Relapsing HCV Associated Glomerulonephritis Despite Sustained Virologic Response (SVR) to Direct-acting Antivirals (DAA)

Joseph Scurozo DO
Lehigh Valley Health Network, joseph.scuorzo@lvhn.org

Wael Hanna MD
Lehigh Valley Health Network, wael_a.hanna@lvhn.org

Grace Chong DO
Lehigh Valley Health Network, Grace.Chong@lvhn.org

Frederick S. Fleszler MD
Lehigh Valley Health Network, Frederick_S.Fleszler@lvhn.org

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Joseph Scuorzo, DO,¹ Wael Hanna, MD,¹ Grace Chong, DO,¹ Frederick Fleszler, MD¹,²

¹Lehigh Valley Health Network, Allentown, PA, ²Valley Kidney Specialists, Allentown, PA

INTRODUCTION
Hepatitis C is a common disease with an increasing incidence in the US. It is closely associated with type II mixed cryoglobulinemia (MC) and its complication of cryoglobulinemic vasculitis (CryoVas). HCV-associated cryoglobulinemic glomerulonephritis (HCV-CryoGN) occurs in 10-35% of those with CryoVas.

CASE PRESENTATION
• A 59-year-old female presented with AKI & proteinuria. In 2011 she was diagnosed with HCV-CryoGN. Complete remission was achieved with IV Cyclophosphamide and steroids. SCr stabilized at 1.4 mg/dL and proteinuria of 0.3 g/g. In 2013 she was treated with DAAs for HCV and attained SVR.
• In September 2017, urinalysis showed proteinuria UPCR 3.5 g/g and microhematuria. Labs revealed SCr 2.5mg/dL, elevated rheumatoid factor (RF) titers 334 IU/L with positive cryoglobulin screen for type II MC.
• Serologic work up for ANA, ANCA and anti-GBM Ab were all negative with normal SPEP, C3 & C4 levels. HCV RNA was undetectable.

DISCUSSION
Reports of HCV-CryoGN despite SVR after DAA therapy are rare. DAAs lead to SVR in the overwhelming majority of HCV patients, but are less consistent in treating CryoVas. A pathological explanation is mixed cryoglobulinemia syndrome is an immune-mediated process that becomes independent from the triggering virus. Rituximab treatment has been found to deplete bone marrow B-cell clonal expansion resulting in a decrease of serum cryoglobulin and RF, correlating with improved treatment success rates with few adverse events.

CONCLUSION
HCV-CryoGN can occur in the absence of ongoing viral replication due to persistence of RF-producing memory B-cell clones. Combining the efficacy of DAAs in eradicating HCV infection along with the immunomodulatory effect of RTX can lead to improved treatment of HCV-CryoVas.

REFERENCES