Dear Sir,

Leukemic patients with acute renal failure (ARF) due to leukemic cell infiltration have been reported to be rare. Furthermore, pathological confirmation has been made only in a few cases with nonlymphoid leukemia. We present a case with acute myelomonocytic leukemia (AMMoL) who developed ARF with prominent interstitial infiltration of leukemic cells in the kidney.

A 17-year-old female with multiple subcutaneous bleedings on the extremities was admitted on May 18, 1991. Her white blood cell count was 77,800/mm³ with 80% blast cells, platelet count 62 × 10³/mm³, fibrinogen 132 mg/dl and fibrin/fibrinogen degradation product 106 mg/ml. Total protein was 5.5 g/dl (albumin 3.0 g/dl), blood urea nitrogen 55 mg/dl, creatinine 9.9 mg/dl, uric acid 32.0 mg/dl, sodium 132 mEq/l, potassium 2.5 mEq/l, inorganic phosphorus 1.0 mg/dl, calcium 4.3 mEq/l and bicarbonate 16.2 mEq/l. Bone marrow aspirate disclosed a nucleated cell count of 1,270,200/mm³ with 98% blast cells. These cells were positive for double staining for both specific and nonspecific esterases. Ultrasound examination disclosed bilateral swollen kidneys without hydronephrosis. She had complete anuria and did not respond to the mannitol test. Diagnosis was made as having AMMoL complicated by disseminated intravascular coagulation (DIC) and ARF. She was started on
hemodialysis and chemotherapy for AMMoL accompanied by oral allopurinol and intravenous anticoagulation including hepa-

Fig. 1. Kidney necropsy of the patient. The renal interstitium is infiltrated by numerous leukemic cells. Tubules show atrophy and degeneration. There are only minor glomerular abnormalities. HE. ×160.

cleated cell count of 18,000/mm3 with 46% blast cells. Due to these findings, the chemotherapy was discontinued. On the other hand, persistent oliguria despite forced diuresis still required dialysis and DIC continued. On day 18, she died of respiratory failure. Tissues from the left kidney were obtained with informed consent. On light microscopy, the necropsy showed massive infiltration of cytoplasm-rich highly atypical mononuclear cells with

fibrosis in the interstitium, predominantly in the cortex (fig. 1). By immunoperoxidase staining using antibody to CD68 (Daco CD68, KP-1), these cells were identified either as myeloid precursor cells or as having a monocyte/macrophage origin. Dilated tubules were frequently seen in the cortex with flattened or degenerated epithelial cells. Tubular atrophies were also observed. In contrast, most of the glomeruli were intact and there were no infiltrations of these cells or microthrombi. Renal vasculature showed no thrombi or leukostasis. Immunohistological examination was negative for immunoglobulins, complements or fibrinogen.

Clinically, acute uric acid deposition and DIC were candidates for being the causes of renal failure. However, neither of the two could explain its persistence when clinical and pathological findings were taken into consideration. A third factor was suggested to have participated in the persistence of renal failure and massive leukemic infiltration in the renal interstitium seemed responsible. As swollen kidneys were confirmed on admission and the second bone marrow aspirate indicated her response to the chemotherapy, the leukemic cells were considered to have infiltrated before the chemotherapy was started, not in her late hospital course. It has been reported that massively infiltrating leukemic cells may compress tubules [1,2] and shut down the microcirculation [1] in the kidney leading to destruction of the normal architecture. This can result in glomerular ischemia and abrogate glomerular filtration preventing diuresis. In the present case, it is conceivable that such an architectural and functional alteration may be a major cause for persistent renal failure with oliguria.

It has been reported in some autopsy studies that leukemic patients in bone marrow remission may show some organs including the kidneys being infiltrated by leukemic cells. For these cases when they are accompanied by renal deterioration, additional intervention including local irradiation may be considered.

References

Renal Failure with Infiltration by Myelomonocytic Leukemia Cells
Nephron 1996;73:728-729
729