Sir,

Antiphospholipid antibodies (APA) are a heterogeneous group of immunoglobulins directed against negatively charged phospholipid, protein-phospholipid complexes, or plasma glycoproteins such as β2-glycoprotein I and include anticardiolipin (ACA) and lupus anticoagulant (LAC) antibodies.

The presence of these antibodies has been associated with the clinical features of the so-called antiphospholipid syndrome (APS), which includes arterial and venous thrombosis, recurrent fetal loss, and thrombocytopenia.1 Prospective studies published by Schved et al.2 and Finazzi et al.,3,4 have shown that hematologic malignancies can develop during follow-up of patients with APA.

A 36-year-old woman with known APS was admitted to our department because of evidence of asymptomatic mediastinal widening and pleural effusion.

Needle biopsy with CT scan of the bulky mediastinal mass disclosed a diffuse large B-cell lymphoma. Staging failed to show other lymphoma localization. Laboratory data were normal except for ESR 38, LAD 674 U/L, copper 25.5 mmol/L, while Coombs’ test and antinuclear antibodies diffuse type (titer 1/320) were positive.

The results of coagulation tests, kaolin clotting time (KCT), lupus anticoagulant Russell’s venom viper time (RVVT), platelet neutralization procedures (PNP) and neutralization with hexagonal phase phosphatidyl ethanolamine test (PE) are shown in Table 1. The patient was treated with MACOP-B followed by mantle-field radiotherapy (39.6 Gy in 22 fractions) obtaining a complete remission. Previously altered laboratory data normalized except for the antinuclear antibodies (positive at the same titer) and coagulation tests (KCT, RVVT, PNP, PE).

An association between APA and hematologic malignancies has been described rarely, even though a prospective Italian study5 revealed that the principal cause of mortality and morbidity in patients with idiopathic APA is lymphoproliferative diseases.2,5,6 Larger studies are necessary to confirm the risk of evolution to hematologic malignancies in patients positive for APA and to clarify the prognostic value of persistent positivity for APA in patients treated for lymphoproliferative disorders.

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Key words
Antiphospholipid syndrome, non Hodgkin’s lymphoma, chemotherapy, immunosuppression

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Table 1. Coagulation tests before and after polychemotherapy.

<table>
<thead>
<tr>
<th>Test</th>
<th>Before polyCT</th>
<th>After polyCT</th>
<th>Normal values</th>
</tr>
</thead>
<tbody>
<tr>
<td>APTT</td>
<td>48 sec</td>
<td>60 sec (on warfarin)</td>
<td>30-40 sec</td>
</tr>
<tr>
<td>APTT diluted</td>
<td>141 sec</td>
<td>130 sec</td>
<td>32-43 sec</td>
</tr>
<tr>
<td>KCT</td>
<td>165 sec</td>
<td>163 sec</td>
<td>60-110 sec</td>
</tr>
<tr>
<td>PNP and PE</td>
<td>positive</td>
<td>positive</td>
<td>negative</td>
</tr>
<tr>
<td>APA</td>
<td>5 UGPL/mL</td>
<td>7.3 UGPL/mL</td>
<td>&lt;5 UGPL/mL</td>
</tr>
<tr>
<td>ACA-IgG</td>
<td>32 UGPL</td>
<td>33 UGPL</td>
<td>&lt;5 UGPL/mL</td>
</tr>
<tr>
<td>ACA-IgM</td>
<td>negative</td>
<td>negative</td>
<td>&lt;5 UGPL/mL</td>
</tr>
</tbody>
</table>

CT = chemotherapy.

the lymphoproliferative disease does not originate from the same lymphoid subset producing APA even though, likely, from the same immune dysregulation causing the APS. This last possibility could explain the different responsiveness to the therapy. Nevertheless, if APA is a risk factor for lymphoproliferative disease, our patient should be at greater risk of relapse.

Larger studies are necessary to confirm the risk of evolution to hematologic malignancies in patients positive for APA and to clarify the prognostic value of persistent positivity for APA in patients treated for lymphoproliferative disorders.

References
New technology and changing parameters of leukapheresis for blood cell transplantation

Sir,

Clinical investigators have recently developed an innovative technique of leukapheresis (LK) referred to as AutoPBSC System. This technique offers the following advantages: a) better collection efficiency of CD34+ hematopoietic progenitor cells; b) higher quality of collection; c) higher yield of platelets; d) higher collection volume; and e) automation. These advantages prompted us to evaluate the effectiveness of the AutoPBSC System and to extend classic parameters for starting LK, i.e., CD34+ cells ≥ 20/µL and platelets ≥ 30 × 10^3/µL, also to poor-mobilizer and/or thrombocytopenic patients. We confirm the advantages of the AutoPBSC System and demonstrate that efficient LK can successfully be performed also in these categories of patients.

Ninety-six leukaphereses were carried out in 65 consecutive patients undergoing BCT for treatment of poor prognosis malignancies (13 multiple myeloma, 12 breast cancer, 8 Ewing's sarcoma family of tumors, 9 non-Hodgkin's lymphoma, 7 Hodgkin's disease, 6 ovarian cancer, 3 rhabdomyosarcoma, 3 desmoplastic small cell tumor, 1 Wilms' tumor, 2 non-small cell lung cancer, 1 yolk sac tumor). The LK procedure implied processing 2.5-fold the individual's blood volume and adaptation of the AutoPBSC software to the specific patient's condition, 5) it yielded mean 1.9 × 10^6 CD34+ cells/kg, median 2.6, range 0.8-14.6 × 10^6; and in thrombocytopenic and poor-mobilizer patients (n = 5) it yielded mean 1.9 × 10^6 CD34+ cells/kg, median 1.8, range 0.8-3.1 × 10^6. Although platelet depletion in thrombocytopenic patients was negligible, a prophylactic platelet transfusion was given after LK to 3 patients.

Results of a single leukapheresis presented here compare favorably with those previously attained with other techniques and confirm for the first time the advantages of the AutoPBSC System in poor-mobilizer and thrombocytopenic patients as well, thus facilitating the clinical application of blood cell transplantation.

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Key words

Leukapheresis, blood cell transplantation, CD34+ cells

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