Liver regeneration following successful treatment of viral hepatitis – a multifactorial pathway

Dr. Oana Săndulescu[1,2], MD, PhD

[1]Assistant Lecturer, Infectious Diseases, Carol Davila University of Medicine and Pharmacy, Bucharest, Romania

[2]Specialist physician, Infectious Diseases, National Institute for Infectious Diseases "Prof. Dr. Matei Balș", Bucharest, Romania

Before the advent of antiviral agents for the treatment of viral hepatitis, liver fibrosis had long been considered a relentlessly progressive process, inevitably leading to liver cirrhosis in time. However, the introduction in the clinic of new antivirals has significantly changed our understanding of liver fibrosis.

In infection with hepatitis C virus, achieving a cure with the new direct acting antivirals is now possible for more than 95% of patients, and following this sustained virologic response, regression of liver fibrosis has been documented. For hepatitis B virus infection, regression of fibrosis occurs following years of successful antiviral treatment, which repressed viral replication and allowed the natural anti-fibrotic mechanisms to come into play.

Liver regeneration is a multifactorial process, triggered by the cessation of antiviral injury following cure in HCV infection or suppression of viral replication in HBV infection, and followed by the accumulation of natural killer cells and dendritic cells in the liver, which in turn eliminate the hepatic stellate cells which had transdifferentiated into myofibroblasts,and degrade the fibrotic extracellular matrix by the concerted action of matrix metalloproteinases.

This presentation will review the complex interactions between hepatic stellate cells and dendritic cells in the liver, and will describe the most important mediators involved in the regression of liver fibrosis following successful treatment of viral hepatitis.