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Atypical Clinical Manifestations of Acute Poststreptococcal Glomerulonephritis

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1. Introduction

Acute poststreptococcal glomerulonephritis (APSGN) is one of the most common and important renal diseases resulting from a prior infection with group A β-hemolytic streptococcus (GAS) (Ash and Ingulli, 2008). Typical clinical features of the disease include an acute onset with gross hematuria, edema, hypertension and moderate proteinuria (acute nephritic syndrome) 1 to 2 weeks after an antecedent streptococcal pharyngitis or 3 to 6 weeks after a streptococcal pyoderma (Ahn & Ingulli 2008; Rodriguez-Iturbe & Mezzano, 2009). Gross hematuria usually disappears after a few days, while edema and hypertension subside in 5 to 10 days (Rodriguez-Iturbe & Mezzano, 2009). Although the incidence of APSGN appears to be decreasing in industrialized countries, more than 472,000 cases with APSGN are estimated to occur each year worldwide, with 97% of them occurring in developing countries (Carapetis et al., 2005; Eison et al., 2010).

APSGN occurs most commonly in children, 5 to 12 years old (Ahn & Ingulli, 2008), although 5 to 10 percent of the patients are more than 40 years old (Yoshizawa, 2000). The immediate and long-term prognoses of APSGN are excellent for children, assuming it is diagnosed in a timely fashion (Kasahara et al., 2001, Rodriguez-Iturbe & Musser, 2008). In contrast, adult patients with APSGN show markedly worse prognoses both in the acute phase and in the long-term (Rodriguez-Iturbe & Musser, 2008).

The most popular theory of the pathogenic mechanism of APSGN has been the immune-complex theory, which involves the glomerular deposition of nephritogenic streptococcal antigen and subsequent formation of immune complexes *in situ* and/or the deposition of circulating antigen-antibody complexes (Oda et al., 2010). Two antigens have been actively investigated as the potential causes of APSGN (Rodriguez-Iturbe & Musser, 2008): the nephritis-associated plasmin receptor (NAPlr) also known as streptococcal glyceraldehyde-3-phosphate dehydrogenase (Yamakami et al., 2000; Yoshizawa et al., 2004), and a cationic cysteine proteinase known as streptococcal pyogenic exotoxin B (SPEB) (Batsford et al., 2005).

Patients with APSGN sometimes exhibit atypical or unusual clinical manifestations, which may lead to diagnostic delay or misdiagnosis of the disorder (Eison et al. 2011; Pais et al. 2008). Recognition of these unusual manifestations in cases of APSGN is important in order to assure that the patient receives adequate treatment. In this chapter, I review the atypical clinical manifestations of APSGN.

2. Atypical manifestations of APSGN

Atypical manifestations of APSGN can be classified as the following: co-occurrence of immune-mediated diseases; non immune-mediated complications; and unusual clinical presentations or courses (Table 1).

Immune-mediated diseases
Acute rheumatic fever (ARF)
Poststreptococcal reactive arthritis (PSRA)
Vasculitis
Immune thrombocytopenic purpura (ITP)
Autoimmune hemolytic anemia (AIHA)
Diffuse alveolar hemorrhage (DAH)
Uveitis
Non immune-mediated complications
Posterior reversible encephalopathy syndrome (PRES)
Thrombotic microangiopathy (TMA)
Gallbladder wall thickening
Unusual clinical presentations or courses
Minimal urinary abnormalities
Recurrence

Table 1. Atypical manifestations of acute poststreptococcal glomerulonephritis

2.1 Immune-mediated diseases

Immune-mediated diseases most likely result from immune-complex formation between streptococcal antigens and their associated antibodies, and include acute rheumatic fever, poststreptococcal reactive arthritis, vasculitis, immune thrombocytopenic purpura, autoimmune hemolytic anemia, diffuse alveolar hemorrhage and uveitis.

2.1.1 Acute rheumatic fever

Acute rheumatic fever (ARF) is an autoimmune disease that follows infection by GAS and is characterized by inflammation of several tissues that gives rise to typical clinical characteristics (the so-called Jones criteria) including carditis/valvulitis, arthritis, chorea, erythema marginatum, and subcutaneous nodules (Steer & Carapetis, 2009). ARF is rare in developed countries, but it remains common in developing countries and some poor, mainly indigenous populations of wealthy countries (Steer & Carapetis, 2009; Carapetis et al., 2005).

Although both ARF and APSGN develop following GAS infection, the two diseases have different epidemiology, immunology and bacteriology, and simultaneous occurrence of them in the same patient is rare (Lin et al., 2007). Since Gibney et al. first reported a patient with co-occurrence of ARF and histologically proven APSGN (Gibney et al., 1981), seventeen patients with concurrent ARF and APSGN have been reported (Akasheh et al., 1995; Ben-Dov et al., 1985; Castillejos et al., 1985; Imanaka et al., 1995; Kakkera et al., 1998; Kujala et al., 1989; Kula et al., 2003; Kwong et al., 1987; Lin et al., 2003; Mastell et al., 1990; Öner, et al., 1993; Said et al., 1986; Sieck et al., 1992; Sinha et al., 2007).

Fourteen patients were children and ten were male. Eight patients initially presented with ARF preceding APSGN, 3 patients suffered from ARF following the development of APSGN and both ARF and APSGN simultaneously occurred in 6 patients. Although many patients showed carditis (16 out of 17 patients) and polyarthritis (13 out of 17 patients), the remaining characteristics (erythema marginatum, chorea and subcutaneous nodules) developed in only 4 patients, 1 patient and 1 patient, respectively.
Although it remains unclear why simultaneous occurrence of APSGN and ARF is so rare, one explanation may be that only few streptococcal strains have both nephritogenic and rheumatogenic antigenic features (Lin et al., 2003).

2.1.2 Poststreptococcal reactive arthritis

Poststreptococcal reactive arthritis (PSRA) is defined as acute arthritis of more than 1 joint following an episode of GAS infection in a patient whose illness does not fulfill the Jones criteria for the diagnosis of ARF (Barash et al., 2008; Gerber M, 2007). It remains controversial whether or not PRSA and ARF are distinct entities or not (Gerber M, 2007). Mackie and Keat reviewed 188 published cases of PSRA and concluded that PSRA was a heterogenous group of clinical entities (Mackie & Keat, 2004). However, two recent studies suggested that PSRA and ARF were separate disease entities on the basis of the differences in clinical presentation and disease course (Barash et al., 2008; van der Helm-van Mil, 2010). Compared to patients with ARF, patients with PSRA are older, respond poorer to salicylates and have non-migratory and persistent arthritis (Tokura et al., 2008; van der Helm-van Mil, 2010). The precise pathogenic mechanism underlying the development of PSRA is unclear, production of antistreptococcal antibodies that cross-react with human epitopes causing inflammation and tissue damage is a likely pathogenic mechanism for PSRA, as has been proposed for ARF (Niewold & Ghosh, 2003).
Simultaneous occurrence of PSRA and APSGN is rare, with only 3 cases having been reported (Niewold & Ghosh, 2003; Sugimoto et al., 2008; Tokura et al., 2008). Niewold & Ghosh described a 44-year-old man who developed severe PSRA and APSGN after a subclinical streptococcal infection (Niewold & Ghosh, 2003). Tokura et al. reported a 16-year-old man who presented with simultaneous occurrence of APSGN and PSRA with symmetric persistent tenosynovitis in hands and feet (Tokura et al., 2008). Sugimoto et al. described a 61-year-old man who exhibited PSRA and APSGN with marked renal interstitial inflammation after bacterial endophthalmitis due to *Streptococcus pyogenes* (Sugimoto et al., 2008). Arthritis of all the patients improved with corticosteroid therapies without any sequellae.

2.1.3 Vasculitis

Vasculitis is not a well-recognized condition associated with GAS infection, but there have been several reports of Henoch-Schönlein purpura (HSP) (al-Sheyyab et al., 1999) or Henoch-Schönlein purpura with nephritis (HSPN) (Masuda et al., 2003), cutaneous leukocytoclastic vasculitis (Chalkias et al, 2010), vasculitic neuropathy (Traverso et al., 1997), polyarteritis nodosa (PN) (David et al., 1993) and unclassified systemic vasculitis (Lucas & Moxham, 1978). Although the precise pathogenic role of GAS infection contributing to the development of vasculitis remains unclear, an immune complex-mediated mechanism triggered by GAS infection has been postulated (Ritt M. et al., 2006).
Vasculitides including cutaneous vasculitis mimicking HSP, cerebral vasculitis, PN, necrotizing vasculitis and Wegener's granulomatosis have been described to occur in patients with APSGN.

HSP is an IgA immune complex-mediated systemic leukocytoclastic vasculitis of small vessels that primarily affects the skin, gastrointestinal tract, joints and kidney (Dedeoglu & Sundel, 2007; Robson & Leung , 1994). Respiratory infections with GAS preceding the onset of HSP have been reported in up to one-third of cases (Dedeoglu & Sundel, 2007). APSGN patients simultaneously presenting with HSP or vasculitis mimicking HSP are rare and only five patients have been reported. Goodyer et al. described two boys with histologically proven APSGN, in whom cutaneous vasculitis and abdominal symptoms mimicked HSP (Goody et al., 1978). Onisawa et al. reported a patient with concurrent APSGN and cutaneous leukocytoclastic vasculitis without IgA deposition (Onisawa et al., 1989). Maruyama et al. presented a patient with congenital complement 9 deficiency exhibiting biopsy-proven APSGN and clinical symptoms mimicking HSP (Maruyama et al., 1995). Matsukura et al. also described a 20-month-old girl with biopsy-proven APSGN, who presented with cutaneous vasculitis mimicking HSP (Matsukura et al., 2003). All patients recovered completely without any sequellae.

Central nervous system abnormalities of APSGN are usually secondary to acute severe hypertension, electrolyte disturbances or uremia, but can also be attributed to cerebral vasculitis (Dursun et al., 2008; Ritt et al., 2006). To date, five cases of cerebral vasculitis associated with APSGN have been reported (Dursun et al., 2008; Kaplan et al., 1993; Ritt et al., 2006; Rovang et al., 1997; Wong & Morris, 2001), in which 4 patients were children. Clinical features of cerebral vasculitis in APSGN include severe headache with nausea and vomiting, transient focal neurological signs, visual disturbances, and seizures. Although the computed tomography of the brain may not detect abnormalities, the magnetic resonance imaging of the brain often demonstrated multiple supratentorial areas of abnormal signal intensity in the white and adjacent grey matter (Dursun et al., 2008). All patients underwent corticosteroid therapy (three of them commenced on methylprednisolone pulse therapy) and recovered without any neurological sequellae.

PN is a necrotizing vasculitis affecting the medium-sized muscular arteries (Dillon et al., 2010). Five patients with co-occurrence with PN and APSGN have been described. Fordham et al. reported three young adult patients with both PN and APSGN, two of whom died secondary to multi-system organ failure (Fordham et al., 1964). Blau et al. described two children with PN who also had serological and clinical evidence of APSGN (Blau et al., 1977).

Although extremely rare, necrotizing vasculitis other than PN (Bodaghi et al., 1987; Ingelfinger et al., 1977) and Wegener's granulomatosis (Garrett et al., 1993) has also been reported in patients with APSGN.

2.1.4 Immune thrombocytopenic purpura

Immune thrombocytopenic purpura (ITP) is an immune-mediated acquired disorder in which antiplatelet antibodies cause accelerated destruction of platelets, resulting in thrombocytopenia and an increase risk of bleeding (Psaila & Bussel, 2007). Childhood ITP often occurs following an infection with viruses such as varicella zoster, rubella, Epstein-Barr, influenza, or human immunodeficiency virus (Tasic & Polenakovic, 2003), but may also be preceded by a bacterial infection (Muguruma et al., 2000). Recently, a number of studies have suggested an association between *Helicobacter pylori* and ITP (Cooper & Bussel, 2006).

Since Kaplan and Esseltine first reported ITP in two patients with APSGN (Kaplan & Esseltine, 1978), five cases of ITP in patients with APSGN have been reported (Muguruma et al., 2000; Rizkallah et al., 1984; Tasic & Polenakov, 2003). All patients were children (4 to 7 years of age) who underwent corticosteroid therapy and fully recovered from APSGN and

ITP. One patient exhibited a marked increase in platelet-associated immunoglobulin G level (Muguruma et al., 2000). Although the precise pathogenic mechanism for the development of ITP in patients with APSGN is unclear, Muguruma et al. speculated about the pathogenesis of associated diseases through production of autoantibodies cross-reactive against GAS and against platelets (Muguruma et al., 2000).

2.1.5 Autoimmune hemolytic anemia

Anemia is common in APSGN and traditionally it has been attributed solely to volume overload (Eison et al., 2011). However, autoimmune hemolytic anemia (AIHA) has recently been reported in patients with APSGN. AIHA is a clinical condition in which IgG and/or IgM antibodies bind to red blood cells (RBC) surface antigens and initiate RBC destruction via the complement system and the reticuloendothelial system (Gehrs & Friedberg, 2002). Subtypes include warm AIHA, cold AIHA, mixed-type AIHA and drug-induced immune hemolytic anemia. Furthermore, cold AIHA has been categorized into cold agglutinin syndrome (CAS) and paroxysmal cold hemoglobinuria (PCH). Infectious agents associated with CAS or PCH include *Mycoplasma pneumonia*, Epstein-Barr virus, adenovirus, cytomegalovirus, influenza viruses, human immunodeficiency virus, measles, mumps, *Escherichia coli*, *Listeria monocytogenes, Haemophilus influenza* and *Treponema pallidum* (Gehrs & Friedberg, 2002).

Greenbaum et al. described three children with both APSGN and cold AIHA, two of whom had an anti-I autoantibody and thereby were diagnosed with CAS (Greenbaum et al., 2003). Two patients were transfused and all patients recovered from AIHA and APSGN. Cachat et al. presented a 10-year-old child with concurrent cold AIHA and APSGN, who developed anuric acute renal failure and profound anemia (Cachat et al., 2003). The patient responded well to corticosteroid therapy and had a full renal and hematological recovery.

2.1.6 Diffuse alveolar hemorrhage

Diffuse alveolar hemorrhage (DAH) is sometimes accompanied by glomerulonephritis, and is often referred to as pulmonary-renal syndrome (Papiris et al., 2007). Pulmonary-renal syndromes include Goodpasture's syndrome, antineutrophil cytoplasmic antibody-associated vasculitis, immune complex-associated glomerulopathy and thrombotic microangiopathy (Papiris et al., 2007).

DAH associated with APSGN is extremely rare and only three patients have been reported. Chugh et al. described a 38-year-old-male with concurrent DAH and crescentic APSGN who progressed to end-stage renal failure (Chugh et al., 1981). Gilboa et al. reported a 12-year-old girl who exhibited a noncrescentic APSGN and DAH (Gilboa et al., 1993). Sung et al. recently described a 59-year-old-woman with APSGN and DAH (Sung et al., 2007). DAH in all three patients subsided after intravenous corticosteroid therapies. The pathogenic mechanism of DAH in APSGN remains unclear.

2.1.7 Uveitis

Uveitis is believed to be an immunological response to exogenous and endogenous antigens (Leiba et al., 1998) and can occur following GAS infection, so-called "poststreptococcal uveitis". Since Cokingtin & Han reported the first case of poststreptococcal uveitis in 1991 (Cokingtin & Han, 1991), several patient reports and case series with poststreptococcal uveitis have been presented (Leiba et al., 1998, Ur Rehman et al., 2006).

Feldon et al. recently reported the first case of a child with concomitant APSGN and uveitis, whose uveitis subsided within a few days with topical corticosteroid and mydriatic treatment (Feldon et al., 2010).

2.2 Non immune-mediated complications

Non immune-mediated complications of APSGN include posterior reversible encephalopathy syndrome, thrombotic microangiopathy and gallbladder wall thickening.

2.2.1 Posterior reversible encephalopathy syndrome

Posterior reversible encephalopathy syndrome (PRES), also known as reversible posterior leukoencephalopathy syndrome (RPLS), is a recently described brain disorder associated with findings on neuroimaging that suggest white-matter edema, mostly in the posterior parietal-temporal-occipital regions of the brain (Hinchey et al., 1996). However, radiological lesions in PRES are rarely isolated to these areas, and often involve the cortex, frontal lobes, basal ganglia and brainstem (Fugate et al., 2010) (Fig. 1).

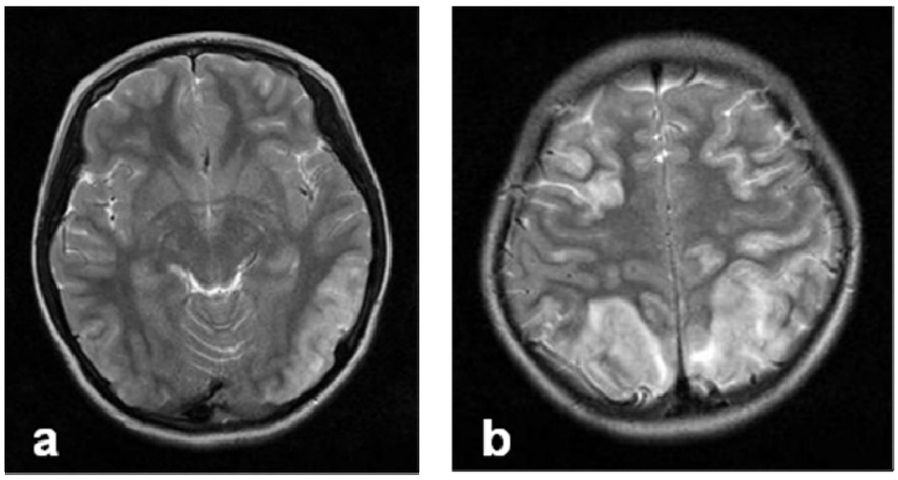

Fig. 1. Magnetic resonance images (MRI) of the brain (T2-weighted image) showing increased intensity in the cortex and subcortical white matter of left occipital (a), bilateral parietal and right frontal lobes (b), consistent with PRES.

The clinical characteristics of the disease include headache, decreased alertness, altered mental functioning, seizures, and visual loss including cortical blindness (Hinchey et al., 1996). The syndrome has been associated with acute hypertension, preeclampsia or eclampsia, glomerulonephritis, sepsis, autoimmune disorders and immunosuppressive or chemotherapeutic treatments (Bartynski, 2008a; Hinchey et al., 1996). Although the underlying pathophysiology of PRES remains elusive, three theories have been proposed: 1) hypertension-induced breakdown in cerebral auto-regulation; 2) cerebrovascular endothelial dysfunction and; 3) vasoconstriction and hypoperfusion with subsequent ischemia and vasogenic edema (Bartynski, 2008b; Fugate et al., 2010). The preferential involvement of the posterior brain in PRES may be caused by its relative paucity of sympathetic innervation in comparison to the anterior circulation (Froehlich et al., 1999). The outcome of PRES is generally favorable, but delay in initiating the appropriate treatment may result in permanent damage to the brain (Fux et al., 2006; Garg, 2001).

While it is estimated that PRES occurs in 5% to 10% of children hospitalized with acute glomerulonephritis of all etiologies, the prevalence of PRES associated with APSGN is unknown (Froehlich et al., 1999). PRES caused by hypertension has been reported in 7 children (from 7 to 15 years of age) with APSGN (Froehlich et al., 1999; Fux et al., 2006; Guputa et al., 2010; Nordby, 1997; Özcakar et al., 2004; Soylu et al., 2001). Six patients complained of headache, 5 exhibited decreased alertness and seizures, and 3 had altered mental functioning and visual loss. All patients exhibited abnormal findings of the brain MRI or CT in the white matter of the parietal and occipital lobes, and recovered without any neurological sequellae following adequate treatment of the associated hypertension.

One patient with APSGN suffered from PRES without severe hypertension (Nordby, 1997). The most important factor in development of pediatric hypertensive PRES is the rapidity of blood pressure elevation and the degree of elevation relative to the patient's baseline pressure (Froehlich et al., 1999). It has been suggested that blood pressures more than 30% above normal for age should alert clinicians to the possibility of hypertensive PRES (Nordby, 1997).

2.2.2 Thrombotic microangiopathy

Thrombotic microangiopathy (TMA) is a pathological term used to describe occlusive microvascular thrombus formation and is most commonly associated with hemolytic uremic syndrome (HUS) and thrombotic thrombocytopenic purpura (TTP) (Keir & Coward, 2010). Pathological features of TMA include vessel wall thickening, swelling and detachment of the endothelial cell from the basement membrane, accumulation of material in the subendothelial space, intraluminal platelet thrombosis, partial or complete vessel luminal obstruction and fragmentation of red blood cells (Keir & Coward, 2010). HUS is defined as the triad of microangiopathic hemolytic anemia, thrombocytopenia, and acute renal injury (Copelovitch & Kaplan, 2008). TTP is characterized by the pentad of microangiopathic hemolytic anemia, thrombocytopenia, fever, acute renal injury, and neurological abnormalities (Copelovitch & Kaplan, 2008).

TMA has been reported in 8 patients with APSGN, consisting of 5 children and 3 adults (Duvic et al., 2000; Izumi, et al., 2005; Laube, et al., 2001; Medani et al., 1987; Proesmans, 1996; Siebels et al., 1995; Tan et al., 1998). All patients exhibited severe hypertension. Hemodialysis or peritoneal dialysis was required in 2 patients. Renal biopsy showed histological features of both APSGN and TMA in 3 patients, and revealed characteristics of APSGN without features of TMA in 5 patients. The outcome in all patients was excellent.

The precise pathogenesis of TMA in patients with APSGN is unclear, although two causes have been postulated: severe hypertension and streptococcal neuraminidase (Duvic et al., 2000; Laube et al., 2001; Izumi et al., 2005). HUS has been reported as a complication of severe hypertension, regardless of the cause (Broyer, 1995). If severe hypertension is transient, histological lesions of TMA are absent. When hypertension becomes malignant, renal histological lesions show features of TMA (Duvic et al., 2000). Another possible cause of TMA in APSGN is alteration of vascular endothelial cells by streptococcal neuraminidase. Circulating neuraminidase causes exposure of the cryptic T-antigen on cell surfaces, to which most people possess a naturally occurring antibody. Therefore antigen-antibody interaction may damage the vascular endothelial cells leading to the clinical manifestations of HUS (Izumi et al., 2005).

2.2.3 Gallbladder wall thickening

Thickening of the gallbladder wall is the most common findings in acute cholecystitis, but it has also been reported in patients with kidney diseases including pyelonephritis (Talarico & Rubens, 1990) and chronic kidney failure (van Breda Vriesman et al., 2007). Only one child with APSGN and gallbladder wall thickening has been reported (Watanabe & Baba, 2009). Although the pathogenesis of this complication is unclear, elevated systemic venous pressure or subclinical vasculitis may have caused edema of the gallbladder wall (Watanabe & Baba, 2009).

2.3 Unusual clinical presentations or courses

Unusual clinical presentations or courses of APSGN include acute nephritic syndrome with minimal urinary abnormalities, and recurrence of the disease.

2.3.1 Minimal urinary abnormalities

Patients with APSGN usually exhibit hematuria and proteinuria. However, Blumberg and Feldman first reported two children with biopsy-proven APSGN without any urinary abnormalities (Blumberg & Feldman, 1962). Thereafter, several authors described biopsy-proven APSGN patients with minimal or no urinary abnormalities including 13 children and 4 adult patients (Albert et al., 1966; Cohen & Levitt, 1963; Dunn, 1967; Fujinaga et al., 2007; Goorno et al., 1967; Grossman et al., 1973; Hoyer et al., 1967; Kandall et al., 1969; Kobayashi et al., 1971). All patients exhibited edema and hypertension, and seven showed pulmonary edema or congestion. Hypertensive encephalopathy occurred in one patient (Hoyer et al., 1967) and acute rheumatic fever developed in another patient (Cohen & Levitt, 1963). All patients recovered completely without any sequellae. The mechanism for the elaboration on normal or minimal urinary abnormalities during the course of APSGN is unclear (Fujinaga et al., 2007; Kandall et al., 1969).

2.3.2 Recurrence

Recurrence of APSGN is a well-recognized, but relatively rare phenomenon, probably due to the relatively limited number of nephritogenic strains of streptococci and the acquisition of protective immunity against a nephritogenic streptococcal antigen after an initial episode of APSGN (Watanabe & Yoshizawa, 2001). Ramberg first mentioned this condition and reported eleven patients with the recurrent attacks out of 152 patients with APSGN (Ramberg, 1947). Thereafter, several clinical studies of APSGN have suggested an incidence of recurrent APSGN that ranges from 0.7% to 7.0% (Baldwin D et al., 1974; Bernstein et al., 1960; Dodge W. et al., 1968; Sanjad et al., 1977; Roy et al., 1969). In addition, a few case reports of recurrent APSGN have been described (Casquero et al., 2006; Derakhshan 2002; Kim et al., 1979; Rosenberg et al., 1984; Velhote et al., 1986; Watanabe & Yoshizawa, 2001). Clinical features and outcomes were well-described in 35 patients including 22 children. Most patients suffered from one recurrent episode, but one patient exhibited 2 recurrent attacks of APSGN (Velhote et al., 1986). Twenty-nine patients recovered completely, whilst 4 patients continued to have some urinary abnormalities and two patients progressed to end-stage renal failure.

Although the exact mechanism leading to recurrence of APSGN has not yet been determined, three possible explanations have been postulated: the suppression of immune response against nephritogenic streptococcal strains due to early antibiotic therapy (Roy et

al., 1969; Sanjad et al., 1977); an absence of natural immune responses against nephritogenic streptococcal components without antibiotic therapy (Watanabe & Yoshizawa, 2001), and; a failure to exclude microbial agents through the digestive and respiratory tract due to IgA deficiency (Casquero et al., 2006).

Sanjado et al. suggested that reinfection with the same type of *Streptococcus* would occur if the patient lacked antibodies against that particular type, and penicillin therapy given in the first ten days after a streptococcal infection suppressed the formation of type-specific immunity conferring antibodies, which might increase the chances of re-infection with the same nephritogenic strain responsible for the initial episode of APSGN (Sanjad et al., 1977).

Recently, Yoshizawa et al. identified a new nephritogenic streptococcal antigen and termed it nephritis-associated plasmin receptor (NAPlr) (Yamakami et al., 2000; Yoshizawa et al., 2004). They demonstrated that most patients with APSGN had high titers of long-lasting antibody against NAPlr and that it was present in glomeruli in 100% of patients with APSGN early in the disease (Yoshizawa et al., 2004). Watanabe and Yoshizawa described an 8-year-old boy with recurrent ASPGN who did not have serum antibodies against NAPlr, even though NAPlr was detected in glomeruli of an early kidney biopsy specimen from the patient during the second attack of APSGN. These results indicated that recurrence of APSGN in some patients might be caused by an absence of a natural immune response to NAPlr (Watanabe & Yoshizawa, 2001).

Recently, Casquero et al. published a patient with selective IgA deficiency who experienced two episodes of APSGN (Casquero et al., 2006), suggesting that a failure of IgA defenses might also predispose to streptococcal re-infection and cause recurrent APSGN.

3. Conclusions

Patients with APSGN sometimes exhibit atypical or unusual clinical manifestations, which are divided into 3 categories: immune-mediated diseases (ARF, PSRA, vasculitis, ITP, AIHA, DAH and uveitis), non-immune mediated conditions (PRES, TMA and gallbladder wall thickening), and unusual clinical presentations or courses (minimal urinary abnormalities and recurrence).

Immune-mediated diseases seem to result from immune-complex formation between streptococcal antigens and their associated antibodies. Hypertension contributes to the development of PRES and TMA, while fluid retention results in PRES and gallbladder wall thickening. Recurrence of APSGN may be the consequence of suppressed immune responses against nephritogenic streptococcal strains caused by early antibiotic therapy, by the absence of natural immune responses against NAPlr, or by selective IgA deficiency.

Because atypical or unusual manifestations of APSGN may lead to diagnostic delays or misdiagnosis of the disorder, recognition of them is important in order to assure that the patient receives adequate treatment.

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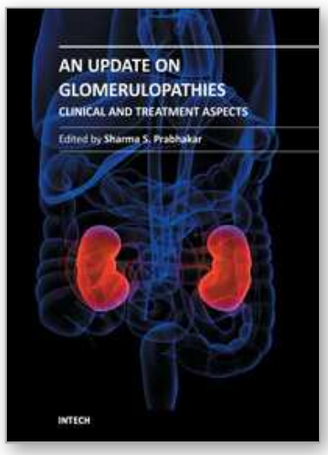

An Update on Glomerulopathies - Clinical and Treatment Aspects
Edited by Prof. Sharma Prabhakar

ISBN 978-953-307-673-7
Hard cover, 468 pages
Publisher InTech
Published online 02, November, 2011
Published in print edition November, 2011

An Update on Glomerulopathies - Clinical and Treatment Aspects is a systemic overview of recent advances in clinical aspects and therapeutic options in major syndromes of glomerular pathology. The book contains twenty four chapters divided conveniently into five sections. The first section deals with primary glomerulopathies, and the second section is devoted to glomerulopathies complicating infectious conditions. The third section deals with systemic autoimmune disorders and vasculitides which constitute major causes of glomerular disease and often renal failure. The fourth section includes chapters discussing the glomerular involvement in some major metabolic and systemic conditions. The final section has chapters which relate to some general aspects of glomerular diseases. This book will form an excellent reference tool for practicing and academic nephrology community.

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