

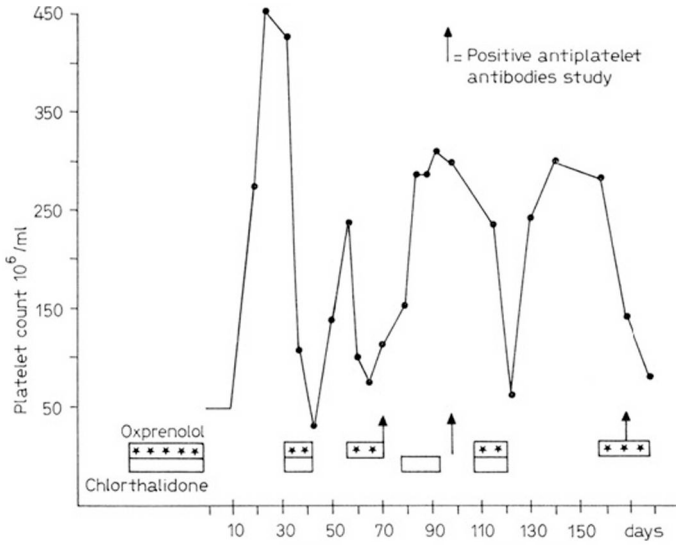
## Thrombocytopenia Secondary to Oxprenolol, a $\beta$ -Blocking Agent

For many years it has been known that a large number of drugs can lead to thrombocytopenia. The compounds most frequently incriminated are diuretics, nonsteroidal anti-inflammatory agents, and the sulfonamides [1]. It is believed that a plasma factor binds the drug to the platelet. It has been shown that this factor is actually an IgG antibody which can cause agglutination, lysis and complement fixation [2]. In this paper we describe a case of drug-induced thrombocytopenia resulting from treatment with the  $\beta$ -blocker, oxprenolol. Antiplatelet antibody titers were found repetitively in our patient. This subject was a 55-year-old male. At the age of 18 he had acute rheumatic fever which was complicated by aortic insufficiency. In the following years, he developed left ventricular hypertrophy and hypertension. The patient was treated in 1979 with digitalis, diuretics and  $\beta$ -blockers for his condition. We saw this patient for the first time in February 1980 when he was admitted to hospital with thrombocytopenia ( $50 \times 10^6/\text{ml}$ ), gingival hemorrhages and petechiae on the limbs. At this time the patient was taking 0.25 mg digoxin/day, and a combination drug containing 10 mg chlorthalidone and 80 mg oxprenolol HCl once a day (Trasitensin, Ciba-Geigy, Basel). The history revealed similar hemorrhagic episodes during previous therapy of this type. Neither lymphadenopathy nor hepatosplenomegaly was present on physical examination. The peripheral blood indices and cellular morphology were normal. Bone-marrow aspiration performed 1 day after admission, showed that megakaryocytes were numerically normal. After suspending the diuretic- $\beta$ -blocker combination therapy we

noted a progressive increase in the platelet count and a rapid disappearance of the hemorrhagic symptoms. We then challenged the patient first with the combination of oxprenolol-chlorthalidone and then with each compound separately, however, maintaining the same dosage schedule. The investigation demonstrated that the  $\beta$ -blocker was the thrombocytopenic agent (fig. 1). During  $\beta$ -blocker treatment the patient complained of paresthesia on the hands, feet and face, loss of memory, weakness, a gingival burning sensation and vague abdominal cramps. In this patient we checked for platelet antibodies by means of the serotonin release method which depends on the capacity of platelets to release serotonin as a result of antibody attachment to the platelet surface [3]. On three occasions this test was positive. In conclusion our patient presented with oxprenolol-induced thrombocytopenia. Furthermore, the presence of antiplatelet antibodies proved that an immunological mechanism was involved. We believe this to be the second published case of thrombocytopenia secondary to oxprenolol therapy [4]. Since  $\beta$ -blocker agents are now widely used in clinical practice, it may be anticipated that other cases with secondary thrombocytopenia will be described in the future.

### References

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**Fig. 1.** Platelet count and antiplatelet antibody positivity in our patient challenged with combination drug oxprenolol-chlorthalidone (Trasitensin), and oxprenolol or chlorthalidone, separately.

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