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Case Report of a Tri-phasic Response of Diabetes Insipidus in a Child with Optic Pathway Glioma, Following a Haemorrhage within the Lesion

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Introduction

There were cases reported in literature, of the occurrence of tri phasic response of Diabetes Insipidus following surgery in supra sellar region [1-6]. After, trans-sphenoidal surgery, diabetes insipidus is more frequent, occurs during the first post-operative day and resolves in majority of cases within 10 days. Few patients were with persistent diabetes insipidus, hyponatraemia occurs at the end of first post-operative week and resolves in most cases within 5 days [7]. There is one case reported in adult literature, where a pituitary apoplexy precipitated diabetes insipidus, patient subsequently underwent trans sphenoidal pituitary surgery with subtotal resection of this mass. Microscopic evaluation of tumor tissue revealed a pituitary adenoma with evidence of recent infarct and hemorrhage [8] but no such cases were reported in paediatric population. Risk factors for persistent DI include an intraoperative CSF leak, a craniopharyngioma, or a Rathke cleft cyst [9]. There was a physiological model which provides a plausible mechanistic explanation for some varieties of postsurgical water and electrolyte disturbances, in which increasing damage to the pituitary potentiates the likelihood of a full triphasic response. However, there was also evidence which show that merely modifying the level of damage does not produce every presentation of water and electrolyte imbalance [10].

Case Presentation

We now report, a case of an 8½ year old boy with known case of optic pathway glioma, he demonstrated a triphasic response of diabetes insipidus, without there being a surgery. No such case is reported in literature so far. He presented to local hospital with altered sensorium. He was then transferred to our tertiary centre. The initial sodium is 158 mmol/l, which did not improve with fluid bolus. Urine output was around 4 ml/kg/hr. He was then started on DDAVP, urine osmolality prior to DDAVP-334 mosm/l, post DDAVP-955 msosm/l. He then went into phase of SIADH for 5 days, (lowest sodium 134 mosm/l). DDAVP stopped and he is advised to drink fluids at his choice, but did not drink because of hypodipsia. He was then encouraged to drink, target set, although the target was met, the sodium levels went high still (156 mmol/l). Hence, DDAVP was started, Urine was better concentrated post DDAVP (prior to restarting DDAVP-urine osmolality-369 msom/l, post DDAVP-889 mosm/l), sodium levels normalized (136 mmol/l at discharge). MRI of Brain done subsequently, showed an increase in the size of tumour mass, likely haemorrhage within the optic pathway glioma, which had contributed to this triphasic response. Progression of the patient is shown in Table 1.

Discussion

Although SIADH is likely in this case, there is also this possibility that this could be a DDAVP toxicity as there are also evidence that prolonged desmopressin bioactivity may increase the risk of water intoxication [11].

Date	Time	Plasma Osmo (mosm/kg)	Urine Osmo (mosm/kg)	Sodium (mmol/l)	Management/DDAVP regime
20.11.17	22:45			158	Fluid bolus
21.11.17	09:00	336	334	161	0.2 mcg iv (12:00 hrs)
21.11.17	18:00		955		0.2 mcg s/c (23.11-01:24 hours
22.11.17	03:30	326	770	160	75 mcg PO tds (started at 07:00 hours)
23.11.17	07:15		388	161	DDAVP 125 mcg PO tds (from 23:00 hours)
24.11.17	07:00		923	161	Additional dose 25 mcg stat given IV fluids stopped
24.11.17	11:45	290	1068	141	150 mcg stat (15:00 hrs) DDAVP stopped after
25.11.17	07:00	268	1066	134	SIADH likely

27.11.17	06:20		529	136	
29.11.17	05:00	310	330	150	Not drinking by himself, hence target set, encouraging him to drink
30.11.17	04:00	317	369	156	100 mcg tds re-started
30.11.17	17:30	315	885	152	
1.12.17	06:50	306	1132	151	Dose reduced to 75 mcg tds
2.12.17	18:50	282	116	136	Afternoon dose omitted, 50 mcg tds started at 23:00 hrs
3.12.17	03:30	301	665	140	
4.12.17	14:45		827	136	Discharged. DDAVP-50 mcg tds

Table 1: Overall progression of the patient.

Conclusion

Mechanisms that underlie the pathophysiology of the triphasic pattern of post-operative diabetes insipidus could be applicable in this case as well. That is, the first phase of diabetes insipidus is initiated by a partial or complete pituitary stalk section, which severs the connections between the cell bodies of AVP secreting neurons in the hypothalamus and their nerve terminals in posterior pituitary gland, which prevents AVP secretion. The second phase of antidiuresis is caused by uncontrolled release of AVP into the blood stream from the degenerating nerve terminals in posterior pituitary. The third phase of Diabetes insipidus develops, when the AVP secreting neuronal cell bodies in hypothalamus have degenerated. But research needed to confirm the same.

References

1. Das B, Ghosh II (2016) The triphasic response: Water imbalance after neurosurgery: A case report. Neurocritical Care 25.
2. Abla AA, Wait SD, Forbes JA, Pati S, Johnsonbaugh RE, et al. (2011) Syndrome of alternating hypernatremia and hyponatremia after hypothalamic hamartoma surgery. Neurosurg Focus 30: E6.
3. Finken MJ, Zwaveling-Soonawala N, Walenkamp MJ, Vulsma T, van Trotsenburg AS, et al. (2011) Frequent occurrence of the triphasic response (Diabetes Insipidus/Hyponatremia/Diabetes Insipidus) after surgery for craniopharyngioma in childhood. Hormone Research in Paediatrics 76: 22-26.
4. Hoorn EJ, Zietse R (2010) Water balance disorders after neurosurgery: The triphasic response revisited 3: 42-44.
5. Finken MJ (2010) High prevalence of diabetes insipidus/hyponatremia after transcranial surgery for childhood craniopharyngioma. Hormone Research in Paediatrics 74: 71.
6. Pratheesh R, Swallow DMA, Rajaratnam S, Jacob KS, Chacko G, et al. (2013) Incidence, predictors and early post-operative course of diabetes insipidus in paediatric craniopharygioma: A comparison with adults. Childs Nerv Syst 29: 941-949.
7. Kristof RA, Rother M, Neuloh G, Klingmüller D (2009) Incidence, clinical manifestations, and course of water and electrolyte metabolism disturbances following transsphenoidal pituitary adenoma surgery: a prospective observational study. J Neurosurg 111: 555-562.
8. Sweeney AT, Blake MA, Adelman LS, Habeebulla S, Nachtigall LB, et al. (2004) Pituitary apoplexy precipitating diabetes insipidus. Endocr Pract 10: 135-138.
9. Nemergut EC, Zuo Z, Jane JA Jr, Laws ER Jr (2005) Predictors of diabetes insipidus after transsphenoidal surgery: a review of 881 patients. J Neurosurg 103: 448-454.
10. Blair ET, Clemmer JS, Harkey HL, Hester RL, Pruett WA (2017) Physiologic Mechanisms of Water and Electrolyte Disturbances After Transsphenoidal Pituitary Surgery. World Neurosurg 107: 429-436.
11. Dehoorne JL, Raes AM, van Laecke E, Hoebeke P, Vande Walle JG (2006) Desmopressin toxicity due to prolonged half-life in 18 patients with nocturnal enuresis. J Urol 176: 754-757.