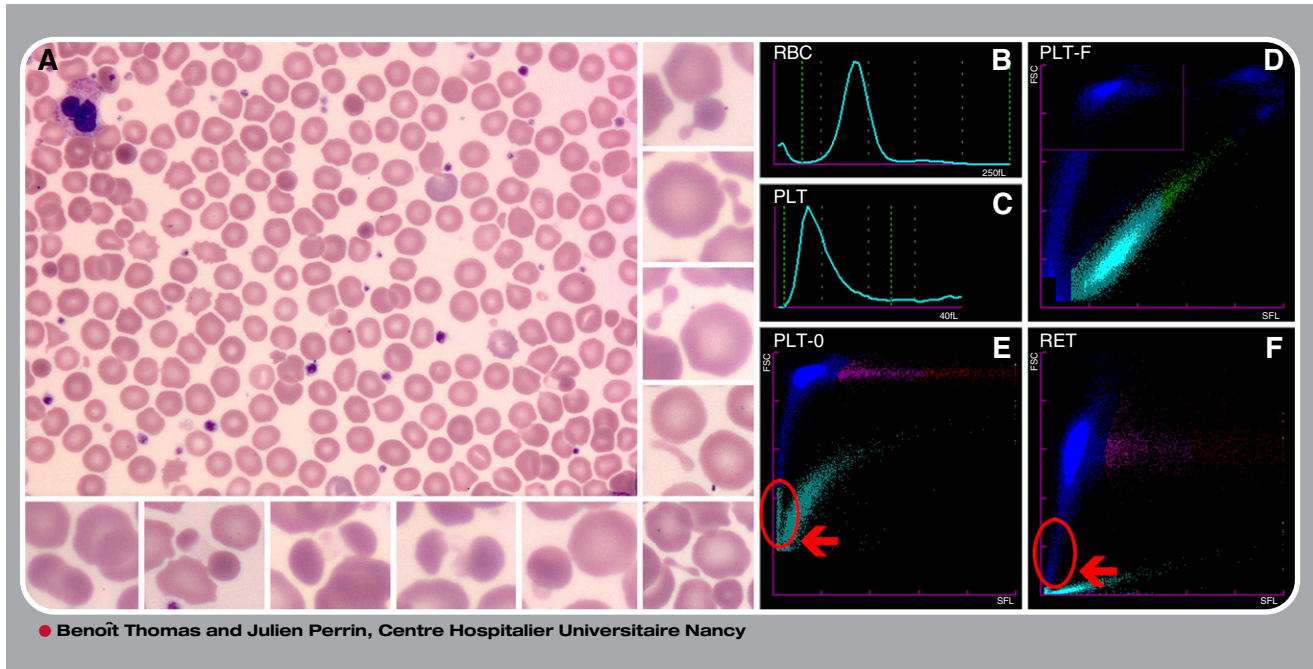


Acquired “pyro”-poikilocytosis



A 3-year-old boy was admitted to the intensive care unit after accidental life-threatening burns affecting 65% of his body surface area. A complete blood count (Sysmex XN-10 analyzer) performed 6 hours later showed marked leukocytosis and thrombocytosis (red blood cells [RBCs], $5.1 \times 10^{12}/L$; hemoglobin, 133 g/L; hematocrit, 42%; mean corpuscular volume, 82 fL; platelets [PLTs], $536 \times 10^9/L$; leukocytes, $36 \times 10^9/L$) without any technical alarm on impedance RBC (panel B) and PLT (panel C) histograms. A routine blood smear (panel A; original magnification $\times 40$, insets, $\times 100$; May-Grunwald Giemsa stain), however, revealed moderate but significant anisopoikilocytosis with numerous echinocytes and spherocytes. Remarkably, many RBC fragments were unusually microcytic, and RBCs undergoing fragmentation were observed. Further analyses revealed that the PLT count by fluorescence (PLT-F, panel D) was 15% lower with impedance because of contamination by microspherocytes; optical measurement of platelets (PLT-O, panel E) and RBCs (RET channel; panel F) estimated the RBC fragments at 3.55%.

Therefore, careful review of the blood smear allowed correction of the impedance count, a usual pitfall in thermal injury cases, by using an additional channel of PLT measurement by fluorescence, which is not accessible with all analyzers. The degenerative changes of RBCs result from thermosensitivity of the cytoskeleton. Such fragmentation may also be seen in hereditary pyropoikilocytosis, a constitutive RBC defect affecting the cytoskeleton, whose name is derived from thermally induced morphological alterations; by contrast, hereditary pyropoikilocytosis is characterized by the presence of numerous elliptocytes.



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