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

Sangeetha Pradeep*

Department of Endocrinology, Great Ormond Street Hospital for Children NHS Foundation Trust, Great Ormond St, London WC1N 3JH, UK

*Corresponding author: Sangeetha Pradeep, Department of Endocrinology, Great Ormond Street Hospital for Children NHS Foundation Trust, Great Ormond St, London WC1N 3JH, UK, Tel: 00447738264810; E-mail: sangeetha.pradeep@rocketmail.com

Received Date: Jan 16, 2018; Accepted Date: Feb 08, 2018; Published Date: Feb 19, 2018

Copyright: © 2018 Pradeep S. This is an open-access article distributed under the terms of the Creative Commons Attribution License, which permits unrestricted use, distribution, and reproduction in any medium, provided the original author and source are credited.

Introduction

There were cases reported in literature, of the occurrence of triphasic 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 transsphenoidal 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 mosm/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 mosm/l, post DDAVP-889 mosm/l), sodium levels normalized (136 mosm/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.

Table 1. Management of Diabetes Insipidus

<table>
<thead>
<tr>
<th>Date</th>
<th>Time</th>
<th>Osmo</th>
<th>Sodium (mmol/l)</th>
<th>Management/DDAVP regime</th>
</tr>
</thead>
<tbody>
<tr>
<td>20.11.17</td>
<td>22:45</td>
<td>158</td>
<td></td>
<td>Fluid bolus</td>
</tr>
<tr>
<td>21.11.17</td>
<td>09:00</td>
<td>336</td>
<td>161</td>
<td>0.2 mcg iv (12:00 hrs)</td>
</tr>
<tr>
<td>21.11.17</td>
<td>18:00</td>
<td>955</td>
<td></td>
<td>0.2 mcg s/c (23.11-01:24 hours)</td>
</tr>
<tr>
<td>22.11.17</td>
<td>03:30</td>
<td>326</td>
<td>160</td>
<td>75 mcg PO tds (started at 07.00 hours)</td>
</tr>
<tr>
<td>23.11.17</td>
<td>07:15</td>
<td>388</td>
<td>161</td>
<td>DDAVP 125 mcg PO tds (from 23:00 hours)</td>
</tr>
<tr>
<td>24.11.17</td>
<td>07:00</td>
<td>923</td>
<td>161</td>
<td>Additional dose 25 mcg stat given IV fluids stopped</td>
</tr>
<tr>
<td>24.11.17</td>
<td>11:45</td>
<td>290</td>
<td>141</td>
<td>150 mcg stat (15:00 hrs) DDAVP stopped after</td>
</tr>
<tr>
<td>25.11.17</td>
<td>07:00</td>
<td>268</td>
<td>134</td>
<td>SIADH likely</td>
</tr>
</tbody>
</table>

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].
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