We have read with interest the recent report by Zoumbos et al. [1] on 4 patients fulfilling some of the criteria for chronic neutrophilic leukemia (CNL), in whom the presence of dysplastic features in blood and bone marrow was a prominent feature. Based on this, as well as on the frequent evolution to acute leukemia, the above authors proposed the term CNL with dysplastic features for this entity, and its inclusion among the myelodysplastic syndromes. We have previously reported [2] a patient who displayed most of the characteristics stressed by Zoumbos et al. [1] in their work.

In brief, the patient was a 63-year-old man with a 6-month history of weakness, weight loss and recurrent gum bleeding, who showed at presentation moderate hepatosplenomegaly. Hb was 9.3 g/dl, MCV 95 fl, platelet count 269×109/1, and WBC count 44.4 × 10^9/l, with 88% neutrophils, 6% lymphocytes, 4% monocytes, and 2% metamyelocytes. The neutrophils exhibited Döhle bodies, marked degranulation and occasional hypersegmentation. The serum levels of vitamin B12, transcobalamin, and uric acid were elevated. A bone marrow aspirate showed myeloid hyperplasia, with dysgranulopoiesis and giant forms, in addition to 6% blast cells and megakaryocytes of hypoploid appearance. At ultrastructural microscopic examination of the marrow aspirate, there were prominent dysplastic changes, not only in the granulocytic but also in the erythroid and the megakaryocytic series. No fibrosis was noted in the bone marrow biopsy, whereas the cytogenetic and DNA studies were normal and the LAP score high. The WBC count progressively increased up to 109 × 10^9/l (with 92% neutrophils), as also did the anemia and the splenomegaly. After evidence for underlying neoplasia or infection could not be obtained, the diagnosis of CNL was established and busulfan subsequently started, that 10 days later was substituted by 6-mercaptopurine due to the appearance of thrombocytopenia. However, at 3 months of diagnosis the disease evolved into monocytic type acute leukemia, which led the patient to death shortly afterwards.

Although our patient shared with those of Zoumbos et al. [1] the main characteristics remarked by these authors (namely, persistent increase in the mature neutrophils, blood and bone marrow dysplastic features, and frequent evolution to acute leukemia), some differences must be pointed out. Among them are noteworthy the presence in the former and the absence in the latter of both Döhle bodies in the neutrophils and a high LAP score, two features considered as typical of CNL.
[3]. This would indicate that the finding of marked dysplastic signs in CNL is not necessarily atypical for this disease.

References