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Infection-Related Glomerulonephritis

Mazdak A. Khalighi[a] Anthony Chang[b]

[a]Department of Dermatology, University of Utah, Salt Lake City, UT, USA; [b]Department of Pathology, University of Chicago Medicine, Chicago, IL, USA

Key Points

- Endocapillary glomerular hypercellularity with abundant neutrophils may provide a first diagnostic clue for infection-related GN.
- Immunofluorescence microscopy in cases of infection-related GN typically shows prominent deposition of C3 with or without staining for immunoglobulins, mainly IgG.
- Electron microscopy is crucial for diagnostic decision-making.
- Subepithelial hump-shaped deposits are a characteristic ultrastructural finding in infection-related GN; they are preferentially seen in mesangial notch/waist regions.
- Infection-related GN may be associated with underlying complement abnormalities and can fall into the spectrum of C3 glomerulopathies.

Keywords
Renal pathology · Kidney biopsy · Complement

Abstract

Background: There has been a long, storied relationship between various bacterial infections and glomerular injury, which is now encompassed under the term of infection-related glomerulonephritis (GN). The clinical and pathologic manifestations vary depending on the duration, magnitude, and underlying pathogen associated with the inciting infectious process. A brief and acute episode may lead to a self-limiting glomerular manifestation while a chronic or repetitive infection can result in persistent and irreversible injury. In this review, we will discuss the clinical and pathologic findings associated with the infection-related glomerulonephritides. ***Summary:*** An acute exudative GN with an influx of neutrophils is the most characteristic morphologic alteration associated with infection-related glomerular injury. The immunofluorescence staining pattern often reveals prominent complement component C3 deposition in both capillary walls and mesangial regions with or without accompanying immunoglobulin. Large subepithelial electron-dense deposits known as "humps" are the hallmark ultrastructural finding; however, these features can also be present in C3 glomerulopathies, which are often triggered by infections and may have similar underlying abnormalities in alternative pathway complement activation. In addition, other glomerular injuries can simultaneously be present along with infection-related GN, such as diabetic nephropathy, lupus nephritis, or immunoglobulin A nephropathy, constituting a true diagnostic challenge for the pathologist. ***Key Messages:*** Bacterial infection-related GN represents a spectrum of glomerular injury with variable clinical and pathologic presentations. The pathologic findings can show overlap with other glomerular diseases, and different forms of infection-related GN vary in terms of prognosis and treatment approach.

karger@karger.com
www.karger.com/gdz

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Correspondence to:
Anthony Chang, anthony.chang@uchospitals.edu

Introduction

Infection-related glomerulonephritis (GN) encompasses the entities of postinfectious (poststreptococcal) GN, immunoglobulin (Ig) A-dominant (Staphylococcus-associated) infection-related GN, endocarditis-associated GN, and shunt nephritis. Poststreptococcal GN has historically represented the prototypical example of infection-related GN; however, an epidemiologic shift has occurred in the past 2 decades in regards to the underlying cause, morphologic and ultrastructural findings, and therapeutic interventions for infection-related glomerular injury [1]. Of note, various infectious agents including some viruses might cause the morphologic phenotype of infection-related GN. Herein, we will review the clinical and pathologic features of bacterial infection-related GN including typical poststreptococcal GN, IgA-dominant infection-related GN, and infectious endocarditis-associated GN with emphasis on ultrastructural findings.

Pathophysiology and Recent Findings

The various forms of infection-related GN are largely driven by deposition of complement with or without associated immunoglobulins followed by the subsequent immune/inflammatory response. Deposition of complement and to a lesser degree immune complexes likely involves multiple mechanisms, including entrapment of preformed circulating immune complexes, in situ immune complex formation related to either embedded bacterial antigens within the glomerular basement membrane, or formation of antibodies directed against endogenous basement membrane antigens due to molecular mimicry and immunoglobulin-independent activation of complement by bacterial antigens [2, 3].

In classic postinfectious/poststreptococcal GN, nephritogenic strains of group A Streptococcus have been implicated with expression of streptococcal pyogenic exotoxin B. This antigen can bind to glomeruli and activate complement via the lectin pathway. It can also elicit an antibody response leading to immune complex formation [4]. Recently, the role of complement dysregulation in the setting of postinfectious GN has been explored with emphasis on its relationship to C3 glomerulopathies [5]. Some patients with infection-related GN have underlying abnormalities in the activation cascade of the alternative complement pathway. Such dysregulation includes the transient and self-limiting formation of autoantibodies (such as C3 nephritic factor and anticomplement factor H antibodies) or genetic abnormalities affecting complement factor H or complement factor H-related protein 5 [6–8]. There is increasing evidence that infection-related GN may represent a milder (and often self-limited) form in the disease spectrum referred to as "C3 glomerulopathies" and that infection may trigger C3 glomerulopathy in patients with underlying complement dysregulation [5, 8].

The mechanism involved in IgA-dominant infection-related GN, which is typically associated with *Staphylococcal aureus* infection, has been less extensively studied. It is thought to involve bacterial superantigens, such as staphylococcal enterotoxin B. These superantigens can induce overactivation of the immune system via interaction with the major histocompatibility complex on antigen-presenting cells, activation of T cells, and related stimulation of B cells with expression of polytypic IgA [2, 9].

Mechanisms causing endocarditis-associated GN and shunt nephritis can vary and depend upon the infectious organism. In addition, some patients with endocarditis-associated GN can develop antineutrophil cytoplasmic antibodies (ANCA) and a pauci-immune vasculitic-type GN [10, 11]. However, it is beyond the scope of this review and will not be further discussed.

Clinical Manifestations

Infection-related GN can be seen with different underlying organisms and stimuli. The prognosis and clinical/treatment implications differ depending on the magnitude and duration of the infection and related activity/chronicity of glomerular injury.

Post-Streptococcal GN

Post-streptococcal GN is typically seen 1–4 weeks following resolution of either streptococcal pharyngitis or impetigo most often in children and young adults; however, older adults can also be affected [2, 3]. Clinical presentation ranges from asymptomatic hematuria and proteinuria to nephritic syndrome. Renal dysfunction with elevated serum creatinine levels is common and can occasionally present with clinical signs of a rapidly progressive GN. Serologic studies for antistreptolysin O or anti-DNAase B antibodies are often positive, indicating a current or past streptococcal infection. Hypocomplementemia is a near universal feature in the active disease phase with typically low C3 and often normal C4 levels.

Recovery of complement levels, typically within 6 weeks, is common as the disease tends to be self-limiting, particularly in children [3, 5]. Most young patients regain normal renal function (>90% of children and young adults) with supportive care only; however, immunosuppression has been used in those cases presenting with a crescentic GN [2, 3]. Unlike children, elderly patients with poststreptococcal GN tend to have less favorable renal outcomes with persistent renal dysfunction. A subset of patients, both children and adults, may develop persistent renal dysfunction, which should prompt further studies of potential alternative complement pathway dysregulation [5, 8].

IgA-Dominant Infection-Related GN

Unlike poststreptococcal GN, IgA-dominant infection-related GN is primarily seen in older adults and commonly occurs in the setting of an active bacterial infection, typically involving *Staphylococcus aureus* [12–14]. The underlying infectious process can range from superficial skin infections to deep-seated infections, including endocarditis and osteomyelitis. Most reports demonstrate a male predominance [15] and an increased incidence in patients with underlying comorbidities including diabetes mellitus, alcoholism, and hypertension [12, 13, 16]. Patients generally present with acute kidney injury, hematuria, and proteinuria, which is often in the nephrotic range. The acute kidney injury is typically severe with the majority of cases showing an acute rise in serum creatinine levels to >3 mg/dL [13, 14, 16–18]. Hypocomplementemia is a common finding reported in up to 60% of cases. Circulating cryoglobulins have rarely been noted [14, 17]. The primary therapeutic strategy involves antibiotic therapy targeting the underlying infectious process. Outcomes vary with progression to ESRD seen in 20–80% of patients and varying degrees of persistent renal dysfunction in the remaining cases [12–18]. Poor outcome is likely related to the severity of acute injury that is often superimposed on other preexisting renal comorbidities, such as diabetic nephropathy. Accurate and timely diagnosis of IgA-dominant IRGN is of paramount importance, both for conservation of renal function and also because GN may be the first clinical clue to an underlying staphylococcal infection, particularly for deep-seated infections.

Endocarditis-Associated GN

GN is a well-documented complication of infectious endocarditis with a prevalence ranging from 2 to 60% [2]. Historically, an infectious endocarditis was primarily seen in patients with injured cardiac valves and caused by organisms of relatively low virulence, such as *Streptococcus viridians* and *Staphylococcus epidermidis*. However, recently, other organisms such as *Staphylococcus aureus* have been more commonly detected, possibly as a result of intravenous drug abuse [19]. Other infectious agents seen in endocarditis-associated GN include *Bartonella henselae*, Streptococcus species, and *Coxiella burnetii* [19, 20]. Clinical manifestations include fever, cardiac murmurs, and hepatosplenomegaly. Some patients may show features of a systemic vasculitis, including purpuric skin lesions, and serologic testing for ANCA is positive in as many as 28% of patients [19–21]. Acute kidney injury and a nephritic urine sediment are common while nephrotic-range proteinuria is uncommon [19]. Laboratory studies often show hypocomplementemia, and in 1 study, low C3 levels were present in 53% of patients, while low C4 levels were identified in 19% [19]. Treatment primarily targets the underlying infection with or without concurrent immunosuppression depending on the severity of the kidney injury. On occasion, the infected valve has to be surgically replaced. The overall prognosis for endocarditis-associated GN varies with the largest retrospective series reporting complete recovery in 32%, persistent renal dysfunction in 37%, ESRD in 10%, and death in 21% of patients [19].

Shunt Nephritis

Shunt nephritis is an uncommon complication of ventriculoatrial shunts used as palliative therapy for hydrocephalus. It represents an immune complex-mediated GN caused by hardware infection [22, 23]. As the use of ventriculoperitoneal shunts is now favored, classical shunt nephritis has become exceedingly rare [24]. Systemic signs and symptoms of shunt infection include fever, malaise, nausea, and hepatosplenomegaly. Patients with renal involvement may present with renal failure, hematuria, and proteinuria [23]. Common pathogens include *Staphylococcus epidermidis*, *Propionibacterium acnes*, and other organisms such as *Staphylococcus aureus*. As with other forms of infection-related GN, hypocomplementemia is a common laboratory finding. A diagnosis of infection can often be made with cultures from both cerebrospinal fluid and blood. Treatment typically involves removal of the infected shunt. Although persistent renal dysfunction can occur, recovery of renal function is common following treatment [23].

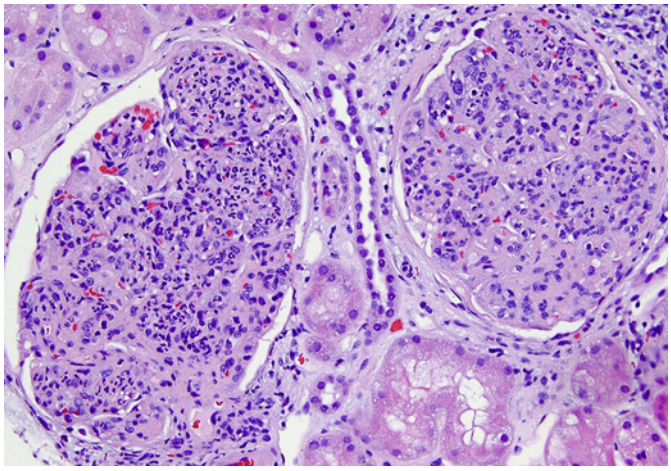

Fig. 1. These glomeruli demonstrate global endocapillary hypercellularity primarily due to a prominent influx of neutrophils, which imparts an accentuation of the lobularity of the glomerular tuft and is descriptively also termed an acute exudative GN (hematoxylin and eosin, original magnification ×400). GN, glomerulonephritis.

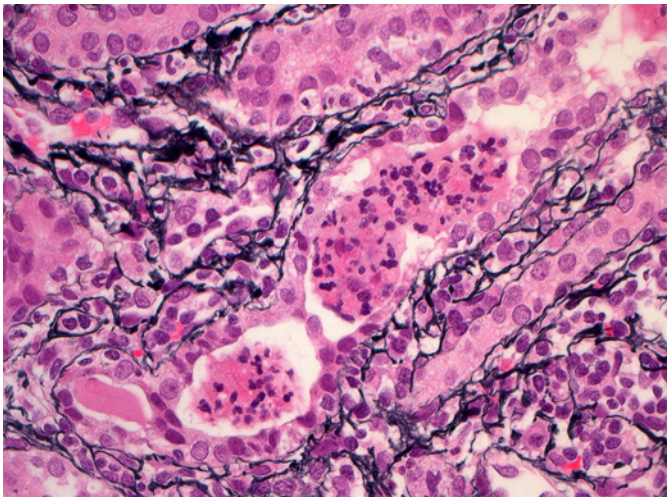

Fig. 2. Aggregates of neutrophils in tubules with neutrophilic tubulitis can be numerous, which may be due to rupture of the glomerular basement membranes and glomerular crescents (not shown) as the possible source. Interstitial neutrophilic infiltration (not shown) may occur when tubular rupture occurs. These findings can mimic acute pyelonephritis (Jones methenamine silver, original magnification ×400).

Light Microscopy

The light microscopic features of poststreptococcal and IgA-dominant infection-related GN are similar. Most cases show prominent endocapillary hypercellularity with numerous neutrophils, a pattern referred to as acute exudative GN (Fig. 1). The glomerular involvement is typically diffuse and global but can be focal and segmental, possibly related to the phase of the injury. In some cases, crescents can be seen but they tend to be focal. Mesangial zones are initially largely unaffected but might expand including mild hypercellularity during disease resolution [2]; mesangiolysis is uncommon. Some cases of IgA-dominant infection-related GN may demonstrate focal cryoglobulin-like features, including scattered subendothelial "wire loop" deposits and intracapillary hyaline pseudothrombi [14, 16, 17]. The presence of an exudative GN is uncommon in cases of typical IgA nephropathy, and this finding should prompt the nephropathologist to favor an infectious process in the context of IgA-dominant deposits by immunofluorescence.

Endocarditis-associated GN presents with a more variable pattern of injury. Often a crescentic GN is found, which can be seen without associated endocapillary or mesangial hypercellularity, followed by glomerular injury presenting as a diffuse proliferative GN, a mesangioproliferative GN [19], or a membranoproliferative GN [20]. Similarly, shunt nephritis shows variable phenotypes such as an endocapillary proliferative, a membranoproliferative, or crescentic GN [22, 23].

The glomerular injury that is seen in infection-related GN is often accompanied by tubulointerstitial changes, including acute tubular injury and interstitial nephritis, which may be rich in neutrophils and include intratubular neutrophil aggregates (Fig. 2), a feature that should not be misdiagnosed as an acute pyelonephritis.

Immunofluorescence Microscopy

Immunofluorescence microscopy characteristically reveals staining for complement component C3 with or without accompanying Ig. Staining along capillary walls typically shows a coarsely granular pattern with numerous large (subepithelial) clumps (representing the subepithelial humps seen by electron microscopy). Granular staining is also found in the mesangium. Described patterns of staining in the setting of IRGN include the "garland," "starry-sky," and "mesangial" patterns. The "garland" pattern demonstrates confluent staining along capillary walls and typically indicates a more active phase of disease (Fig. 3). The "starry-sky" pattern shows randomly scat-

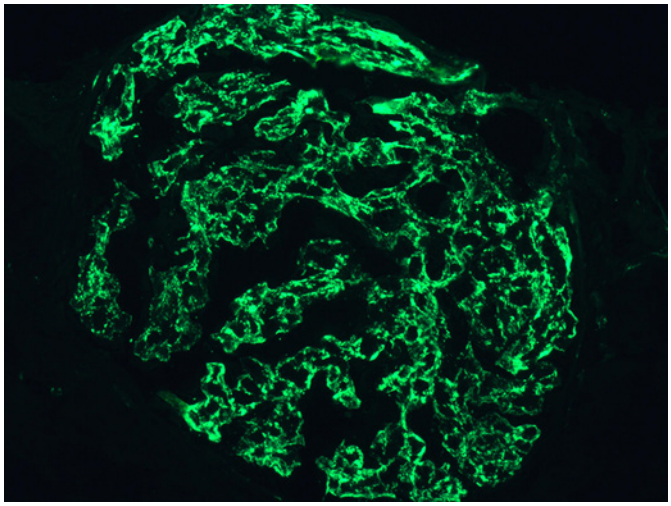

Fig. 3. Diffuse granular C3 immunofluorescence staining along the glomerular capillaries can impart a "garland" pattern as it outlines the capillary walls and the larger globules may offer the first clue regarding the presence of subepithelial "humps" that can be subsequently visualized by electron microscopy (original magnification ×600).

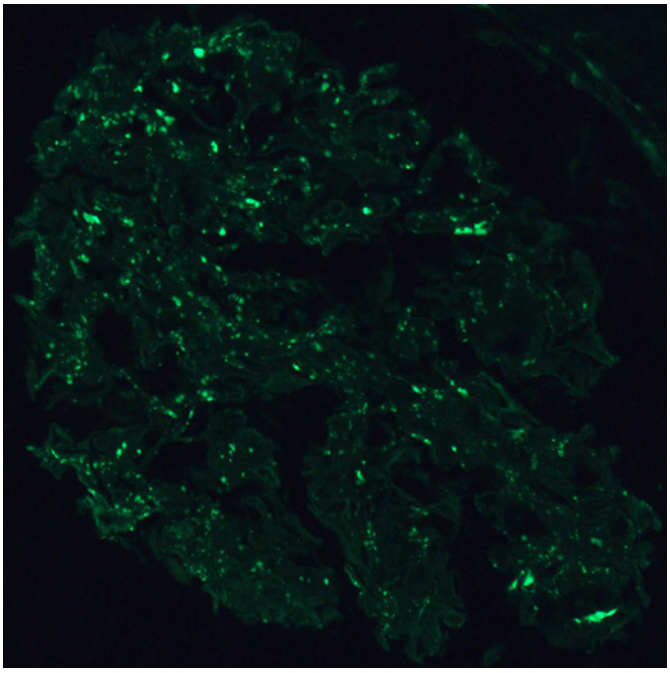

Fig. 4. Immunofluorescence microscopy for C3 can reveal a "starry-sky" pattern when there is both mesangial and capillary staining, but the degree of staining is not as prominent as the "garland" pattern seen in Figure 4 (original magnification ×600).

tered fine and coarse granular staining within the mesangium and capillary walls (Fig. 4) and the "mesangial" pattern demonstrates granular staining primarily localized within mesangial areas. The latter 2 patterns are more indicative of less active disease as the confluent staining seen in the "garland" pattern corresponds to large and often confluent subepithelial hump deposits. In poststreptococcal GN, the accompanying immunoglobulin, if present, is most commonly IgG, whereas a GN related to a *Staphylococcal aureus* infection frequently shows dominant or codominant staining for IgA (IgA-dominant infection-related GN) [1, 2, 12]. Lambda light chain bias, as is frequently noted in IgA nephropathy, may not be seen in IgA-dominant infection-related GN, which may aid in differentiation between these 2 entities. Of note, the chronic phase of infection-related GN may reveal no significant immunofluorescence staining, and a diagnosis might only be rendered based on typical findings made by electron microscopy [25]. An endocarditis-associated GN or shunt nephritis often shows a more variable pattern with dominant staining for C3 and IgM, followed by IgG and IgA [2, 19]. In the absence of significant immunoglobulin deposits, sole staining for complement component C3 raises the differential diagnosis of a C3 GN that can in some patients be associated with an underlying infection. Occasional cases of an endocarditis-associated GN can show a so-called pauci-immune staining pattern with no or only minor immunoglobulin, complement components, and light chain deposits. Such cases can show glomerular tuft necrosis by light microscopy and elevated ANCA serum levels. Potential pitfalls in the interpretation of immunofluorescence staining patterns include scarred glomeruli or those with fibrinoid necrosis, which can reveal irregular and nondiagnostic staining for IgG, C3, or C1q. Such areas should be avoided during diagnostic decision-making and rather a significant staining pattern established in glomeruli with perfused tufts.

Electron Microscopy

Subepithelial hump-shaped deposits are considered to be the ultrastructural hallmark of infection-related GN. "Humps" have a predilection for mesangial folds/mesangial waist zones (Fig. 5, 6). They are often large with a broad base, protrude into the overlying podocytes (that are activated and enlarged), and generally lack a significant basement membrane reaction. Some subepithelial deposits have a divot rather than a broad base (Fig. 7, 8) and electron-

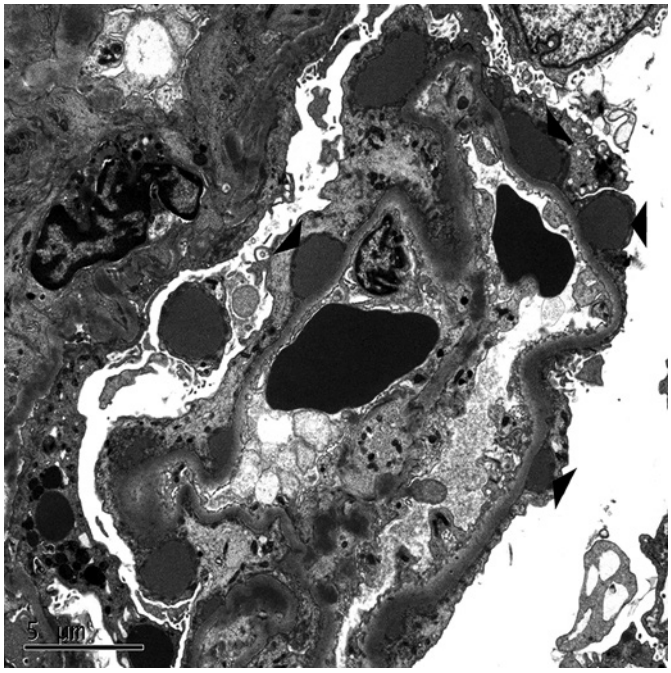

Fig. 5. Numerous large subepithelial electron-dense deposits (arrowheads) with broad bases and an absence of basement membrane reaction to the deposit are located along the glomerular basement membrane. Some appear to be floating in the urinary space but simply represent tangential sectioning of the subepithelial hump-shaped deposits. Scale bar, 5 µm.

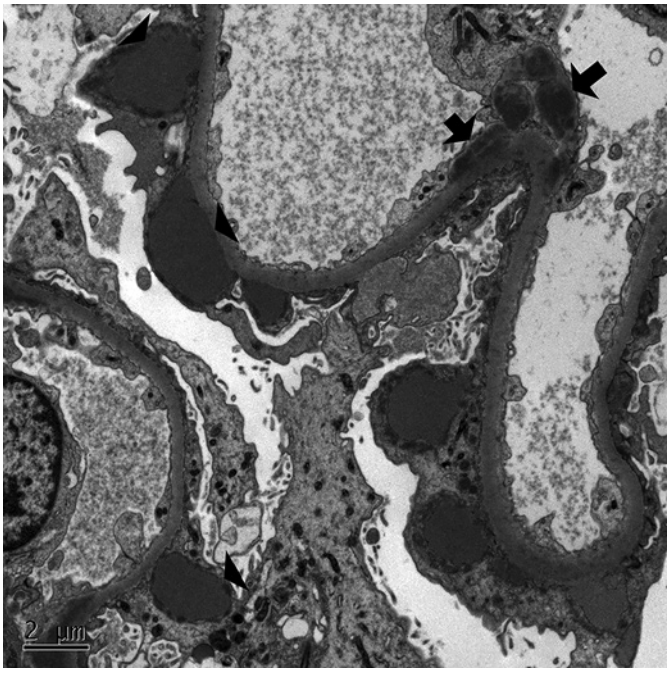

Fig. 6. Many large subepithelial electron-dense deposits (arrowheads) often known as "humps" due to their shape as they protrude into the overlying podocyte cytoplasm. Additional aggregates of mesangial electron-dense deposits (arrows) are also noted. Scale bar, 2 µm.

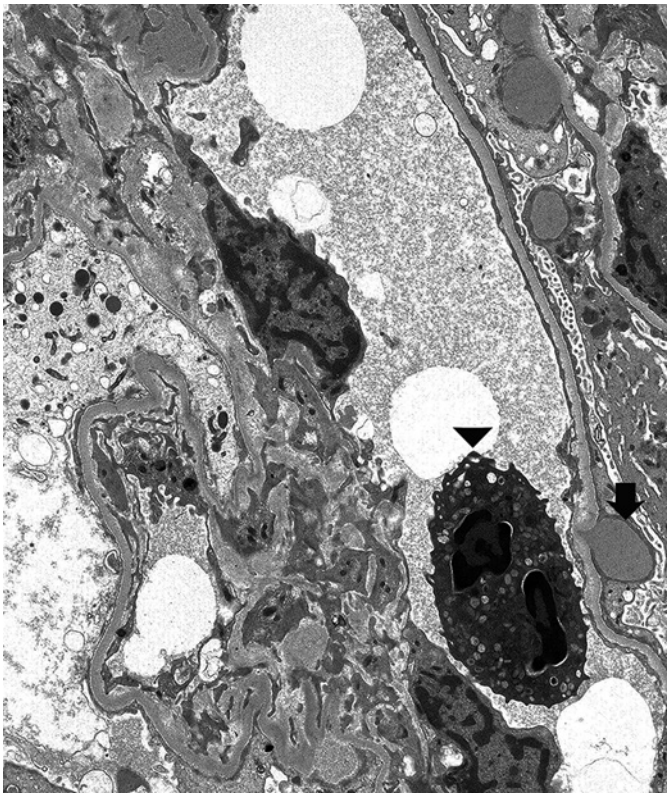

Fig. 7. This subepithelial "hump" (arrow) has a divot instead of a broad base, which can often be observed with infection-related GN. An adjacent neutrophil (arrowhead) may be responding to the subepithelial deposit, which may not be completely shielded from the circulation unlike the subepithelial deposits of membranous nephropathy that often do not elicit an inflammatory response. GN, glomerulonephritis.

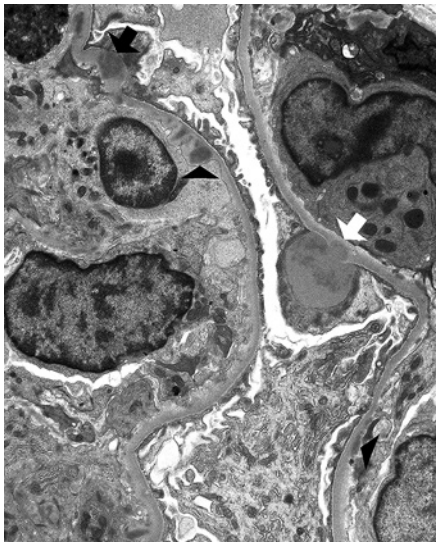

Fig. 8. Another subepithelial "hump" with variable electron density that has an unusually shaped divot at the base which appears to insert into the glomerular basement membrane (white arrow). A transmembranous electron-dense deposit (black arrow) and subendothelial (arrowhead) deposits are also present in adjacent glomerular capillaries.

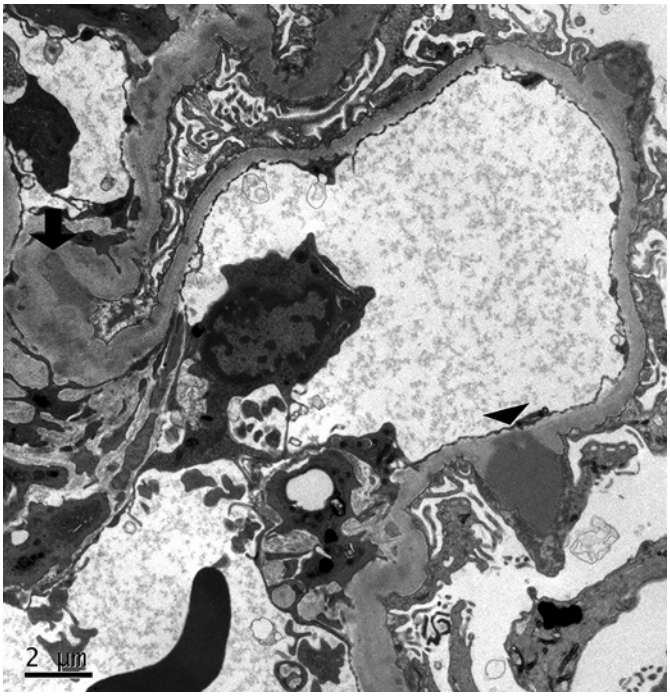

Fig. 9. An electron-dense deposit (arrow) is located in the mesangial notch while another large subepithelial "hump" (arrowhead) has a divot that traverses the entire width of the glomerular basement membrane as the base of the deposit appears to rest on the endothelial cell. Scale bar, 2 µm.

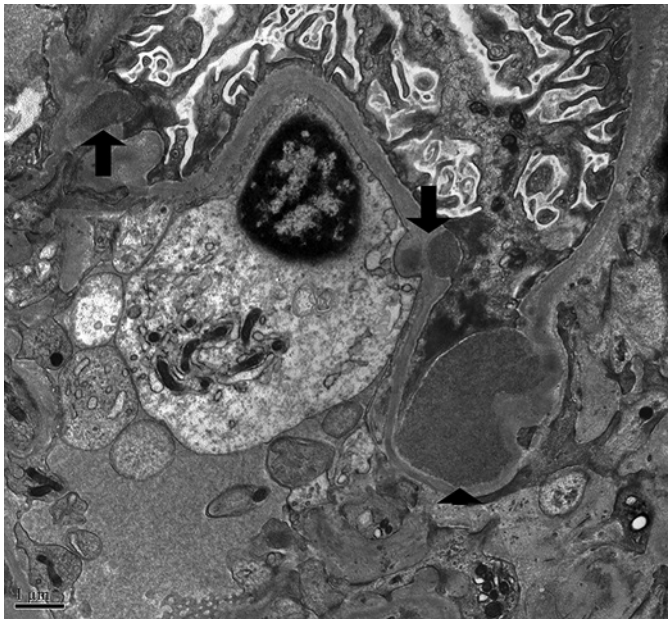

Fig. 10. A large subepithelial "hump" (arrowhead) is located in the notch region where the glomerular basement membrane inserts in the mesangium. A few transmembranous electron-dense deposits (arrows) also accompany the large subepithelial "hump." Scale bar, 1 µm.

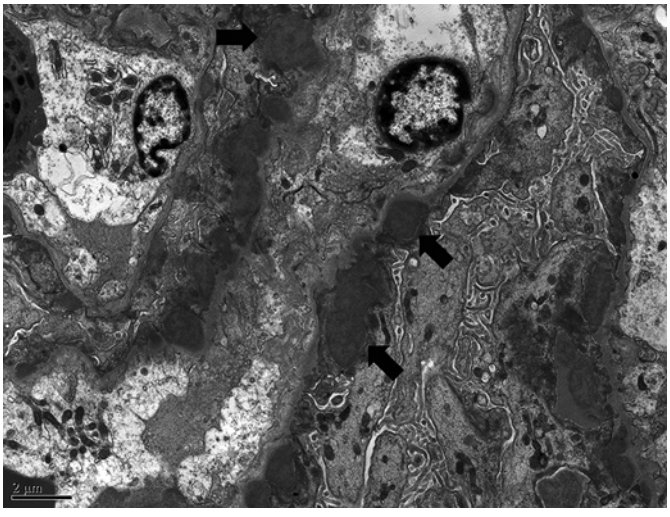

Fig. 11. Many large subepithelial electron-dense deposits (arrows) have a vague substructural appearance. Scale bar, 2 µm.

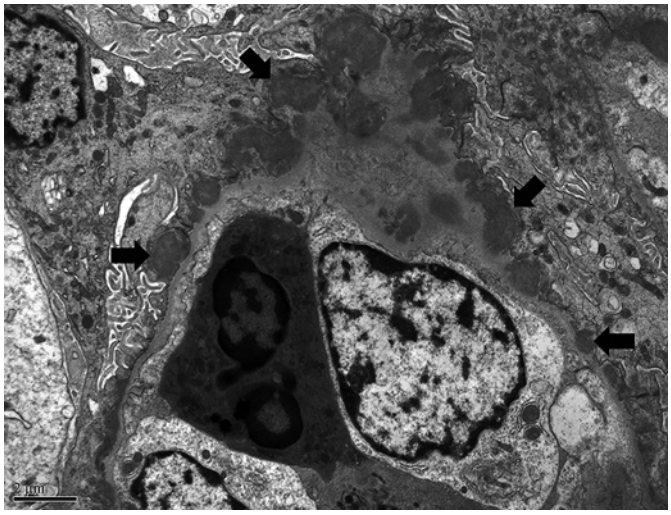

Fig. 12. Several subepithelial electron-dense deposits (arrows) reveal variable electron lucency with a vague substructural organization of the deposits. A neutrophil is present in the capillary lumen. Scale bar, 2 μm.

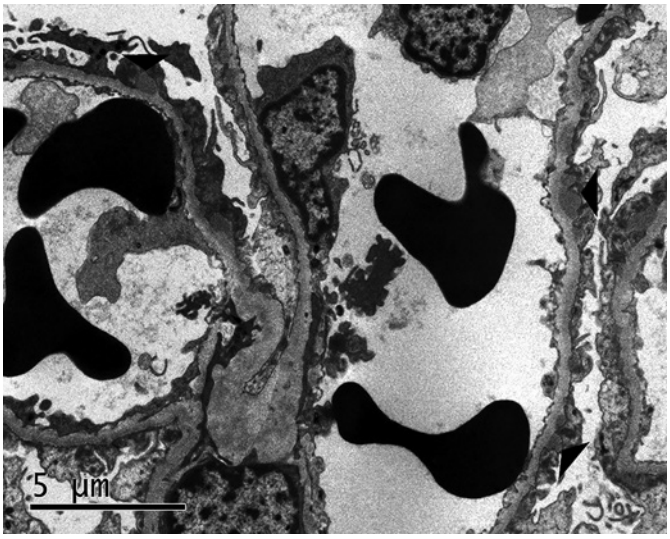

Fig. 13. Many small subepithelial electron-dense deposits (arrowheads) are scattered along the glomerular basement membrane in these glomerular capillaries and some deposits have a hint of basement membrane reaction, which can be very difficult to distinguish from the early stage of membranous nephropathy. Scale bar, 5 μm.

dense immune complex-type deposits may also be found embedded within the GBM (Fig. 9). Subepithelial "humps" tend to be present in greater numbers in the acute phase of injury and may become scarce in chronic or resolving disease stages and may only be identified in mesangial waist zones (Fig. 10, arrowhead) [25]. In some cases, the finding of these scarce subepithelial "humps" in mesangial waist zones may be the only piece of evidence indicating infection-related GN, particularly when a clear history of infection is not identified. Deposits are often very osmiophilic with a density similar to the intramembranous deposits of dense deposit disease. On occasion, a variegated appearance is noted (Fig. 11, 12) although a clearly discernible substructure is lacking. The presence of subepithelial "humps" can be more variable in other forms of infection-related GN including IgA-dominant/staphylococcal infection-related GN where they have been reported in 31–100% of cases; "humps" are less common in endocarditis-associated GN and shunt nephritis.

When the subepithelial electron-dense deposits are small and numerous (Fig. 13), the differential diagnosis may include an early stage of membranous nephropathy, which can be difficult to distinguish as patients presenting with such GBM alterations often have nephrotic-range proteinuria. Immunofluorescence studies showing dominance of IgG (in cases of membranous GN), knowledge about the PLA2R serum/staining status, the ultrastructural detection of "humps" in mesangial folds, or the presence of an underlying infection can all provide helpful clues for diagnostic decision-making: infection-related GN versus membranous nephropathy.

Small mesangial and scattered subendothelial deposits are identified in nearly all cases of infection-related GN in both the active and resolving phases (Fig. 6, 8, arrowhead). Glomerular capillaries with subendothelial deposits can contain polymorphonuclear leukocytes.

Recently, IgA-dominant/staphylococcal infection-related GN with cryoglobulin-like features has been reported [17]. In these cases, the subendothelial and intraluminal deposits ("hyaline thrombi") may be massive with a "wire loop" ultrastructural appearance (Fig. 14, 15).

Conclusion

Infection-related GN can manifest with a wide range of morphologies. The ultrastructural identification of characteristic subepithelial "humps" greatly increases the confidence in making a diagnosis. Other types of immune-complex electron-dense deposits are less diagnostic since they are commonly seen in a variety of glomerulonephritides. Infection-related GN shares some features

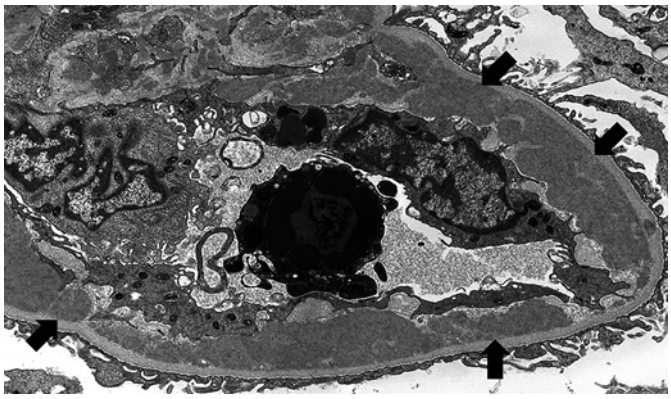

Fig. 14. Massive accumulation of subendothelial electron-dense deposits (arrows) with focal duplication of the glomerular basement membrane correlates with the presence of "wire loops" in this unusual variant of IgA-dominant infection-related GN. GN, glomerulonephritis.

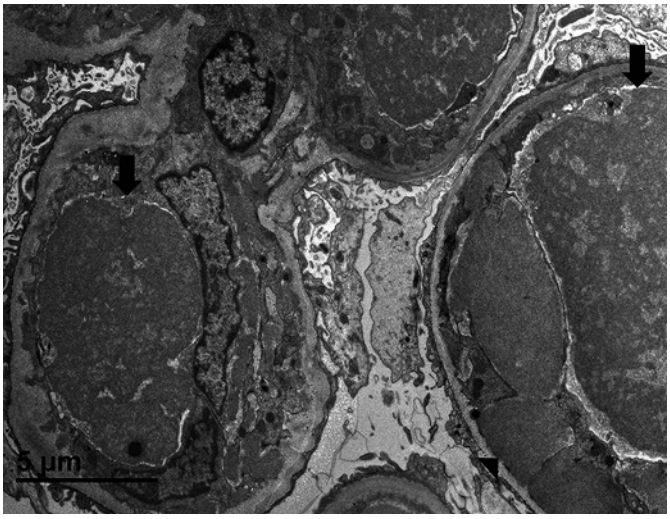

Fig. 15. Numerous electron-dense deposits within several adjacent glomerular capillaries occupy the subendothelial region and extend into the lumina, which correlate with the presence of hyaline "thrombi" or pseudothrombi (arrows) in an unusual variant of IgA-dominant infection-related GN. Scale bar, 5 µm. GN, glomerulonephritis.

and pathophysiologic pathways with C3 GN underscoring the close relationship of these disease entities. The detection of an infection-related GN is crucial to spur clinical investigations into underlying possibly occult infections including an endocarditis.

Conflict of Interest Statement

Anthony Chang has served as a consultant for Amicus Therapeutics, Alexion Pharmaceuticals, GlaxoSmithKline, and PathAI. He is a member of the speakers bureau for Alexion Pharmaceuticals. He receives royalties from Elsevier.

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Author Contributions

The authors (Mazdak Khalighi and Anthony Chang) contributed equally to the production/writing of the manuscript.

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