



Mitochondrial complex I defect resulting from exercise-induced lower limb ischemia in patients with peripheral arterial disease

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This study aims to compare the structural and mitochondrial alterations between muscle segments affected by exercise-induced ischemia and segments of the same muscle without ischemia, in the same subject. In a prospective analysis, 34 patients presenting either peripheral arterial disease or chronic coronary syndrome without any evidence of peripheral arterial disease were eligible for inclusion based on findings indicating a need for either a femoro-popliteal bypass or a saphenous harvesting for coronary bypass. Before surgery, we assessed the level of exercise-induced ischemia in proximal and distal sections of the thigh by the measurement of transcutaneous oxygen pressure during an exercise treadmill test. Distal and proximal biopsies of the sartorius muscle were procured during vascular surgical procedures to assess mitochondrial function and morphometric parameters of the sartorius myofibers. Comparisons were made between the distal and proximal biopsies, with respect to these parameters. Thirteen of the study patients that initially presented with peripheral arterial disease had evidence of an isolated distal thigh exercise-induced ischemia, associated with a 35% decrease in the mitochondrial complex I enzymatic activity in the distal muscle biopsy. This defect was also associated with a decreased expression of the manganese superoxide dismutase enzyme and with alterations of the shapes of the myofibers. No functional or structural alterations were observed in the patients with coronary syndrome. We validated a specific model ischemia in peripheral arterial disease characterized by muscular alterations. This "Distal-Proximal-Sartorius Model" would be promising to explore the physiopathological consequences specific to chronic ischemia. NEW & NOTEWORTHY We compared proximal versus distal biopsies of the sartorius muscle in patients with superficial femoral artery stenosis or occlusion and proof of, distal only, regional blood flow impairment with exercise oximetry. We identified a decrease in the mitochondrial complex I enzymatic activity and antioxidant system impairment at the distal level only. We validate a model to explore the physiopathological consequences of chronic muscle ischemia.

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