
To the Editor:

The recent paper by Czapek et al. emphasizes the possibility that a population of patients exists who have mild abnormalities of platelet function, risk of posttraumatic bleeding, and an increased sensitivity to aspirin as measured by the template bleeding time. Their study, however, did not completely rule out either von Willebrand's disease or drug-induced inhibition of prostaglandin synthetase which existed in their patients prior to the aspirin tolerance test.

Of importance would have been an initial assessment of platelet aggregation in response to arachidonic acid. Arachidonic acid-induced platelet aggregation is apparently unrelated to the release reaction and may be a specific measure of the activity of the cyclooxygenase system. Unsuspected aspirin ingestion would explain the initial aggregation abnormalities that these authors demonstrated in their patients.

The authors ruled out von Willebrand's disease based upon normal and near-normal bleeding times, normal aggregation of platelet-rich plasma to ristocetin, normal platelet adhesiveness, and normal levels of factor VIII-related antigen.

The bleeding time in von Willebrand's disease is sensitive to aspirin ingestion. Ristocetin-induced aggregation of platelet-rich plasma is known to be dependent upon ristocetin concentration. Even at the concentration of ristocetin used in this study (1.2 mg/ml), aggregation response may vary. Platelet adhesiveness may also be normal in mild von Willebrand's disease. Normal levels of factor VIII-related antigen is now recognized to occur in von Willebrand's disease patients whose von Willebrand factor is qualitatively abnormal. Of interest would have been measurement of the ability of these patient's platelet poor plasma to support ristocetin-induced platelet agglutination in either a washed gel-separated or formalin-fixed platelet suspension.

We feel that it is common to obtain from patients a falsely negative history of drug exposure. There are a great number of proprietary drugs which contain aspirin. Drug-induced platelet abnormalities and/or von Willebrand's disease must be ruled out before an intermediate platelet dysfunction syndrome is widely accepted.

RONALD S. WEINGER, M.D.
PHILIP L. CIMO, M.D.
Gulf States Hemophilia Center
UT Medical School at Houston
Houston, Texas
REFERENCES


To the Editor:

In reply to the letter by Weinger and Cimo, it was our intent to draw to attention a group of patients with minimal or no hemostatic difficulties unless they ingested drugs known to interfere with platelet functions.

All our patients were aware of the multitude of drugs that interfere with platelet function. Furthermore, all were tested on multiple occasions with similar results, making the ingestion of inhibitors of platelet function unlikely.

Our patients form an important clinical group in that they do have excessive bleeding if they ingest drugs that interfere with platelet function, especially if they undergo a surgical procedure. We do not suggest they all have the same basic underlying defect; this is in fact, unlikely. The normal or borderline bleeding times, normal platelet adhesiveness, factor VIII activity: antigen ratios, and ristocetin aggregation, rule out “classical von Willebrand’s disease.” They could certainly have a functional abnormality in von Willebrand factor, a defect in the cyclo-oxygenase system, or other metabolic defects of platelet function that are still in the process of being elucidated. No matter what the underlying defect in platelet function may be, it is important that the clinician be aware that mild forms of platelet dysfunction exist and do increase patient morbidity.

EMILY E. CZAPEK, M.D.
DANIEL DEYKIN, M.D.
EDWIN SALZMAN, M.D.

ERRATUM

In the article Atypical Lymphoid Leukemia in Ataxic Telangiectasia, by Levitt, Pierre, White, and Siekert, published in the November 1978 issue [Blood 52: 1003–1011, 1978], Figure 2, the ideogram, depicts a tandem translocation of chromosomes 13 and not 14 as stated. The chromosome abnormality in the patient is as stated in the article, a tandem translocation of chromosome 14. The chromosome terminology should be corrected to t(14q11:14q32).
Platelet function abnormalities [letter]
RS Weinger and PL Cimo