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Oral presentation

HCV-specific T-cell responses during acute Hepatitis C Virus infection in pregnancy

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Background

While T-cell immune responses are recognized as important determinants of acute hepatitis C virus (HCV) infection outcome in non-pregnant adults, little is known about HCV-specific T-cell immunity during pregnancy or its impact on vertical transmission. Here we describe the temporal course of T-cell responses and HCV replication in a patient with acute HCV infection during pregnancy.

Materials and methods

A 34 year old pregnant woman was diagnosed with acute genotype 1a HCV infection when she presented with jaundice at 24 weeks gestation. She delivered at 34 weeks gestation. T-cell immunity, plasma viremia, and liver function (ALT) were assessed serially from week 28 gestation through week 26 post-partum. HCV-specific T-cell activity in peripheral blood was quantified by ex vivo interferon-gamma ELISpot using overlapping peptides spanning the entire HCV polyprotein.

Results

During pregnancy, HCV viral load (IU/mL) rose from 1.3 million at 24 weeks gestation to 25.4 million at 34 weeks gestation (delivery). High viremia during pregnancy was associated with low frequency T-cell responses restricted to HCV NS3 and NS5b proteins. At week 8 post-partum the viral load fell sharply to 11,400, but rebounded to 16.6 million at week 26. The transient 1000-fold drop in viremia in the early post-partum period was accompanied by a transient 5-fold increase in T-cell frequency with broadening of the response to most viral proteins. T-cell activity returned to low levels by week 26 post-partum. Failing T-cell immunity accompanied by a climb in viral load in the months after delivery may predict a persistent course of infection for the mother. Preliminary data also indicate that the baby was infected in the perinatal period. Comparisons of HCV evolution and immune responses in the mother and child are ongoing.

Conclusions

In this case of acute HCV infection during pregnancy, T-cell responses increased prominently in frequency and breadth in the early post-partum period, and were associated with a sharp fall in viral load. This surge occurred several months after the onset of symptoms, suggesting that it was not the initial primary T-cell immune response to HCV, but perhaps related to a post-partum phenomenon. This finding needs to be corroborated in further studies of HCV infection during pregnancy, as it may afford new insights into T-cell control of HCV and maternal influences on vertical HCV transmission.