ORIGINAL ARTICLE

IgA NEPHROPATHY IN NORTH WEST FRONTIER PROVINCE OF PAKISTAN

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ABSTRACT

Background: IgA nephropathy is the most common form of glomerulonephritis in the world. The extent to which IgA nephropathy is diagnosed depends upon the local attitude towards urine testing and renal biopsy. This study was conducted to know the frequency and behavior of IgA nephropathy in our setup.

Material and Methods: This study was conducted at Khyber Teaching Hospital Peshawar, from July 2005 to December 2006. Kidney biopsy was performed in 120 patients having glomerulonephritis. Those with IgA nephropathy were further studied.

Results: Out of 120 patients with glomerulonephritis, 25 (20.83%) were having IgA nephropathy. Among these 21 (84%) were males and 4 (16%) females. Majority of patients were 20-39 years old and had microscopic haematuria with non-nephrotic range proteinuria. Six (24%) patient had impaired renal function and they were also hypertensive. Only one (4%) patient had nephrotic range proteinuria and none had gross hematuria.

Conclusion: IgA nephropathy is not uncommon in our setup. Majority of patients have microscopic hematuria with non-nephrotic range proteinuria. Hypertension and renal insufficiency occurs in 24% of these patients.

Key words: IgA, Nephropathy, Proteinuria, Hematuria.

INTRODUCTION

IgA nephropathy is considered to be the most common form of glomerulonephritis in the world.[1] It is unique among glomerular diseases in being defined by immunohistochemical findings rather than by light microscopy. Although it is prevalent in all ethnic groups, Japan and Korea have the highest recorded incidence. Approximately 50% of new cases of glomerulonephritis and 40% of all end stage renal disease in Japan are due to IgA nephropathy. This is in contrast to United States and Western Europe, where IgA nephropathy accounts for 10-30% of glomerulonephritis. Although these disparities may reflect differences in the public health awareness and the willingness of nephrologists to perform diagnostic biopsies, certain populations seem to have a genetic predisposition to the development of IgA nephropathy.[1,2]

Patients with IgA nephropathy typically present in one of the three ways. Approximately 40-50% present with one or recurrent episodes of gross hematuria, usually following an upper respiratory infection. These episodes can be provoked by bacterial tonsillitis or by other viral upper respiratory infections. It is presumed, although not proven, that the first episode represents the onset of the disease. Patients may complain of flank pain during acute episode, which reflects stretching of the renal capsule. Low grade fever may also be present. These features can mimic urinary tract infection or urolithiasis. Another 30-40% have microscopic hematuria and usually mild proteinuria and are incidentally detected on a routine examination.[3,4,5] In these patients, the disease is of uncertain duration. Gross hematuria will eventually occur in 20-25% of these patients. Less than 10% patients present with either nephrotic syndrome or acute rapidly progressive glomerulonephritis picture, characterized by edema, hypertension and renal insufficiency as well as hematuria.

Rarely IgA nephropathy may present with malignant hypertension. It is usually presumed that these patients have longstanding disease not detected earlier because the patient did not have frank hematuria or routine urinalysis. Rarely, patients develop acute renal failure with or without oliguria. This may be due to crescentic IgA nephropathy, or to heavy glomerular hematuria leading to tubular occlusion and/or damage by red cells. The later is usually a reversible phenomenon, although incomplete recovery of renal function may occur.[6,7]

The extent to which IgA nephropathy is diagnosed depends upon the local attitude toward urine testing and renal biopsy. This study was conducted to know the frequency and behavior of IgA nephropathy in our setup.

MATERIAL AND METHODS

This study was conducted at Medical Unit B, Khyber Teaching Hospital Peshawar, Pakistan, from July 2005 to December 2006. Kidney biopsy was performed in 120 patients having glomerulonephritis. Those with IgA nephropathy were further studied. A written informed consent was obtained from all patients undergoing a kidney biopsy. They had their full blood count, renal function, prothrombin time and APTT checked and blood pressure controlled (BP<140/90 mmHg) before subjecting them to a kidney biopsy. All patients had anaemia corrected (Hemoglobin brought to 10g/dl). Ultrasound was performed by the operator on the bedside. Biopsy was deferred if there was hydronephrosis, solitary kidney, multiple cysts or small shrunken kidneys. Biopsy was performed in prone position with patients lying with the abdomen on a firm pillow. Lower pole of the kidney was localized by ultrasound and the site of insertion of biopsy needle was marked with ink. Kidney biopsy was performed with automated disposable gun (Monopty needle) under local anesthesia (2% lidocaine) and ultrasound guidance. Minimum two cores of renal tissue were obtained. Following biopsy, the patients were confined to bed for 6 hours and if remained stable were allowed to sit up and walk to the toilet. Pulse and BP was recorded every 15 minutes for 2 hours and then hourly for 6 hours. Two tablets of Distalgesic (Paracetamol + Dextropropoxiphen) were given routinely after the procedure and on need basis. After 24 hours the biopsy site was checked for hematoma and an ultrasound performed to check for perinephric hematoma, hydronephrosis and urinary bladder clot. They were discharged after 24 hours if remained hemodynamically stable with no evidence of local hematoma, macroscopic hematuria or urinary bladder clot with the advice to avoid exertion for the next seven days and to report any problem like pain, syncopal attacks or macroscopic hematuria.

RESULTS

Over this period 120 kidney having glomerulonephritis had renal biopsy, out of which 25 (20.83%) turned to have IgA nephropathy; 21 (84%) males and 4 (26%) females. Majority of patients were 20-39 years old. (Table-1)

All the 25 patients had microscopic hematuria and 24 had non-nephrotic range proteinuria. Six (24%) patients had impaired renal function and they were also hypertensive while the rest were normotensive with normal renal function. There was only one (4%) patient with nephrotic range proteinuria and none had gross hematuria. (Table-2)

The blood pressure of these patients is given below. Table-3

Table-1: Age distribution of patients with IgA nephropathy. (n=25)

11-19 (years)	20-29 (years)	30-39 (years)	40-49 (years)
4 (16%)	10 (40%)	8 (32%)	3 (12%)

Table-2: Indications for kidney biopsy.

Hematuria with non-nephrotic range proteinuria	Hematuria with nephrotic range proteinuria	Impaired Renal Function (Creatinine >1.5 mg/dl)
18 (72%)	1 (4%)	6 (24%)

Table-3: Blood pressure distribution.

120-140/ 80-90 (mm Hg)	141-160/ 90-100 (mm Hg)	161-200/ 101-110 (mm Hg)
16 (64%)	3 (12%)	6 (24%)

DISCUSSION

IgA nephropathy is the most common lesion found to cause primary glomerulonephritis.[1,2,6,7] Patients may present at any age but the peak incidence is in the second and third decades of life. There is approximately a 2:1 male to female predominance in North American and Western European populations, although this difference is not observed among populations in the Pacific Rim. IgA nephropathy occurs with greatest frequency in Asians and Caucasians and is relatively rare in blacks.[6,7,8] In a Chinese study of 13,519 renal biopsies, IgA nephropathy constituted 45% of all the cases of primary glomerulonephritis.[7,9,10] However, IgA deposits may also be seen on renal biopsy in individuals with no evidence of renal disease.[11] Variations in the disease prevalence may in large part reflect regional differences in the screening for kidney disease and kidney biopsy practices.[7,12] Many patients with IgA nephropathy are detected on routine urine screening since their

only clinical manifestation is asymptomatic hematuria and/or proteinuria. Prevalence may therefore appear to be higher in countries with an active urine testing program, such as Japan, where testing is routinely performed in schools and workplaces. Conversely, clinicians in North America seldom biopsy a patient with isolated hematuria or mild proteinuria, resulting in apparently lower disease prevalence. Our results show prevalence of 21% with male predominance and clustering of cases in the second and third decade of life.

Praga et al[6] reviewed the clinical course of 29 patients with gross hematuria secondary to IgA nephropathy and demonstrated that 37% developed transient renal failure. Although early renal dysfunction was more common in patients with gross hematuria, complete recovery was observed in all patients. These results are consistent with the previous observations that patients with intermittent gross hematuria do not develop proteinuria and generally have a better overall prognosis.[13] The vast majority of our patients had microscopic hematuria and non-nephrotic range proteinuria. Surprisingly none of our patients had gross hematuria. It may be due to the reason that patients with gross hematuria usually consult a urologist and may have escaped renal biopsy.

Renal insufficiency and hypertension is less common in IgA nephropathy but high in our study (24%). It may be due to the fact that the vast majority of our patient had a silent disease as compared to the international trend of recurrent gross hematuria.[7]

CONCLUSION

IgA nephropathy is not uncommon in our setup. Majority of patients have microscopic hematuria with non-nephrotic range proteinuria. Hypertension and renal insufficiency occurs in 24% of patients.

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