MODERN OPTICAL METHODS OF INVESTIGATION OF ACUTE ISCHEMIC MYOCARDIAL INJURY

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The high mortality rate of circulatory system diseases establishes their leading position among the major medical problems in the world. Ischemic heart disease and one of its forms - acute coronary insufficiency, is one of the most frequent causes of mortalities of the circulatory system diseases. The peak of mortality is observed in the working age. Currently, methods for establishing lesions of acute ischemia used in forensic practice require a substantial upgrade. This is why researching, and improving methods of diagnosis and monitoring parameters of the myocardium to develop objective criteria for the forensic determination of acute ischemic myocardial injury is relevant. Nowadays there is a trend of morphological studies that are based on fundamental research in physics, mathematics, chemistry and biology. The development of laser technology allows for the advancement of a major research direction - the interaction of coherent electromagnetic radiation with biological systems. Using lasers makes it possible to study two-dimensional distribution of optical parameters of images of biological objects to monitor their changes associated with structural alterations in various pathological processes. We suggest using laser polarimetric techniques for the study of human myocardium for the diagnosis of acute ischemic damage. During numerous studies we have found a number of features and patterns of changes in the properties of laser radiation as a result of passing through biological tissues. A set of objective forensic methods and criteria for the diagnosis of acute myocardial ischemia were described. Thus, we came make conclusions about the advisability of continuing studies of these fields to increase the number of new specific, objective indicators and criteria for their further use in forensic practice. In the future a database of images of biological tissues and fluids of the human body can be created, as a theoretical framework to address issues of forensic practice.

MODULATION OF THE ANTITUMORAL EFFECT OF DOXORUBICIN BY QUERCETIN-MENADIONE COMBINATIONS IN HUMAN LEUKIMIA JURKAT CELLS

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Introduction: The combination of the flavonoid quercetin (QC) and menadione (MD, vitamin K3) at clinically relevant levels has been reported to induce cell death at highly efficient rates in human leukemia Jurkat T-cells. We investigated the antitumoral effect of the anticancer drug doxorubicin (DOX) and its modulation by QC:MD combinations in vitro. Materials and Methods: Cellular viability, apoptosis and necrosis were assessed by flow cytometry, using propidium iodide or Annexin V-FITC/7-AAD labelling. Mitochondrial polarisation and oxidative stress were determined by flow cytometry on Jurkat cells labelled with JC-1/Annexin V-APC/7-AAD and CM-H2DCFDA/7-AAD, respectively. Results: In 48-h treatments, doxorubicin decreased cell viability dose-dependently and in a cooperative manner, with an IC50 of 93 nM and Hill coefficient of 1.8. Apoptotic and necrotic rates were closely similar. All these effects were accompanied by consistent, dose-dependent mitochondrial depolarisation and generation of oxidative stress. The combination 7.5 □M QC/7.5 □M MD exerted protective effects against DOX-induced cell death, by increasing IC50 to 255 nM, whereas the combination 15 □M QC/7.5 □M MD enhanced the antitumoral effect of doxorubicin by decreasing IC50 to 56 nM. Neither combination affected the cooperativity of the DOX antiproliferative effect. The two QC:MD combinations induced a significant, DOX-independent apoptotic rate of ~50%, as well as a low and a DOX-dependent necrotic rate, respectively. They also enhanced considerably the oxidative stress and mitochondrial depolarising effect of doxorubicin. Conclusions: Inclusion of the QC:MD combination in doxorubicin-based treatment schemes for leukemia could improve significantly the efficiency of the therapeutic drug. The primary mechanism responsible for this effect appears to be the increase in the affinity of doxorubicin towards the DNA, which could allow for a reduction of the therapeutic DOX dose.
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