INTRODUCTION
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Nephrotic syndrome is the most frequently encountered disease among all nephrologic entities in pediatric age group with incidence reported to be 4-15/1ac, under 16 years of age.

It is the mere clinical manifestation of large number of morphologically distinct glomerular disorders, which in approximately 95% of children result from primary glomerular disease and in 5% due to secondary systemic diseases. Among the primary glomerular, MCNS is the commonest underlying pathology estimated around 52-78% followed by focal glomerulosclerosis and mesangial proliferative around 9-15% (Habib et al, 1971). Children with these glomerular lesions are clinically indistinguishable at presentation with MCNS but show a relative lack of response to the usual regimen of steroid therapy. Except for membranous nephropathy, less common causes like membranoproliferative glomerulonephritis and post streptococcal glomerulonephritis are rarely confused with MCNS. As cause is not identifiable this group, combinely is also known as idiopathic nephrotic syndrome which is predominantly a disease of young children. Sixty percent of the children being between 2-6 years of age suffer from idiopathic nephrotic syndrome. Absence of azotemia, hypocomplementemia, hypertension, hematuria and age 1 to 6-7 years favours the diagnosis of MCNS.
Though most of the patients of MCNS can be separated with characteristic clinical presentation and dramatic response to corticosteroid therapy exact identification of underlying pathology is possible only by microscopic studies. Clinico-pathologic correlation is significant for evaluation of prognostic value of specific pathologic and clinical features as well as management of patients. Association of mesengial hypercellularity and focal segmental sclerosis with clinical presentation of hematuria and hypertension and poor therapeutic response to steroids have been observed by various workers (Habib et al, 1971; White, 1971, ISKDC, 1981 and Saxena et al, 1988).

Various regimens of steroid therapy have been worked out but alternate day regime is said to be associated with least toxicity and relapses.

These patients showed a variable clinical course. Ninety five percent of them respond favourably to corticosteroid therapy and 90-95% of those who respond, have MCNS by renal biopsy but majority of them (— 85%) relapse within one year. Still many (— 25%) relapse frequently (72/6 months or 73/year) and some become dependent to it. A small group do not respond to corticosteroids either initially (initial non responders = 5%) or during a relapse (late non responder 5 = 10%) and have to be treated with cytotoxic drugs. Like cyclophosphamide, cyclosporin A, and chlorambucil. Immunoregulatory drugs like levamisol are also under trial.
Response to steroids is slow in presence of infection, which should include tuberculosis as well as most widely prevalent infection in India and many patients of nephrotic syndrome showing delayed response should be investigated for tuberculosis in addition to other infections.

Minimal change nephrotic syndrome as an immunologically induced disease was first seriously considered in 1907 by Bell and Clawson. Pointers towards immunologic basis are clinical association with respiratory infections and prophylactic immunizations, other atopic disorders (eczema, rhinitis) increased incidence of MCNS in patients with hodgkin's disease (in whom defects in T cell mediated immunity are well recognized), response to corticosteroid and immunosuppressive therapy and spontaneous remission following measles infection or immunization.

Giangiacoma (1975) observed decreased levels of IgG in patients with idiopathic nephrotic syndrome irrespective of the course of disease. This depression in IgG cannot be explained on urinary losses alone. Increased catabolism may play a part. It has been postulated that T cell mediated conversion from IgM to IgG may be defective. Distributed T helper cell fail to direct the B cells, these in turn would continue to produce IgM and no synthesis of IgG would occur. Increased suppressor cells may have a similar role.
It has not been established whether the depression in cell mediated immunity is a primary event or secondary to hypoalbuminuria, hyperlipidemia or zinc deficiency that coexists in these patients, as all these factors are known to depress cell mediated immunity. Abnormal immunogenic response to some unknown stimuli may be a primary event in patients with nephrotic syndrome leading to suppression of T cell function.

The most difficult problem in the care of children with MCNS continues to be the occurrence of frequent relapses in patients who respond initially to steroids. Repeated or continuous administration of steroids although effective is not always free from severe toxicity. MCNS in India differs from west one, in that majority are frequent relapers and second, that regular presence of alternate day steroid therapy in inducing longer remission may not be impressive.

Clinically, presence of hematuria, azotemia or hypertension is not helpful or their identification but increased incidence of atopic disorders (asthma, eczema) or rhinitis, less relapses in first few months of induction of first remission, biochemically impaired concentration capacity of urine (as evident by low urinary osmolality) and immunologically persistently low IgG levels and raised IgM levels are among the features linked with early identification of frequent relapers.

Although etiology of MCNS is still obscure, the
association of nephrotic syndrome with certain morphological immunological and biochemical abnormalities have lead to a better understanding of this disease in recent times. Attempts to correlate the course of the disease with the histologic findings have met with only limited success. The presence of either mild mesangial proliferation or focal tubular changes on renal biopsy is associated with a decreased rate of initial response to intensive prednisolone treatment. At present therefore potential frequent relapsers cannot be identified from histopathologic characteristics.

Preventing the occurrence of frequent relapsers and until that is possible, finding more effective and safer methods of treating them thus remains the major unsolved problem in case of patients with minimal change nephrotic syndrome (MCNS).

It is in the light of those observations, that the present venture has been undertaken to study incidence, mode of onset, clinical presentation, biochemical, histopathological and immunological changes and to search for clinical or immunological basis to identify frequent relapsers early, which shall permit investigations that might explain the striking mysterious variations in course of patients with MCNS, who at the onset of their disease appear to share similar characteristics. Future needs are the identification of markers for predicting methods for preventing and safe effective drugs for treating the frequent relapses of MCNS.