Case report

Splenic histiocyte-rich pseudotumor following chemotherapy for non Hodgkin diffuse large B cell lymphoma

A.G. ABDOU, M. KANDIL, M.S. ELDIEN, R. ABDALLAH
Pathology Department, Faculty of Medicine, Menofiya University, Shebein Elkom, Egypt

Key words
Spleen • Postchemotherapy • Xanthomatous pseudotumor

Summary
Chemotherapy may induce mass lesion in rare conditions, which can be easily mistaken as a residual tumor mass. In this report, we describe a mass affecting spleen in a patient received chemotherapy for non Hodgkin diffuse large B cell lymphoma. This mass proved histologically to be non neoplastic formed of sheets of histiocytes and xanthoma cells, which is called histiocyte-rich pseudotumor. This report describes this rare lesion and the possible differential diagnosis.

Introduction
Histopathological response to chemotherapeutic agents is manifested by several changes including induction of necrosis. The extent of both induced necrosis and residual viable tumor cells determine the efficiency of the chemotherapeutic regimen. Extensive necrosis and absence of viable tumor cells are indicators of complete response and other than this, is considered as a partial or no response. Rarely, chemotherapy could induce mass lesion, which may be easily mistaken as a residual tumor mass. Several reports showed mass lesions after chemotherapy as in breast, intestine, mediastinum and spleen (Table I).

<table>
<thead>
<tr>
<th>Author</th>
<th>Site</th>
<th>Age/y</th>
<th>Sex</th>
<th>Type of original tumor</th>
<th>Type of chemotherapy</th>
</tr>
</thead>
<tbody>
<tr>
<td>Chandra et al (1)</td>
<td>Spleen</td>
<td>74</td>
<td>Male</td>
<td>DLBCL</td>
<td>R-CHOP</td>
</tr>
<tr>
<td>Cases 2</td>
<td>Spleen</td>
<td>67</td>
<td>Male</td>
<td>DLBCL</td>
<td>CHOP</td>
</tr>
<tr>
<td>Ashfaq et al, (5)</td>
<td>Small intestine</td>
<td>9</td>
<td>Male</td>
<td>Burkitt lymphoma</td>
<td></td>
</tr>
<tr>
<td>Tan et al, (4)</td>
<td>Breast</td>
<td>46</td>
<td>Female</td>
<td>high-grade invasive ductal carcinoma</td>
<td>Neoadjuvant (doxorubicin 60 mg/m² and cyclophosphamide 600 mg/m.</td>
</tr>
<tr>
<td>Ford et al, (7)</td>
<td>Spleen</td>
<td>58</td>
<td>Male</td>
<td>Burkitt</td>
<td>MaGrath regimen</td>
</tr>
<tr>
<td>Cases 2</td>
<td>Spleen</td>
<td>45</td>
<td>Male</td>
<td>DLBCL</td>
<td>R-CHOP</td>
</tr>
<tr>
<td>Otto et al, (6)</td>
<td>Mediastinum</td>
<td>15</td>
<td>Female</td>
<td>DLBCL</td>
<td>FAB/LMB-96 regimen with radiation</td>
</tr>
<tr>
<td>The present case</td>
<td>Spleen</td>
<td>51</td>
<td>Male</td>
<td>DLBCL</td>
<td>CHOP</td>
</tr>
</tbody>
</table>

Correspondence
Asmaa Gaber Abdou, Department of Pathology, Faculty of medicine, Menofiya University, Shebein Elkom, Egypt - Tel. (002) 048 2282939 - Fax:(002) 0482233521 - E-mail: Asmaa_elsaidy@yahoo.com
Case report

This case is for a male patient 51 years old presented with a single yellowish splenic focal lesion. The patient had a history of non hodgkin diffuse large B cell lymphoma, and had completed the course of chemotherapy. Clinically, there is a great suspicion of lymphoma of this splenic mass. Microscopic examination of the mass revealed multiple nodules with central necrosis and cholesterol crystals formation (Fig. 1 A, B and C), which were surrounded by sheets of xanthoma cells (Fig. 1 D) and foamy macrophages (Fig. 2 A and B). Areas of dystrophic calcification were also seen (Fig. 2 C). The infiltrate was histiocytic in nature proved by diffuse CD68 immunoreactivity (Fig. 2 D). The necrotic cells were highlighted by CD20 (Fig. 3), however, no viable lymphoma cells were recognized. The histiocytic cells were negative for CD1a.

Discussion

In the present case, the lesion appeared yellowish grossly similar to that described in the reported case for breast carcinoma. Histologically, the lesion is formed of sheets of xanthoma cells and lipidized macrophages, which give the lesion the yellow color. According to Chandra et al, 2009, they explained the formation of this lesion by recruitment of monocytes by chemokines released from necrotic tissue, which will then be activated into macrophages with increasing phagocytic lysosomal activity, especially in a site like spleen. Accentuation of lipidized macrophages or xanthoma cells is due to engulfment of membranous debris due to extensive cell lysis in the process of necrosis. This picture is enhanced in spleen because of its rich vascular blood supply and it is a major repository of phagocytic cells. According to our knowledge, only two reports demonstrated the presence of histiocytic rich pseudotumor in spleen. According to the latter authors, they preferred to call this lesion as a postchemotherapy histiocyte-rich pseudotumor rather than xanthomatous pseudotumor. The main differential diagnosis at the clinical level is residual lymphoma. The present case showed absence of viable lymphoma cells excluding residual tumor mass. At the microscopic level, the presence of these sheets of histiocytes in the spleen arouse several diagnostic
categories that included storage disease, histiocytic sarcoma and langerhans’ cell histiocytosis. Storage disease is usually characterized by certain manifestations such as hepatosplenomegally in Gaucher disease for example, but our patient is presented with focal splenic lesion and not diffuse enlargement and the history does not support storage disease. Histiocytic sarcoma is a rare malignant disease, which may involve spleen, the neoplastic histiocytic cells in this neoplasm exhibit atypical features and mitoses, which were not seen in our case. Furthermore, this disease is an aggressive one with fatal outcome, while our patient is still alive after splenectomy. With regards to langerhans’ cell histiocytosis, the histiocytes are characterized by grooved nuclei and the expression of CD1a together with the presence of eosinophils, which were not seen in our case. Finally, our case demonstrates a very rare splenic mass induced by previous chemotherapy for non Hodgkin, which necessitates careful histological examination for exclusion of residual tumor.

References


