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Resuscitation Measures for Myocardial Injury During Chemotherapy in Patients with Acute Leukemia

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Abstract: The essence of this scientific article is that general concepts about chemotherapy, about its therapeutic goals, about the use of drugs, about traditional chemotherapeutic agents, dosage of chemotherapy and many other information have been given.

Key words: Chemotherapy, medical oncology, pharmacotherapy, anticancer drugs, chemotherapeutic agents, chemotherapy regimen, mitosis, estrogens, androgens.

INTRODUCTION

Chemotherapy (often abbreviated to chemotherapy and sometimes CTX or CTx) is a type of cancer treatment that uses one or more anticancer drugs (chemotherapeutic agents or alkylating agents) as part of a standardized chemotherapy regimen. Chemotherapy can be given for a curative purpose (which almost always involves a combination of drugs) or it can be aimed at prolonging life or reducing symptoms (palliative chemotherapy).

Chemotherapy is one of the main branches of medicine that deals with pharmacotherapy for cancer, called medical oncology. Chemotherapy (often abbreviated to chemotherapy and sometimes CTX or CTx) is a cancer that uses one or more anticancer drugs (chemotherapeutic agents or alkylating agents) as part of a standardized chemotherapy regimen. a type of disease treatment. Chemotherapy can be given for a curative purpose (which almost always involves a combination of drugs) or it can be aimed at prolonging life or reducing symptoms (palliative chemotherapy).

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MAIN PART

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A woman being treated with docetaxel chemotherapy for breast cancer. Cold mittens and cold booties are placed on her hands and feet to reduce harm to her nails.

Other names chemo, CTX, CTx

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The term chemotherapy refers to the nonspecific use of intracellular poisons to inhibit mitosis (cell division) or induce DNA damage, so inhibition of DNA repair can enhance chemotherapy.[3] The term chemotherapy excludes more selective agents that block extracellular signals (signal transduction). The development of therapies with specific molecular or genetic targets that inhibit the growth-promoting signals of classical endocrine hormones (primarily estrogens for breast cancer and androgens for prostate cancer) is called hormonal therapy. In contrast, other inhibitions of growth signals associated with receptor tyrosine kinases are called targeted therapies.

Importantly, the use of drugs (chemotherapy, hormonal therapy, or targeted therapy) constitutes a systemic treatment of cancer, since they are introduced into the bloodstream and therefore, in principle, are able to treat cancer in any anatomical location of the body. Systemic therapy is often used in combination with other modalities that constitute local therapy (that is, treatments that are effective in the anatomic area where they are applied), such as radiation therapy, surgery, or hyperthermia therapy for cancer.

Conventional chemotherapeutic agents are cytotoxic by interfering with cell division (mitosis), but cancer cells vary widely in their sensitivity to these agents. In many ways, chemotherapy can be seen as a way of damaging or stressing cells, which can lead to cell death if apoptosis is triggered. Many of the side effects of chemotherapy can damage normal cells that

divide rapidly and are therefore sensitive to antimitotic drugs: cells in the bone marrow, digestive system, and hair follicles.

This leads to the most common side effects of chemotherapy: myelosuppression (decreased production of blood cells, hence immunosuppression), mucositis (inflammation of the lining of the digestive tract), and alopecia (hair loss). Because of their effects on immune cells (especially lymphocytes), chemotherapy drugs are often used in many diseases caused by the immune system's harmful overactivity against itself (so-called autoimmunity). These include rheumatoid arthritis, systemic lupus erythematosus, multiple sclerosis, vasculitis, and others.

Dosing chemotherapy can be tricky: if the dose is too low, it will be ineffective against the tumor, and if the dose is too high, the toxicity (side effects) will be unbearable for the person receiving it. The standard method for determining chemotherapy dose is based on calculated body surface area (BSA). BSA is usually calculated by a mathematical formula or nomogram using the recipient's weight and height rather than by direct measurement of body surface area. This formula was originally derived from a study conducted in 1916 and attempted to translate drug doses established with laboratory animals into equivalent doses for humans. The study included only nine people. When chemotherapy was introduced in the 1950s, the BSA formulation was adopted as the official standard for chemotherapy doses for lack of a better alternative.

The accuracy of this method for calculating single doses has been questioned, as the formula only takes into account the individual's weight and height. Absorption and clearance of drugs are affected by many factors, including age, sex, metabolism, disease state, organ function, drug interactions, genetics, and obesity, which affect the actual concentration of the drug in the individual's bloodstream. has a great impact.

As a result, there is high variability in systemic chemotherapy drug concentrations in individuals dosed by BSA, and this variability has been shown to be greater than tenfold for many drugs. In other words, if two people receive the same dose of a drug based on BSA, the concentration of that drug in the blood stream of one person may be 10 times higher or lower than that of the other. This variability is characteristic of many chemotherapy drugs dosed by BSA and, as shown below, 14 shown in studies of common chemotherapy drugs.

Alkylating agents are the oldest group of chemotherapeutics in use today. Originally derived from mustard gas used in World War I, there are many types of alkylating agents in use today.They are so named because of their ability to alkylate many molecules, including proteins, RNA, and DNA. The ability to covalently bind to DNA through the alkyl group is the main reason for their anticancer effects. DNA consists of two strands, and molecules can link twice to one strand of DNA (intrastrand crosslinking) or link once to both strands (interstrand crosslinking).

If a cell tries to replicate or repair cross-linked DNA during cell division, DNA strands can break. This leads to a programmed cell death called apoptosis. Alkylating agents act at any point in the cell cycle and are therefore known as cell cycle-independent drugs. Therefore, the effect on the cell is dose-dependent; The percentage of dead cells is directly proportional to the dose of the drug.

CONCLUSION

Subtypes of alkylating agents are nitrogen mustards, nitrosoureas, tetrazines, aziridines, cisplatins and derivatives, and non-classical alkylating agents. Nitrogen mustards include

mechlorethamine, cyclophosphamide, melphalan, chlorambucil, ifosfamide, and busulfan. Nitrosoureas include N-Nitroso-N-methylurea (MNU), carmustine (BCNU), lomustine (CCNU) and semustine (MeCCNU), fotemustine, and streptozotocin. Tetrazines include dacarbazine, mitozolomide, and temozolomide. Aziridines include thiotepa, mitomycin, and diazicone (AZQ). Cisplatin and its derivatives include cisplatin, carboplatin, and oxaliplatin. They form a covalent bond with amino acid, carboxyl, sulfhydryl and phosphate groups in biologically important molecules and disrupt the activity of cells. Non-classical alkylating agents include procarbazine and hexamethylmelamine.

Anti-microtubule agents are plant-derived chemicals that block cell division by preventing microtubule function. Microtubules are an essential cellular structure composed of two proteins, α-tubulin and β-tubulin. They are hollow, rod-shaped structures that are essential for cell division, among other cell functions. Microtubules are dynamic structures, meaning they are constantly being assembled and disassembled.

Vinca alkaloids and taxanes are two major classes of anti-microtubule agents, and although both classes of drugs cause microtubule dysfunction, their mechanisms of action are completely opposite: Vinca alkaloids prevent microtubule assembly, whereas taxanes prevent microtubule disassembly. Thus, they can induce mitotic catastrophe in cancer cells. This is followed by cell cycle arrest, which causes programmed cell death (apoptosis). These drugs can also affect blood vessel growth, an important process that tumors use to grow and metastasize.

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