Intestinal macrophages in liver cirrhosis produce NO, disrupt intestinal barrier function

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A South African study presented today determines the importance of bacterial infections, which commonly occur in cirrhosis and can alter the natural history of the condition, possibly leading to loss of liver function and decompensation. It is now recognised that many infections in cirrhotic patients result from bacterial translocation (BT) from the intestine.

Results show the presence of activated CD14+Trem-1+iNOS+ intestinal macrophages, as well as increased levels of NO, IL-6 and claudin-2 levels in the duodenum of patients with decompensated liver cirrhosis .(1) The data suggests these observed factors enhance intestinal permeability of bacterial products.

Increased plasma LPS levels were observed, as well as increased numbers of activated duodenal macrophages expressing CD33+/CD14+/Trem-1+, synthesising iNOS and functionally secreting NO.

Upregulation at the mRNA level of IL-8, CCL-2, CCL-13, NOS2 was found. Increased levels of IL-8 and IL-6 levels were detected in the serum and culture supernatant of patients with cirrhosis.

The epithelial barrier was not structurally altered by electron microscopy but increased expression of the "pore"-forming tight junction claudin-2

levels were detected by western blot.

More information: du Plessis J, Activated intestinal macrophages in liver cirrhosis produce nitric oxide and disrupt intestinal barrier function. Abstract presented at the International Liver Congress 2012

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