

318. GENETIC MECHANISMS OF DRUG RESISTANCE IN CANCER CHEMOTHERAPY

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Introduction. One of the major problems in cancer chemotherapy is the development of drug resistance during treatment. Currently, 90% of failures in chemotherapy are during the invasion and metastasis of cancers related to drug resistance that can develop in different mechanisms.

Aim of the study. To study genetic mechanisms of drug resistance in cancer chemotherapy.

Materials and methods.: This paper is a descriptive research, based on retrospective analysis. Analysis of statistical data, current management documents, reports, studies, bibliographic and digital sources have been carried out with reference to the topic.

Results. The study of the genetic mechanisms of drug resistance in the treatment of cancer has identified the presence of different extracellular and intracellular mechanisms: tumor heterogeneity, tumor microenvironment, cancer stem cells, inhibition of cell death, inactivation of anticancer drugs, multi-drug resistance (MDR), changing drug metabolism, changing chemotherapeutic agents targets, enhancing DNA repair, gene amplification, epigenetic changes, microRNA. Responsible for multiplication, growth and metastasis have been shown to be some genes that encode for kinases. According to the latest studies, use of kinase inhibitor preparations is effective in both stopping progression of cancer and increasing the intracellular concentration of the preparation in MDR cells.

Conclusions. Cancer drug resistance is a complex phenomenon determined by numerous mechanisms and some genes. Gaining knowledge about these particularities and performing genetic tests make it possible to avoid the misuse of the preparations, in order to prevent the chemotoxicity on the organism and affect the systems with a high division rate.

Key words: Cancer, multi-drug resistance, epigenetic changes, kinases.

319. DEFECTS IN SPERMATOGENESIS OF MEN WITH Y CHROMOSOME MICRODELETIONS

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Introduction. Male infertility has multiple etiology, most commonly caused by spermatogenesis disorders, clinically manifested by oligo/azoospermia. Until recently, Y microdeletion had little clinical significance since men with a deletion were considered unable to reproduce. However, by utilizing of Intracytoplasmic sperm injection (ICSI) and Testicular sperm extraction (TESE) it is now possible for oligo/azoospermic men with Y microdeletion to father children.

Aim of the study. To analyze the type of defect in spermatogenesis associated with specific Y deletions found in our IVF program, for prevention the transmission of these deletions through ICSI to offspring.