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Cellular abnormalities in patients with alcoholic liver disease

Ashwani K Singal
University of Alabama at Birmingham, USA

Alcoholic liver disease (ALD) is second most common cause of liver cirrhosis in the US but remains under researched. Alcoholic hepatitis (AH) is a unique syndrome in ALD with a high short-term mortality and its diagnosis often requires invasive liver biopsy. Further, only about 50% of patients with severe AH respond to corticosteroids which can only be identified at 1 week of treatment. Unpredictable tresponse and fear of adverse effects of corticosteroids preclude their liberal use. Inflammation and oxidative stress with mitochondrial dysfunction are major components in pathogenesis of ALD with crucial role of neutrophils, lymphocytes, and monocytes. Abnormalities in peripheral blood cells have been described in ALD patients with T cell activation, decrease in oxidative burst, and impaired phagocytic capacity. Peripheral blood cells isolated by MACS technique are plated on the Seahorse Bioscience equipment for measuring their mitochondrial oxygen consumption rate (OCR) in pmol/mcg of cellular protein (mitochondrial bioenergetics). After measuring the basal OCR, serial injections are made with oligomycin (blocks ATP synthase), FCCP (uncoupler of proton exchange), antimycin (blocks complex III) and PMA (activates protein kinase with increase in oxidative burst). These experiments provide various components of mitochondrial respiration: ATP linked, proton leak, maximal respiration, reserve capacity, and oxidative burst. Mitochondrial OCR are reduced in 10 alcoholic cirrhosis (AC) patients compared to 10 healthy controls. Of AC patients, those with AH (N=5) showed decrease in OCR measurtements compared to 5 patients with AC decompensation without AH (N=5). Among patients with AH, corticosteroids restored the mitochondrial bioenergetics abnormalities. Assessment of mitochondrial bioenergetics of peripheral blood cells may be a novel biomarker in ALD patients for diagnosis of AH and for personalizing corticosteroid use to likely responders in patients with AH.

Biography

Ashwani K Singal has completed his MD in Gastroenetrology with MS in Clinical Research Scxience from the UTMB, Galveston. He then obtained advanced fellowship in Transplant Hepatology from the Mayo Clinic. He is currently faculty as an Assistant Professor at the UAB in AL, USA. His clinical research interests include alcoholic liver disease, NASH, porphyria, and transplantation. At the UAB, he is actively involved in clinical-translational research in alcoholic liver disease patients. He has published more than 65 papers in reputed journals and serving as an editorial board member in many of these journals.

aksingal@uab.edu