A 58-year-old woman presented with asthenia, anorexia, fever and mild hepatomegaly. The blood cell count showed WBC 25.1x10^9/L with 30% neutrophils, 19% lymphocytes, 6% monocytes, 45% blasts, Hb 12 g/dL, platelets 38x10^9/L. Bone marrow biopsy revealed M2 acute myeloid leukemia (AML). A large mass was found in the left kidney by ultrasonography. Before starting chemotherapy the patient underwent nephrectomy. Hematologic remission was obtained with daunorubicin and cytarabine. A year later she suffered a marrow relapse, was treated with chemotherapy and died during the aplastic phase.

Peripheral blood blasts were large with numerous azurophilic granules and frequent nuclear indentations; Auer rods were seen in a few cells. Binucleated blasts and morphologically abnormal neutrophils were also present (Figure 1).

Bone marrow was hypercellular with few erythroblasts and megakaryocytes with granuloblastic hyperplasia without hiatus and 40% blast cells were prominent. Blasts were polymorphous; many were large with abundant cytoplasm. They presented an eccentric nucleus often indented or cleft; a clear cytoplasmic area corresponding to the nuclear indentation, many azurophilic granules, needle-like Auer rods and vacuoles; a minority, however, were small and agranular. Myelocytes and metamyelocytes showed evident N/C asynchronism with the persistence of nucleoli and a loose chromatin pattern even in the presence of numerous secondary granules in the cytoplasm. Neutrophils were often agranular and exhibited abnormalities of nuclear segmentation. An increase of morphologically normal eosinophils was evident (Figure 2).

Blasts were PAS and ANAE negative and Sudan positive, with the reactivity being localized in a limited cytoplasmic area, in the nuclear cleft. Immunophenotyping showed expression of the myeloid antigens CD13 and CD33 as well as HLA-DR and stem cell antigen CD34, whereas lymphoid antigens were lacking.

The bone marrow karyotype was 46, XX t(8;21) (q22;q22) in all 7 mitoses suitable for analysis.

A large mass involved the middle and inferior portions of the left kidney, with invasion of the capsule. Histological examination revealed a picture of primary renal cell adenocarcinoma associated with wide areas infiltrated by myeloid blasts (Figures 3-5).

AML with t(8;21) is a well-known, distinct clinicopathological entity recognized by the MIC group and characterized by a constellation of pathognomonic morphological features, indicative of diagnosis even before cytogenetic and/or molecular results are known.1-5 The two genes involved in the t(8;21) have recently been isolated and the cDNA...
of the AML1/ETO fusion gene identified.6-8 This AML subtype occurs chiefly in young patients and is characterized by high complete remission rates and good survival.9,10 Local tumors are often present at diagnosis or at relapse and may involve deep sites. The leukemic nature of the tumor may be established by biopsy.

Very surprisingly in this case, a leukemic infiltrate was found to coexist with a primary renal cell carcinoma in the left kidney. Histochemical and immunohistochemical criteria were useful in confirming the morphological diagnosis.

References