EFFECT OF SULFONPYRAZONE ON PLATELET LOSS INDUCED IN VITRO BY ACTIVATED CHARCOAL

H. Yamazaki, T. Notohira, N. Sonoda and N. Miyagawa. The Tokyo Metropolitan Institute of Medical Science, Tokyo, Japan.

Substantial clinical evidence indicates that large doses of estrogen frequently result in thrombotic disorders. Effects of estrogen on platelet aggregability were examined in women with uterine myomas before and after oophorectomy. Bilateral oophorectomy on 15 cases (48-74.74 yrs), unilaterial oophorectomy on 18 cases (control group: 22.0±1.8 yrs) were performed with myomectomy of the uterus. One day before and one day, one week and one month after the operation, platelet counts by Coulter counter, platelet volume by Coulter channelizer and platelet aggregability by biaxial aggregometer were measured. 24 hrs total estrogen in urine was also determined. In the control group, platelet counts were 85.1±4.5% of the preoperated value one day after, 127.7±9.0% one week after and 108.4±7.6% one month after. In the unilaterial oophorectomy group, these were 82.4±5.2% one day after, 124.0±5.7% one week after and 96.1±4.8% one month after. Both the groups showed the same change. Platelet aggregability by 3 URF ADP were 76.0±14.3% one day after, 203.0±7.2% one week after and 193.4±59.0% one month after in the control, while 55.0±13.6% one day after, 102.5±12.9% one week after and 60.6±14.7% one month after in the operation group. There was a statistically significant difference in the values obtained one month after the operation between the groups (p<0.05). Characteristic changes in platelet volumes were also observed. A significant correlation was observed between the platelet aggregability and the daily urinary estrogen excretion levels. The above results suggest that estrogen may enhance platelet aggregability in vivo.

DECREASE IN PLATELET AGGREGABILITY AFTER TOTAL OOPHORECTOMY. N. Sonoda, T. Notohira, N. Miyagawa. The Tokyo Metropolitan Institute of Medical Science, Tokyo, Japan.

We recently examined a possible role and extent of involvement of plasmodium falciparum parasitemia in our earlier report of significantly lower circulating platelet numbers in healthy adult Nigerians (100-200x10^9/L) compared with matched Caesians (350-1400x10^9/L) (P<0.001), and in view of recently reported association of plasmodium parasitemia with thrombocytopenia, cytotoxic changes (TII) and other hemorrhagic syndromes. Ninety-eight febrile children aged between 6 months and 10 years whose fever was attributed to malaria parasitemia only were admitted into the study. Platelet count, platelet aggregation, Factor VIII and FVIII were determined on each. A sample which was collected before treatment was started. Tests were repeated 10-14 days later. It was found that the mean platelet count of 320,000/g, 604×10^9/L (1.29) during illness and immediately after treatment was significantly lower than the count of 254, 490×10^9/L in healthy control subjects (1.6, 506) (P<0.001). Significant thrombocytopenia (Platelets 70,000/μl) was observed only in 5% of the subjects and none had any hemorrhagic syndromes. The effect was independent of age or degree of parasitemia. Pre- and post-treatment leucocyte counts were similarly different (792±200 vs. 522±179; t = -2.61; P<0.05). Hematocrit values did not change significantly. However, in the response to platelet aggregating agents, it was found that the second wave of ADP-induced aggregation was regularly abolished during the parasitemia.

It is concluded that in hyperendemic plasmodium falciparum environment, the malaria may often lead to mild depression of platelet count but that severe thrombocytopenia is uncommon. The platelets may not be optionally functional.