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Opinion Article

Treating Tumors through Chemotherapy

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DESCRIPTION

Chemotherapy plays a major part in the treatment of patients with gynecological malignancies. In general, chemotherapy has a lower remedial window compared with medicines of other types; hence, the potential for severe adverse effects associated with chemotherapy has made appropriate patient and medicine selection critical.

There are certain issues that need to be considered before initiating treatment to any patient with chemotherapeutic agent which include:

Tumor characteristics

• The primary malignant opinion should be confirmed histologically.

• Ideally, intermittent disease should be confirmed by cytology or rather histology; it's conceded that this isn't always possible especially in ovarian cancer where the diagnosis of recurrent disease is generally grounded on clinical examination, determination of tumor labels, and imaging.

• The extent of disease.

• The liability of tumor response (e.g. type of cancer, rate of disease progression, interval since last treatment).

• Molecular biology if available.

• Tumor labels if applicable.

Patient characteristics

• The patient's age.

• The patient's general state of health (performance status).

• Comorbidities (similar as heart, liver, and order conditions).

• Response and adverse effects in the previous cancer treatments.

• The patient's psychosocial status.

Purposes of treatment

• Cure

• Tumor control to protract survival

• Justification of symptoms

Cytokinetic studies have shown how the kinetics of cellular growth defines the characteristics of tumor growth and, in part, explain the natural behavior and heterogeneity of tumors. Essential to cytokinetic principles is the conception of the cell cycle. Daughter cells formed as a result of mitosis correspond of three subpopulations cells that are non-dividing and terminally differentiated cells that are continually proliferating, and cells that are resting but may be recruited into the cell cycle (i.e; stem cells). All three populations exist simultaneously in tumors. The cell cycle is composed of four phases during which the cell prepares for and goods mitosis. The cells enter the G1 Phase again after they are committed to divide. Primary synthetic cellular processes do that prepare the cell to enter the DNA synthetic (S) phase. The cell cycle is regulated by specific protein signals which allow replication of the genome where the DNA content becomes tetraploid (4N). After completion of the S phase, the cell enters an alternate resting phase, G2, prior to undergoing mitosis. The cell progresses to the mitotic (M) phase, in which the chromosomes condense and separate and the cell which further divides, producing two daughter cells.

Chemotherapeutic agents can be classified according to the phase of the cell cycle in which they're active. Agents that are cell- cycle-phase – nonspecific (eg; alkylating agents) have a direct cure-response wind; that is, the lesser the dose of medicine, the greater is the fraction of cell kill. Still, cell- cycle- phase – specific medicines have a plateau with respect to cell killing ability, and cell kill won't increase with further increases in medicine dosage.

The rate of growth of a tumor is a reflection of the proportion of actively dividing cells (the growth fraction), the length of the cell cycle (doubling time), and the rate of cell loss. Variations in these three factors are responsible for the variable rates of tumor growth observed among tumors of differing histologies, as well as among metastatic and primary tumors of the same histology. Sigmoid-shaped Gompertzian growth curve is characteristically exhibited by tumors, in which tumor doubling time varies with tumor size. Tumors grow most fast at small tumor volumes. As tumors become larger, growth slows grounded on a complex process dependent on cell loss and tumor blood and oxygen supply. In order to have the best chance for cure, chemotherapy must achieve a fractional cell kill in a logarithmic fashion (ie, 1-log-kill is 90 of cells, 2-log-kill is 99 of cells). From these concepts, chemotherapy models have been developed exercising alternating non –cross-resistant therapies, induction-intensification approaches, and adjuvant chemotherapy

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regimens.

There are multiple reasons for chemotherapy failure in cancer patients, involving a variety of anatomic, pharmacologic, and biochemical mechanisms. Tumor sanctuary sites (brain, testes) and blood flow to the tumor represent anatomic walls; pharmacologic and biochemical explanations include altered medicine activation/inactivation in normal tissues, depleted drug accumulation, increased repair of medicine- induced damage to the cell, altered medicine targets, and altered gene expression. Overexpression of the MDR1 (multidrug resistance) gene is the most notable mediator of drug resistance and encodes a 170-kd transmembrane p-glycoprotein. p-Glycoprotein is an energy-dependent pump that serves to remove toxins or endogenous metabolites from the cell. A high level of MDR1 expression is reliably correlated with resistance to cytotoxic agents. Tumors that naturally express the MDR1 gene prior to chemotherapy characteristically display poor durable responses.

The effective use of cancer chemotherapy requires an understanding of the principles of tumor biology, cellular kinetics, pharmacology, and drug resistance. Thanks to the development of new, effective chemotherapeutic agents, coupled with our expanding knowledge about the administration and combination of these agents, we now are suitable to cure nearly 20 of all new cases of cancer through chemotherapy alone.