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Hemoperfusion leads to impairment in hemostasis and coagulation process in patients with acute pesticide intoxication

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Objectives: Hemoperfusion (HP) is one of the important treatment modalities in extracorporeal therapy for patients with acute intoxication. Its use has declined during the past 20 years despite its efficacy, because of its side effects, especially an increased risk of bleeding. Mechanisms of hemostasis impairment have not been clearly elucidated, and studies demonstrating the mechanism are lacking.

Methods: Acute pesticide intoxicated patients who underwent HP were participated. The changes in complete blood counts, platelet function markers, and anticoagulation profiles were measured. The changes in platelet shape were observed by scanning electron microscopy (SEM). After exposure to activated charcoal, changes of platelet glycoprotein expression on the platelet surface were evaluated using flow cytometry *in vitro*.

Results: After HP, the platelet count decreased rapidly during the first 30 minutes from 242.4 ± 57.7×10^3/μL to 184.8 ± 49.6 ×10^3/μL, then gradually decreased even lower to 145.4 ± 61.2 ×10^3/μL over time ($p < 0.001$). Among representative markers of platelet activation, platelet distribution width increased significantly during HP; however, mean platelet volume did not show significant change, nor delayed closure time measured using PFA-100 test suggested that platelet dysfunction occurred during HP. Although these conflicting results, activated platelets adhered to activated charcoal were observed in SEM.
Platelet expression of CD61, fibrinogen receptor, significantly decreased, while those expressing CD42b, von Willebrand factor receptor, did not show significant change; however, platelet expression of CD49, collagen receptor, significantly increased. Thrombin-antithrombin complex, a marker for thrombin generation, appeared to decrease, however, it was not statistically significant. Fibrin degradation products and d-dimers, markers for fibrinolysis, increased significantly during HP.

Conclusions: Our data suggests that hemoperfusion leads to impairment of platelet aggregation with incomplete platelet activation, which was associated with reduced thrombin generation, accompanied by increased fibrinolysis.