ORIGINAL ARTICLE

Ten-year review of disease pattern from percutaneous renal biopsy: an experience from a paediatric tertiary renal centre in Hong Kong

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Objective To study the childhood renal disease pattern based on the renal biopsy histology in a local paediatric tertiary renal centre.

Design Retrospective study.

Setting Department of Paediatrics and Adolescent Medicine, Princess Margaret Hospital, Hong Kong.

Patients All patients who underwent real-time ultrasound-guided closed renal biopsy from 1 April 1997 to 31 March 2007 were included.

Results A total of 209 renal biopsies were performed, 162 on native kidneys and 47 on grafts. In the native group, major indications were renal manifestations secondary to systemic diseases (34%), followed by idiopathic nephrotic syndrome (28%) and haematuria (27%). In 94% the histopathology revealed glomerular diseases. Among the primary glomerular diseases, thin glomerular basement membrane disease, immunoglobulin A nephropathy, minimal change disease, and focal segmental glomerulosclerosis accounted for most. In all, 37% of patients with steroid-resistant nephrotic syndrome had focal segmental glomerulosclerosis and its relative incidence was increased when compared to previous studies. Minimal change disease and minimal change disease with mesangial immunoglobulin M deposits accounted for the majority of steroid dependent and frequent relapsers. Among patients with isolated microscopic haematuria, 73% had thin glomerular basement membrane disease, while patients with concomitant haematuria and proteinuria had a wide variety of pathology. In the kidney graft group, acute graft dysfunction was due to acute rejection in 38% of the patients, followed by calcineurin inhibitor toxicity in 14%. Chronic allograft nephropathy caused chronic allograft dysfunction in the majority of cases. Post-transplant proteinuria was caused by recurrence of the primary renal disease in all of our patients.

Conclusion This study provides updated epidemiological information for childhood renal disease and a change in the pattern of disease was observed.

Key words
Biopsy; Glomerulonephritis, IGA; Glomerulosclerosis, focal segmental; Kidney transplantation; Nephrotic syndrome

Hong Kong Med J 2008;14:348-55

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Introduction

Percutaneous renal biopsy is a commonly performed procedure in the assessment of kidney diseases in children. It can provide diagnostic precision, especially in glomerular diseases, and also provides important information of prognostic value and about treatment options.

Data of childhood renal diseases were available from studies of renal biopsy in different countries.[1-3] A review of paediatric renal biopsies in 1991 to 1993 was undertaken in Hong Kong by Wong et al,[4] and an update of such information was necessary. Therefore the objectives of this study were: (1) to study the epidemiology of childhood renal disease based on renal biopsy histology in a local paediatric tertiary renal centre; (2) to analyse specific groups including those with idiopathic nephrotic syndrome, immunoglobulin A (IgA) nephropathy, lupus nephritis, and haematuria, and (3) to review the histology in biopsies from graft kidneys.

Methods

Patients

We reviewed the histology from all patients admitted to the Department of Paediatrics and Adolescent Medicine at Princess Margaret Hospital (Hong Kong) from 1 April 1997 to 31 March 2007, who had undergone real-time ultrasound-guided closed renal biopsy. Renal biopsy was performed using gauge-16 or -18 biopsy needles, and two cores of tissue were taken each time. Conventional light microscopy, immunofluorescent and ultrastructural studies were performed on each specimen.

Data

Data were collected retrospectively from records. Patients were divided into the native kidney biopsy and kidney graft biopsy groups. Failure of renal biopsy was defined as inadequate numbers of glomeruli obtained for diagnostic purposes.

Native kidney biopsy group

Indications for native kidney biopsy were classified as: (1) nephrotic syndrome (defined as spot first-voided urine albumin-to-creatinine ratio greater than 2 mg/mg or 24-hour urine protein greater than 40 mg/m^2/h; hypoalbuminaemia, hyperlipidaemia, and oedema); (2) persistent microscopic haematuria; (3) recurrent gross haematuria; (4) haematuria with proteinuria (proteinuria was defined as spot first-voided urine albumin-to-creatinine ratio greater than 0.2 mg/mg); (5) significant proteinuria (defined as spot first-voided urine albumin-to-creatinine ratio greater than 0.2 mg/mg); (6) acute renal failure; (7) chronic renal failure; and (8) secondary to systemic diseases.

Indications for biopsy in idiopathic nephrotic syndrome were: (1) steroid resistant (SR) [defined as persistent proteinuria of more than 40 mg/m^2/h after 4 weeks of treatment]; (2) steroid dependent and frequent relapser (SDFR) before using potent steroid-sparing drugs such as cyclophosphamide or cyclosporine A (steroid dependent was defined as two consecutive relapses within 14 days after complete withdrawal of steroid treatment or during the tapering phase of treatment; frequent relapser was defined as two or more relapses in 6 months or four or more relapses in any 1 year); and (3) atypical features including age less than 1 year or more than 10 years, gross haematuria, hypertension, and renal impairment. The characteristics of specific groups including idiopathic nephrotic syndrome, IgA nephropathy, lupus nephritis, and haematuria were analysed.

Kidney graft biopsy group

Indications for kidney graft biopsy were: (1) acute graft dysfunction (defined as more than 10% increase in baseline creatinine in preceding 2 weeks); (2) chronic graft dysfunction (defined as more than 10% increase in baseline creatinine for 6 months or more); (3) delayed graft function (defined as dialysis dependence immediately after kidney transplantation); and (4) proteinuria (defined as spot first-void urine albumin-to-creatinine ratio greater than 0.2 mg/mg). There was no protocol biopsy (defined as a biopsy at a predefined time, without any clinical indication) in our series.

Statistical analysis

Data were analysed using the Statistical Package for the Social Sciences (Windows version 15.0; SPSS Inc, Chicago [IL], US). Mean serum IgA was compared between IgA nephropathy and non-IgA nephropathy groups, using the Student's *t* test. A statistically significant difference was assumed when the P value was less than 0.05.

Results

Over the 10-year period of our study, 209 renal

利用十年經皮腎活檢案例檢視腎病病譜：香港一所兒童三級腎科醫療中心的經驗

目的 利用腎組織病理活檢資料，研究兒童腎病病譜。

設計 回顧研究。

安排 香港瑪嘉烈醫院兒科及青少年科。

患者 1997年4月1日至2007年3月31日期間，所有接受過實時超聲引導閉合性腎活檢的病人。

結果 腎活檢案例共有209宗，其中162例為原腎，47例屬移植腎。在原腎一組病例中，主要徵狀為系統性疾病繼發腎損害(34%)，次之分別為腎病綜合徵(28%)和血尿症(27%)。組織病理分析數據顯示94%有腎小球疾病。在原發性腎小球疾病類中，腎小球薄基底膜病、IgA腎病高血壓、輕微腎小球病變和局灶節段性腎小球硬化症佔大部分。整體而言，37%出現激素耐藥腎病綜合徵的病人同時患有局灶節段性腎小球硬化症，與過往多個研究相比，比較發生率有所增加。激素依賴型和經常復發型案例多數出現輕微腎小球病變與輕微腎小球病變兼有系膜IgM沉積。在出現單純鏡下血尿的患者中，73%兼有腎小球薄基底膜病，而衍生型血尿症和蛋白尿症的病人則出現大量不同的病理現象。在移植腎一組的病例中，造成急性腎功能衰竭的原因，急性排斥的佔38%，鈣調素抑制劑中毒佔14%。大部分病例均為慢性移植腎腎病導致慢性移植腎功能減退。所有病人都因為原發性腎病復發以致接受移植手術後患上蛋白尿。

結論 本研究就兒童病提供了更新的流行病學資料，並發現病譜有所變化。

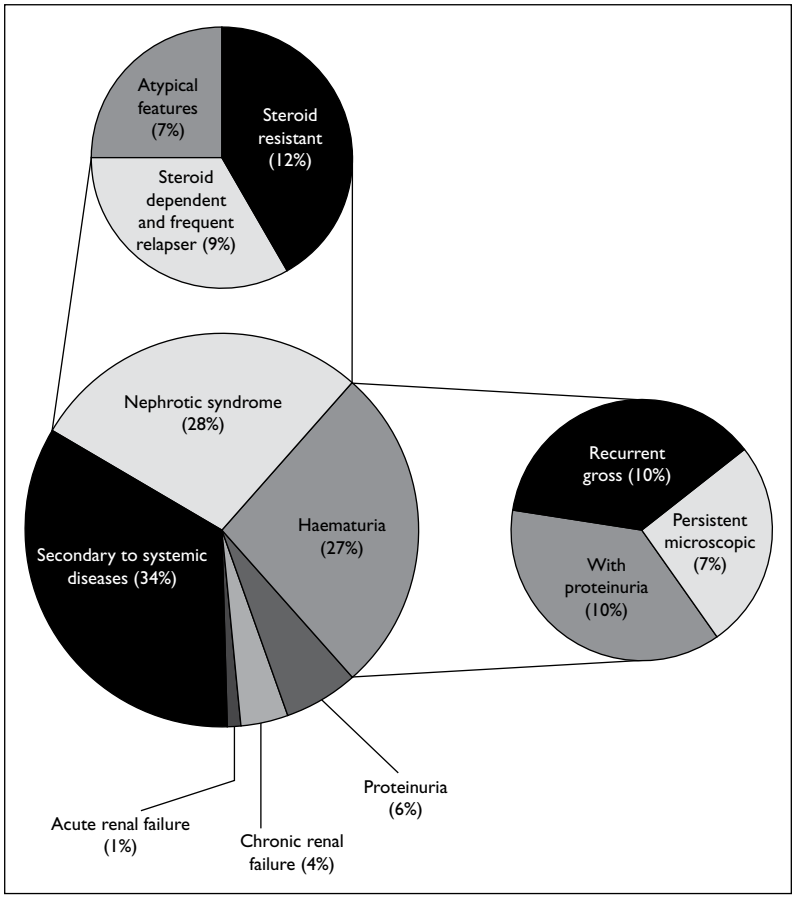

FIG. Main indications for biopsy of native kidneys

biopsies were performed in 171 patients, with an average of 21 per year; 162 (78%) were performed on native kidneys and 47 (22%) on grafts.

Native kidney biopsy group

A total of 162 renal biopsies in 150 patients (mean age, 11 years; standard deviation [SD], 5 years; range, 2-24 years) were performed on native kidneys during the 10-year study period. Among these, 13% were aged 0 to 4 years, 31% 5 to 9 years, 33% 10 to 14 years, 18% 15 to 18 years, and 6% 19 years or more. The male-to-female ratio was 1 to 1.1. The success rate was 99% (161/162). The Figure shows the different indications for renal biopsy; the most common being renal manifestations of systemic diseases, followed by idiopathic nephrotic syndrome.

Table 1 shows the pattern of histopathological diagnoses. Glomerular diseases accounted for 94% (152/161) of the cases; 66% (100/152) were primary glomerular diseases while 34% (52/152) were secondary. Common causes of primary glomerulonephritis (GN) were minimal change disease (MCD), IgA nephropathy, and thin glomerular basement membrane (GBM) disease, while lupus nephritis was the most common secondary cause. Patient age at biopsy and the clinical presentations for the commonest pathologies are shown in Table 2.

For patients with idiopathic nephrotic syndrome, mean (± SD) age at biopsy was 8±5 years (range, 2-17 years), and the male-to-female ratio was 2.3:1. When comparing patients with SR nephrotic syndrome and SDFR nephrotic syndrome, the mean age at biopsy was similar (7±5 years vs 7±4 years, respectively), while a male predominance was observed in SDFR group (male-to-female ratio was 0.6:1 vs 9:1, respectively). Focal segmental glomerulosclerosis (FSGS) patients had a younger mean age (6±4 years) and a male-to-female ratio of 1.2:1. C1q nephropathy patients had a mean age of 8±5 years and a male-to-female ratio of 4:1. Histopathology findings in patients with idiopathic nephrotic syndrome are shown in Table 3, and commonly included MCD (50%, 22% with IgM deposits) and FSGS (17%). Most SR patients had FSGS (37%) or MCD (37%), and C1q nephropathy (16%), while MCD with IgM deposits was uncommon (5%). On the other hand, most SDFR patients had MCD (73%, of whom 33% had IgM deposits), while only 7% had FSGS.

For patients with IgA nephropathy, mean (± SD) age of symptom onset was 11±4 years (range, 4-20 years) and the mean age at biopsy was 13±4 (range, 4-21) years, and the male-to-female ratio was 1:1.1. Indications of biopsy were recurrent gross haematuria (25%), haematuria with proteinuria (35%), nephrotic syndrome (20%) or microscopic haematuria (5%). Renal impairment, acute renal failure, or chronic renal failure each accounted for about 5% of the indications. Serum IgA was available for analysis from 16 of the 20 patients; the mean (± SD) value was 2.9±1.1 g/L with a range of 1.2 to 4.9 g/L (reference range, 0.8-3.0 g/L). Among these, 38% had levels exceeding 3.0 g/L. Analysis of all patients with the indication of haematuria with or without proteinuria for biopsy revealed that the mean IgA level was 3.0±1.1 g/L in those with confirmed IgA nephropathy (n=13, data were missing for one) and 1.6±0.9 g/L in those with non-IgA nephropathy (n=26, data were missing for five) [P<0.005].

In our 11 patients with isolated microscopic haematuria, thin GBM disease accounted for eight cases while the other three were due to IgA nephropathy, minor glomerular abnormalities, and mesangioproliferative GN. In those with recurrent gross haematuria, nine of them had thin GBM disease and five had IgA nephropathy. However, in patients having concomitant haematuria and proteinuria, there was a wide variety of pathology; only two of 17 patients had thin GBM disease, whilst the others had MCD, thrombotic microangiopathy, mesangioproliferative GN, crescentic GN, and diffuse proliferative GN.

Graft biopsy group

A total of 47 renal graft biopsies were carried out in 26 Chinese patients, varying from one to five biopsies per patient. The mean (± SD) age of the patients at the time of biopsy was 17±6 years (range, 5-26 years); the male-to-female ratio was 1.5:1. The time interval from transplantation ranged from 4 days to 119 months. The success rate was 94% (44/47). There were four major indications for biopsy: (1) acute graft dysfunction (29/47); (2) chronic graft dysfunction (11/47); (3) delayed graft function (4/47); and (4) significant proteinuria (3/47). Biopsy pathologies are shown in Table 4. A wide spectrum of pathologies was identified for patients with acute graft dysfunction. Proteinuria after transplantation was secondary to recurrence of primary disease in all three patients.

Discussion

From the histopathology of the renal biopsy, disease patterns and changes over time can be analysed. In Hong Kong, limited data from 1991 to 1993 and 1998 to 2000 were reported by Wong et al.[4,5] Our study aimed to provide updated epidemiological data of childhood renal disease from renal biopsies in a local paediatric tertiary renal centre in Hong Kong.

The new automated ultrasound-guided percutaneous renal biopsy is safe in children. Success rate is high and failure to reach a diagnosis is rare, especially in experienced hands. In our centre, the failure rate was 0.6% in native kidneys and 6% in renal grafts, as compared to 3.3% and 2.2% respectively in Feneberg et al's series[6] and 1.3% in native kidneys reported from Japan.[7] Our success rate was encouraging in native kidneys, but limited for grafts, which may be related to surrounding fibrosis and a few difficult cases.

TABLE 1. Histopathology encountered in native kidney biopsies

Reported pathology*		Patients (n=161)† No. (%)
Primary glomerular disease	Minimal change disease • with mesangial IgM deposits	23 (14) 10 (6) out of 23
	IgA nephropathy	20 (12)
	Thin glomerular basement membrane disease	19 (12)
	Focal segmental glomerulosclerosis	11 (7)
	Other GN • Mesangioproliferative GN • Membranous GN • Crescentic GN • Diffuse proliferative GN • Focal proliferative GN • Immune complex GN	13 (8) 6 1 3 1 1 1
	C1q nephropathy	5 (3)
	Alport's syndrome	5 (3)
	Minor glomerular abnormalities	4 (2)
Secondary glomerular disease	Lupus nephritis • WHO type II • WHO type III • WHO type IV • WHO type V • WHO type VI • WHO type III and V • WHO type IV and V • Thrombotic microangiopathy	37 (23) 1 11 12 2 1 4 5 1
	Henoch-Schönlein purpura nephropathy	13 (8)
	Diabetes nephropathy	1 (1)
	Hepatitis B–related membranous GN	1 (1)
Acute tubular necrosis		1 (1)
Tubulointerstitial nephritis		1 (1)
Thrombotic microangiopathy		1 (1)
Goodpasture syndrome		1 (1)
Chronic interstitial nephritis		1 (1)
No abnormalities		4 (2)

* IgM denotes immunoglobulin M, IgA immunoglobulin A, GN glomerulonephritis, and WHO World Health Organization

† Total number was 161 because one biopsy yielded an inadequate sample

Native kidney biopsy

Different centres may differ with respect to selection criteria for biopsies. Nephrotic syndrome was the most common indication for renal biopsy in our study, and was similar to findings from other series.[1,8]

Idiopathic nephrotic syndrome

Focal segmental glomerulosclerosis occurred in 8% of those biopsied for idiopathic nephrotic syndrome from 1991 to 1993 in Wong et al's study,[4] while it accounted for 17% in our series. Overall incidence of FSGS was increasing in Hong Kong, which is similar to the worldwide trend.[9-11] The International Study of Kidney Disease in Children reported in the 1970s that FSGS was observed in only 5 to 7% of biopsies of idiopathic nephrotic syndrome patients.[12] However, recent studies show that its incidence is increasing, for example, a study in US by Bonilla-Felix et al[10] showed that FSGS was noted in 31% of their biopsies from children with idiopathic nephrotic syndrome; African American ethnicity was conferred an even stronger predominance of FSGS. In our study, this trend was also evident for Chinese ethnicity, and we can expect to encounter more difficulties in managing cases with FSGS in the future.

Minimal change disease with mesangial IgM deposits comprised one third of our cases in SDFR groups. Compared to the previous studies by Wong et al,[4,5] the incidence of MCD with mesangial IgM deposits has increased. There is still controversy as to whether cases with IgM deposits should be regarded as a spectrum of MCD or a separate entity such as IgM nephropathy. In recent years IgM nephropathy is being increasingly identified and recognised, in which it is defined immunohistochemically by diffuse

TABLE 2. Age distribution and clinical presentations of common pathological diagnosis

Pathology*	Age at time of biopsy (years) Mean±SD (range)	Clinical presentation†
Minimal change disease		
• Without IgM deposits	8±5 (2-17)	Nephrotic syndrome (11/13) ARF (1/13) Haematuria with proteinuria (1/13)
• With IgM deposits	10±5 (4-16)	Nephrotic syndrome (10/10)
IgAN	13±4 (4-21)	Haematuria with proteinuria (7/20) Recurrent gross haematuria (5/20) Nephrotic syndrome (4/20) ARF/CRF/renal impairment (1/20 for each) Persistent microscopic haematuria (1/20)
Thin glomerular basement membrane disease	10±4 (5-17)	Recurrent gross haematuria (9/19) Persistent microscopic haematuria (8/19) Haematuria with proteinuria (2/19)
Focal segmental glomerulosclerosis	6±4 (2-12)	Nephrotic syndrome (8/11) Proteinuria (2/11) CRF (1/11)
C1q nephropathy	8±5 (2-17)	Nephrotic syndrome (5/5)
Alport's syndrome	9±4 (3-12)	Haematuria with proteinuria (2/5) Nephrotic syndrome (1/5) Proteinuria (1/5) Family history of Alport's syndrome (1/5)
Lupus nephritis	15±4 (8-24)	Nephrotic range proteinuria (23/37) Haematuria with proteinuria (8/37) Non-nephrotic range proteinuria (5/37) Renal impairment (1/37)
HSP nephropathy	10±5 (4-22)	Nephrotic syndrome (9/13) Haematuria with proteinuria (4/13)

* IgM denotes immunoglobulin M, IgAN immunoglobulin A nephropathy, and HSP Henoch-Schönlein purpura
† ARF denotes acute renal failure and CRF chronic renal failure

TABLE 3. Histopathology reported for various indications of renal biopsy in nephrotic syndrome

Pathological diagnosis*	Indications for biopsy					
	Steroid resistant	Steroid dependent, frequent relapser	Atypical features			
			Gross haematuria	Atypical age	Family history of hereditary GN	Renal failure
Minimal change disease						
• Without IgM deposits	6 (32%)	6 (40%)	-	-	-	1
• With IgM deposits	1 (5%)	5 (33%)	1	2	-	1
Focal segmental glomerulosclerosis	7 (37%)	1 (7%)	-	-	-	-
C1q nephropathy	3 (16%)	2 (13%)	-	-	-	-
GN						
• Mesangioproliferative GN	-	1 (7%)	1	-	-	-
• Mesangial GN	-	-	-	-	-	-
• Crescentic GN	-	-	-	1	-	-
• Immune complex GN	1 (5%)	-	-	-	-	1
IgAN	1 (5%)	-	2	1	-	-
Alport's syndrome	-	-	-	-	1	-
Total	**19**	**15**	**4**	**4**	**1**	**3**

* IgM denotes immunoglobulin M, GN glomerulonephritis, and IgAN immunoglobulin A nephropathy

mesangial staining of glomeruli for IgM. Varying morphology is characteristic, and can vary from normal glomeruli to mesangial hyperplasia of varying degrees with or without segmental or global sclerosis. Some studies showed that IgM nephropathy has a poorer prognosis and patients were less likely to respond to steroids and immunosuppressive agents than those with MCD.[13-16] In our series, patients with IgM deposits presented at atypical ages, and most were SDFRs. Considering this entity as a separate disease has important prognostic implications. However, we have no data of the incidence of IgM

TABLE 4. Histopathological diagnosis in allograft biopsies

Biopsy result*	Delayed graft function	Acute graft dysfunction	Chronic graft dysfunction	Significant proteinuria
Acute rejection (cellular or humoral)	1	11	1	-
Chronic allograft nephropathy	-	1	7	-
Acute tubular necrosis	3	1	-	-
CNI toxicity	-	4	-	-
Recurrence of FSGS	-	3	1	1
Recurrence of IgAN	-	-	-	2
Polyomavirus-associated nephropathy	-	3†	-	-
Tubulointerstitial nephritis	-	1	-	-
Acute pyelonephritis	-	1	-	-
No abnormalities	-	3	-	-
Inadequate for diagnosis	-	1	2	-
Total	**4**	**29**	**11**	**3**

* CNI denotes calcineurin inhibitor, FSGS focal segmental glomerulosclerosis, and IgAN immunoglobulin A nephropathy

† Three serial results from one patient for monitoring of possible rejection

deposits in steroid-sensitive patients, because biopsy was not performed in them.

The histology was different in the SR group when compared to the SDFR group. The former tended to have more FSGS (37%) and C1q nephropathy (16%), while the latter tended to have more MCD with IgM deposits (33%). Also notable was the remarkably different sex ratios in our series; marked male predominance was noted in SDFR and C1q nephropathy cases. Our FSGS patients were younger than the whole group with idiopathic nephrotic syndrome, which was different from other series reporting FSGS to be more common in older patients.[10,17]

Secondary glomerulonephritis, lupus nephritis

One third of our biopsies were performed because of systemic diseases and one third of the glomerular diseases turned out to be secondary GN. Systemic lupus erythematosus is common in the Asian population and clinically evident lupus nephritis occurred in at least 75% of children with systemic lupus erythematosus,[18] therefore, in our series a high proportion of lupus nephritis was expected. Lupus nephritis is second only to infection as the most common cause of mortality in lupus patients. The 5-year renal survival in childhood-onset lupus nephritis ranged from 44 to 93% in different studies. Type IV lupus nephritis was shown to have worse renal survival than other types.[19,20] Therefore, in lupus patients with renal involvement, renal biopsy is very important as a guide to treatment options and prognosis counselling.

In our series, only one hepatitis B virus–related GN was identified, compared to seven in Wong et al's series in the early 1990s.[4] Our patient was an adolescent boy born in Mainland China who had not received hepatitis B vaccine at birth. The incidence of hepatitis B–related GN has been decreasing in recent years, due to implementation of newborn hepatitis B virus vaccination since 1988. Immunisation history, especially for immigrants, is particularly important.

Immunoglobulin A nephropathy

The most common primary GN was IgA nephropathy, which is consistent with other Asian and European series.[1,2] Among those presenting with haematuria with or without proteinuria, serum IgA levels were shown to be higher in IgA nephropathy as opposed to other patients; the difference being statistically significant. Serum IgA was reported to be high in only 10 to 15% of children in other studies.[21] In our series, 38% of patients with confirmed IgA nephropathy had a raised serum IgA level (>3.0 g/L), indicating that a raised level seemed to be a slightly more sensitive indicator in our locality. Thus, whilst serum IgA cannot be used as a diagnostic test, it is a useful adjunct in the diagnostic process, whenever a raised level is encountered in a patient with haematuria. Renal biopsy is still the gold standard for confirmation of IgA nephropathy.

Microscopic haematuria

The utility of renal biopsy in children with isolated microscopic haematuria is controversial. In a Japanese cohort, it was found that 37% of patients with persistent microscopic haematuria for more than 6 months had normal histology; while 34% had thin GBM disease and 16% had IgA nephropathy.[22] In the

same study, if there was coexisting proteinuria, 46% had IgA nephropathy, 25% had normal histology, and 18% had thin GBM disease. In our centre, children with isolated microscopic haematuria biopsy would have been considered in conditions associated with a positive family history, hypertension, impaired renal function, patient preference, or parental anxiety. In all, 73% had the pathology of thin GBM disease, while IgA nephropathy, minor glomerular abnormalities, or mesangioproliferative GN each accounted for 9%. When compared to the Japanese cohort, we had a greater proportion of patients with thin GBM disease and a smaller percentage with normal pathology and IgA nephropathy. In our patients with concomitant haematuria and proteinuria, we also encountered much less thin GBM disease and normal pathology than the Japanese cohort, though we did come across other pathologies (eg diffuse proliferative GN, crescentic GN) mandating more aggressive treatment. In conclusion, renal biopsy may not be clinically useful in isolated microscopic haematuria patients. However, close follow-up is important and biopsy is indicated if other renal manifestations (eg proteinuria or hypertension) should develop.

Graft biopsy

Renal biopsy is often needed to confirm the cause of graft dysfunction as a clinical diagnosis is difficult to establish.[23] Compared to other studies,[23,24] in our series, delayed graft function was rarely an indication for renal biopsy. In patients with delayed graft function, biopsy is useful for differentiating acute rejection from acute tubular necrosis. In acute graft dysfunction, the diverse pathological causes can be reflected in the renal biopsy, and is important for the purpose of confirmation and as a guide to further treatment options. In our series, pathological calcineurin inhibitor (CNI) toxicity is not common and is sometimes difficult to correlate clinically and pathologically. For the four pathologically confirmed patients with CNI toxicity, they all presented with acute graft dysfunction and only three of them showed a raised CNI level. Other pathologies were demonstrated in three patients with suspected CNI toxicity (raised CNI levels and acute graft dysfunction).

Overall

There were several limitations to our study. The sample size was relatively small because our patients were from a single centre. Most paediatric renal transplantations in Hong Kong were performed in our centre, therefore our results for kidney graft biopsy were representative. However, our native kidney sample may represent a more severe spectrum of the disease, owing to only difficult cases being referred to us. This may explain the increased incidence of FSGS in our series, although a rising trend has also been noted worldwide. Finally, data were collected retrospectively, mainly from records and missing data were encountered. A prospective study is necessary to standardise data measurement, biopsy staining and interpretation.

Conclusion

This study provides updated epidemiological data for childhood renal disease patterns in a paediatric tertiary renal centre in Hong Kong and demonstrates a change in disease pattern.

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