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## TREATING SLE NEPHRITIS: SOME GUIDELINES

While data concerning the use of total lymphoid radiation and cyclosporine show promise in the treatment of lupus nephritis, evidence is still anecdotal. A conservative approach that combines vigorous early treatment with long-term low-dose therapy is still the preferred method of treatment. The following points outline the basic tenets of this approach:

- Ideally, early treatment of lupus nephritis should start when the patient has diffuse proliferative lupus glomerulonephritis and a serum creatinine  $\leq 1.2$ .

- An accurate renal biopsy will help avoid over-treatment. Patients with mild involvement will respond well to a relatively modest therapeutic regimen.

- Short-term oral cyclophosphamide has proven to be as efficacious as other immunosuppressive regimens that have been added to prednisone.

- Intravenous cyclophosphamide should be used instead of oral cyclophosphamide if there is a possibility of poor patient compliance.

- Early treatment (for the first 4 to 8 weeks) should be as vigorous as possible, followed by a judicious tapering of prednisone with a long-term goal of alternate-day steroid therapy.

- The induction of a remission of glomerulonephritis, with a serum creatinine  $\leq 1.4$  mg/dL and a 24-hour urine protein  $\leq 300$  mg, is an important determinant of long-term prognosis.

- Prednisone may be required indefinitely for patients who have had severe lupus nephritis. Such patients should be followed closely, both systemically and nephrologically, since they are susceptible to recurrence.

- Systemic arterial hypertension and drug-associated renal damage and dysfunction are as much a

concern among these patients as in any population with chronic renal disease.

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### SUGGESTED READING

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## HEMODIALYSIS-INDUCED HYPOTENSION: THE SEARCH FOR A CAUSE

Often during the course of dialysis, episodes of hypotension occur that can be severe enough to require interruption of dialysis and treatment with saline infusion. Among the proposed etiologic factors in dialysis-induced hypotension are (1) excessive fluid removal; (2) venodilation with venous sequestration of blood volume; (3) autonomic neural dysfunction, with the inability to counterbalance cardiovascular instability induced by fluid removal; and (4) autonomic disequilibrium with predominant vagal stimulation induced by the reduction of left ventricular volume.

Autonomic dysfunction in hemodialysis has been extensively studied as a cause of dialysis-induced hypotension; however, evidence suggests that the pathogenesis is more related to hemodynamic and volume factors. Studies at the Cleveland Clinic have shown that although the efferent parasympathetic pathway was abnormal in hemodialysis patients, adrenergic responses were generally normal. These findings could not be linked to the occurrence of dialysis-induced hypotension and, in fact, there were no differences between dialysis patients with and without hemodialysis-related hypotension.

Echocardiographic studies of dialysis patients at the Cleveland Clinic point out the importance of