Diffuse Endocapillary Proliferative Glomerulonephritis

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Case scenario

These are representative images of a kidney biopsy from a 50-year-old female patient presenting with acute nephritic syndrome. A history of cough for 3 months and fever for 1 month was also elicited. The biopsy was adequate with both cortex and medulla. Up to 17 glomeruli were included. All glomeruli were abnormal, enlarged, and hypercellular. Some of the representative glomeruli with pathologic lesions are shown in the following images.

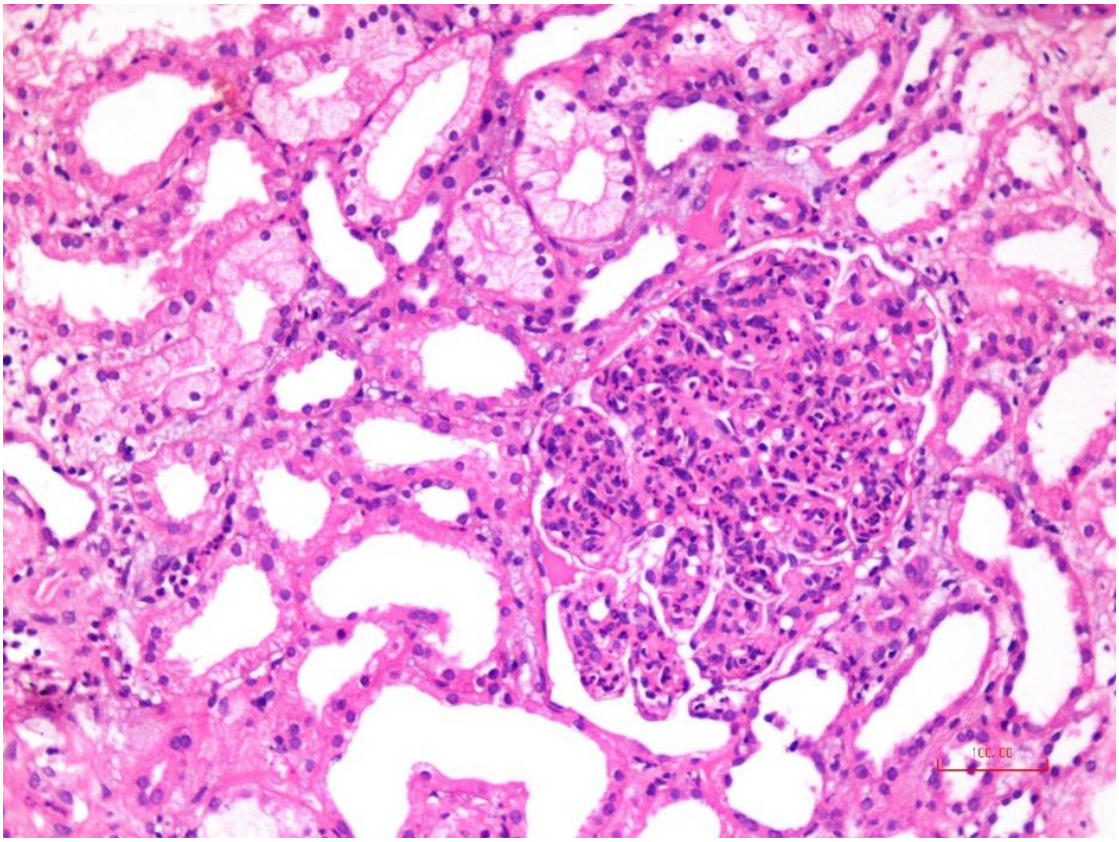

Figure 1. A medium-power view of renal biopsy from the above case shows one glomerulus exhibiting global endocapillary hypercellularity. The background parenchyma shows acute tubular injury but no inflammation or fibrosis. (H&E stain, × 200).

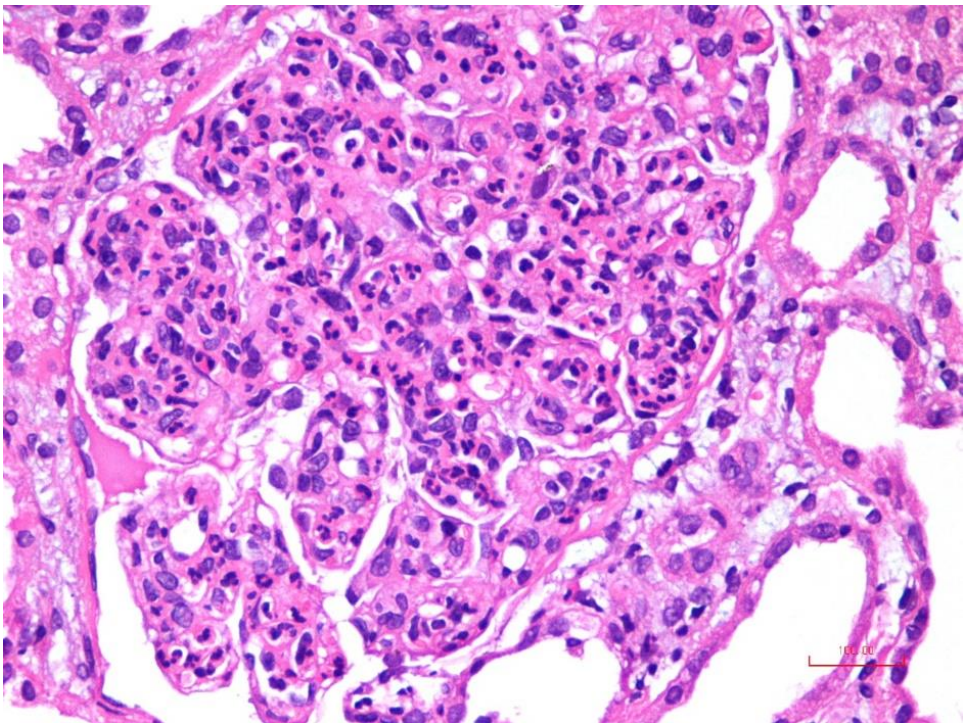

Figure 2. A high-power view of the same glomerulus as shown in Figure 1. The glomerulus is enlarged and hypercellular, resulting in marked narrowing/obliteration of the capillary lumina and the Bowman's space. The latter stems from a combination of mesangial proliferation, endothelial cell proliferation, and infiltration by neutrophils. Mild acute tubular injury is seen in the background. (H&E stain, × 400).

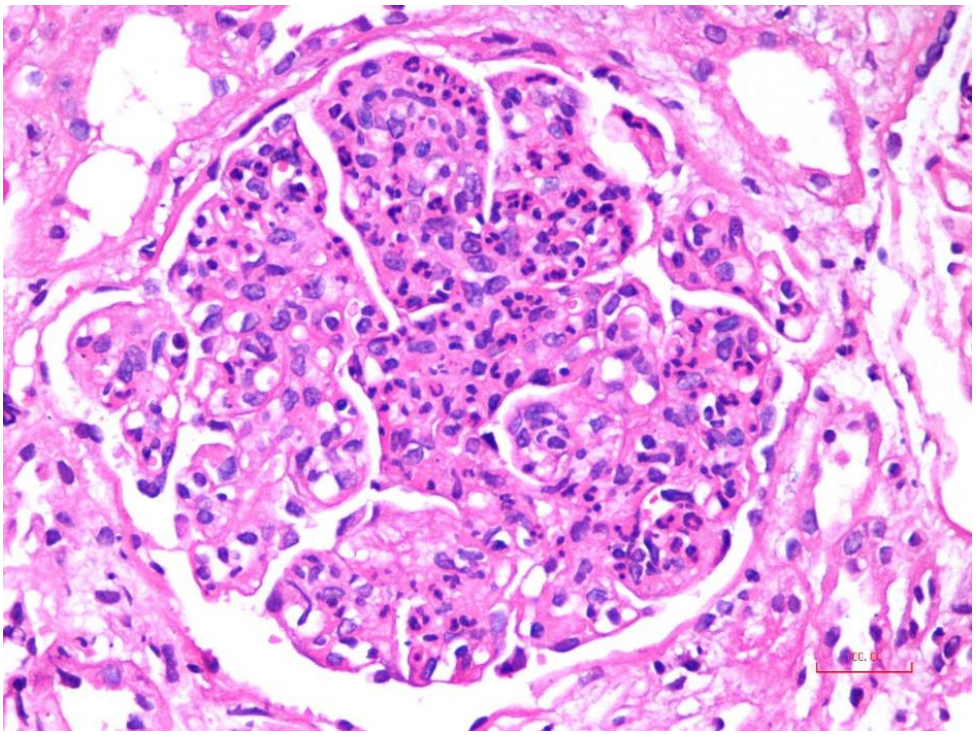

Figure 3. A high-power view of another glomerulus from the same renal biopsy from the above case shows one glomerulus with diffuse endocapillary proliferation with exudation in the right half or so of the glomerulus, whereas the left half shows less exudation. (H&E, × 400).

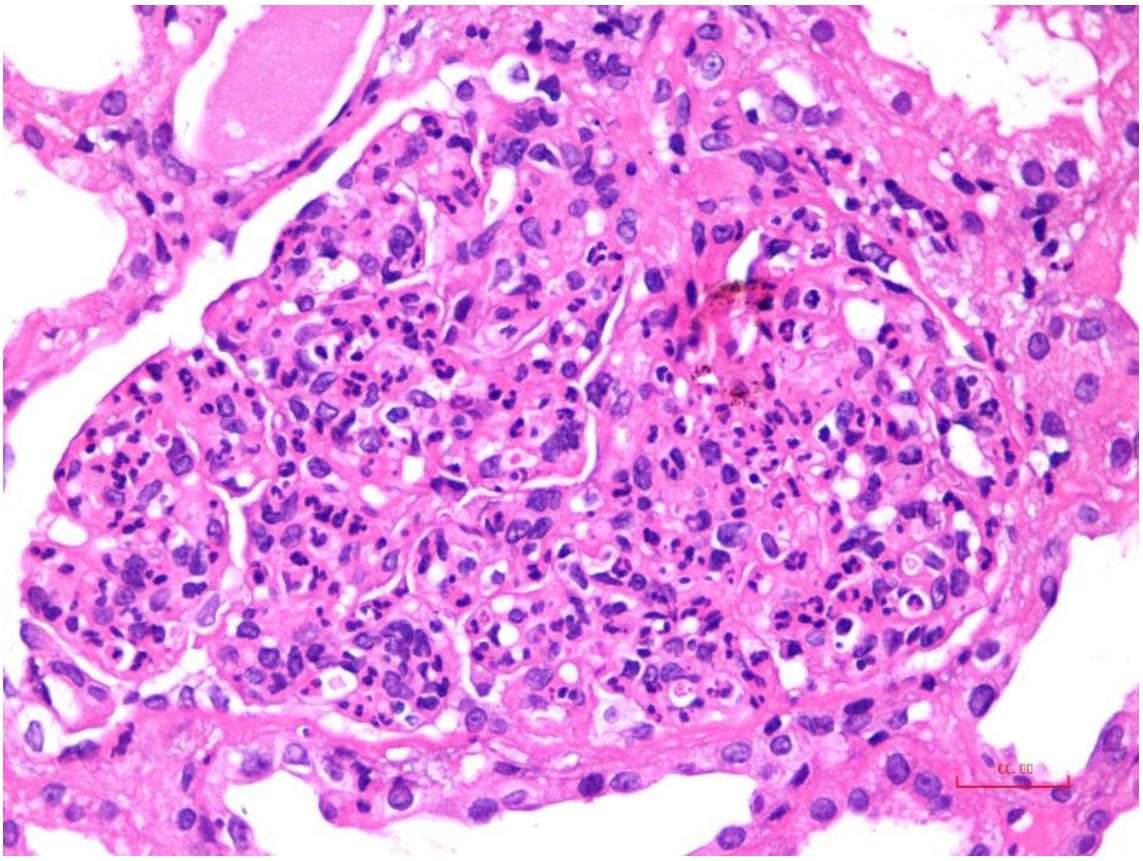

Figure 4. A high-power view of another glomerulus from the same renal biopsy from the above case showing global endocapillary hypercellularity and obliteration of almost all capillary lumina. Bowman's space is markedly narrowed as a result of endocapillary hypercellularity. (H&E, × 400).

Questions

Q1. What is the significance of this morphological pattern of glomerular injury observed in this biopsy?

Q2. What is the pathogenesis of this condition?

Q3. What is the underlying disease in this case?

Answers on Next Page

Answers:

Ans 1. This morphologic pattern of injury reflects a one-shot immune complex-mediated insult to almost all glomeruli in the kidney, hence the use of the term diffuse in its name. The condition is often self-limited and resolves on its own. As the name implies, the lesion is usually diffuse, but in some cases, it can be segmental and focal. When there are abundant neutrophils in the tuft, particularly in early stages of the disease, it is called exudative GN and is almost always associated with infections. In cases with severe injury, it is possible to find tuft necrosis with karyorrhexis, capillary thrombosis and crescent formation.

In this particular case, this response was secondary to some bacterial infection as suggested by history, marked exudation, and immunofluorescence findings.

Ans 2. This pattern of glomerulonephritis can occur in a variety of primary and secondary glomerulonephritides, including infections, lupus nephritis (LN), and IgA nephropathy (IgAN). Although the pathogenesis of this condition is variable, the fundamental mechanism is believed to be deposition of immune complexes in the mesangium and capillary walls, activation of the complement and other mediators, and recruitment of neutrophils and other inflammatory cells. Complement activation and deposition alone can also lead to this form of glomerular injury, as in C3GN.

Ans 3. This pattern of glomerular injury does not represent a specific disease. It can occur in a variety of primary and secondary glomerulonephritides, including infections, LN, and IgAN. Specific diseases can only be diagnosed by the correlation of all available clinical, serological, laboratory, immunofluorescence, and electron microscopy findings.