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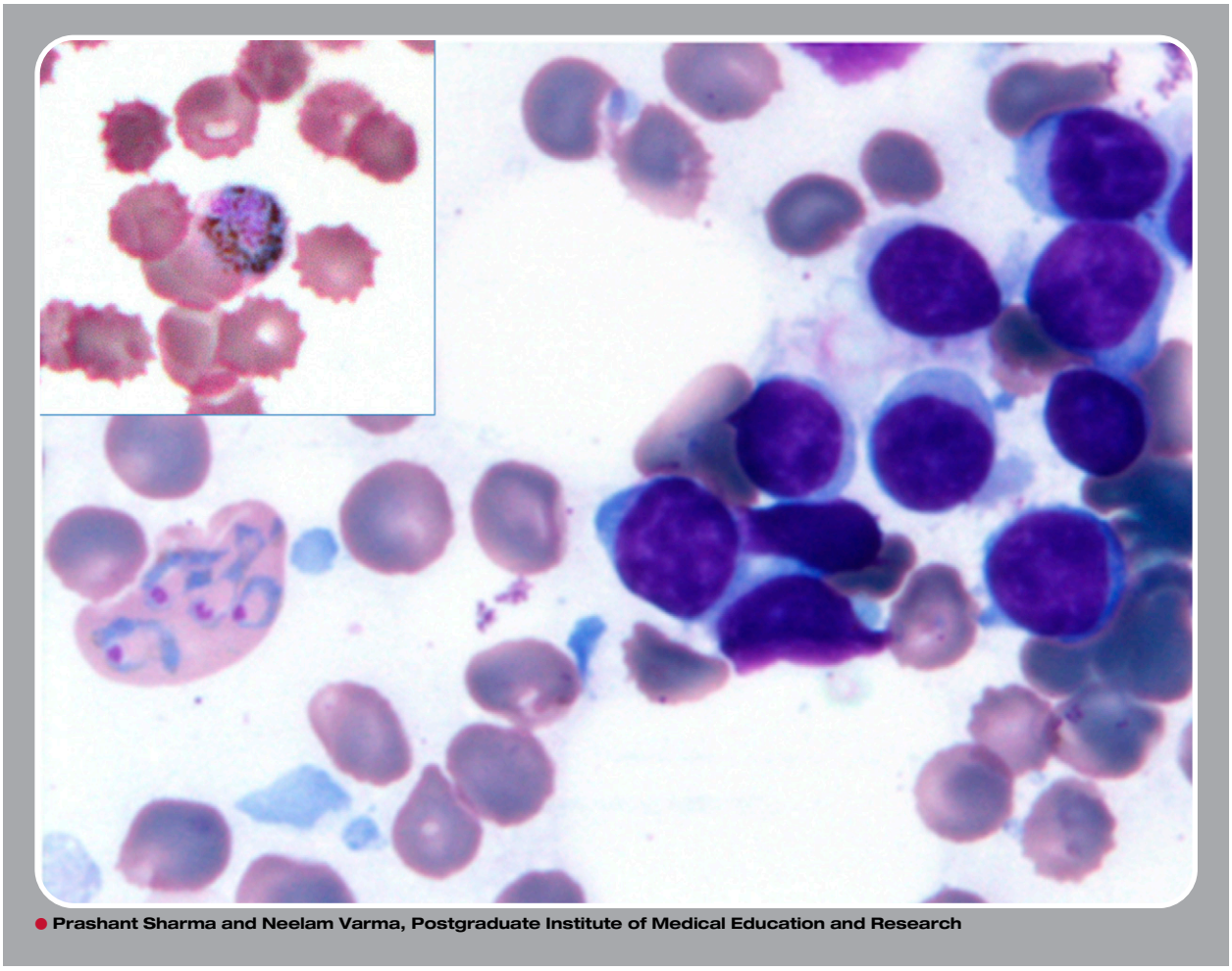
blood work
IMAGES IN HEMATOLOGY

Pancytopenia following *vivax* malaria in a CLL patient



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Routine blood counts in a 65-year-old man revealed marked lymphocytosis (hemoglobin, 115 g/L; total leukocyte count [TLC], 70.2 × 10^9/L; absolute lymphocyte count [ALC], 65.5 × 10^9/L; platelets, 142 × 10^9/L). Examination revealed enlarged cervical lymph nodes and hepatomegaly 2 cm below costal margin without splenomegaly. A week later, without any therapy/intervention, he developed high-grade fever, breathlessness, and diarrhea over 3 to 4 days. Hemograms revealed pancytopenia (hemoglobin, 95 g/L; TLC, 3.3 × 10^9/L; ALC, 1.8 × 10^9/L; platelets, 30 × 10^9/L). Bone marrow showed 86% lymphoid cells with typical chronic lymphocytic leukemia (CLL) immunophenotype ($CD19^+$/$CD5^+$/$CD23^+$/$CD20^{dim}$/κ^{dim}). Additionally, blood and marrow films showed amoeboid trophozoites and schizonts of *Plasmodium vivax* (see inset), confirmed on immunochromatographic testing. Many red blood cells contained multiple rings. Hemophagocytosis was inconspicuous. Serum urea was 110 mg% (preinfection result, 44 mg%), creatinine was 2.4 mg%, and lactate dehydrogenase (LDH) was 1050 IU/L, with normal sodium and potassium levels. Artesunate therapy cleared the parasite. Five days later, TLC was 16.2 × 10^9/L and after 11 days was 47.7 × 10^9/L. The typical CLL blood picture returned. LDH was 880 IU/L. Urea, creatinine, and uric acid normalized. Hemoglobin and platelets returned slowly to pretherapy levels.

Vivax malaria may cause pancytopenia via hemophagocytic lymphohistiocytosis, myelosuppression, hypersplenism (all clinically/morphologically unlikely in our case), or tumor lysis by infection-related steroid release. Implications of infection-lowered counts remain open to exploration by future clinical/therapeutic mechanistic studies.

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