Antibodies. Clinical characteristics such as fulfillment of three criteria items in patients with idiopathic thrombocytopenic purpura (ITP). However, we have two major questions on this article. First, they defined anti-phospholipid syndrome (APS) by adopting thrombocytopenia as one of the clinical features. Therefore, “Group A” patients in this study should be diagnosed as having APS at the entry of their study. Their statement, “Three patients in Group A were diagnosed later to have APS because they had cerebral thrombosis or habitual abortion.” is confusing. Moreover, it is still controversial that thrombocytopenia can be suitable for one of the criteria items of APS (2). Second, they did not show any serological tests for syphilis (BFP-STS) and anti-nuclear antibodies (ANA) were found in ITP patients with IgG anti-cardiolipin-β2-GPI complex antibodies, which are generally referred to as anti-cardiolipin-β2-GPI complex antibodies. These clinical features were compared between group (a) and group (b) patients (Table 1). These features in our group (a) patients can be included in the concept of lupus-like disease by Asherson and Cervera (7). Therefore, the diagnosis of ITP should be carefully made to exclude definite SLE and primary APS in those who do not have any underlying diseases. It is suggested that thrombocytopenia should not be listed in the criteria items of APS, and that both thrombosis and intrathecal fetal death are important in the clinical characteristics of APS. Also, these results are compatible with our previous reports indicating that anti-phospholipid antibodies in patients with APS who have a history of thrombosis or pregnancy loss. On the other hand, some patients with idiopathic thrombocytopenic purpura (ITP) may have aPL in the circulation and the turn-over of platelets may be shorter than unstimulated thrombocytes. Chronic thrombocytopenia associated with aPL is heterogeneous (5). aPL may function like anti-thrombocyte antibodies and immunological thrombocytopenia may ensue. Platelets may be constantly activated by aPL in the circulation and the turn-over of platelets may be shorter than unstimulated thrombocytes. Chronic thrombocytopenia caused by aPL can increase the consumption of platelets, which can also lead to secondary thrombocytopenia. In patients with SLE, a more heterogeneous origin of thrombocytopenia, such as bone marrow dysfunction or suppression, haemophagocytic syndrome, and thrombotic thrombocytopenic purpura, can be present. In some cases, thrombocytopenia is life-threatening while in other cases it is mild. Some of those mechanisms can co-exist. Heterogeneity must be taken account when treating patients with thrombocytopenia and aPL.

Table 1. Clinical Characteristics of Idiopathic Thrombocytopenic Purpura (ITP) Patients with IgG Anti-cardiolipin-β2-GPI Complex Antibodies

<table>
<thead>
<tr>
<th></th>
<th>n</th>
<th>Fulfillment of Three Criteria Items</th>
<th>BFP-STS</th>
<th>ANA</th>
</tr>
</thead>
<tbody>
<tr>
<td>Group a</td>
<td>6</td>
<td>3 (50%)</td>
<td>3 (50%)</td>
<td>5 (83%)</td>
</tr>
<tr>
<td>Group b</td>
<td>77</td>
<td>4 (5%)</td>
<td>2 (3%)</td>
<td>24 (31%)</td>
</tr>
</tbody>
</table>

P < 0.005 < 0.005 < 0.05

Group a consisted of ITP patients with IgG anti-cardiolipin-β2-GPI complex antibodies. Group b included ITP patients who did not have IgG anti-cardiolipin-β2-GPI complex antibodies.

References