Occurrence Rate of Polyarteritis nodosa in Chronic Renal Diseases

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Dear Sir,

Polyarteritis nodosa is a systemic necrotizing vasculitis characterized histologically by inflammation and fibrinoid necrosis of medium-sized or small arteries. It has 2 histological forms: classical or macroscopic and microscopic. However, overlapping between these groups occurs.

The classical type refers to systemic vasculitis, predominantly affecting muscular arteries at branch points with varying ages and various sizes of focal aneurysms. Hypertension and ischemia of glomerular lesions are commonly found in this form of polyarteritis.

Microscopic polyarteritis is characterized by a necrotizing inflammation of the smaller arteries, and venules. In this type, vascular lesions occur usually at similar ages and aneurysm formation is uncommon. Hypertension is rarely seen and glomeruli are typically involved by segmental necrotizing and crescentic glomerulonephritis [1].

The overlapping form refers to cases in which combined features of both macroscopic and microscopic forms of polyarteritis are present. In this form both small and larger blood vessels might be involved; these may be caused by vasculitis of larger arteries associated with glomerulonephritis. ■

Polyarteritis has varied clinical manifestations, depending on the location of the involved arteries. The kidneys, heart and gastrointestinal tract are commonly involved in macroscopic forms; lungs and skin are often spared. Whereas in the microscopic forms, lungs and dermis are the most frequent sites of involvement. In the overlapping form, both large and small arteriolar lesions are seen [2].

In polyarteritis, patients might present with only renal findings which might be confused with other primary glomerular diseases. Therefore patients with primary renal disease should always be examined for other systemic diseases.

Owing to the problems in classification and overlapping with other disease entities, the incidence of renal involvement in polyarteritis has been reported to vary greatly. It has been given as 80% by Droz et al. [3], 70% by Fujimoto and Yamamoto [4], and 26% by Guillemin et al. [5]. However, studies on the occurrence rate of polyarteritis among chronic renal diseases are very few. It is reported to be 2.4% by Cohen et al. [6]. We therefore planned a study to find the incidence of polyarteritis among patients presenting with glomerular diseases.

834 renal biopsies obtained from patients with chronic renal disease have been reviewed. 27 patients were found to have histopathological lesions of polyarteritis nodosa giving an incidence of 3.2%. Of these 27 patients, 11 (40.7%) were females and 16 (59.2%) were males with a mean age of 34 ± 2 years (range 13-58). The duration of the disease was about 11 ± 2 months (range 1 month to 7 years). The clinical and laboratory findings are presented in tables 1 and 2. Hypertension was present in 66.6% of cases. Renal function was found to be less than 25% of normal in 33.3% (9 cases). Of 27 cases of polyarteritis, 18 (66.6%) showed nephritic syndrome and 33.3% (9

cases) nephrotic syndrome. Histological study of the kidney revealed mesangioproliferative glomerulonephritis in 13 cases (48%), focal segmental proliferative glomerulonephritis in 6 cases (22.2%), membranoproliferative nephritis in 5 cases (18.5%), membranous nephropathy in 3 cases (11%).

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Table 2. Laboratory findings in polyarteritis nodosa

Cases %

ESR	25	92.5
Total protein	9	33.3
Total lipids	7	25.9
Cholesterol	6	22.2
Hct	6	22.2
WBC	9	33.3
Eosinophils	9	33.3
Massive	9	33.3
1-1.5 g/day	5	18.5
Trace	13	37.5
Hematuria	11	40.0
Leukocytes or leukocyte casts	5	18.5
Telescopic appearance	8	29.6

18 cases (66.6%) showed fibrinoid necrosis, 5 cases (18.5%) hyalin thrombus, 11 cases (40.7%) showed a dense inflammatory infiltrate of lymphocytes, plasma cells, neutrophils and occasionally eosinophils.

Segmental crescentic glomerulonephritis accounted for about 88% (23 cases) of all cases of polyarteritis. In these cases, there was typical fibrin deposition in the tuft with dense inflammatory infiltrate of leukocytes and segmental rupture of the glomerular basement membrane. Although the vascular changes appear to be of the same age, the glomerular lesions varied from acute to more chronic and sclerosing lesions in the same specimen.

The mechanisms of vascular damage in polyarteritis are poorly understood at present. Cellular immunity, vascular immune complex deposition, antiendothelial antibodies, intravascular coagulation, anti-neutrophil cytoplasmic antibodies may all be mediators of the vascular damage. Recently attention has been drawn to the possible roles of cell-mediated injury and endothelial cell surface antigens.

In conclusion, it can be said that polyarteritis occurs in 3.2% of all cases of glomerulopathies. Nephrotic syndrome is not infrequent; hypertension, fever, arthralgia and visual disturbances were the commonest clinical findings. Fibrinoid necrosis, segmental necrotizing glomerulopathy with crescents and mesangioproliferative glomerulonephritis were found to be the most frequently seen renal lesions.

References

D'Agati V, Chander P, Nash M, Manilla-Jimenez RM: Idiopathic microscopic polyarteritis nodosa: Ultrastructural observations on the renal vascular and glomerular lesions. *Am J Kidney Dis* 1986;7:95.

Crocker BP, Lee T, Gunnells JC: Clinical and pathologic features of polyarteritis nodosa and its renal limited variant: Primary crescentic and necrotizing glomerulonephritis. *Hum Pathol* 1987; 18:38.

Droz D, Noel LH, Leibowitch M: Glomerulonephritis and necrotizing angiitis; in Hamburger J, Crosnier J, Maxwell MH (eds): *Advances in Nephrology*. Chicago Year Book Medical, 1979, p 343.

Fujimoto S, Yamamoto Y: Renal failure associated with polyarteritis nodosa. *Nippon Jinzo Gakkai Shi* 1990;32:739.

Guillemin L, Le Thi Huong Du, Godeau P: Clinical findings and prognosis of polyarteritis nodosa and Churg Strauss angiitis: A study in 165 patients. *Br J Rheumatol* 1988;27:258. Cohen L, Guillemin L, Meyrier A: Malignant arterial hypertension in polyarteritis nodosa. Incidence, clinicobiologic parameters and prognosis based on a series of 165 cases. *Arch Mal Coeur Vaiss* 1986;79:773.

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Nephron 1996;72:365-366

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