Amiodarona provoca inchaço mitocondrial em células do câncer de pulmão e de próstata

Effect of mitochondrial metabolism-interfering agents on cancer cell mitochondrial function and radio/chemosensitivity.

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Abstract

Abnormal mitochondrial function is common in cancer cells and activates metabolic pathways suppressed in normal tissues. Experimental and clinical studies suggest that mitochondria might serve as targets for novel anticancer therapies. We investigated whether mitochondrial metabolism-interfering agents (MMIAs) available currently in clinical practice affect cancer cell mitochondrial metabolism and synergize with chemotherapy and radiotherapy. Two cancer cell lines A549 (lung cancer) and DU145 (prostate cancer) were treated with a variety of MMIAs (metformin, nimodipine, memantine, oxytetracycline, amiodarone, and sodium azide) and their response was assessed using a resazurin reduction method and confocal microscopy. Focusing on amiodarone and metformin, we investigated their potential sensitizing effect on cancer cells when treated with ionizing radiation, cisplatin, and docetaxel. Resazurin reduction was increased by metformin and decreased by amiodarone at nontoxic concentrations. Amiodarone induced mitochondrial swelling, whereas metformin exerted no apparent effect on their morphology. Amiodarone and metformin exerted a weak radiosensitization effect on A549, whereas a synergetic activity with cisplatin and docetaxel was evident in both cell lines. It can be concluded that amiodarone and metformin, being well-established drugs in clinical practice, constitute two potential drugs for further experimental and clinical evaluation as cancer cell sensitizers to chemotherapy and radiotherapy.

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