A clinical syndrome of ‘thrombocytopenic pur-pura’ has become recognized as a manifestation of HIV infection [8]; however, the mechanism of platelet destruction is still controversial. Several reports show evidence of platelet autoantibodies in drug addicts [6], whereas the nonspecific influence of immunocomplexes has been demonstrated in homosexuals [7]. Only few reports are available on the occurrence of thrombocytopenia in hemophiliacs with HIV infection [1, 3, 4]. The diagnosis of immune thrombocytopenia in these cases has been based on an increased number of megakaryocytes, short platelet life-span and a high level of platelet-associated IgG (PAIgG). However, liver damage and secondary spleen enlargement are common features in hemophiliacs who are exposed to viral infections. Furthermore, secondary hypergammaglobulinemia [3] and immunocomplexes [4] may also account for thrombocytopenia besides an autoimmune mechanism. Immunocomplexes are detected in a large percentage of patients with AIDS or ARC who also present the rheumatoid factor, antinuclear and anti-cardiolipin antibodies [9].

We have investigated the serum-bindable platelet IgG (SPB IgG), platelet-associated IgG (PAIgG), circulating immunocomplexes (CIC) and serum IgG in 16 patients with severe hemophilia (15 hemophilia A, 1 hemophilia B) and mild thrombocytopenia (below 200 × 10^9/1). All patients had been infected by hepatitis B and 8 patients had HIV seropositivity (Western-blot). The staging of HIV infection was carried out according to the Walter-Reed classification [5]: 2 patients were in stage 1, 2 in stage 2, 2 in stage 3, 1 in stage 4 and 1 in stage 6 (full-blown AIDS). SPB IgG and PAIgG were performed by ELISA [2] and CIC as C1Q consumption. The results are summarized as follows:

<table>
<thead>
<tr>
<th>HIV+ (8 cases)</th>
<th>HIV- (8 cases)</th>
<th>Normal values</th>
</tr>
</thead>
<tbody>
<tr>
<td>Platelet count, × 10^11</td>
<td>143 ± 31</td>
<td>148 ± 30</td>
</tr>
<tr>
<td>SPB IgG</td>
<td>all positives* 1 out of 8 positive negatives</td>
<td></td>
</tr>
<tr>
<td>PAIgG, fg/platelet</td>
<td>0.8 ± 0.6</td>
<td>1.8 ± 1.4</td>
</tr>
<tr>
<td>CIC, µg/ml</td>
<td>5.7 ± 4.4*</td>
<td>2 ± 0.9 &lt; 2</td>
</tr>
<tr>
<td>Serum IgG g/l</td>
<td>134 ± 16*</td>
<td>14 ± 5 &lt; 15</td>
</tr>
</tbody>
</table>

Values are means ± SD.

* Statistical significance (p < 0.05) between HIV+ and HIV- hemophiliacs.
There is no evidence of HIV-related immune thrombocytopenia in our patients. Homosexuals with HIV-related immune thrombocytopenia had platelet-bindable immunocomplexes, high level of PAIgG and severe thrombocytopenia [7, 8]. Our HIV+ hemophiliacs had mild thrombocytopenia but the platelet count was not statistically different from HIV- ones. Nevertheless, HIV+ subjects had the presence of CIC, high serum IgG and SPB1gG with normal amounts of PAIgG. In conclusion, our study confirms the appearance of CIC in HIV+ hemophiliacs. Its role in the occurrence of severe thrombocytopenia should be evaluated in a long-term follow-up.

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References


