Case Study of the Month

Complete Response to the Combination Therapy with Androgen Blockade and Somatostatin Analogue in a Patient with Advanced Prostate Cancer: Magnetic Resonance Imaging with 1H-Spectroscopy

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1. Case history

In October 2005, a 74-yr-old man was diagnosed at prostate biopsy as having prostatic adenocarcinoma with a Gleason score 7 (4 + 3); his serum prostate-specific antigen (PSA) level was 680 ng/ml and the determination of serum chromogranin A (CgA, 121 ng/ml; normal, <90 ng/ml) suggested neuroendocrine (NE) activity. Performance status (Eastern Cooperative Oncology Group) score was 3 and the bone pain and analgesic requirement score was 3. A bone scan showed several areas of diffuse skeletal metastases (Fig. 1A). In October 2005, the patient had a magnetic resonance 1H-spectroscopic imaging (1H-MRSI) of the prostate (Fig. 2A). The 1H-MRSI showed an area of low signal intensity (1.8 cm)
involving the left lobe of the prostate and infiltrating the left seminal vesicle (Fig. 2A). At spectroscopy, in this area the metabolic ratio modification was highly suggestive for neoplastic tissue (choline + creatine/citrate > 1; Fig. 2A). Considering that the patient was at first treatment and on the basis of a possible NE activation at the prostate adenocarcinoma level, he underwent (November 2005) combination therapy using complete androgen blockade (CAB; leuprolin acetate 3.75 mg every 4 wk plus bicalutamide 50 mg daily) and a somatostatin analogue (lanreotide acetate 60 mg every 4 wk). After 2 mo of combination therapy (January 2006), PSA and CgA levels were 0.09 ng/ml and 82 ng/ml, respectively. At follow-up, the reduction of serum PSA and CgA levels progressively continued (PSA nadir = 0.05 ng/ml at 6 mo; CgA nadir = 9 ng/ml at 12 mo; Table 1). After 6 mo of therapy (May 2006),

Table 1 – Serum PSA and CgA variations at different intervals of follow-up during therapy

<table>
<thead>
<tr>
<th></th>
<th>Baseline</th>
<th>1-mo FU</th>
<th>2-mo FU</th>
<th>3-mo FU</th>
<th>6-mo FU</th>
<th>12-mo FU</th>
</tr>
</thead>
<tbody>
<tr>
<td>PSA, ng/ml</td>
<td>680</td>
<td>102</td>
<td>0.09</td>
<td>0.07</td>
<td>0.05</td>
<td>0.05</td>
</tr>
<tr>
<td>CgA, ng/ml</td>
<td>121</td>
<td>106</td>
<td>82</td>
<td>70</td>
<td>48</td>
<td>9</td>
</tr>
</tbody>
</table>

FU = follow-up; PSA = prostate specific antigen; CgA = chromogranin A.

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an objective complete response to therapy was demonstrated by a normal PSA level (PSA = 0.05 ng/ml) and bone scan imaging (Fig. 1B) response. At the 12-mo follow-up (November 2006) the normal serum markers (PSA = 0.05 ng/ml, CgA = 9 ng/ml) were accompanied by a 1H-MRSI response (Fig. 2B). In particular, the normal choline plus creatine-to-citrate ratio (<0.2) at 1H-MRSI confirmed a complete response to the combination therapy at the prostate level. The clinical response was associated with concomitant normal ECOG (1 score) and bone pain scores (0 score), beginning at 2 mo of follow-up. Therefore, after 12 mo of follow-up the patient is alive without disease progression and with a complete objective and symptomatic response.

Conflicts of interest

Authors disclose any commercial relationship such as: consultancies, stock ownership or other equity interests, patents received and/or pending, or any commercial relationship which might be in any way considered related to the submitted article.

EU-ACME question

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Question:

Neuroendocrine differentiation could be considered one of the factors related to the progression of prostate adenocarcinoma in hormone-refractory disease. How has it been proposed to manage this neuroendocrine hyperactivation in prostate cancer?
A. To continue complete androgen blockade (CAB).
B. To discontinue androgen-deprivation therapy and start chemotherapy.
C. To discontinue androgen-deprivation therapy and start somatostatin analogues.
D. To add a somatostatin analogue to androgen-deprivation therapies.