Gliomas. O mecanismo proliferativo requer ativação da glicólise anaeróbia

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Nos gliomas o número de mitocôndrias está diminuído o que diminui a produção de ATP via fosforilação oxidativa e facilitando a produção de ATP via Embden-Meyerhof. Este fato poderia explicar porque alguns casos de gliomas evoluem tão bem quando se provoca dieta cetogênica a qual promove a biogênese das mitocôndrias cerebrais, isto é aumenta o número de mitocôndrias no tecido cerebral. Vide biblioteca de câncer neste site. JFJ

Gliomas are driven by glycolysis: putative roles of hexokinase, oxidative phosphorylation and mitochondrial ultrastructure.


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To elucidate the reasons for glycolytic deviation commonly found in brain tumors, hexokinase (HK) activity, mitochondria-HK binding, oxidative phosphorylation and mitochondrial ultrastructure were studied in 4 human xenografted gliomas. Lactate/pyruvate ratios were increased 3-4 fold and HK activity was of 2-4 fold lower than that of normal rat brain tissue, used as the control. The mitochondria-bound HK (mHK) fraction varied considerably and represented 9 to 69% of the total HK of that normal rat brain. The respiratory activity of glioma mitochondria, assessed by polarography and spectrophotometry, was within the normal range. However, the mitochondrial content of gliomas was lower than in the rat brain tissue, as revealed by the markedly decreased, activities of two unrelated mitochondrial enzymes, cytochrome c oxidase and citrate synthase in glioma homogenates. Electron microscopical studies confirmed the reduced number of mitochondria in 3 out of the 4 gliomas. Profound alterations of mitochondrial ultrastructure, namely of cristae and matrix densities, were observed in the 4 gliomas. The intercrista space was wider in all gliomas and the crista area was larger in 3 out of the 4 gliomas than in normal rat brain. Finally, the outer membrane of glioma mitochondria interacted intimately and extensively with the rough endoplasmic reticulum (RER) and/or nuclear membrane. These results suggest that, because of the very low content of normally functioning mitochondria, gliomas shift their energy metabolism towards a high-level glycolysis to generate their cellular ATP supply, probably through RER-mitochondria interactions and transformation-dependent redistribution of particulate HK from non-mitochondrial to mitochondrial receptors.

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