Abstract

The signs of ROSS-associated autophagy have been shown in the varicocele (VCL) condition. Considering the cross-talk between metabolic stress and ROS generation in testicular tissues, we investigated the expression levels of metabolic mediators, their transporters, and oxidative stress (OS) biomarkers to examine one of the main pathophysiologies leading to supraphysiologic ROS generation and autophagy in response to ROS. For this purpose, 18 mature Wistar rats were divided into control, control-sham, and VCL-induced (left-hand side) groups (n=6/each group). Following 4 months, the general testicular histological characteristics, intracytoplasmic carbohydrate (ICC) and lipid/fatty acids (LP/AC) storage, the expression levels of glucose transporters (GLUT-1 and GLUT-3), monocarboxylate transferase (MCT-1 and MCT-4) in the germ and Sertoli cells, testicular lactate, and lactate dehydrogenase (LDH) levels, total antioxidant capacity (TAC), malondialdehyde (MDA) content and relative $NADP^+$/NADPH ratio were investigated. The VCL-induced animals showed classical VCL-induced histological damages, including impaired spermatogenesis and spermiogenesis development and reduced sperm count. The expression levels of GLUT-1, GLUT-3, MCT-1, and MCT-4 were significantly ($p<0.05$) decreased at the mRNA level. Moreover, the mean distributions of $GLUT\text{-}1^+$, $GLUT\text{-}3^+$, $MCT\text{-}1^+$, and $MCT\text{-}4^+$ germ and somatic cells/seminiferous tubule were decreased in the VCL-induced group. The VCL-induced group showed a remarkable ($p<0.05$) reduction in the lactate, LDH, TAC, MDA, and relative $NADP^+$/NADPH ratio versus control and control-sham groups. Despite various stimulators triggering massive ROS generation in VCL conditions, impaired production and transition of the metabolomics and metabolic mediators in VCL condition significantly affects $NADP^+$/NADPH balance and partially boosts ROS generation in the testicles.

Key Words: Varicocele, Metabolomics, Oxidative stress