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ORIGINAL ARTICLE

Clinicopathological Analysis of 155 Patients with Persistent Isolated Hematuria

Rong-rong Li[1], Hang Li[2]*, Yu-bin Wen[2], Qing-yuan Huang[2], Lin Duan[2], and Yan Li[2]

[1]Department of Clinical Nutrition, [2]Department of Nephrology, Peking Union Medical College Hospital, Chinese Academy of Medical Sciences & Peking Union Medical College, Beijing 100730, China

Key words: isolated hematuria; renal biopsy; pathology; IgA nephropathy; thin basement membrane nephropathy

Objectives To reveal etiologies of persistent isolated hematuria (PIH) through ultrastructural pathological examination, to disclose clinicopathological correlation in cases with PIH, and to summarize appropriate management of patients with PIH.

Methods we retrospectively studied 155 PIH patients receiving renal biopsy between January, 2003 and December, 2008 in Peking Union Medical College Hospital. All the clinical data and follow-up result were analyzed.

Results All subjects included 38 children and 117 adults, with mean age of 11.38±3.25 years for children and 35.17±8.44 years for adults. Thin basement membrane nephropathy (TBMN) was the most common pathology (55.3% of children and 49.6% of adults), followed by IgA nephropathy (18.4% of children and 32.5% of adults, mainly grade 2-3) and mesangial proliferative glomerulonephritis (MsPGN) without IgA deposition (13.2% of children and 12.8% of adults). Besides, Alport syndrome (2.6% of children) and membrane nephropathy (2.6% of children and 0.9% of adults) were demonstrated as other causes of PIH. Elevated mean arteral pressure or protein excretion rate, as well as episodic macrohematuria, indicated higher risk for MsPGN rather than TBMN. On the other hand, severity of microhematuria was irrelevant to pathological types of PIH. Totally, 86 patients were followed up and 37 cases therein stayed on track for long term (mean duration 41.11±28.92 months, range 8-113 months). Most cases had benign clinical course except 3 cases with TBMN, 5 cases with IgA nephropathy, 1 case with MsPGN (without IgA deposition), and 1 case with Alport syndrome, who developed hypertension or proteinuria. All of them were administered timely intervention.

Conclusions Close follow-up should be required as the primary management for PIH. Equally important is careful monitoring for early identification of undesirable predictors; while renal biopsy and other timely intervention are warranted if there is hypertension, significant proteinuria or renal impairment.

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*Corresponding author Tel: 86-13911364903, E-mail: lihang9@hotmail.com

PERSISTENT isolated hematuria (PIH) is a common urinary abnormality frequently detected in health examination by routine urinalysis and phase-contrast microscopic examination, indicating dysmorphic erythrocytes originating from glomerules. The patients barely had other clinical symptoms including hypertension, renal insufficiency, proteinuria, or any other extrarenal causes of hematuria. [1] As a prevalent condition, PIH has been demonstrated to be present in 0.1% to 2.7% of adolescents and adults, [2, 3] while in 0.008% to 2.0% of children by many large-sample studies. [4-10]

Despite the frequency of PIH, evidence-based guidelines have been lacking for its proper evaluation, management and prognosis. Some nephrologists consider PIH as an early sign of chronic renal disease for which renal biopsy is necessary for early detection and treatment to reduce risk of end-stage renal disease. [11, 12] While others assert that PIH has a benign course so that it will be more rational to take a "wait and watch" approach rather than perform renal biopsy. [13] We conducted a retrospective study to analyze the clinical and pathological features of PIH, and provide practical implication of management for such patients.

PATIENTS AND METHODS

Patients

From January, 2003 to December, 2008, 4360 patients underwent renal biopsy at the Department of Nephrology in Peking Union Medical College Hospital (PUMCH), which included 155 consecutive individuals (3.56%) presenting with PIH. The diagnosis of PIH was defined as: (1) an erythrocyte excretion rate ≥3 erythrocytes per high-power field (HPF) revealing dysmorphic red blood cells from centrifuged urine obtained on 3 or more separate occasions over 4 or more weeks; (2) urinary protein excretion <0.3 g/24 h prior to renal biopsy; (3) serum creatinine values within the normal range (≤132.6 μmol/L) and the estimated glomerular filtration rate ≥60 ml/(min·1.73 m^2) by Modification of Diet in Renal Disease (MDRD) equation; (4) normal blood pressure (systolic pressure <140 mm Hg and diastolic pressure <90 mm Hg). All the clinical and pathological data (including light microscopy, immunofluorescence, and electron microscopy results) were intact and complete.

Patients with PIH were divided into 4 groups according to various clinical features: (1) persistent isolated microscopic hematuria with family history of hematuria, nephropathy or renal failure (PIMH+FH); (2) persistent isolated microscopic hematuria with episodic gross hematuria (PIMH+GH); (3) persistent isolated microscopic hematuria with episodic gross hematuria as well as positive family history (PIMH+FH+GH); (4) persistent isolated microscopic hematuria as the sole manifestation (PIMH).

Fifty-four patients (including 14 children and 40 adults) received quantification of urinary erythrocyte count in urinalysis. The severity of hematuria was defined as three levels: 3-5/HPF, 6-10/HPF, and >10/HPF.

All the PIH patients were scheduled for periodic follow-up visits to nephrologists. Urinalysis was recommended to be performed at 1- to 3-month intervals, and assessment of blood pressure and renal function was also required at follow-up visits.

Renal pathology evaluation

Renal biopsies were obtained through a standard percutaneous technique. Tissue containing more than 10 glomeruli was fixed and embedded into paraffin sections for light microscopy, immunofluorescence, and electron microscopy examination. In electron microscopic examination, ultrastructure of renal tissue as well as electron-dense immune deposits were reviewed and photographed by JEOL-1010 transmission electron microscope (Japan Electron Optics Laboratory Co., Ltd.). The diagnosis of mesangial proliferative glomerulonephritis (MsPGN) is made when light microscopy reveals an increase in mesangial matrix, hypercellularity and other glomerular lesions including focal necrosis, segmental scarring, and crescents in Bowman's space; while electron microscopy shows electron-dense material mainly in the mesangial and paramesangial areas corresponding to immune deposits on immunofluorescence microscopy. Predominance of IgA deposits in the glomerular mesangium is the diagnostic hallmark of IgA nephropathy (IgAN). [14] Features of IgAN on light microscopy is classified according to Lee's histological grading system [15] as follows: grade I, mostly normal glomeruli; grade II, <50% glomeruli show segmental mesangial hypercellularity and sclerosis; grade III, diffuse mesangial proliferation with occasional adhesions and small cresents; grade IV, diffuse marked mesangial proliferation and ≤45% glomeruli having cresents with frequent segmental and global sclerosis; grade V, similar to IV but more severe with >45% glomeruli showing cresents. Thin basement membrane nephropathy (TBMN) was diagnosed when diffuse thinning of the glomerular basement membrane (GBM, GBM thickness less than 200 nm [16] for children or less than 280 nm for adults) existed in the majority of capillary loops with at least 80% of the GBM involved in individual capillaries.

Statistical analysis

Data were analyzed by SPSS 16.0 statistical software.

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