A 61-year-old man was admitted for anemia in 1994. Physical examination revealed generalized lymphadenopathy and hepatosplenomegaly. The erythrocyte sedimentation rate was 184 mm/h. The hemoglobin was 8.5 g/dl, the leukocyte count was 3,500/mm$^3$ with normal differential, and the platelet count was 181,000/mm$^3$. The bone marrow was hyperplastic and contained 60.2% of lymphoplasmacytoid cells. There was marked rouleaux formation of the erythrocytes. The serum protein was 10.6 g/dl with IgM 8,660 mg/dl, IgG 930 mg/dl, and IgA 16 mg/dl. A monoclonal IgM spike was seen on serum protein electrophoresis and Bence Jones $\kappa$ protein was detected in the urine. A biopsy of the liver revealed dense periportal infiltration of neoplastic cells. He was treated with plasmapheresis and intermittent chemotherapy with fluctuation of serum IgM levels for the past 10 years. The most recent leukocyte count was 7,100/mm$^3$ with 30% of lymphoplasmacytoid cells that showed $\kappa$ light chain monoclonality. These abnormal cells had pale to bluish cytoplasm, oval to reniform nuclei, and nucleoli in some (Fig. 1). The neoplastic cells in Waldenström’s macroglobulinemia have hybrid features of lymphocytes and plasma cells, and can be readily mistaken for lymphocytes, especially by automatic blood cell analyzers. To obviate this error, it should be noted that some macroglobulinemia cells often appear in the peripheral blood with progression of the disease, although frank leukemic conversion is rare.

Key words: Waldenström’s macroglobulinemia, circulating tumor cells

Figure 1. Peripheral blood smears prepared on January 5, 2004 (right) and February 12, 2004 (left) each showing 2 macroglobulinemia cells (Wright’s stain).