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Viral-Associated GN: Hepatitis C and HIV

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Abstract

Viruses are capable of inducing a wide spectrum of glomerular disorders that can be categorized on the basis of the duration of active viremia: acute, subacute, or chronic. The variable responses of the adaptive immune system to each time period of viral infection results mechanistically in different histologic forms of glomerular injury. The unique presence of a chronic viremic carrier state with either hepatitis C (HCV) or HIV has led to the opportunity to study in detail various pathogenic mechanisms of viral-induced glomerular injury, including direct viral infection of renal tissue and the development of circulating immune complexes composed of viral antigens that deposit along the glomerular basement membrane. Epidemiologic data show that approximately 25%–30% of all HIV patients are coinfecting with HCV and 5%–10% of all HCV patients are coinfecting with HIV. This situation can often lead to a challenging differential diagnosis when glomerular disease occurs in this dual-infected population and requires the clinician to be familiar with the clinical presentation, laboratory workup, and pathophysiology behind the development of renal disease for both HCV and HIV. Both of these viruses can be categorized under the new classification of infection-associated GN as opposed to being listed as causes of postinfectious GN as has previously been applied to them. Neither of these viruses lead to renal injury after a latent period of controlled and inactive viremia. The geneses of HCV- and HIV-associated glomerular diseases share a total dependence on the presence of active viral replication to sustain renal injury so the renal disease cannot be listed under "postinfectious" GN. With the new availability of direct-acting antivirals for HCV and more effective combined antiretroviral therapy for HIV, successful remission and even regression of glomerular lesions can be achieved if initiated at an early stage.

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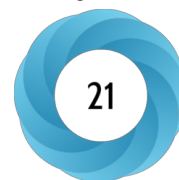
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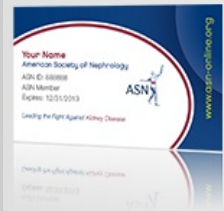
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