This report emphasizes the importance of monitoring hypothalamic pituitary functions, during the illness and later in the follow-up of central nervous system infection, as the clinical features are often non-specific and easily confused with the post-encephalopathy picture.
spastic hemiparesis, roving eye movement with bilateral optic atrophy.

The patient was eventually transferred to the pediatric ward after two months of presentation. At that time, the patient developed classic DI with a triad of polyuria with urine output more than 6 ml/kg/hr, low urine osmolality and high blood osmolality and serum sodium 160 mg/dl. The child, therefore, did not require water deprivation test. He received a treatment with oral DDAVP. The anterior pituitary function tests at that time were normal.

At the age of 3 years, he started to gain weight; serum thyroid hormone level (T4) was done and it was low (8 pmol/l) and TSH (1.5 mIU/l). We investigated him further for secondary adrenal insufficiency and the results showed low serum cortisol and low ACTH level. So the patient was started on hydrocortisone (6 mg/m²/day) and thyroxin (37.5 mcg/day) replacement therapies. In the last six months his growth velocity was < 25th centile, IGF-1 was low and formal growth hormone stimulation test was planned to be done. MRI pituitary showed an absent bright spot of the posterior pituitary.

**Table 1: Pituitary function tests**

<table>
<thead>
<tr>
<th>Test</th>
<th>T4 (pmol/l)</th>
<th>TSH (mIU/l)</th>
</tr>
</thead>
<tbody>
<tr>
<td>Without treatment</td>
<td>7.720</td>
<td>2.8</td>
</tr>
<tr>
<td>With treatment</td>
<td>9.960</td>
<td>0.14</td>
</tr>
<tr>
<td></td>
<td>8.43</td>
<td>0.66</td>
</tr>
<tr>
<td></td>
<td>8.5</td>
<td>2.6</td>
</tr>
<tr>
<td></td>
<td>17</td>
<td>0.05</td>
</tr>
</tbody>
</table>

* With treatment

**Discussion**

Hypothalamic-pituitary dysfunction can be caused by diverse etiologies. Pituitary tumor, including craniopharyngioma, glioma and germinoma, postoperative and radiotherapy, vasculitis and vascular condition, and head-trauma are the commonest [1-3]. Infectious causes such as mycobacterium tuberculosis and non-mycobacterial agent, such bacteria, as as *streptococcus pneumonia, haemophilus influenzae* and *group B streptococcus*, fungi, spirochetes, viruses, protozoa are not that uncommon and involve the pia matter, arachnoid and cerebrospinal fluid [4-12].

The mechanism of the pituitary dysfunction after acute meningitis is not clearly defined, but the pituitary hormone deficiency pattern is suggestive of an ongoing dynamic process. It is reasonable to assume that the incidence and the pattern of hormone deficiency may vary with the type of positive causative agent, and the localization of brain lesion, as well as with the severity of the disease as evident by hydrocephalus and cerebritis. Tantivierdi et al [14] postulated that, an autoimmune hypothalamic-pituitary process could be triggered by acute meningitis.

In the previous studies, isolated posterior pituitary insufficiency has been reported post infections, as well as isolated corticotrophin deficiency. The insufficiency of hydrocortisone and thyroid hormone might interfere with the water excretion, and hence mask the clinical picture of diabetes insipidus. Also both prospective and retrospective studies and case reports demonstrated a variable degree of hypothalamic-pituitary insufficiency and also showed that either transit or permanent hormonal deficiency may occur [4-21]. Further extended studies needed to clarify this important issue. In our patient’s illness, with its stormy course, we demonstrated a permanent panhypopituitarism: hypothyroidism, hypoadrenalism, growth hormone deficiency and diabetes insipidus. The scenario suggested that the severity of the disease could be reflected on the degree of hormonal deficiency.

**Conclusion**

As the symptoms of pituitary insufficiency post infections are non-specific, and the sequelae are devastating and can be life threatening, screening for hypothalamic-pituitary dysfunction is warranted in those cases. Further studies are required to evaluate the hypothalamic-pituitary access using dynamic tests and
MRI brain imaging by if needed. Further workup of autoimmunity needs to be explored in future cases.

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