Anticoagulant pseudothrombocytopenia with platelet satellitism
MONICA MORSELLI, GIUSEPPE LONGO, GORETTA BONACORSI, LEONARDO POTENZA, GIOVANNI EMILIA, GIUSEPPE TORELLI
Department of Medical Sciences, Section of Internal Medicine, Oncology and Haematology, University of Modena and Reggio Emilia, Italy

There has been a discussion recently in the literature about platelet-leukocyte interactions in vivo and their well recognized importance in modulating inflammation and hemostasis. Occasionally, and only under particular conditions, platelets may interact with polymorphonuclear neutrophils forming complexes that may lead to a spurious thrombocytopenia.1-3 We observed a 35-year-old healthy man in whom repeated routine laboratory tests showed a mild thrombocytopenia (70-90,000/mm³). The blood film revealed scattered platelet rosetting around polymorphonuclear neutrophils; the phenomenon was evident only when the blood was treated with EDTA as anticoagulant and incubated at room temperature (Figure 1). A blood capillary film performed without EDTA did not reveal platelet satellitism and the direct count using capillary blood with ammonium oxalate was normal. Of interest, platelet satellitism with polymorphonuclear eosinophils or basophils, was never observed (Figure 2). Rosetting was not evident when citrate or heparin was used. Platelet phagocytosis by monocytes was sometimes seen. In this case, the in vitro interaction between platelets, neutrophils and EDTA was the likely cause of the spurious thrombocytopenia.

The mechanism of platelet satellitism is not completely understood. Ig autoantibodies direct against the glycoprotein IIb/IIIa complex of the platelets and the Fc (γ) receptor III (FcγRIII) of neutrophils have been implicated. EDTA at low temperature might alter the conformation of platelet glycoproteins and neutrophils Fc receptor by chelation of calcium ions. Bridge formation between platelets and neutrophils may take place through the binding of the Fab fragment to the IIb/IIIa platelet glycoprotein and the Fc fragment to the FcγRIII of the PMNs. Alternatively, but less likely, autoantibodies may recognize the same epitope in the two structures.4 A non-immunologic mechanism has also been proposed involving thrombospondin (or other α-granule proteins such as P-selectin) that, when an activation stimulus is present, is rapidly expressed on platelet surface favoring adhesion to neutrophils.3 Physicians should be aware that some thrombocytopenias of uncertain etiology may be artifactual in particular conditions; spurious thrombocytopenia due to EDTA-induced platelet satellitism is an example of such a phenomenon.

References

Figure 1. Platelet satellitism around 2 neutrophils.

Figure 2. Platelet satellitism around the neutrophil but not around the eosinophil.