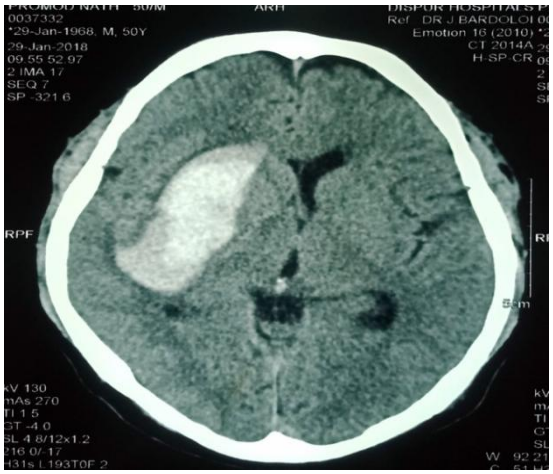

Figure 1. NCCT brain showing right capsuloganglionic bleed with ventricular compression.

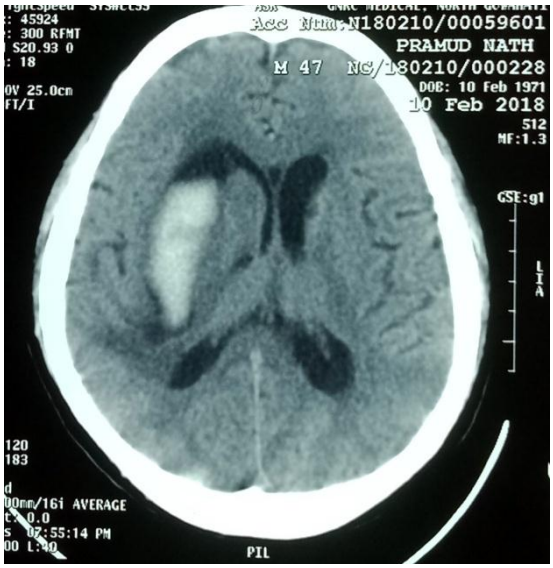

Figure 2. NCCT brain showing resolving right capsuloganglionic hematoma on the day of presentation

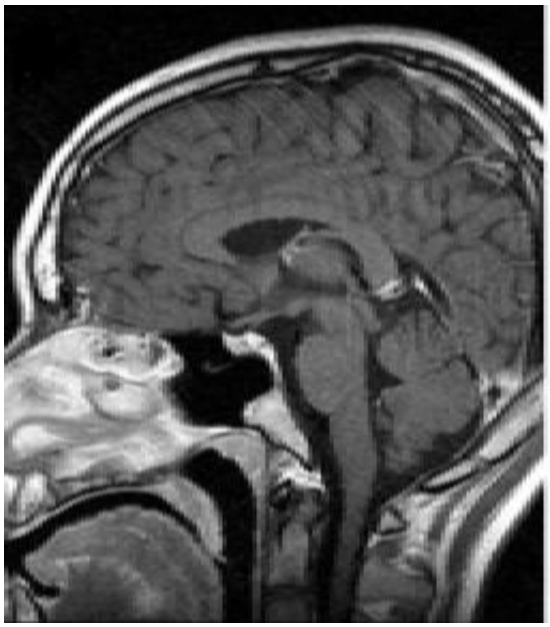

Figure 3. MRI of brain (Sagittal section) showing normal pituitary with intact posterior pituitary hyperintensity

III. Discussion

CDI is a rare complication of intraparenchymal hemorrhage Of Brain.[1] Muthukrishnan Jayaraman Et Al described a young male who presented with bilateral ischemic infarcts of brain who developed transient CDI two weeks into his illness, requiring desmopressin and recovered completely in five days with no other pituitary hormone deficiency. [3]Ischemia of posterior pituitary due to inferior hypophyseal arteries has also been proposed as a cause of idiopathic CDI.[4] Transient CDI has also been described following cardiopulmonary bypass.[5] In the setting of hemorrhagic stroke, CDI is a rare manifestation, which can be explained by involvement of osmoreceptors in hypothalamus and posterior pituitary by hypoperfusion in a setting of hemorrhagic stroke. Normal pituitary gland on imaging and normal anterior pituitary hormones ruled out any significant trauma or persistent ischemic or hemorrhagic damage to the pituitary gland. Late onset of polyuria after the onset of stroke can be due to residual stored AVP in the posterior pituitary nerve endings.

IV. Conclusion

This case highlights a rare complication of a common condition like hemorrhagic stroke. Confirmation requires water deprivation test modified in view of the neurological status. Desmopressin replacement therapy requires to be administered with a close watch on fluid intake, electrolyte status and likely resolution of DI with neurological recovery.

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