

## Clinical Reports

# Post-traumatic diabetes insipidus combined with primary polydipsia

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**Summary:** We describe a case of diabetes insipidus after head injury in which thirst persisted despite treatment with DDAVP and normal plasma osmolality. Symptoms were only completely relieved when plasma osmolality was below 270 mosmol/kg. We believe that this might have been due to hypothalamic injury causing resetting of the thirst osmostat. To our knowledge, this type of primary polydipsia has not been described before in association with diabetes insipidus following head injury.

### Introduction

Primary polydipsia may occur in patients with severe mental or emotional disturbances.<sup>1</sup> It may also occur in patients with organic hypothalamic pituitary disease,<sup>2</sup> or in association with cerebral damage. It has not been described in association with diabetes insipidus due to head injury.

We report a patient who developed diabetes insipidus after head injury, whose symptoms were only completely relieved when his plasma osmolality was reduced below 270 mosmol/kg. We interpret this case as one of combined diabetes insipidus and primary polydipsia due to post-traumatic hypothalamic injury.

### Case report

A 20 year old man was admitted following a motorcycle accident in which he fractured his left frontal, parietal and temporal bones. He had bilateral periorbital haematomas with proptosis for which he underwent bilateral decompression. After 4 weeks he developed polydipsia and polyuria. Drinking reduced his thirst for only a few minutes and diabetes insipidus was suspected. His 24 hour urine volume was 9100 ml with a urine osmolality of 60 mosmol/kg and his plasma osmolality was 277 mosmol/kg. The osmolality was measured by freezing point depression using a HALBBIKRO osmometer (Knauer & Co., West

Germany). The reference range for normal healthy individuals under basal conditions in our laboratory is 280–295 mosmol/kg. The coefficient of variation is 0.83% at 286 mosmol/kg and 0.78% at 325 mosmol/kg.

An 8 hour water deprivation test showed a maximum plasma osmolality of 298 mosmol/kg with a simultaneous urine osmolality of 317 mosmol/kg which increased to 590 mosmol/kg after 2 µg desmopressin (DDAVP) intramuscularly. In normal individuals, urine osmolality after 8 hours' water deprivation exceeds 750 mosmol/kg and does not increase after DDAVP. In patients with cranial diabetes insipidus the urine osmolality is usually lower than plasma osmolality after dehydration but increases to > 750 mosmol/kg after DDAVP. In our patient urine osmolality was higher than plasma osmolality after dehydration and showed a further increase after DDAVP but only to 590 mosmol/kg. These results were interpreted as being due to partial diabetes insipidus together with a degree of polyuria-induced nephrogenic diabetes insipidus. The patient was put on a therapeutic trial of 2 µg DDAVP intramuscularly daily. Twelve hours after commencing DDAVP plasma osmolality decreased to 283 mosmol/kg but he remained extremely thirsty. Consequently, fluid intake was restricted to the previous 24 hour output in an attempt to maintain plasma osmolality above 280 mosmol/kg. For the following 10 days urine volumes ranged between 3.0 and 4.5 litres/day and despite plasma osmolalities between 280 and 285 mosmol/kg he remained extremely thirsty. He was

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**Table I** Prolonged water deprivation test (19 hours)

	Plasma osmolality (mosmol/kg)	Urine osmolality (mosmol/kg)	Cumulative volume (ml)	Plasma AVP (pmol/l)	Weight (kg)
Pre-water deprivation	270	70			56
Post-water deprivation					
9 hours	292	95	2700	0.12	
11 hours	295	95	3370		
13 hours	305	230	3492		
15 hours	305	185	3767		
17 hours	302	295	3872	0.08	
19 hours	300	338	3977		52
After 2 µg DDAVP i.m.					
12 hours	283	730			

then allowed free access to water for 2 days and plasma osmolality dropped to 262 on the first day and 265 mosmol/kg on the second. Compulsive water drinking was suspected. Because of the atypical response to DDAVP, the drug was stopped and a prolonged water deprivation test was performed (see Table I). The starting plasma osmolality was 270 mosmol/kg. Nineteen hours of water deprivation produced plasma hyperosmolality (305 mosmol/kg) and a 7% weight loss. Maximum urine osmolality was 338 mosmol/kg rising to 730 mosmol/kg after 2 µg DDAVP i.m. Plasma arginine vasopressin (AVP) was determined, at the SAS Laboratory, Middlesex Hospital, by radioimmunoassay,<sup>3</sup> and was low for the corresponding plasma osmolality (Table I). The results confirmed diabetes insipidus, but this diagnosis did not explain the low starting plasma osmolality (270 mosmol/kg). Treatment with DDAVP 2 µg i.m. daily was recommenced and the patient allowed free access to water. After 24 hours plasma osmolality had dropped to 269 mosmol/kg and the patient reported satisfactory relief of his thirst. For the following 4 weeks, plasma osmolality remained between 263 and 270 mosmol/kg (i.e. hyposmolar), mean daily urine output was 2 litres and he no longer complained of thirst.

## Discussion

Diabetes insipidus is a well recognized sequel of head injury. It presents with polydipsia and polyuria and a plasma osmolality which is usually slightly increased. This contrasts with the low plasma osmolality of primary polydipsia. In our patient, despite confirmation of diabetes insipidus plasma osmolality was low.

Antidiuretic therapy, rarely, if ever, leads to significant hyposmolality in patients with diabetes insipidus because their thirst mechanism is intact. On the other hand, in patients with compulsive water drink-

ing, antidiuretic therapy may cause severe hyposmolality because they do not reduce their water intake. Our patient reacted atypically to the antidiuretic therapy in that he was still extremely thirsty when his plasma osmolality was normal. Thirst was completely relieved only when plasma osmolality fell below 270 mosmol/kg. In contrast to patients with psychogenic polydipsia, plasma osmolality never fell below 263 mosmol/kg at which level his thirst was quenched. Thirst is normally absent when plasma osmolality is below 290 mosmol/kg. Therefore, we interpret the disturbance in this patient as being due to abnormal resetting of his thirst osmostat rather than to compulsive water drinking. His thirst sensation did respond to plasma osmolality but at a lower level than normal. We believe that he has a variety of primary polydipsia which has recently been called 'the syndrome of inappropriate thirst'.<sup>4</sup>

The osmoreceptors that control thirst have many properties in common with those regulating the secretion of AVP. Anatomically, the thirst osmoreceptors appear to be located in similar, if not identical areas in the hypothalamus. Clinical and experimental data show that destructive lesions in the hypothalamus can abolish osmoregulation of thirst and/or AVP secretion.<sup>5</sup> It is surprising that despite the well-known association between head injury and diabetes insipidus, no case of diabetes insipidus combined with primary polydipsia to our knowledge, has been so far reported after head injury.

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