Insipid Diabetes and Acute Myeloid Leukemia: Genotypic/Phenotypic Correlation?

Maria Manuel Costa1,2,3, Sandra Belo1,2,3, Pedro Souto1, Davide Carvalho1,2,3

1Department of Endocrinology, Diabetes and Metabolism, Centro Hospitalar São João, Porto, Portugal; 2Faculty of Medicine, University of Porto, Porto, Portugal; 3Instituto de Investigação e Inovação da Saúde da Universidade do Porto

INTRODUCTION

- Central diabetes insipidus (CDI) is a rare complication of Acute Myeloid Leukemia (AML) occurring in less than 0.6% of patients.
- CDI may precede, occur simultaneously or after the diagnosis of AML.
- It is associated with genetic changes in chromosomes 3 and 7 and these alterations are predictors of a poor prognosis.

CASE REPORT

72 anos year-old man

Past Medical History:
Thyroid nodule, Inguinal hernia
No therapy

Study findings suggested AML:
- Analytical Study: Hb 8.8g/dL, leucocytes 13.03x10^9/L, neutrophils 0.87, blasts 4.6%
- Karyotype: 45,XY,inv (3) (q21q26), -7 (20)
- Abdominal ultrasound: hepatomegaly (21.2 cm) and mild splenomegaly (13.5 cm). Hepatic parenchyma with diffuse increase in echogenicity related to abnormal cell infiltration.
- Immunophenotyping: 53% of myeloid blasts, CD34+

He was admitted to the Hematology Department and began chemotherapy

Endocrinology evaluation was requested due to analytical alterations and patient clinical:

- Hypernatremia: 159 mEq/L (135-145)
- Serum osmolality: 332 mOsm/Kg (282-300)
- Urine osmolality in the lower limit of normal: 187 mOsm/kg (50-1200)
- Negative water balance with weight loss and dehydration

- Pituitary CT: Pituitary with normal morphology and dimensions, although a low uptake area in the median/right paramedian region was assumed

He was discharged with oral desmopressin 0.06 mg twice a day

CONCLUSIONS

- In this case the symptoms of diabetes insipidus led to the diagnosis of AML.
- There are descriptions in the literature that these cytogenetic changes are associated with the development of DCI in AML, although the causes of this association are not fully understood.

References:

Table 1: Evolution of the patient

<table>
<thead>
<tr>
<th></th>
<th>3.08.2015</th>
<th>5.08.2015</th>
<th>6.08.2015</th>
<th>7.08.2015</th>
</tr>
</thead>
<tbody>
<tr>
<td>Na (135-145 mEq/L)</td>
<td>159</td>
<td>149</td>
<td>147</td>
<td>142</td>
</tr>
<tr>
<td>Serum Osm (50-1200 mOsm/kg)</td>
<td>288</td>
<td>288</td>
<td>288</td>
<td>288</td>
</tr>
<tr>
<td>Water Balance</td>
<td>Negative</td>
<td>Negative</td>
<td>Positive</td>
<td>Positive</td>
</tr>
<tr>
<td>Resolution of hypotremia, polydipsia and polyuria</td>
<td></td>
<td></td>
<td></td>
<td></td>
</tr>
</tbody>
</table>

As the patient did not show response to induction chemotherapy, he started salvage chemotherapy.

Given the patient’s clinical context, we decided not to conduct water restriction test and pituitary MRI was also delayed.