Brief Communication

Transient central diabetes insipidus following ischemic stroke

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ABSTRACT

Central Diabetes Insipidus (CDI) following ischemic infarction 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 bilateral extensive ischemic infarction sustained at high altitude, who had transient polyuria due to central diabetes insipidus, requiring desmopressin therapy. DI completely resolved during the course of his neurological recovery.

Key words: Diabetes insipidus, stroke, polyuria

Introduction

Central Diabetes Insipidus (CDI) following ischemic infarction of the brain has been described as a rare presentation.[1] Posterior pituitary ischemia has also been postulated as a possible cause of idiopathic CDI.[2] Complete resolution during the course of neurological recovery suggests vascular mechanism of this defect.

Case Report

A 35 years old male, presented with sudden onset left hemiparesis. Over the next 24 h, he developed generalized tonic-clonic seizure and progressive obtundation of sensorium. There was no history of preceding transient ischemic attack, 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 bilateral frontal, temporopareital and occipital ischemic infarcts with hemorrhagic transformation. Magnetic resonance imaging (MRI) of brain confirmed these findings. [Figure 1] MR Arteriography (MRA) and venography (MRV) did not show any evidence of vascular thrombosis.

He was managed with anti-cerebral edema measures (Mannitol), statins, antiplatelets and supportive care. Over the next two days, he developed worsening level of consciousness due to increasing mass effect. As a life saving measure, decompressive craniectomy with expansile duroplasty was done. Subsequently, he developed increasing urine output (volume 6900 ml, 5525 ml, 5500 ml in 24 h) with corresponding fluid intake of 3000-4000 ml over the next three days and serum sodium levels increased from 136 mMol/l to 145 mMol/l (N 135-144 mMol/l). 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 775 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

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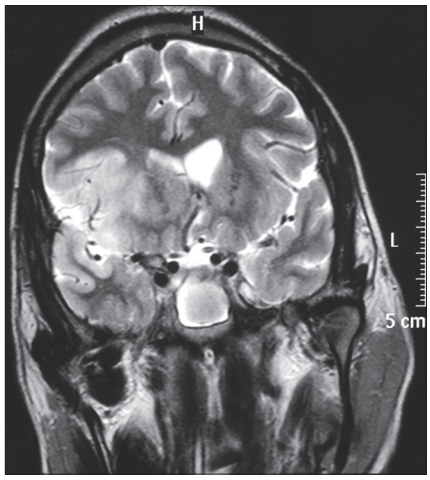

Figure 1: MRI brain (Coronal section) showing right sided temporopareital infarction

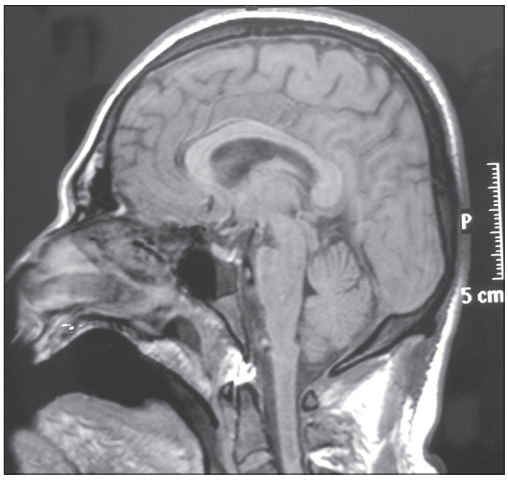

Figure 2: MRI of brain (Sagittal section) showing normal pituitary with intact posterior pituitary hyperintensity

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 1050 ml and serum sodium levels decreased to 131 mMol/l, when dose of desmopressin was reduced to 10 mcg once a day and subsequently withdrawn in view of persistent hyponatremia. Following withdrawal of desmopressin, he continued to have normal urine output and serum sodium levels. Urine osmolality was 762 mOsm/kg. He regained full consciousness, but had persistent aphasia, facial paresis and bilateral limb weakness (L > R) with bilateral extensor plantars. 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 2].

Discussion

CDI is a rare complication of ischemic infarction of brain.[1] We treated 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. Osmotic causes of polyuria were excluded. Clinical setting did not permit a standard water deprivation to be performed hence; a limited test was done with satisfactory predictive value for DI. Urinary osmolality above 300 mOsm/kg with plasma hyperosmolality and persistent polyuria during the test confirmed CDI. Response to desmopressin with normalization of urine output and concentration of urine confirmed central DI. Post-recovery evaluation excluded persistent CDI, urinary concentrating defect or other pituitary hormone deficiency.

Ischemia of posterior pituitary due to inferior hypophyseal arteries has also been proposed as a cause of idiopathic CDI.[2] Transient CDI has also been described following cardiopulmonary bypass.[3] In the setting of ischemic infarction, CDI is a rare manifestation, which can be explained by Involvement of osmoreceptors in hypothalamus and posterior pituitary by hypoperfusion in a setting of ischemic stroke. Normal pituitary gland on imaging and normal anterior pituitary hormones ruled out any significant trauma or persistent ischemic or haemorrhagic damage to the pituitary gland. [Figure 1] Various pathophysiological mechanisms have been postulated underlying transient central DI due to cerebral ischemia. Resolution of CDI during recovery of his neurological status suggests vascular cause for CDI in this patient. Late onset of polyuria after the onset of stroke can be due to residual stored AVP in the posterior pituitary nerve endings.

This case highlights a rare complication of a common condition like ischemic 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.

References

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