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ORIGINAL ARTICLE

Outcome of severe acute post-streptococcal glomerulonephritis in New Zealand children

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Abstract Post-streptococcal glomerulonephritis (PSGN) is the commonest cause of severe acute glomerulonephritis in New Zealand children, with the majority (85%) of the patients being of either Pacific Island or Maori ethnicity. We have performed a retrospective study on 27 pediatric patients with acute PSGN. Of these patients, those with crescentic glomerulonephritis (n=11) had a greater tendency (72.7%) for needing acute dialysis and were left with persistent urinary sediment abnormalities after a mean follow-up of 3.2 years (95% confidence interval 2.1–4.3). The efficacy of immunosuppression in the group with crescentic disease was uncertain. The severity of renal histopathological abnormalities as judged by the total biopsy score did not correlate with either presentation or eventual outcome. Severe childhood acute post-streptococcal glomerulonephritis, although uncommon, results in significant long-term renal morbidity, particularly among Maori and Pacific Island children.

Keywords Acute glomerulonephritis · Crescentic · Infection · Post-streptococcal · Rapidly progressive children

Introduction

Over the past 30 years, there has been a progressive decline in the incidence of acute post-streptococcal glomerulonephritis (APSGN) in developed countries [1–5]; however, among New Zealand children, APSGN is the commonest cause of acute glomerulonephritis. In a recent unpublished study undertaken between January 2006 to April 2007, 111 cases were identified in two children's inpatient facilities in Auckland, New Zealand, resulting in a mean annual admission rate of 30–62 per 100,000 children under the age of 14 years between these two centers. These two pediatric inpatient facilities serve a total population of just over 1.2 million where the local population comprises 61.5% Europeans, 11% Maori, 13.2% Pacific Island people and 10% Asians.

The severity of APSGN varies from subclinical disease [6, 7] to rapidly progressive glomerulonephritis (RPGN) needing acute dialysis [8–10]. In most pediatric studies of crescentic glomerulonephritis (CGN) or RPGN, post-infectious or post-streptococcal disease constitute a small proportion of cases. In one of the largest pediatric series of CGN, the South West Pediatric Nephrology Study Group reported 50 children with CGN, of whom only six were due to APSGN [8]. In another series of CGN by Jardim [9], only two of 30 children had APSGN as the primary diagnosis. In these two series vasculitides due to Henoch–Schönlein purpura or systemic lupus erythematosus were collectively the most common cause of CGN. In contrast, our own experience has been very different, with APSGN being the leading cause of crescentic or rapidly progressively glomerulonephritis.

We report here a retrospective study of children who had been referred to our institution on the basis of a renal biopsy between 1995 and 2007 for investigation and management of APSGN. This report describes the management and outcome of APSGN in these patients for up to 8 years after initial diagnosis.

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Patients and methods

All children had been diagnosed with APSGN on the basis of renal biopsy were identified from a database at our institution. The diagnostic criteria for APSGN were: (1) elevated streptococcal titers; (2) transiently depressed C3 complement; (3) absence of past history of chronic renal disease. The diagnosis of APSGN was made if the patients' clinical and laboratory findings and subsequent progression of the symptoms were consistent with the disease. Clinical details from the case records of the patients were extracted. The patient group was analyzed as a whole and also as separate groups consisting of those with biopsies that showed 50% or more crescents (group 1, $n=11$) and those with fewer than 50% crescents in their glomeruli (group 2, $n=16$) The histopathology was reviewed by a single histopathologist (JZ) who had no previous knowledge of the clinical history or outcome of the group. The biopsies were scored according to a modification of the method of Clark et al. [11]. Scores were assigned semi-quantitatively on a scale of 1–4 for the following five microscopic features: mesangial cellularity, mesangial matrix expansion, neutrophil infiltration, tubular atrophy and interstitial inflammatory cell infiltration. A score for cellular crescents (scale 1–4) was also obtained, with one point for each 25% of glomeruli affected. The total score of all six features for each case was then calculated (maximum 24 points). All renal biopsies were examined by light microscopy, direct immunofluorescence and electron microscopy. Indications for renal biopsy were: (1) anuric renal failure; (2) acute glomerulonephritis with a severe reduction in renal function and creatinine more than fourfold higher than upper limit or normal; (3) mixed nephritic nephrotic syndrome; (4) delay in recovery of renal function within 2–3 weeks after the onset of acute nephritis. All children had either gross hematuria with cellular casts or 3+ to 4+ blood on urinalysis and proteinuria at the time of their renal biopsies. Nephrotic range proteinuria was defined as either 4+ protein on dipstick testing or a urine protein to creatinine ratio greater than 200 mg/mmol. A mixed nephritic nephrotic syndrome was defined as patients who had features of both illnesses with gross hematuria, impaired renal function and nephrotic proteinuria.

Streptococcal titers were compared with previous published data and were considered to be clinically significant if they exceeded the following upper limits: anti-streptolysin O (ASO) ≥ 480 Todd units ($n<170$), anti-DNAase B≥ 680 ($n<170$), and anti-hyaluronidase (AHT) ≥ 1024 ($n<256$) (New Zealand Heart Foundation Guide for Diagnosis of Acute Rheumatic Fever; www.nhf.org.nz.

Statistical analysis was performed using the software package GraphPad Instat 3.0 (GraphPad Software, San Diego, USA). The non-parametric t test and Fisher exact test were used to assess significance, and p values less than 0.05 were considered to be statistically significant.

The study was performed in accordance with the ethical standards laid down by the Declaration of Helsinki.

Results

Demographics and clinical features

Twenty-seven children, of whom 16 were males, were identified. The median age at diagnosis was 9.4 years (range 3.5–13.2). The majority of children with severe APSGN were either of Maori (11/27, 41%) or Pacific Island (12/27, 44%) origin with more than two-thirds being over the age of 6 years at time of presentation The 2006 census for New Zealand reported that 64.7% of the population were Europeans, 14% Maori and 6.6% Pacific Islanders. A comparison of the census with the ethnic identity of the pediatric patients indicated that Maori and Pacific Island children were overrepresented in that they comprised only 20.6% of the total population but 85% of the cases of APSGN. The demographics and clinical features of the 27 pediatric patients with APSGN are presented in Table 1.

Seven (26%) children presented with rapid onset of anuric renal failure with a clinical picture indicative of rapidly progressive glomerulonephritis with severe uremia. All children presenting with anuric renal failure required urgent dialysis because of severe electrolyte derangement and volume overload. Interestingly, these children were predominantly New Zealand Europeans. Older children were treated with hemodialysis and the younger ones with peritoneal dialysis. Two patients (cases 16, 22) developed hemoptysis secondary to pulmonary hemorrhage—in one child as a presenting symptom and the other during the course of her illness. Seventeen patients had nephrotic syndrome or nephrotic range proteinuria during their illness.

Renal biopsy

Twenty-seven children with APSGN had 28 renal biopsies. The indications for biopsy were anuric renal failure (seven patients; 26%), acute severe glomerulonephritis (11, 41%), mixed nephritic nephrotic syndrome (five, 18%) and unexpected delay in recovery from acute glomerulonephritis (four, 15%). One child (case 8) had two biopsies because of persisting acute renal failure. At presentation, the patient was anuric and was treated with urgent hemodialysis. The first biopsy performed on day 2 of hospitalization showed diffuse proliferative glomerulonephritis. The patient remained in dialysis-dependent renal failure, and a further

Table 1 Demographic, laboratory features and outcome of patients

Case no.	Age (years)	Ethnicity[a]	Bx time	Anti-streptolysin O titer	C3 (ug/l)	Initial plasma creatinine (umol/l)	Plasma creatinine pre-Bx	Biopsy reason[b]	Percentage crescents present	Bx score	Nephrotic	Treatment[c]	Dialysis		Recent plasma creatinine (umol/l)	Follow-up duration (years)
													Yes/no	Duration (days)		
1	13.2	PI	4	530	0.94	670	640	Anuric	60	17	No	St+CYC	Yes	ESRD	ESRD	8
2	11.7	NZE	2	515	0.83	1320	1400	Anuric	100	16	Yes	St	Yes	28	87	7.5
3	5.6	PI	19	288	0.97	230	270	Delayed recovery	100	17	Yes	St	No			5
4	9	Maori	2	546	0.19	600	620	Anuric	100	16	Yes	St	Yes	14	95	6
5	13.1	Maori	2	514	0.23	890	580	AGN	60	14	Yes	No	Yes	2	80	5
6	9.7	PI	15	2690	1.3	110	430	Delayed recovery	70	14	Yes	St	Yes	4	60	4
7	9.4	NZE	12	126	0.14	80	70	Nephritic/nephrotic	40	9	Yes	No	No			Lost
8	10.3	NZE	2	543	0.26	520	660	Anuric	50	8	No	St+CYC	Yes	18		2
9	13.9	PI	10	880	0.45	3520	1010	Anuric	75	7	No	St+CYC	Yes	ESRD	ESRD	3
10	5.9	Maori	2	1650	0.3	340	360	ARF/AGN	75	14	Yes	St	No			1.7
11	3.5	PI	120	1750	0.3	620	80	Nephrotic/nephritic	80	14	Yes	No	No			1
12	5.6	NZE	3	158	0.43	600	726	Anuric	80	8	Yes	St+CYC	Yes	11	48	1
13	3.6	Maori	3		1.4	70	50	AGN	0	12	Yes	No	No	0		Lost
14	12	PI	1	317	1.24	50	50	AGN	0	6	No	No	No	0		Lost
15	9.4	Maori	0			60	60	Nephritic/nephrotic	0	5	Yes	No	No	0		Lost
16	9.2	PI	21	1150	0.5	60	60	Nephrotic/pulm H	0	7	Yes	St+CYC	Yes	14	100	8
17	9.1	PI	2	236		90	90	Nephritic/Nephrotic	0	8	Yes	No	No	0		Lost
18	12.3	Maori	0	861	0.2	580	580	ARF/AGN	25	8	Yes	St	Yes	7	70	5
19	5.6	Maori	4	498	0.1	50	290	AGN	20	6	Yes	No	No	0		2.3
20	8	Maori/PI	2	696	1.4	550	590	Anuric	0	8	No	St	Yes	1		3
21	13.2	Maori	2	110	0.13	320	490	ARF/oliguria	0	8	No	No	Yes	3	50	1.3
22	8.6	Maori	5	346	0.14	120	50	AGN/pulm H	0	10	No	St for pulmH	No	0		Lost
23	8.7	Maori	4	80	1	180	210	AGN	10	5	Yes	No	No	0		1.5
24	11.7	PI	1	95	1	150	150	AGN	0	7	Yes	No	No	0	61	1.5
25	10.4	PI	7	590	0.36	186	247	AGN	10	6	No	St	No	0	61	1.4
26	5.5	PI	17	263	0.1	130	100	Delayed recovery	20	7	Yes	St	No	0	50	1.6
27	10	PI	23	171	0.1	259	135	Delayed recovery	0	8	No	No	No	0	57	1.7

Bx (biopsy) time, Time to biopsy from admission (days); ESRD, end stage renal disease

[a] M, Maori; NZE, New Zealand European; PI, Pacific Islander

[b] ARF, Acute renal failure; AGN, acute glomerulonephritis; pulm H, pulmonary hemorrhage

[c] CYC, cyclophosphamide; St, steroid

biopsy on day 12 showed CGN with 70% of glomeruli showing cellular crescents. Four patients (cases 3, 6, 26, 27) were biopsied because their serum creatinine failed to improve to normal levels within the expected period of 2–3 weeks. Most of the children (18/27) had renal biopsies performed within 5 days of presentation to hospital due to severe acute or anuric renal failure. Two patients were biopsied after 20 days after initial presentation; one patient was referred from the Pacific Islands 4 months after onset of the acute illness for investigation of persistent renal failure and the other was admitted after the development of acute pulmonary hemorrhage during her illness. There was no significant relationship between the total biopsy score and the need for acute dialysis, the pre-biopsy serum creatinine, duration of dialysis, the presence of anuria at initial presentation or the development of nephrotic range proteinuria. All renal biopsies showing glomerular crescents had cellular crescents with no evidence of fibrocellular changes to indicate chronicity. Direct immunofluorescence on all the renal biopsies showed C3 and immunoglobulin (Ig)G deposition in a pattern consistent with post-infectious glomerulonephritis.

Serological studies

Of the 27 patients, 25 had streptococcal titers measured at the time of presentation; 14 of these patients had significantly elevated anti-streptolysin O titers (>480 Todd units). Four patients had elevated anti-DNAse B titers (>680 Todd units) while having ASO titers within the normal range (data not shown). Seven patients had normal initial streptococcal titers; however, follow-up titers 10–14 days later were not done or available. Streptococcal titers were not available for two patients. There was no correlation between the degree of ASO elevation and the severity of clinical presentation or depression of the C3 complement. C3 complement was significantly reduced (<0.8 mg/l) in 16 of 26 patients in whom complement studies were performed. No patient had reduced C4 complement. Serology for antinuclear factor and antineutrophil cytoplasmic antibody were all negative in those patients for whom these factors were measured.

Comparison of crescentic (Group 1) with non-CGN (Group 2)

As a whole, group 1 patients ($n=11$) had significantly higher serum creatinine values than group 2 patients (556 vs. 210 umol/l, respectively; $p=0.006$). This difference could not be accounted for by a difference in age as the mean ages of the two groups were 9.2 and 9.1 years, respectively. There was also a tendency for group 1 patients to require acute dialysis more frequently than those without crescentic changes, although this was not statistically significant ($p=0.1$). There was no difference in the duration of dialysis between the two groups. Anuric renal failure was the most common presenting clinical syndrome patients in group 1 patients (6/11); in contrast, this condition was only observed in one of the 16 patients in group 2. One patient (case 1) presented with severe renal failure and progressed to end stage renal disease within 1 month despite immunosuppression therapy with methylprednisolone and cyclophosphamide. Another patient (case 9) had a delayed presentation (approx. 1 month) and had been anuric for some days before eventual hospitalization. A renal biopsy 10 days after initial stabilization of his clinical condition showed severe CGN with 80% glomeruli affected. Despite methylprednisolone and cyclophosphamide therapy, this patient remained in anuric renal failure and was dialysis dependent. He is now awaiting a renal transplant.

Treatment and outcome

Twelve patients of the 27 patients (seven in group 1, five in Group 2) required acute dialysis. Follow-up data were available in 18/27 patients with a mean follow-up time of 3.2 years (95% confidence interval 2.1–4.3; median 2 years, range 1–8 years). Patients with renal failure severe enough to need acute dialysis tended to have persistent proteinuria after up to 8 years of follow-up. Patients that had a large percentage of crescents on biopsy had a greater likelihood of needing acute dialysis. At last follow-up, eight of the 12 patients who needed dialysis are either in end stage renal disease or chronic renal failure or they have persistent proteinuria of 2 to 4+ on casual urinalysis. Approximately one-half (7/15) of the group who did not have dialysis have been lost to long-term follow-up

Fifteen patients were treated with immunosuppression therapy, with either methylprednisolone pulses followed by oral prednisone alone or in combination with cyclophosphamide. Overall, those that had 50% or more crescents on biopsy were offered either methylprednisolone alone or in combination with cyclophosphamide. No clear conclusions on the efficacy of immune suppression on long-term outcome can be drawn from this study.

Discussion

This retrospective study describes the outcome of severe post-streptococcal glomerulonephritis over a 12-year period in New Zealand where this disease is still a major cause of acute renal failure. The extent of the problem is currently being studied in a prospective nation-wide study. The study reported here focused on patients who have had renal biopsies and hence were at

the severe end of the clinical spectrum. It is important to accurately consider the impact of acute post-streptococcal glomerulonephritis as a cause of acute illness in childhood in the New Zealand context. It is a common disease in New Zealand, with the New Zealand Health Information System reporting 768 cases of either confirmed or probable APSGN in children under age 15 between 2000 and 2006, giving an average of 109 cases per year for the whole country (personal communication, with permission of the Ministry of Health, New Zealand). This study reviewed 27 cases over a 12-year period that were severe or atypical enough to warrant a renal biopsy. This gives an average of two cases of severe APSGN per year or, otherwise, approximately 2% of cases of APSGN have a severe course. This is an important observation which puts into perspective both the relative rarity of severe APSGN and the fact that most children make a good recovery. This is similar to other studies from overseas [12, 13].

Almost three-quarters of our patient group presented with either anuria or severe acute glomerulonephritis with significant renal failure. Two children had pulmonary hemorrhages at the onset or during their clinical course. One patient had a bronchoscopy which showed diffuse bleeding consistent with pulmonary capillaritis, with a clinical picture similar to that of Goodpasture's syndrome. Interestingly, the renal biopsies of both of these patients showed no evidence of crescent formation, which might be more typical of Goodpasture's syndrome, and their direct immunofluorescence showed a coarse granular C3 complement and IgG in glomerular capillary walls consistent with post-streptococcal glomerulonephritis. In the clinical setting of rapid onset of acute anuric renal failure, the differential diagnosis of RPGN includes systemic lupus erythematosus, IgA nephropathy, membranoproliferative glomerulonephritis, Goodpasture's disease, vasculitis, Henoch–Schönlein purpura and post-streptococcal glomerulonephritis.

The recommended treatment of severe APSGN is supportive. The role of immunosuppressive therapy is thought to be limited. There have been no recent randomized controlled trials of immune suppression, particularly in children where the disease is more common. A number of published studies [9, 13–15] have not been able to demonstrate conclusively the benefit of immune suppression on long-term outcome. We also conclude from our study that there is no clear benefit of immune suppression on renal outcome. The indications for immunosuppressive therapy were not based on pre-determined criteria, and its administration was dependent on the practice of the physician caring for the patient at the time of admission. A general principle in the treatment of severe CGN (>75% crescents) regardless of etiology is immune suppression with corticosteroids with or without an alkylating agent. This treatment strategy is frequently utilized to suppress the severe extra-capillary inflammation.

The role of plasmapheresis is currently unknown, despite a case report of a pediatric patient being treated by such a regimen. In that patient, methylprednisolone and antiplatelet therapy were also used [16]. We currently do not advocate the use of plasmapheresis in this clinical setting as we have seen a number of patients with CresPSGN who have a made a good recovery without this therapy.

A recent report from Ralf et al. [17] suggested that CresPSGN should receive more aggressive treatment, especially if associated with nephrotic syndrome. Nephrotic syndrome in adult APSGN is a predictor of poor renal survival and is more than fivefold more likely to progress to uremia within 2 years of the acute illness compared with those without nephrotic proteinuria [18]. In Chugh's series [18] of 142 adults, 14% had nephrotic proteinuria and, on a long-term follow-up, 15% had persistent hypertension, 1.9% end stage renal failure, and 3.8% chronic renal failure. In Vogl's series of 98 adults [19], 21.5% had nephrotic proteinuria and 16% developed chronic renal insufficiency. In the series reported here, 18 (66%) patients had nephrotic syndrome or nephrotic range proteinuria and eight had CGN. Two patients progressed to end stage renal failure and only 3/21 (14%) patients still have persistent proteinuria after 4–6 years of follow-up. This result suggests that children with nephrotic range proteinuria from APSGN is more common than previously described and that this age group may possibly have a better long-term prognosis than adults, although our follow-up time is still relatively short, and only a long-term prospective study will answer this issue with any degree of certainty.

Limitations of this study are those of a retrospective study. Incomplete data were a problem in that only 18 or 27 patients had significantly elevated streptococcal titers, and follow-up or convalescent titers were not performed in a proportion of patients. This raises the possibility that not all patients had post-streptococcal glomerulonephritis even though all of the renal biopsies showed features typical of the disease. In our clinical setting, acute glomerulonephritis following a streptococcal infection is by far the most common cause of post-infectious glomerulonephritis [20]. This clinical impression is substantiated by the high incidence of rheumatic fever in the same ethnic groups who have acute glomerulonephritis. We are therefore confident that all the cases reported in this study were due to post-streptococcal disease.

Acute post-streptococcal glomerulonephritis during childhood continues to be a major health issue in New Zealand Maori and Pacific Island children. Children from both Maori and Pacific Island backgrounds are over-represented in this report. The major reason for the high incidence of this disease in these groups is their

lower socioeconomic status in the community. Other infectious diseases, such cellulitis and respiratory illness, are more frequent among Maori and Pacific Island children [21]. This relates mainly to poverty, poor education, crowding and, occasionally, cultural attitudes.

In conclusion, acute post-streptococcal glomerulonephritis during childhood continues to be major health problem in New Zealand, affecting predominantly Maori and Pacific Island children. A subgroup of these children presented with rapid onset of severe disease that required a kidney biopsy to exclude other causes of rapidly progressive glomerulonephritis. A significant proportion of this subgroup needed a period of acute dialysis. Some of these patients have persistent proteinuria following the acute illness and progress to chronic renal insufficiency. A worrying aspect of our retrospective study is that a quarter of this group was lost to follow-up despite attempts by the authors to encourage attendance at follow-up clinics.

References

1. Yap HK, Chia Muragasu B, Saw AH, Tay JS, Ikshuvanam M, Tan KW, Cheng HK, Tan CL, Lim CH (1990) Acute glomerulonephritis changing patterns in Singapore. Pediatr Nephrol 4:482–484
2. Haverkorn MJ, Valkenburg HA, Goslings WR (1971) *Streptococcal pharyngitis* in the general population I. A controlled study of streptococcal pharyngitis and its complications in the Netherlands. J Infect Dis 124:339–347
3. Roy S, Stapleton FB (1990) Changing perspectives in children hospitalised with post-streptococcal glomerulonephritis. Pediatr Nephrol 4:585–588
4. Bodaghi E, Vazarian SH, Abtahi M, Honarmand MT, Madani A, Zia Shamsa AM (1989) Glomerular disease in children, the Iranian experience. Pediatr Nephrol 3:213–217
5. Berrios X, Lagomarsino E, Solar E, Sandoval G, Guzmán B, Riedel I (2004) Post-streptococcal glomerulonephritis in Chile—20 years of experience. Pediatr Nephrol 19:306–312
6. Tasic V, Polenakovic M (2003) Occurrence of subclinical post streptococcal glomerulonephritis in family contacts. J Paediatr Child Health 39:177–179
7. Yoshizawa N, Suzuki Y, Oshima S, Takeuchi A, Kondo S, Ishida A, Nakabayashi I, Nishiyama J, Tazawa K, Sagel I (1996) Asymptomatic acute poststreptococcal glomerulonephritis following upper respiratory tract infections caused by Group A streptococci. Clin Nephrol 46:296–301
8. Roy S III, Murphy WM, Arant BS (1981) Post streptococcal crescentic glomerulonephritis in children: Comparison of quintuple versus supportive care. J Pediatr 98:403–410
9. Southwest Pediatric Nephrology Study Group (1985) A clinicopathologic study of crescentic glomerulonephritis in 50 children. A report by the Southwest Pediatric Nephrology Study Group. Kidney Int 27:450–458
10. Jardim HMPF, Leake J, Risdon A, Barratt TM, Dillon MJ (1992) Crescentic glomerulonephritis in children. Pediatr Nephrol 6:231–235
11. Clark G, White RHR, Glasgow EF, Chantler C, Cameron JS, Gill D, Comley LA (1988) Poststreptococcal glomerulonephritis in children: clinicopathological correlations and long term prognosis. Pediatr Nephrol 2:381–388
12. Nissenson AR, Mayon-White R, Potter EV, Mayon-White V, Abidh S, Poon-King T, Earle DP (1979) Continued absence of clinical renal disease seven to 12 years after poststreptococcal acute glomerulonephritis in Trinidad. Am J Med 67:255–262
13. Popovic-Rolovic M, Kostic M, Antic-Peco A, Jovanovic O, Popovic D (1991) Medium and long term prognosis of patients with acute post streptococcal glomerulonephritis. Nephron 58:393–399
14. Srivastava R, Mudgil A, Bagga A, Bhuyan UN, Vasudev AS, Sundraem KR (1992) Crescentic glomerulonephritis in children: A review of 43 cases. Am J Nephrol 12:155–161
15. Couser WG (1988) Rapidly progressive glomerulonephritis—classification, pathogenetic mechanisms, and therapy. Am J Kidney Dis 11:449–464
16. Suyama K, Kawasaki Y, Suzuki H (2007) Girl with garland pattern post streptococcal glomerulonephritis presenting with renal failure and nephrotic syndrome. Pediatr Int 49:115–117
17. Ralf A, Hebert T, Pullma J, Coco M (2005) Crescentic post streptococcal glomerulonephritis with nephrotic syndrome in an adult: is aggressive therapy warranted. Clin Nephrol 63:375–380
18. Chugh KS, Malhotra HS, Sakhuja V, Bhusnurmath S, Singhal PC, Unni VN, Pirzada R, Kapoor MM (1987) Progression to end stage renal disease in post streptococcal glomerulonephritis—Chandigarh Study. Int J Artif Organs 10:189–194
19. Vogl W, Renke M, Mayer-Eicherger D, Bohle A (1986) Long term prognosis for endocapillary glomerulonephritis of post streptococcal type in children and adults. Nephron 44:58–65
20. Thornley C, McNicholas A, Baker M, Lennon D (2001) Acute rheumatic fever registers in New Zealand. N Z Public Health Rep 8:41–44
21. Mills CF, Tobias M, Baker M (2002) A re-appraisal of the burden of infectious disease in New Zealand: aggregate estimates of morbidity and mortality. NZ Med J 115:1–8