CASE DESCRIPTION

A 69-year-old man presented with a long-standing thrombocytopenia and microcytic anemia. The automated platelet count was $67 \times 10^3/\mu L$ (reference interval, 150–450 $\times 10^3/\mu L$); therefore, EDTA-dependent pseudothrombocytopenia was excluded. The hemoglobin concentration was 10.5 g/dL (reference interval, 14–18 g/dL). The patient denied having petechiae and reported no excessive bleeding in daily life. The patient had no family history of coagulation disorders. The following image was seen on a blood smear (Fig. 1).

Fig. 1. Blood smear from the patient.
Shown are giant thrombocytes (red arrow) and cytoplasmic inclusion in neutrophilic leukocytes (black arrow).

QUESTIONs

1. Why should the automated platelet count be interpreted cautiously in this patient?
2. What techniques are still able to provide a reliable platelet count?
3. What do the cytoplasmic inclusion bodies found in neutrophilic leukocytes suggest?

The answers are on the next page.
**Answers**

The blood smear revealed giant thrombocytes (red arrow). These cells are often not recognized as platelets by automatic blood count analyzers, hence the thrombocytopenia. Confirmation comes from manual platelet counts, either by using a hemocytometer or via indirect estimation from the blood smear (e.g., according to the Fonio method).

The abundant cytoplasmic inclusions in neutrophilic leukocytes (black arrow) morphologically resemble Döhle bodies, which are frequently found in macrothrombocytopenias, such as May–Hegglin anomaly (1). Indeed, that was the final diagnosis for our patient. The aberrant erythrocyte morphology is due to iron-deficiency anemia, a common phenomenon in patients with May–Hegglin anomaly.

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**Reference**


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**News & Views**

**Vitamin D and Prevention of Cardiovascular Disease and Diabetes: The Potential Sunny Vitamin D Effects Are Clouded by Unclear Evidence**

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What are the recommended intakes for vitamin D? What concentration of 25-hydroxyvitamin D delineates “normal” from vitamin D deficient? These recurring questions have received considerable attention and debate. The Institute of Medicine (IOM) used a systematic, evidence-based review approach to make its recommended dietary allowances for vitamin D, including a critical evaluation of the plethora of evidence relevant to the role of vitamin D in human health and disease. According to the 2011 IOM report (1), despite a large body of scientific evidence supporting the potential role of vitamin D in preventing cardiovascular disease and type 2 diabetes, these data were deemed insufficient to inform nutritional requirements. How could such substantial evidence have had minimal influence on the IOM recommended intakes for vitamin D?

In a recent commentary in the *Journal of the American Medical Association* (2), 2 members of the IOM committee, Drs. Sue Shapses and JoAnn Manson, discussed the committee’s assessment of available evidence and subsequent conclusions that led to the IOM recommended intakes for vitamin D. The principle that association does not prove causation was highlighted as a key criterion, which contended that a cause-and-effect relationship between a nutrient and a health outcome is essential. The IOM committee reviewed several observational studies and uncovered both uncertainty and inconsistency among the studies that associated 25-hydroxyvitamin D concentrations with subsequent cardiovascular events. Select studies further suggested a nonlinear association with potential increased risk of cardiovascular dis-

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