

Hyponatremia initial presenting feature of normal pressure hydrocephalus in elderly patient: a rare case report

S. Kumar, P. Bhayani, D. Hathi, J. Bhagwati

Jawahar Lal Nehru Medical College, Datta Meghe Institute of Medical Sciences (Deemed to be University): Wardha, Maharashtra, India

A 70 year old male presented with unsteadiness, declining cognitive functions and urinary incontinence, with serum sodium level of 108 meq/L and serum osmolality of 268 mosm/kg. His computerized tomography scan of Brain showed ventriculomegaly which made us to keep the possibility of Normal Pressure Hydrocephalus (NPH). This case report highlights about suspecting syndrome of inappropriate antidiuretic hormone secretion (SIADH) due to hyponatremia as an important differential in a case of Normal Pressure Hydrocephalus. Theoretically, hyponatremia may occur in NPH, but only three associated cases of SIADH have previously been reported in the literature and first in India.

Key words: Hyponatremia, Normal Pressure Hydrocephalus, Elderly, SIADH

INTRODUCTION

Normal pressure hydrocephalus is a rare condition which is clinically characterized by triad of gait disturbance, incontinence of urine and decline in cognitive function resulting from ventriculomegaly without elevated CSF pressure. Etiology of NPH is unknown; current theory suggests there is diminished vascular compliance¹. The disorder of SIADH is due to non physiological release of hormone from the neurohypophysis due to low plasma osmolality. Theoretically hyponatremia may occur in NPH, but till late only three associated cases of SIADH has been reported in literature¹⁻³. Here a case of elderly male with typical clinical features and brain imaging consisting with NPH accompanied by hyponatremia due to SIADH is presented.

CASE REPORT

A 70 year old male patient was admitted in medicine intensive care unit of our hospital with complaint of urinary incontinence since 6 month, generalized weakness, unsteadiness and giddiness. Patient's relatives also gave

history of forgetfulness on and off. There was no history of swelling in lower limbs. There was no history of head trauma, cerebrovascular episodes and chronic illness like hypertension or diabetes. There was no history of chronic urinary tract infection, benign prostatic hypertrophy and diuretic use. He was non smoker and non alcoholic.

On examination, the patient was conscious, disoriented to time, place and person. His BP was 110/70 mmHg, pulse of 110 beat per minute, normal Jugular Venous Pressure, and respiratory rate of 20 per minute. There was no edema in lower limb. His pupils reacted appropriately to light and there was no papilledema on fundus examination. There was no postural hypotension. Rest of the systemic examination was normal. Neurological examination showed mild cognitive function impairment with ataxic gait. His speech was normal and there was no evidence of any sensory deficit.

On routine investigations his Complete blood count was within normal limit. Renal function test was done which showed blood urea of 40 mg/dl, serum creatinine of 0.9 mg/dl, serum sodium levels of 108 mmol/L, potassium of 4.5 mmo/L. His thyroid and hepatic profile was within normal limits. His chest X-ray was normal. In view of low

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■ Correspondence: Sunil Kumar, Department of Medicine, Jawahar Lal Nehru Medical College, Datta Meghe Institute of Medical Sciences (Deemed to be University), Wardha, Maharashtra, India ,442001 - E-mail: sunilkumarmed@gmail.com

serum sodium, we investigated for serum Osmolality which was 268 mOsm/kg, urine Osmolality was 158 mOsm/kg and urine sodium was 24 mmol/L. In this patient, hyponatremia associated with decreased osmolality with inappropriately increased urine osmolality, natriuresis, normal thyroid and adrenal function, absence of renal, hepatic, or cardiac disease makes the diagnosis of SIADH.

His computerized tomography scan of Brain revealed disproportional enlargement of the temporal horns of the lateral ventricles with diffuse periventricular and confluent white matter signal changes (Fig. 1).

His sodium was corrected with 3% and normal saline infusion in due course. He became oriented, as well as unsteadiness and giddiness also improved. He did not undergo neurosurgical intervention due to financial constraint, hence was subjected to therapeutic lumbar puncture where 20 ml of CSF was drained which was under normal pressure after which there was subjective improvement in the patient's symptoms. There was no recurrence of hyponatremia after treatment with 3% and normal saline along with CSF drainage by lumbar puncture. Patient had partial improvement in cognition, his gait and urinary incontinence also improved. Blood chemistry became normal.

DISCUSSION

This patient developed hyponatremia because of SIADH which was part of NPH, a relatively rare form of hydrocephalus especially in elderly. The temporal profile of symptomatology of this patient like forgetfulness, ataxia and urinary incontinence in the presence of characteristic ventriculomegaly from the brain imaging was diagnostic of NPH.

Essential diagnostic criteria for the diagnosis of SIADH are:

- serum sodium < 135 mmol/L;
- decreased measured plasma osmolality (< 275 mOsm/kg);
- urinary osmolality > 100 mOsm/kg during Hypotonicity;
- clinical euvolemia;
- no clinical signs of contraction of extracellular fluid (e.g. no orthostasis, tachycardia, decreased skin turgor or dry mucous membranes);
- no clinical signs of expansion of extracellular fluid (e.g. no edema or ascites);
- increased urinary sodium excretion > 30 mmol/L with normal dietary salt and water intake;
- normal thyroid and adrenal function determined by both clinical and laboratory assessment;
- no recent use of diuretic agents.

Supporting diagnostic criteria are:

- plasma uric acid < 4 mg/dL;

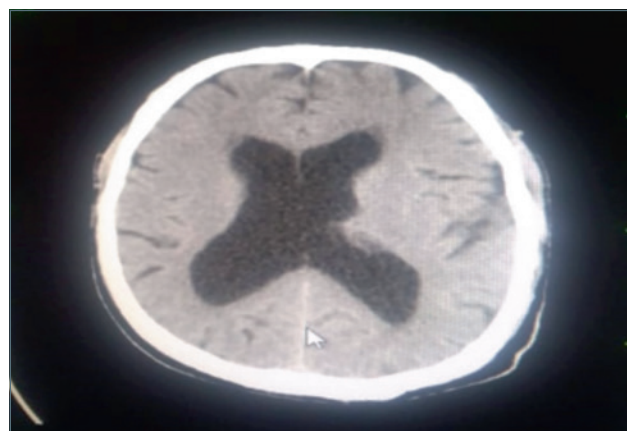


Figure 1. Computerized tomography scan of Brain showing disproportional enlargement of the temporal horns of the lateral ventricles.

- blood urea nitrogen < 10 mg/dL;
- fractional sodium excretion > 1%;
- fractional urea excretion > 55%;
- failure to improve hyponatremia after 0.9% saline infusion;
- improvement of hyponatremia with fluid restriction⁴.

In this patient hyponatremia was accompanied by decreased osmolality with inappropriately increased urine osmolality, natriuresis, normal thyroid function, absence of renal cardiac and hepatic disease, a classical feature of SIADH and NPH was tenable.

Hyponatremia due to SIADH in a patient with NPH is thought to be result from the mechanical pressure on the hypothalamus from the third ventricle. Possible explanation for this hypothesis is that many patients with NPH show response to CSF shunting¹³.

In conclusion SIADH, an extremely rare metabolic manifestation of NPH should be considered whenever a hyponatremia is considered especially in elderly.

CONFLICT OF INTEREST

The authors declare no conflict of interest.

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