A remission spectroscopy system for *in vivo* monitoring of hemoglobin oxygen saturation in murine hepatic sinusoids, in early systemic inflammation

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Outline Abstract

Background

During the early stages of systemic inflammation, the liver integrity is compromised by microcirculatory disturbances and subsequent hepatocellular injury. Little is known about the relationship between the hemoglobin oxygen saturation ($HbsO_2$) in sinusoids and the hepatocellular mitochondrial redox state, in early systemic inflammation. In a murine model of early systemic inflammation, we have explored the association between the sinusoidal $HbsO_2$ detected with a remission spectroscopy system and 1.) the NAD(P)H autofluorescence (an indicator of the intracellular mitochondrial redox state) and 2.) the markers of hepatocellular injury.

Results

Animals submitted to 1 hour bilateral hindlimb ischemia (I) and 3 hours of reperfusion (R) (3.0 h I/R) exhibited lower $HbsO_2$ values when compared with sham. Six hours I/R (1 hour bilateral hindlimb ischemia and 6 hours of reperfusion) and the continuous infusion of endothelin-1 (ET-1) further aggravated the hypoxia in $HbsO_2$. The detected NAD(P)H autofluorescence correlated with the detected $HbsO_2$ values and showed the same developing. Three hours I/R resulted in elevated NAD(P)H autofluorescence compared with sham animals. Animals after 6.0 h I/R and continuous infusion of ET-1 revealed higher NAD(P)H autofluorescence compared with 3.0 h I/R animals. Overall the analysed $HbsO_2$ values correlated with all markers of hepatocellular injury.

Conclusion

During the early stages of systemic inflammation, there is a significant decrease in hepatic sinusoidal $HbsO_2$. In parallel, we detected an increasing NAD(P)H autofluorescence representing an intracellular inadequate oxygen supply. Both changes are accompanied by increasing markers of liver cell injury. Therefore, remission spectroscopy in combination with NAD(P)H autofluorescence provides information on the oxygen distribution, the metabolic state and the mitochondrial redox potential, within the mouse liver.