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A Case with Membranous Glomerulonephritis and Myelodysplastic Syndrome

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Dear Sir,

We described a case of membranous glomerulonephritis (MGN) and anemia-thrombocytopenia in a male who was subsequently diagnosed as hypoplastic-type myelodysplastic syndrome (MDS).

A 65-year old man was admitted for uremia and anemia. He has had history of edema and lumbal pain for 8 months. Ten days before admission, he complained of melena. On physical examination, pallor and splenomegaly were detected. Investigations were as follows: hematocrit 15%, hemoglobin 4.9 g/dl, WBC 10, 100/mm^3, platelet count 75,000/mm^3, peripheral blood smear disclosed anisocytosis, poikilocytosis, polychromasia, WBC were normal, BUN 90 mg/dl, creatinine 8.7 mg/dl, phosphorus 8.1 mg/dl, total protein 6.6 g/dl, albumin 4.1 g/dl, total iron 140 μg/dl, unsaturated iron-binding capacity 230 μg/dl, total iron-binding capacity 37 μg/dl, acid phosphatase 10.8 mg/dl, prostatic acid phosphatase normal, prostate-specific antigen normal, vitamin B_{12} 465 pg/ml, folic acid 4.20 ng/ml, erythrocyte sedimentation rate 115 mm/h. Antinuclear antibody, rheumatoid factor, HB_sAg, anti-HCV and anti-HAV were negative. Urine protein excretion was 3.75 g/day and creatinine clearance 10.8 ml/min. There was no Bence Jones proteinuria. Urine microscopy showed granular and cellular casts but no organisms.

Ultrasonographic and computerized tomographic exam of the abdomen and pelvis were negative except for prostatic hypertrophy ($38 \times 22 \times 27$ mm). Chest x-ray showed a dilated aorta. Upper and lower gastrointestinal endoscopy were negative. Dry tap was found in bone marrow aspiration. Bone marrow biopsy was consistent with hypoplastic-type MDS. A renal biopsy was performed. Light microscopy of the biopsy showed membranous glomerulonephritis (fig. 1). Immunofluorescent microscopy using antisera against human immunoglobulins and complement revealed granular IgG and C3 deposition along glomerular capillary walls.

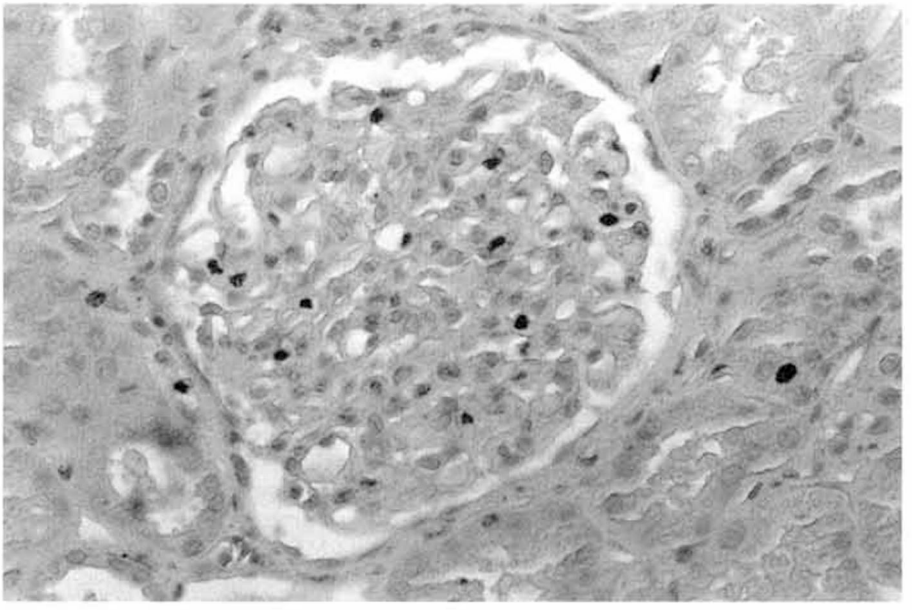


Fig. 1. Biopsy No. 1339/91: diffuse thickening of the glomerular capillary walls (membranous glomerulonephritis). HE. × 600.

With supportive therapy, BUN was decreased (36 mg/dl). The origin of gastrointestinal bleeding could not be detected. After 4 weeks follow-up, the regimen consisted of prednisolone (60 mg/day), cyclophosphamide (100 mg/day), dipyridamole (225 mg/day) and famotidine (20 mg/day). Three months later, urea level and urine protein excretion were 30 mg/dl and <0.3 g/day respectively; but anemia persisted (Hct 14–16%). A second bone marrow biopsy was performed. It was found that the bone marrow was infiltrated by monocytic cells (fig. 2), suggesting blastic transformation from hypoplastic bone marrow. Low-dose cytosine arabinoside was infused 20 mg/m^2 daily for 14 days every 4 weeks.

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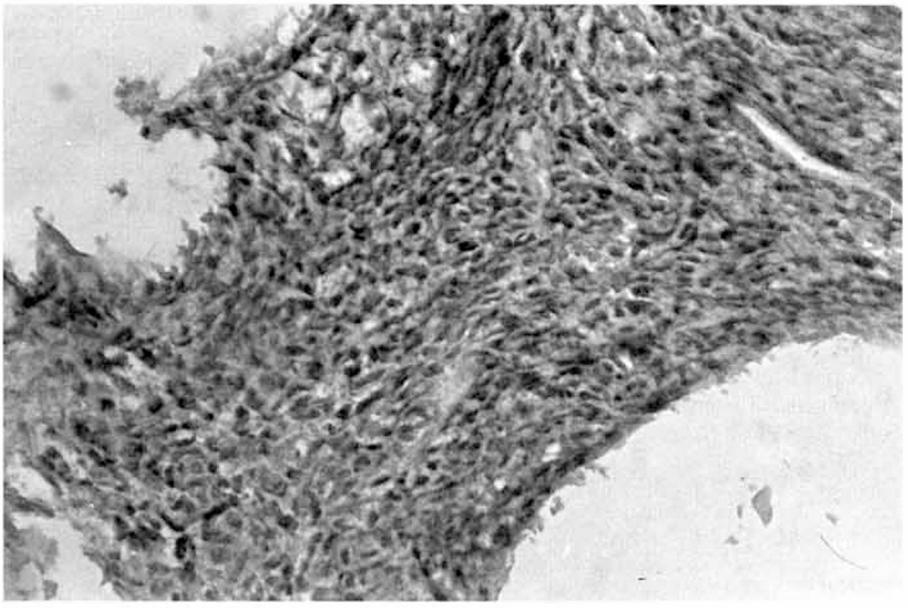


Fig. 2. Biopsy No. 9441/91: bone marrow infiltrated by monocytic cells, suggesting blastic transformation. HE. × 480.

MGN is the most common cause of the idiopathic nephrotic syndrome in adults. Most cases of MGN are idiopathic. An underlying disorder or antigen was determined in approximately one third of patients with MGN [1]. The most common causes of tumor-related MGN are solid lung and gastrointestinal tumors [2]. We didn't find any solid tumor for 6 months. At first, we found that bone marrow was hypoplastic, and myeloblastic transformation was determined 4 months later. These findings suggest the transformation of hypoplastic-type MDS to blastic type.

In the literature, MGN was not described in MDS. It is known that immune complex deposition and T lymphocyte dysfunction were major pathogenetic factors in MGN [3]. Although circulating immune complex is not known to be involved in MDS, there is cellular dysfunction at the pluripotent stem cell [4]. Cyclophosphamide may be the cause of blastic transformation in our cases. But 3 months are too short for these changes. It is possible that blastic transformation is a natural course of MDS. The coexistence of MDS and MGN was of interest.

References

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