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Cancer patient advocacy – The role of the nurse

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The cancer patient advocacy (CPA) is frequently mentioned as a way to reduce the burden of cancer. Although the responsibility of being a patient advocate is clear in the nurse's code of ethics, there is still a need to clarify it in the practice of care, and so it can be used as a strategy for quality care in an oncologic setting. Nursing patient advocacy, related with the empowerment of the patient, can be defined as an interactive process of analyzing, counseling and responding to a patient's care and self-determination preferences, but the respective activities should be promoted in the oncologic setting. Firstly, it is important to be aware that acting as a CPA cannot be left to the individual wishes of each nurse, but based on a team decision to improve their intervention as cancer patient advocates as a whole. But CPA is complex and nurses interpret this concept in different ways. With these premises, in our organization we decided to characterize the knowledge of nursing team of 60, and then improve that knowledge through a plan of continual education. Furthermore, our plan includes setting goals that communicate what we want to achieve as a result of our advocacy efforts, observe and evaluate the existing evidence based on the subject, analyze the environment in which the care is being provided, evaluate the team capacity and establish a framework for monitoring and evaluation of our advocacy project.

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Reactive oxygen species promote doxorubicin-induced P-gp expression in colon cancer cells

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Multidrug resistance (MDR) could be one of the most important problems associated to chemotherapy. P-glycoprotein (P-gp) and BCRP (breast cancer resistant protein) are ABC transporters that could be expressed in different cancer cell lines resistant to various drugs used for the treatment of cancer. Many of the anti-tumor chemotherapeutics like doxorubicin, induces reactive oxygen species (ROS) at a high level. In this study we investigated if doxorubicin-induced ROS generation could be involved on P-gp and BCRP expression promoting MDR in colon cancer cells. We analyzed ROS accumulation, Pgp and BCRP expression by qPCR and the relationship between doxorubicin uptake and ABC transporters expression by confocal microscopy using HT-29 WT and HT-29 Doxorubicin-resistant colon cancer cells (HT-29Doxo) treated with doxorubicin. We used N-acetyl cysteine (NAC) as a control of ROS and APE-1 endonuclease inhibitor E-3330 as a modulator of oxidative stress activity. We showed that doxorubicin-induced ROS generation occurs in a time-dependent manner. Increasing ROS accumulation induced a slowly increased Pgp expression in HT29-WT treated whit doxorubicin and NAC similar to cells treated whit doxorubicin alone. In contrast BCRP expression had a significant reduction whit doxorubicin use and NAC. Furthermore APE-1 inhibitor E3330 induced Pgp and BCRP downregulation and at the same time an increased on doxorubicin accumulation higher than cells no treated. Our results suggest that MDR could be modulated by a mechanism associated to oxidative stress produce by use of doxorubicin in colon cancer cells. Additionally disrupting ROS accumulation could favor cancer therapy.

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