Acute Renal Failure and Nephrotic Syndrome in a Patient with T-Cell Lymphoma

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Dear Sir,

Hodgkin's disease is representative of the neoplasms which cause nephrotic syndrome [1]. Non-Hodgkin's lymphoma (NHL) is also reported to induce nephrotic syndrome, but rarely to result in acute renal failure (ARF) [2]. Herein we report on a case of T-cell type NHL presenting with pleural effusion, proteinuria and acute renal failure, probably associated with vascular permeability factors which may be released by abnormal T cells.

A 59-year-old man was admitted because of fever, weight loss and systemic lymphadenopathy. On admission, laboratory tests revealed negative urinary protein, hematocrit 38.7%, serum albumin 3.5 g/dl and normal levels of serum creatinine.

Immunohistological examination of biopsy specimens from the lymph nodes revealed T cell NHL of the diffuse, large-cell type. After admission, he developed systemic eruption, followed by a gradual decrease in urine output, resulting in the development of edema and bilateral pleural effusion (fig. 1). Serum creatinine acutely rose from 0.90 to 6.60 mg/dl accompanied by 7 kg of weight gain and a rise in blood pressure from 84/50 to 150/80 mm Hg. At this time urinalysis showed a 3+ test for protein and serum albumin decreased to 2.6 g/dl. The fractional excretion of urinary sodium (FENa) was 0.08%. Ultrasonographic study and a computed tomographic scan revealed neither compression of the renal artery or ureter by lymphadenopathy nor enlargement of the kidneys. Chemotherapy resulted in diuresis and recovery of renal function. Urinary protein, however, rapidly increased, amounting to 2.5-3.2 g/day and he became anuric.

Chemotherapy X
Fig. 1. Clinical course. WBC = Leukocyte count; S-Cr = serum creatinine; UP = urinary protein; BW = body weight; UV = urine volume; HD = hemodialysis; RBx = renal biopsy; P.Effusion = pleural effusion; FENa = fractional excretion of urinary sodium.

Renal biopsy performed at this time showed no infiltration of atypical lymphoma cells. The enhanced vascular permeability factors released by abnormal T cells are suggested to cause nephrotic syndrome [3,4]. Symptoms observed in our case such as eruption, nephrotic with serum albumin levels of 2.5-2.9 g/dl. When the leukocyte count recovered from the nadir, the second episode of acute renal failure occurred with findings similar to the first one, though to a lesser extent, and resolved after chemotherapy.

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edema and pleural effusion seem to be derived from factors which enhance vascular permeability. At the onset of ARF in our case, there were no findings suggestive of hypovolemia, whereas the findings of low FENa, elevated blood pressure and increased body weight were compatible with findings of ARF in patients with minimal change nephrotic syndrome (MCNS) described by Lowenstein et al. [5]. Lowenstein et al. [5] demonstrated that the reduced filtration rate in MCNS is due to a reversible alteration in glomerular hemodynamics which is related to fluid retention and associated intrarenal edema. This mechanism appeared to be involved in the development of

ARF in our patient, because peripheral edema and ARF were reversed by chemotherapy, probably associated with a reduction in enhanced vascular permeability as a result of destruction of tumor cells. This vascular permeability factor seems to participate in urinary protein excretion by enhancing glomerular permeability probably via the same pathomechanism described in patients with MCNS [6]. We concluded that the factors released by abnormal T cells would result in an increase in vascular permeability including glomerular capillary, leading to eruption and pleural effusion, proteinuria and intrarenal edema which may cause acute renal failure as well as nephrotic syndrome.

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ARF and Nephrotic Syndrome in a Patient with T-Cell Lymphoma *Nephron* 1996;72:326-327 327