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Salt Loosing Nephropathy in Lymphoplasmacytic Lymphoma Cases

Neelesh Jain*

*Department of Transfusion Medicine, Balco Medical Centre, Chhattisgarh, India.

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ABSTRACT

In lymphoplasmacytic lymphoma (LPL) also known as Waldenstrom's macroglobulinaemia (WM) renal disease is less common than in multiple myeloma. WM accounts for about 5% of all malignant B-cell disorders associated with a monoclonal protein spike in the serum or urine and about half of patients who have a monoclonal Immunoglobulin M (IgM) spike in the serum have this disorder. Plasma cell dyscrasias often are associated with kidney diseases due to the production of monoclonal immunoglobulin but with a diverse set of pathologic renal patterns. While many patients undergoing a renal biopsy and showing a cast nephropathy have multiple myeloma (MM), kidney involvement associated with pathological immunoglobulin light chains and lymphoma is rare. Thus a therapeutic approach that decreases light chain production appears to be warranted in such patients.

Keywords: Waldenstrom's macroglobulinaemia, Lymphoma, Nephropathy

INTRODUCTION

Multiple myeloma is very commonly associated with renal failure. A renal biopsy in such patients shows a cast nephropathy but the association of cast nephropathy with lymphoma is quite rare [1]. Some of these patients develop salt losing nephropathy as a renal complication in lymphoplasmacytic lymphoma and then subsequently there is a diagnostic dilemma in distinguishing the causes for renal dysfunction. The incidence of renal complication in waldenstrom's macroglobulinemia (WM) is very less compared to the incidence in those of myeloma patients. This is probably due to the rare occurrence of Bence-Jones proteinuria in WM which may also explain why tubular casts and so called 'myeloma kidney' are rare in WM [2]. In WM renal disease is usually caused by IgM deposits along the glomerular basement membrane, infiltration of the interstitium with lymphoid cells or amyloidosis [3].

DISCUSSION

We have had an experience of few patients who presented with renal failure with a slight proteinuria with kappa and lambda light chains on immunoelectrophoresis. Renal involvement in WM presents as usually a mild non-selective proteinuria and microscopic hematuria. Massive proteinuria and a nephrotic syndrome may develop and in most cases is caused by amyloidosis [3]. Bence Jones proteinuria is present in 80-90% of the patients, but the quantity is much smaller than in MM. Cryoglobulinemia and acute renal failure are rare in WM. In most cases renal failure is chronic, but due to inadequacy of follow-up data, not much is known about the course of renal failure in WM [3,4]. The two major classes of light-chain proteins are kappa and lambda, which are being synthesized in bone marrow plasma cells. Each major type can be further classified by the use of appropriate antisera into several subtypes, four kappas and five lambdas [5].

Kidney is the major site of metabolism of light-chain proteins. Even though the complete Ig (molecular weight 160,000 to 900,000) and heavy chains do not pass through the normal glomerular filtration barriers, the small light chains can freely pass through. These filtered proteins, reabsorbed by proximal tubular cells are then catabolized by lysozymal enzymes. Normally, this highly efficient process leaves only a minute amount of light-chain protein, which then appears in the urine. Thus, metabolism depends on the function of the proximal tubular cell and damage to these cells can result in increased excretion of light-chain proteins in the urine. In diseases in which the production of light-chain proteins is markedly increased, the ability of the proximal tubules to reabsorb all the filtered protein is

Corresponding author: Dr. Neelesh Jain, MD, Consultant-Transfusion Medicine, Balco Medical Centre, (A unit of Vedanta Medical Research Foundation), Atal Nagar, Raipur, Chhattisgarh, India, Tel: +91-9874592738; E-mail: drneeleshjain@gmail.com

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exceeded and light-chain proteins appear in the urine in high concentrations in this setting as well [4].

Light-chain proteinuria is common in WM; its prevalence ranging from 30% to 40%. Patients with WM have a plasma cell dyscrasia and they produce IgM paraproteins. As IgM has a tendency to form a pentamer, patients with the disease are much more likely to develop high serum viscosity than are patients with MM. The overall prevalence of Nephrotic Syndrome (NS) in Hodgkin disease is about 0.4% and is even lower in non-Hodgkin lymphomas (NHLs) [4]. Both idiopathic and malignancy-associated NS are thought to be mediated by a soluble permeability factor, still unidentified, which causes loss of selective capillary permeability and allows albumin and other negatively-charged molecules to cross the glomerular barrier. In lymphoma-associated NS, the permeability factor is supposed to be paraneoplastic in origin [4,5].

CONCLUSION

Lymphoma-associated kidney involvement occurs by a variety of mechanisms, which differ widely in prevalence and clinical presentation. One of our patient with WM and Salt losing nephropathy caused by IgM deposits, who subsequently treated with chemotherapy and resulted in an improvement to a normal renal function and disappearance of proteinuria.

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