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CLINICAL STUDY

The Spectrum of Adult Postinfectious Glomerulonephritis in the New Millennium

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Background. Postinfectious glomerulonephritis is rare in adults. The characteristics of the disease now differ from what were described decades ago. The goal of this study is to illustrate the clinicopathological spectrum of the disease in the modern era. *Methods.* Between July 2000 and June 2008, 20 adult cases of postinfectious glomerulonephritis were identified at a medical center in Taiwan. The patients' records were retrospectively reviewed with respect to clinical presentation, microbiology, serology, morphology of renal biopsy, and clinical course. *Results.* There were 14 males and 6 females. The mean age was 61 years. All patients developed acute renal failure, and the majority (65%) required dialysis support during the disease course. Hypocomplementemia was present in 60% of patients. The most frequently identified infectious agent was *Staphylococcus* (60%). Histological characteristics showed two distinct patterns of glomerulonephritis: diffuse endocapillary proliferative glomerulonephritis (65%) and focal mesangial proliferative glomerulonephritis (35%). There were no significant differences in the clinical presentation and outcome between the two groups. However, glomerular neutrophil infiltration was more commonly present in diffuse endocapillary proliferative pattern (p = 0.017). The percentage of patients with focal mesangial proliferative pattern significantly increased over time ($p < 0.001$). At the last follow-up, 6 patients (30%) had died, 6 (30%) were in complete remission, 4 (20%) had partial remission with renal insufficiency, and 4 (20%) were on chronic dialysis. *Conclusions.* Our data suggested that *Staphylococcus* had become the leading pathogen in adult postinfectious glomerulonephritis over the past 10 years. Furthermore, atypical histological feature with focal mesangial proliferative pattern was increasingly identified over time. The prognosis was still guarded, carrying a considerable mortality rate and risk for developing chronic renal failure.

Keywords acute renal failure, infections, glomerulonephritis, renal biopsy, renal prognosis

INTRODUCTION

Infection has been well documented to cause glomerulonephritis. In the past, the vast majority of cases typically followed streptococcal upper respiratory tract or skin infections, and the disease most frequently affects children. Typical histological findings include diffuse endocapillary proliferative glomerulonephritis accompanied by infiltration with neutrophils within the capillary lumens (exudative features) on light microscopy, C3-dominant granular deposits with or without IgG co-deposition on immunofluorescence, and characteristic subepithelial hump-shaped deposits on electron microscopy. Abrupt symptoms of acute nephritis, hypocomplementemia, and rising anti-streptolysin O (ASLO) titers are diagnostic. Renal biopsy is rarely required. Most children with poststreptococcal glomerulonephritis (PSGN) recover completely. Over recent decades, the pattern of the disease has changed. Not only *Streptococcus* but also other bacterial, viral, and parasitic agents have been implicated in the pathogenesis of glomerulonephritis.[1] In developed countries, glomerulonephritis associated with non-streptococcal infections is assuming greater important. This is thought to be secondary to a decline in the incidence of group A streptococcal infections in children and a relative increase in the incidence of glomerulonephritis associated with other infections in adults. Furthermore, an increasing number of adult cases have been observed in alcoholics, diabetics, and intravenous drug abusers.[2,3] Atypical clinical presentation often presents complex diagnostic challenges and highlights the

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important diagnostic role of renal biopsy. The more extensive use of renal biopsy has demonstrated the presence of atypical histological features of the disease.[4,5] While several studies performed before 2000 evaluated the course of adult postinfectious glomerulonephritis,[2,3,6] there have been few reports in the new millennium. The aim of this study was to investigate whether the spectrum of the disease had changed over the past 10 years.

METHODS

Between July 2000 and June 2008, 20 adult cases of postinfectious glomerulonephritis were identified at a medical center in Taiwan, in which there was close temporal relationship between infection and first appearance of renal manifestations, and there was no clinical or laboratory evidence of systemic disease that might cause glomerulonephritis. All patients were submitted to renal biopsy analyzed by light microscopy after hematoxylin-eosin, periodic acid-Schiff, and Masson trichrome stains and by immunofluorescence using antisera against IgG, IgA, IgM, C3, and C1q. The extent of mesangial and endocapillary proliferation was graded as focal (involving <50% of glomeruli) and diffuse (involving ≥50% of glomeruli). Scoring of the chronic tubulointerstitial injury was based on the percentage of tubular atrophy and interstitial fibrosis, and was graded as mild (1+) if involving <25%, moderate (2+) if involving 25–50%, and severe (3+) if involving >50%. Other histological parameters were also scored semiquantitatively on a scale from 0 to 3+. The intensity of immunofluorescence staining was graded on a scale of 0 to 3+. In 11 out of the 20 cases, electron microscopy was performed. Patients' medical records were reviewed for age, sex, type and source of infection, clinical presentation, parameters of renal function, and outcome. Remission was defined as complete if last follow-up serum creatinine was ≤1.2 mg/dL and partial if last follow-up serum creatinine was >1.2 mg/dL.

Statistical analyses were performed using SPSS version 12 (SPSS Inc., Chicago, Illinois, USA) for Windows and produced by simple non-parametric test (Mann-Whitney *U* test and Fisher's exact test). A p value < 0.05 was considered significant.

RESULTS

There were 14 males and 6 females, with mean age of 61 ± 15 years (range 30–80 years). Clinical characteristics at presentation are summarized in Table 1. All patients developed acute renal failure. The mean 24-hour urinary protein excretion was 3.2 g, and 41.1% of patients had nephrotic range proteinuria. Hematuria was present in all patients. Leukocyturia was seen in the majority of patients. The mean peak serum creatinine was 6.65 mg/dL. Hypocomplementemia was present in 60% of patients. Thirteen out of 20 patients (65%) required dialysis support during the disease course.

All but one patient (case 9) had renal manifestations during the course of intercurrent infections. In case 9, there was no clinical evidence of infection at presentation, and the diagnosis was made according to rising ASLO titers and classic histological criteria. The sites of infection included skin (20%), heart/endocarditis (20%), lung (15%), bone/joint (15%), urinary tract (15%), and deep-seated abscess (10%). The two most frequently identified infectious agents were *Staphylococcus* (60%) and *Streptococcus* (15%). In a total of 12 patients with staphylococcal infection, 8 (67%) had methicillin-resistant *Staphylococcus aureus* (MRSA) and 4 (33%) had methicillin-sensitive *Staphylococcus aureus* (MSSA).

Histological characteristics are summarized in Table 2. Two distinct patterns of glomerulonephritis were identified: diffuse endocapillary proliferative glomerulonephritis (65%) and focal mesangial proliferative glomerulonephritis (35%). Glomerular crescents were present in 45% of cases (affected ≥50% of glomeruli in 20% of cases). Glomerular neutrophil infiltration was seen in 55% of cases. Segmental glomerular necrosis was seen in one case. Acute tubular injury with luminal ectasia, epithelial simplification, loss of brush border, and nuclear enlargement was a common feature, identified in 65% of cases. All but two cases had interstitial infiltration with inflammatory cells. Only four biopsies showed characteristic subepithelial hump-shaped deposits.

All but six patients (30%) who died within three months after diagnosis were followed for a mean of 27 ± 23 months. Six patients (30%) entered complete remission. Four patient (20%) had partial remission, with serum creatinine levels ranging from 1.9 to 4.1 mg/dL, and four patients (20%) had to be submitted to chronic dialysis.

By comparison, there were no significant differences in the clinical presentation and outcome between patients with diffuse endocapillary proliferative pattern and focal mesangial proliferative pattern. However, glomerular neutrophil infiltration was more commonly present in diffuse endocapillary proliferative pattern ($p = 0.017$; see Table 3). Our data suggested that the percentage of patients with focal mesangial proliferative pattern significantly increased over time ($p < 0.001$; see Figure 1).

DISCUSSION

Classically, postinfectious glomerulonephritis occurs after streptococcal infections, typically involving upper

Table 1

Clinical details of 20 adult patients with postinfectious glomerulonephritis

Case number	Sex/age (year)	Infectious agent	Infectious origin	Time relationship between nephritis and infection	Peak serum creatinine (mg/dl)/ dialysis support	Proteinuria (g/24 h)	Hematuria (RBC/HPF)	Complement (mg/dl)*	Follow-up (month)	Last serum creatinine (mg/dl)
1	M/31	MSSA	Endocarditis	During infection	9.8/Y	0.5	35 to 40	C3: 40.9	36	1.2
2	M/70	MRSA	Intra-abdominal abscess	During infection	9.4/Y	9.8	Numerous	Normal	3	Died
3	M/73	MRSA	Urinary tract infection	During infection	9.5/Y	>300 mg/dl	Numerous	Normal	1	Died
4	M/74	MSSA	Osteomyelitis of spine	During infection	11.8/Y	7.9	Numerous	Normal	4	Dialysis
5	F/72	*Nontuberculosis mycobacterium*	Pneumonia	During infection	5.7/Y	1.9	Numerous	Normal	3	Died
6	M/46	*Streptococcus viridans*	Endocarditis	During infection	11.1/Y	>300 mg/dl	Numerous	C3: 37.4	6	Dialysis
7	M/63	*Streptococcus pyogenes*	Intra-abdominal abscess	During infection	6.8/N	0.7	Numerous	C3: 39.6	74	2.6
8	F/66	*Proteus mirabilis*	Cellulitis of leg	During infection	1.7/N	2.1	Numerous	C3: 31.5	42	1.1
9	M/59	*Streptococcus*	Unknown	Post-infection	2.0/N	5.2	Numerous	C3: 25.7	15	1.2
10	M/55	*Klebsiella pneumoniae*	Urinary tract infection	During infection	8.9/Y	2.2	Numerous	C3: 31.9	1	Died
11	M/30	MSSA	Endocarditis	During infection	3.3/N	2.5	Numerous	C3: 26.0	41	0.9
12	F/75	MRSA	Lung abscess	During infection	3.3/Y	>300 mg/dl	Numerous	C3: 66.6	2	Died
13	F/70	Not available	Urinary tract infection	During infection	8.7/Y	4.8	35 to 40	Normal	36	Dialysis
14	M/45	MRSA	Endocarditis	During infection	2.8/N	3.8	20 to 30	C3: 65.9	68	0.8
15	M/80	MRSA	Central line infection	During infection	4.3/Y	3.3	Numerous	Normal	1	Died
16	F/70	MSSA	Osteomyelitis of spine	During infection	4.8/Y	1.2	Numerous	C3: 66.0	25	2.6
17	M/73	*Mycobacterium tuberculosis*	Pneumonia	During infection	8.8/Y	0.6	Numerous	Normal	12	4.1
18	M/44	MRSA	Septic arthritis of hip	During infection	9.6/N	2.9	Numerous	C3: 70.5	5	1.9
19	F/65	MRSA	Infected surgical wound	During infection	3.0/N	1.5	Numerous	C3: 52.0	10	1.0
20	M/57	MRSA	Infected diabetic foot	During infection	7.7/Y	3.0	Numerous	Normal	4	Dialysis

*Normal range: C3, 72–136 mg/dl; C4, 12–47 mg/dl.

Abbreviations: M = male, F = female, MSSA = methicillin-sensitive *Staphylococcus aureus*, MRSA = methicillin-resistant *Staphylococcus aureus*, MSSE = methicillin-sensitive *Staphylococcus epidermidis*.

Table 2
Histological features of 20 adult patients with postinfectious glomerulonephritis

Case number	Light microscopy	Immunofluorescence	Electron microscopy
1	Diffuse endocapillary proliferative GN with neutrophils, acute tubular necrosis, tubular atrophy 1+	C3 (1+); GCW	Not available
2	Diffuse endocapillary proliferative GN with 50% cellular crescents, acute tubular necrosis, interstitial infiltration 3+, tubular atrophy 1+	IgA (1+), IgG (1+), C3 (3+); MES	Not available
3	Diffuse endocapillary proliferative GN with neutrophils and 50% cellular crescents, glomerular fibrinoid necrosis 1+, interstitial infiltration 1+, interstitial fibrosis 1+, tubular atrophy 1+	IgA (2+), IgG (1+), C3 (2+); GCW	Not available
4	Diffuse endocapillary proliferative GN with 30% cellular crescents, acute tubular necrosis, interstitial infiltration 3+, tubular atrophy 1+	IgA (1+), IgG (1+), C3 (1+); MES	Not available
5	Diffuse endocapillary proliferative GN with neutrophils and 40% cellular crescents, acute tubular necrosis, interstitial infiltration 3+, tubular atrophy 1+	IgA (1+), IgG (1+), C3 (2+), C1q (1+); MES/GCW	Subendothelial and mesangial deposits
6	Diffuse endocapillary proliferative GN with 100% cellular crescents, acute tubular necrosis, interstitial infiltration 3+	IgM (1+), C3 (2+), C1q (1+); GCW	Not available
7	Diffuse endocapillary proliferative GN with neutrophils, interstitial infiltration 3+, interstitial fibrosis 1+, tubular atrophy 1+	C3 (1+); MES/GCW	Not available
8	Diffuse endocapillary and focal mesangial proliferative GN with neutrophils, interstitial infiltration 1+, interstitial fibrosis 1+, tubular atrophy 1+	C3 (1+); GCW	Subendothelial and mesangial deposits, subepithelial humps
9	Diffuse endocapillary proliferative GN with neutrophils, interstitial infiltration 1+, interstitial fibrosis 1+	IgA (2+), IgG (1+), IgM (2+), C3 (3+), C1q (2+); GCW	Subendothelial and mesangial deposits, subepithelial humps
10	Diffuse endocapillary proliferative GN with neutrophils and 35% cellular crescents, acute tubular necrosis, interstitial infiltration 3+, tubular atrophy 1+	IgA (1+), IgM (1+), C3 (2+), C1q (2+); GCW	Not available
11	Diffuse endocapillary proliferative GN with neutrophils	IgA (1+), IgG (1+), IgM (1+), C3 (2+), C1q (1+); GCW	Subendothelial and mesangial deposits, subepithelial humps
12	Diffuse endocapillary proliferative GN with neutrophils, acute tubular necrosis, interstitial infiltration 3+	IgA (2+), IgG (1+), C3 (2+); GCW	Not available
13	Diffuse endocapillary proliferative GN with neutrophils and 60% cellular crescents, acute tubular necrosis, interstitial infiltration 3+, tubular atrophy 1+	IgG (1+), IgM (1+), C3 (2+), C1q (1+); MES/GCW	Not available
14	Focal mesangial proliferative GN, interstitial infiltration 1+	IgA (2+), IgG (1+), C3 (2+);	Mesangial deposits
15	Focal mesangial proliferative GN, acute tubular necrosis, interstitial infiltration 1+, interstitial fibrosis 1+, tubular atrophy 1+	IgA (2+), IgG (1+); MES	Mesangial deposits
16	Focal mesangial proliferative GN, diabetic glomerulosclerosis, acute tubular necrosis, interstitial infiltration 1+, tubular atrophy 1+	IgA (2+), IgG (1+), IgM (1+), C3 (2+), C1q (1+); MES	Mesangial deposits
17	Focal mesangial proliferative GN with neutrophils and 10% cellular crescents, acute tubular necrosis, interstitial infiltration 3+, tubular atrophy 1+	IgA (1+), IgG (1+), IgM (1+), C3 (2+); MES	Mesangial deposits, subepithelial humps
18	Focal mesangial proliferative GN with 30% cellular crescents, acute tubular necrosis, interstitial infiltration 1+, tubular atrophy 1+	IgA (1+), IgG (1+), IgM (1+), C3 (1+); MES/GCW	Mesangial deposits
19	Focal mesangial proliferative GN, interstitial infiltration 1+, tubular atrophy 1+	IgA (2+), IgG (1+), IgM (1+), C3 (2+); MES	Mesangial deposits
20	Focal mesangial proliferative GN, acute tubular necrosis, interstitial infiltration 3+, tubular atrophy 1+	IgA (2+), C3 (2+), C1q (1+); MES	Mesangial deposits

Abbreviations: GN = glomerulonephritis, GCW = glomerular capillary wall, MES = mesangial.

Table 3
Comparison of clinicopathological features between diffuse endocapillary and focal mesangial proliferative glomerulonephritis associated with infection in adults

Variables	Diffuse endocapillary proliferative GN (n = 13), mean ± SD or number (%)	Focal mesangial proliferative GN (n = 7), mean ± SD or number (%)	*p*
Age (years)	60 ± 16	62 ± 14	
Gender (male/female)	9/4	5/2	
Proteinuria (g/24 h)*	3.8 ± 3.1	2.3 ± 1.2	0.558
Hematuria	12 (100)	6 (100)	
C3 (mg/dl)*	56.6 ± 28.3	79.6 ± 21.2	0.122
C4 (mg/dl)*	20.1 ± 8.1	23.4 ± 10.0	0.362
Glomerular crescents (%)*	28 ± 32	8 ± 11	0.118
Glomerular neutrophil infiltration†	10 (77)	1 (14)	0.017
Acute tubular necrosis	8 (62)	5 (71)	
Interstitial infiltration (2+ or 3+)†	8 (62)	2 (29)	0.350
Subepithelial humps	3 (23)	1 (14)	
Dialysis support requirement†	9 (69)	4 (57)	0.651
Death†	5 (38)	1 (14)	0.354
Remission in survivors†	5/8 (63)	5/6 (83)	0.580
Complete remission	4/8 (50)	2/6 (33)	0.627
Partial remission	1/8 (13)	3/6 (50)	0.245
Long-term dialysis in survivors†	3/8 (38)	1/6 (17)	0.580

*Data were analyzed by Mann-Whitney *U* test.
†Data were analyzed by Fisher's exact test.
Abbreviation: GN = glomerulonephritis.

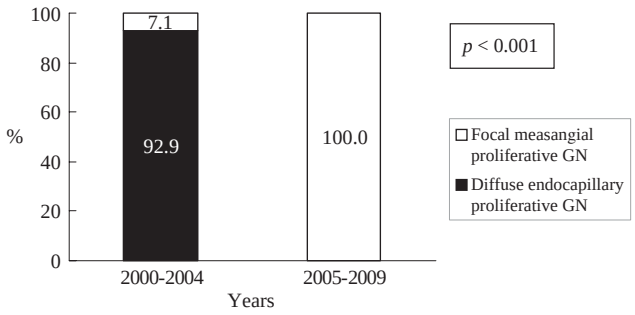

Figure 1. Percentage of patients with diffuse endocapillary proliferative glomerulonephritis and patients with focal mesangial proliferative glomerulonephritis in different time periods.

respiratory tract or skin. The disease most frequently affects children and is relatively uncommon in adults. In developed countries, the incidence of typical PSGN has declined sharply over the last 50 years, probably due to the improved environmental sanitation and better public health service. On the contrary, adult cases of glomerulonephritis associated with non-streptococcal infections are increasingly recognized in the recent years. Adults with an immunocompromised background are particularly at risk for developing the disease.[2,3] More diverse sites of infection and microorganisms have been linked to adult postinfectious glomerulonephritis. Previous studies have demonstrated that *Staphylococcus* was responsible for an increasing number of cases.[3,7] Our series also showed that *Staphylococcus* was the most commonly identified infectious agent. Moreover, MRSA accounted for the majority of cases (see Table 1). In our series, the site of infection could be indentified in the majority of cases; the most common sites were skin (20%), heart/endocarditis (20%), lung (15%), bone/joint (15%), and urinary tract (15%). Considering that all 242 patients compiled from four modern studies,[2,3,6,7] the most common sites of infection included upper respiratory tract (24%), skin (16%), lung (14%), heart/endocarditis (9%), and teeth (7%). In these studies, 7–16% of patients had no clinical evidence of infection preceding the renal disease, and in 24–59% of patients, the offending microorganism could not be identified. These data suggested that postinfectious glomerulonephritis should be included in the differential diagnosis of nephritic/nephrotic syndrome in adults, even in the absence of a history of infection.

It has been demonstrated that there is a broad spectrum of glomerular histological findings in adult postinfectious glomerulonephritis.[4,5] The classic glomerular

pattern is diffuse endocapillary proliferative glomerulonephritis. This pattern was present in approximately two-thirds of patients in our series. Focal mesangial proliferative glomerulonephritis was present in the remaining seven cases. Importantly, our data suggested a significant escalation of focal mesangial proliferative pattern over time (see Figure 1). The cause was probably also related to a growing number of cases of glomerulonephritis associated with staphylococcal infection, particularly MRSA infection. Over the decades, two histological patterns of glomerulonephritis associated with staphylococcal infection are well-defined. One exhibits a diffuse endocapillary and exudative pattern, resembling classic PSGN, in patients with *Staphylococcus aureus* infection; the other displays a pattern identical to membranoproliferative glomerulonephritis in patients with *Staphylococcus epidermidis* infection secondary to ventriculovascular shunts. In recent years, however, a third form of *Staphylococcus*-associated glomerulonephritis characterized by mesangial proliferation with IgA-dominant or co-dominant deposits has been increasingly recognized, which generally occurs in patients with infections caused by MRSA.[8–10] It is speculated that enterotoxins produced by MRSA may serve as superantigens that contribute to this type of glomerulonephritis.[11] In our series, all seven patients (cases 14–20, 1 identified within 2000–2004 and 6 identified within 2005–2008) with mesangial proliferative glomerulonephritis had IgA mesangial staining (see Table 2). Among them, five patients had MRSA and one patient had MSSA (see Table 1). The remaining one patient (case 17) had unusual pathogen of *Mycobacterium tuberculosis*.

The prognosis of adult postinfectious glomerulonephritis has not been well defined, but there is general agreement that the prognosis is less favorable than PSGN in children. Before the 1990s, complete remission was reported in 60–80% of adults.[12–15] More recent studies found complete remission in only 26–56% of adults.[3,6,7] In our series, complete recovery was found in 30% of patients. Moreover, short-term mortality was found in 30% of patients. All these data strongly suggest that the prognosis of postinfectious glomerulonephritis is worsening in adults. This is probably due to typical PSGN becoming rarer and the number of patients with severe underlying diseases progressively increasing. This study also showed that patients with less severe glomerular change with focal mesangial proliferative glomerulonephritis had no better outcome than patients with more severe glomerular change with diffuse endocapillary proliferative glomerulonephritis (see Table 3). Previous reports of *Staphylococcus*-associated mesangial glomerulonephritis had the same results.[10,11] This was probably due to other histological findings, such as the severity of tubulointerstitial damage, and the clinical underlying diseases were also found to affect the prognosis.[6]

In conclusion, this study suggested that the clinicopathological spectrum of postinfectious glomerulonephritis had changed over the past 10 years. Atypical clinical and histological features may present diagnostic challenges. *Staphylococcus* had become the leading infectious agent. Furthermore, atypical histological feature with mesangial proliferative pattern was increasingly identified over time. This was probably due to a growing number of cases of glomerulonephritis associated with MRSA infection, which is now endemic in most hospitals and is increasingly identified from community-acquired infections.[16] Our data showed an unfavorable prognosis in adult postinfectious glomerulonephritis. However, this result was probably influenced by the fact that only patients with severe disease received renal biopsy and were enrolled into the study.

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REFERENCES

1. Sotsiou F. Postinfectious glomerulonephritis. *Nephrol Dial Transplant.* 2001;16(Suppl. 6):68–70.
2. Keller CK, Andrassy K, Waldherr R, Ritz E. Postinfectious glomerulonephritis—is there a link to alcoholism? *Q J Med.* 1994;87:97–102.
3. Montseny JJ, Meyrier A, Kleinknecht D, Callard P. The current spectrum of infectious glomerulonephritis: Experience with 76 patients and review of the literature. *Medicine (Baltimore).* 1995;74:63–73.
4. Edelstein CL, Bates WD. Subtypes of acute postinfectious glomerulonephritis: A clinico-pathological correlation. *Clin Nephrol.* 1992;38:311–317.
5. Sotsiou F, Dimitriadis G, Liapis H. Diagnostic dilemmas in atypical postinfectious glomerulonephritis. *Semin Diagn Pathol.* 2002;19:146–159.
6. Moroni G, Pozzi C, Quaglini S, et al. Long-term prognosis of diffuse proliferative glomerulonephritis associated with infection in adults. *Nephrol Dial Transplant.* 2002;17: 1204–1211.
7. Nasr SH, Markowitz GS, Stokes MB, Said SM, Valeri AM, D'Agati VD. Acute postinfectious glomerulonephritis in the modern era: Experience with 86 adults and review of the literature. *Medicine (Baltimore).* 2008;87:21–32.
8. Nasr SH, Markowitz GS, Whelan JD, et al. IgA-dominant acute poststaphylococcal glomerulonephritis complicating diabetic nephropathy. *Hum Pathol.* 2003;34:1235–1241.

9. Satoskar AA, Nadasdy G, Plaza JA, et al. Staphylococcus infection-associated glomerulonephritis mimicking IgA nephropathy. *Clin J Am Soc Nephrol.* 2006;1:1179–1186.
10. Nasr SH, Share DS, Vargas MT, D'Agati VD, Markowitz GS. Acute poststaphylococcal glomerulonephritis superimposed on diabetic glomerulosclerosis. *Kidney Int.* 2007;71: 1317–1321.
11. Koyama A, Kobayashi M, Yamaguchi N, et al. Glomerulonephritis associated with MRSA infection: A possible role of bacterial superantigen. *Kidney Int.* 1995;47:207–216.
12. Hinglais N, Garcia-Torres R, Kleinknecht D. Long-term prognosis in acute glomerulonephritis: The predictive value of early clinical and pathological features observed in 65 patients. *Am J Med.* 1974;56:52–60.
13. Lien JW, Mathew TH, Meadows R. Acute post-streptococcal glomerulonephritis in adults: A long-term study. *Q J Med.* 1979;48:99–111.
14. Vogl W, Renke M, Mayer-Eichberger D, Schmitt H, Bohle A. Long-term prognosis for endocapillary glomerulonephritis of poststreptococcal type in children and adults. *Nephron.* 1986;44:58–65.
15. Chugh KS, Malhotra HS, Sakhuja V, et al. Progression to end stage renal disease in post-streptococcal glomerulonephritis (PSGN)—Chandigarh Study. *Int J Artif Organs.* 1987;10:189–194.
16. Fridkin SK, Hageman JC, Morrison M, et al. Methicillin-resistant *Staphylococcus aureus* disease in three communities. *N Engl J Med.* 2005;352:1436–1444.