Cardiac mitochondria alteration and peripheral vessel morphology in female diabetes

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One of diabetes complications is chronic cardiomyopathy and fibrotic alteration of aorta and peripheral vessels. Diabetes has been correlated to ROS superproduction (Lashin et al., 2006) and an alteration of mitochondrial chain complexes function in the whole heart (Herelin et al., 2011). Despite diabetic cardiomyopathy is most frequent in women, to date studies on female experimental diabetic models are lacking. Our aim was to investigate on heart mitochondrial oxygen phosphorylation (OXPHOS), and on aorta and portal vein morphology in female Wistar rats, after 4 and half months of streptozotocin-induced (65 mg/kg) diabetes. Mitochondrial OXPHOS was assessed by means of a Clark-type electrode on the following isolated mitochondrial subpopulations: left and right ventricles subsarcomemmal (SSM) and interfibrillar (IFM) mitochondria. Morphology and extracellular matrix composition of aorta and portal vein were investigated in light microscopy on paraformaldehyde fixed samples, stained with Masson Trichrome method (for collagen fibers) and Weigert’s stain (for elastic fibers). Evaluation of OXPHOS revealed an impairment of complex II in mitochondrial diabetic female rats in left and right IFM, but not in SSM. Interestingly, administration of the substrate glutamate resulted in an improvement of complex I efficiency in left IFM only, while association of complex I and II substrates displayed a reduced efficiency both in left and right IFM. Neither administration of glucidic substrates on SSM or of lipidic substrates on both SSM and IFM resulted in any change of mitochondrial OXPHOS efficiency.

The study of connective fibrous composition in aorta and vena porta revealed a slight more abundant collagen production in the aorta’s wall and a disorganized and fragmented aspect of elastic bundles in the portal vein.

Taken together, these data suggest a peculiar unknown development of diabetic cardiopathy in female rats.

Keywords

Diabetes, heart mitochondria, OXPHOS, aorta, portal vein.