Thrombocytopenic Effect of Ethanol Infusion

By Robert M. Post and Jane F. Desforges

Transient depression of the platelet count with temporary sequestration of platelets for several hours occurs with a variety of agents including adenosine diphosphate infusion, hypothermia and blood transfusion. Many other agents are capable of causing platelet sequestration under experimental conditions. Ethanol given intravenously was shown to result in transient lowering of the platelet count of a patient who presented initially with thrombocytopenic purpura following excessive alcoholic intake. The case report and special studies are presented with speculations as to mechanisms and their possible significance.

Case Report, Materials and Methods

The patient was a forty-year-old white woman with a fifteen-year history of chronic alcoholism. On three occasions, she had been admitted to the hospital with thrombocytopenia and purpura following several months of very heavy drinking. She had none of the stigmata of chronic liver disease, and her spleen was never palpable or enlarged on abdominal x-ray examinations. Hemoglobin, leukocyte count and differential count were consistently normal except when gastrointestinal bleeding and pulmonary infection were present. (Table 1). Bone marrow aspiration on two occasions showed numerous megakaryocytes and normoblastic maturation of erythroid and myeloid elements. L.E. Preparations and antinuclear antibody tests were repeatedly negative. There was slight elevation of the SCOT, but all other liver function tests were normal. Remission was spontaneous on two occasions after withdrawal of ethanol, and on one occasion she was given a short course of prednisone.

On three separate occasions, 2,000 ml. of five percent ethanol in a five percent dextrose solution were administered intravenously at a rate of 11 ml per minute and serial platelet counts were done using phase contrast microscopy (Table 2). In one of these studies, the patient's platelets were labeled with Cr51 and reinfused and platelet radioactivity was measured in a well-type scintillation counter. Precordial, hepatic and splenic radioactivity were estimated by surface scanning.

Results

Intravenous infusion of ethanol resulted in a fall in platelet count beginning four to six hours after the start of the infusion. The lowest platelet counts were noted at five to seven hours and were 10 percent, 25 percent and 40 percent respectively, of the pre-infusion level. Platelet radioactivity paralleled the plate-
Table 1.—Hematological Data

<table>
<thead>
<tr>
<th></th>
<th>Hct.</th>
<th>WBC</th>
<th>Platelet Count</th>
<th>Resolution Time (Days)</th>
</tr>
</thead>
<tbody>
<tr>
<td>1st Admission</td>
<td>41</td>
<td>4700</td>
<td>32,000</td>
<td>7</td>
</tr>
<tr>
<td>2nd Admission</td>
<td>41</td>
<td>7550</td>
<td>40,000</td>
<td>4</td>
</tr>
<tr>
<td>3rd Admission</td>
<td>35</td>
<td>6800</td>
<td>Decreased on smear*</td>
<td>6</td>
</tr>
</tbody>
</table>

*1st platelet count done 3 days after admission — 100,000 with subsequent rise to 248,000 in 3 days.

Table 2.—Response of Platelet Count to Ethanol Infusion Platelet Counts ($\times 10^3$)

<table>
<thead>
<tr>
<th></th>
<th>Pre-Infusion</th>
<th>4 hrs.</th>
<th>5 hrs.</th>
<th>6 hrs.</th>
<th>7 hrs.</th>
<th>10 hrs.</th>
<th>24 hrs.</th>
</tr>
</thead>
<tbody>
<tr>
<td>1st Infusion</td>
<td>210</td>
<td>152</td>
<td>96</td>
<td>22</td>
<td>48</td>
<td>68</td>
<td>220</td>
</tr>
<tr>
<td>2nd Infusion</td>
<td>242</td>
<td>250</td>
<td>148</td>
<td>100</td>
<td>62</td>
<td>95</td>
<td>243</td>
</tr>
<tr>
<td>3rd Infusion</td>
<td>258</td>
<td>229</td>
<td>173</td>
<td>122</td>
<td>102</td>
<td>184</td>
<td>264</td>
</tr>
</tbody>
</table>

Fig. 1.—Concomitant change in platelet count and Cr$^{51}$ labeled platelets following ethanol infusion.

Fig. 1.—Concomitant change in platelet count and Cr$^{51}$ labeled platelets following ethanol infusion. There was no change in hepatic or splenic radioactivity throughout the experiment. During the thrombocytopenia, the platelets appeared swollen when examined with phase microscopy. The morphology returned to normal as the platelet count rose.
The mechanism and location of the platelet sequestration in this case are unknown. Intravascular thrombosis is unlikely because of the reappearance of intact labeled platelets in the circulation. Intravenous infusion of adenosine diphosphate causes aggregation and transient splenic sequestration of platelets. However, surface monitoring during ethanol infusion with Cr\(^{51}\) labeled platelets present showed no increase in hepatic or splenic activity when the platelet count fell. Other possible sites are intramedullary or diffuse intravascular sequestration.

Ethanol affects several metabolic pathways which may influence circulating platelets. Transient alteration in the pathway of serotonin degradation has been demonstrated after a test dose of ethanol, and the time of onset and duration of this effect are similar to that of the thrombocytopenic effect of ethanol observed in this patient. Epinephrine metabolism is also affected by ethanol. Both of these catechol amines aggregate platelets in vitro. It is likely that other pathways are similarly affected and that a metabolic alteration or alterations were responsible for changes in the platelet membrane leading to temporary aggregation and sequestration. In support of this is the swelling of the platelets noted during the thrombocytopenia. This is similar to the morphologic changes prior to the aggregation of platelets exposed to adenosine diphosphate.

There is no proof of a relationship between clinical thrombocytopenia and the thrombocytopenia following the experimental ethanol infusion. In fact, transient platelet sequestration does not appear at present to have any clinical significance. However, the pathologic effects of alcohol, direct or indirect, are extensive and vary markedly even among individuals with similar exposure. The data gained from this patient suggest that prolonged exposure to ethanol in a sensitive individual might affect platelet lifespan in the circulation.

**SUMMARY**

Transient thrombocytopenia was induced by ethanol infusion in an alcoholic patient with transient thrombocytopenia. A fall in activity of Cr\(^{51}\) labeled platelets paralleled the fall in platelet count, but no hepatic or splenic sequestration could be demonstrated. It is postulated that ethanol produced a metabolic alteration in the platelets which led to aggregation and temporary sequestration at an unknown site.

**SUMMARIO IN INTERLINGUA**

In un patiente de alcoholismo con thrombocytopenia transiente, il esseva trovate que iste thrombocytopenia poteva esser inducite per medio de infusiones de ethanol. Un declino in le activitate de plachettas marcate con Cr\(^{51}\) esseva in parallela con le declino del numeratio thrombocytic, sed un sequestration hepatic o splenic non poteva esser demonstrate. Es postulate que ethanol produciva un alteration metabolic in le plachettas e que iste alteration causava lor accumulation e sequestration in un sito no identificate.

**ACKNOWLEDGMENTS**

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REFERENCES


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