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A REVIEW ON: CARBOPLATIN DOSE OPTIMISATION BY USING AUC AND CALVERT FORMULA METHOD

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Abstract:
Cancer remains a significant global health challenge, driving the need for enhanced treatment options. Chemotherapy, particularly for advanced stages, is a cornerstone of cancer management. Among the chemotherapeutic agents, platinum-based compounds have demonstrated considerable efficacy. Carboplatin, a second-generation platinum analog of cisplatin, was developed to offer a more favorable safety profile and is employed in the treatment of a variety of malignancies. Its mechanism of action involves the formation of platinum-DNA cross-links, which impede DNA replication and protein synthesis, ultimately leading to cancer cell death. While generally better tolerated than cisplatin, carboplatin can still elicit adverse effects, including myelosuppression and gastrointestinal disturbances.
Renal function is a critical determinant of carboplatin dosage, as the kidneys are the primary route of drug elimination. The Area Under the Curve (AUC) method, employing the Calvert formula, is the standard for individualizing carboplatin doses, aiming to optimize therapeutic benefit while minimizing toxicity. Carboplatin is predominantly used for ovarian cancer but also shows efficacy in lung, head and neck, and bladder cancers.
This review delves into chemotherapy strategies and platinum-based anticancer agents, with a specific focus on carboplatin's characteristics, mechanism of action, dosing principles, and clinical application.

Keywords: Area under the Curve, Cancer chemotherapy, Carboplatin, Calvert formula, Glomerular filtration rate, Platinum-based anticancer agents, Pharmacokinetics, Renal function assessment.

INTRODUCTION

Cancer, a complex disease influenced by genetic and environmental factors, has emerged as the second leading cause of mortality worldwide, surpassed only by heart disease. The American International Cancer Agency reported approximately 18.1 million new cancer cases and nearly 9.6 million deaths in 2018, underscoring the urgent requirement for improved therapeutic interventions. The body's immune system is equipped to combat cancer by identifying and eliminating malignant cells. However, cancer can evade immune surveillance by manipulating the tumor microenvironment (TME), a complex ecosystem comprising various cellular components such as white blood cells, fibroblasts, cancer stem cells (CSCs), tumor-associated macrophages (TAMs), and others. These cells can release cytokines and chemokines, fostering inflammation, which can, in turn, modulate the adaptive immune response. A diverse array of cancer treatments exists, including surgery, immunotherapy, hormone therapy,

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radiotherapy, and chemotherapy. Chemotherapy is particularly valuable for managing metastatic disease.[1]

TREATMENT STRATEGIES:

Chemotherapeutic agents are utilized in various clinical contexts to manage cancer. They can be employed to prolong survival or for targeted therapeutic effects.

A) Combined chemotherapy involves the concurrent administration of multiple treatment modalities, such as radiation, surgery, or thermal therapy.

B) Induction chemotherapy typically represents the initial application of chemotherapeutic drugs in cancer treatment.

C) Consolidation chemotherapy is often administered following remission to enhance long-term survival prospects and delay disease recurrence.

D) Intensification chemotherapy is analogous to consolidation but employs different drug regimens.

E) The synergistic use of diverse chemotherapy drugs can lead to varied outcomes and distinct side effect profiles. A significant advantage of combination chemotherapy is its potential to mitigate the development of drug resistance and permit lower medication dosages, thereby reducing the incidence of adverse events.

F) Neoadjuvant chemotherapy is administered prior to surgery to reduce tumor size and is also indicated for cancers with a high propensity for metastasis.

G) Neoadjuvant chemotherapy may also be employed in cases of suspected or confirmed cancer recurrence to eradicate disseminated cancer cells.

H) Maintenance chemotherapy involves the regular administration of low-dose agents to achieve sustained disease control.

I) Salvage chemotherapy is utilized to reduce tumor burden and extend patient survival.[2]

PLATINUM BASED ANTI CANCER AGENTS:

Chemotherapy has become an indispensable component of cancer treatment paradigms. Prior to the 1960s, cancer therapies were largely based on organic compounds. The serendipitous discovery of cisplatin in the late 1960s, a simple metal compound exhibiting anticancer properties, marked a turning point. Its observed antimicrobial activity further fueled interest in its potential for cancer treatment. Platinum-based drugs, including oxaliplatin, carboplatin, and cisplatin, exert their effects through distinct mechanisms and have proven effective against various cancers. Cisplatin, the first platinum-based chemotherapy agent, was discovered in the late 1960s and approved for clinical use in 1978. Despite its efficacy in treating cancers such as breast, ovarian, and colorectal cancers, its non-specific nature leads to significant toxicity and damage to healthy tissues. Consequently, carboplatin, a second-generation platinum drug, underwent extensive development before its clinical introduction.[3]

OVERVIEW OF CARBOPLATIN:

Carboplatin is classified as an alkylating agent, a type of chemotherapy drug that damages DNA. Structurally, it differs from cisplatin by the presence of a carboxy-cyclobutane group instead of chloride atoms, rendering it more stable in vivo and potentially less toxic. Both agents function by inducing DNA damage in cancer cells, forming intrastrand and interstrand cross-links that disrupt DNA, RNA, and protein synthesis, leading to the demise of rapidly proliferating cells. Carboplatin received FDA approval in the United States in 1989 and is primarily indicated for advanced ovarian cancer, as well as other solid tumors like lung and head and neck cancers. Available in powder or liquid formulations (50, 150, and 450 mg under the brand name Paraplatin), carboplatin shares some side effects with cisplatin, including

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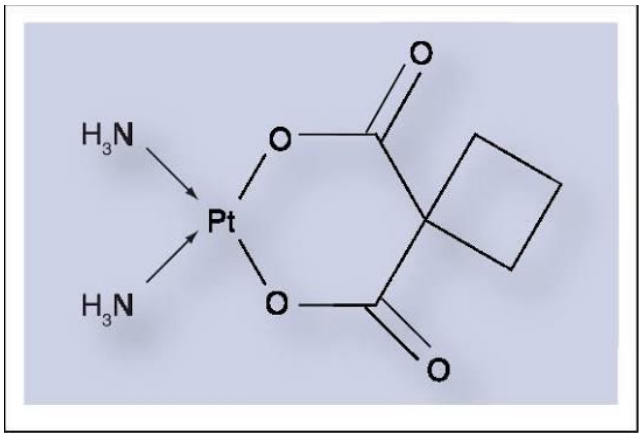
nausea, vomiting, diarrhea, and hematologic, neurologic, ototoxic, and nephrotoxic effects. However, it is generally better tolerated. Potential risks include secondary malignancies, teratogenicity, and genetic mutations.[4]

STRUCTURE OF CARBOPLATIN:[5]



MECHANISM OF ACTION:

Carboplatin, an alkylating agent, induces DNA damage. Its chemical structure, featuring two amine groups and two carboxylate groups surrounding the platinum core, contrasts with cisplatin's chloride ligands. This structural difference confers greater stability and reduced reactivity in the bloodstream, contributing to diminished systemic toxicity. Upon entering a cancer cell, carboplatin is activated in the intracellular environment, which is characterized by lower chloride concentrations and mild acidity. This activation releases a reactive platinum species that binds to purine bases (adenine and guanine) primarily at the N7 position of guanine on DNA. The resulting platinum-DNA cross-links distort the DNA helix, interfering with DNA replication and transcription. While cellular DNA repair mechanisms are initiated, the extensive cross-linking induced by carboplatin often overwhelms these repair pathways, leading to cell cycle arrest, particularly in the G2/M phase, and ultimately triggering programmed cell death (apoptosis).[6]

INDICATIONS:

Carboplatin is FDA-approved for the treatment of advanced ovarian cancer, both as a first-line therapy and for subsequent treatment of recurrent disease. It is also utilized in palliative care for recurrent ovarian cancer. Off-label indications, supported by clinical data and NCCN guidelines, encompass non-small cell lung cancer, small cell lung cancer, head and neck cancers, bladder cancer, cervical cancer, endometrial cancer, germ cell tumors, and specific pediatric malignancies such as retinoblastoma and neuroblastoma. Carboplatin is frequently administered in combination with other chemotherapeutic agents, most notably paclitaxel, for ovarian and lung cancers.[7]

PHARMACOKINETICS PROPERTIES OF CARBOPLATIN:

In circulation, carboplatin exists as total platinum, unbound platinum species, and a decarboxylated platinum degradation product. These fractions can be quantified using techniques like high-performance liquid chromatography and flameless atomic absorption spectrophotometry. The unbound platinum species exhibit similar pharmacokinetic profiles for the initial 12 hours post-administration. Carboplatin generally displays minimal drug interactions when co-administered with other chemotherapeutic agents

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and exhibits linear pharmacokinetics at standard clinical doses. Its disposition is typically described by a two-compartment model, with elimination from the central compartment. The volume of distribution in the central compartment correlates with extracellular fluid volume, and its clearance is directly proportional to the glomerular filtration rate (GFR). In individuals with normal renal function, the elimination half-life ranges from 2 to 6 hours, whereas in patients with impaired renal function, it can extend up to 18 hours.[9]

AREA UNDER THE CURVE BASED CARBOPLATIN DOSING:

In pharmacology, the Area Under the Curve (AUC) quantifies the total systemic exposure to a drug over time. It is derived by integrating the drug's plasma concentration-time profile and serves as a measure of drug exposure and clearance. AUC is instrumental in determining optimal drug dosages. A prospective study utilizing the Calvert formula established target AUC values between 3 and 8 mg·min/mL. For previously treated patients, an AUC of 4–6 mg·min/mL was found to be manageable, while 6–8 mg·min/mL was tolerable for previously untreated patients. Consequently, AUC values of approximately 5 mg·min/mL are commonly employed in monotherapy, 6 mg·min/mL in combination regimens, and higher values (7–8 mg·min/mL) in more aggressive treatment strategies.

Calvert formula:
Total dose (mg) = Target AUC × (GFR + 25)[10],[11]

RENAL FUNCTION ASSESSMENT IN CARBOPLATIN DOSING:

Carboplatin, a platinum-containing chemotherapy agent, is employed for treating various solid tumors and hematologic malignancies. Renal excretion is the predominant elimination pathway. Earlier dosing strategies based solely on body surface area led to considerable interpatient variability in drug exposure and toxicity. Given the pivotal role of the kidneys in drug clearance, a more precise method for assessing drug exposure was necessitated. This approach, introduced in 1989, integrates pharmacokinetic and pharmacodynamic principles to predict the AUC, thereby enabling individualized dosing and improving the predictability of side effects, such as thrombocytopenia.

Glomerular filtration rate (GFR) is a key metric for evaluating kidney function. The Calvert equation was developed in the 1980s to estimate AUC using 51Cr-EDTA. However, due to the limited availability of radioactive tracers in many clinical settings, the Cockcroft-Gault equation, which estimates creatinine clearance based on age, weight, sex, and serum creatinine levels, is widely used. This equation has become a standard in clinical practice.

Creatinine clearance (CrCl) is another essential measure for assessing renal function and guiding drug dosage, particularly for renally excreted medications. While a 24-hour urine collection provides the most accurate CrCl measurement, its practical limitations often lead to the reliance on the Cockcroft-Gault equation in clinical practice. Accurate renal function assessment is paramount for drug dosing, as the kidneys eliminate over half of all medications. Therefore, understanding the accuracy and reliability of estimation methods like the Cockcroft-Gault equation is crucial for optimizing patient outcomes.[12],[13]

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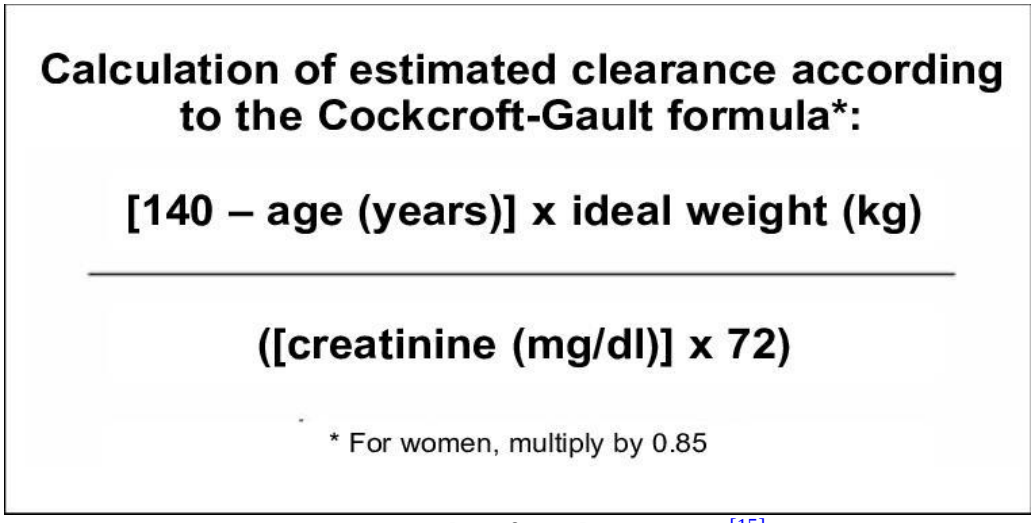

Figure 1: Cockcroft gault Equation.[15]

ALTERNATIVE EQUATIONS TO ESTIMATE GFR:

Various formulas are employed in clinical practice and research to estimate GFR, typically utilizing readily available patient data such as serum creatinine levels, body weight, sex, race, and age (as presented in Table 1).

Table 1Various equations developed to estimate GFR based on serum creatinine

Name	Formula	Validation Study	Strengths	Limitations
Cockcroft and Gault	Ccr = (140 – age) (wt [kg]) /72 × S_{cr} (mg/dL)	Published in 1976	Based on readily available clinical data	Based on 24-hour urine creatinine clearance rather than a measured GFR
	(15% less in females)	249 hospitalized male, white patients aged 18–92 (17 patients aged >80)	Ease of use	Based on older non-IDMS methods of serum creatinine measurement
			Described in a range of clinical scenarios	Developed in hospitalized patients who may have more complicated metabolism
				Statistical methods used in its derivation weak
Jeliffe	Ccr = (98 j (0.8 × (age [yrs] – 20)))/serum creatinine (mg/dL)	Published in 1973	Based on readily available clinical data	Based on 24-hour urine creatinine clearance rather than a measured GFR
	(10% reduction for	128 serial	Ease of use	Based on non-IDMS

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	females)	observations in 15 patients after renal transplantation		creatinine
		Assumes a stable creatinine in an adult, nonobese, nonemaciated patient with normal muscle mass who is not on hemodialysis	Used in many ovarian cancer trials	Not applicable at extremes of variables Underestimates GFR when low, and overestimates in renal impairment with an error rate of 20%
Modification of diet in renal disease (MDRD)	186 × Serum creatinine$^{-1.154}$ × age$^{-0.203}$	A series of studies looking at the diagnosis and complications of renal disease	Based on readily available clinical data	Expresses the GFR against 1.73 m^2 which maybe no longer be an "average" person
			Ease of use	Not validated specifically for drug dosing or in oncology patients
	Multiply by a factor of 1.21 if black and a further factor of 0.742 if female	Derived from the GFR of 1070 patients on clinical trials	More accurate than Cockcroft-Gault and Jeliffe	80% of its variability is derived from the serum creatinine
		Validated with 558 patients	Based on measured GFR as the standard comparator	Expresses the GFR against a standard BSA of 1.73m^2
		Compared the calculated GFR against ^{125}I-iothalamate	Further validated in chronic renal disease populations Refined formula based on IDMS creatinine	
Chronic Kidney Disease Epidemiology Collaboration	GFR = 141 × min (serum creatinine/*k* or 1)^{a} × max (Serum creatinine/*k* or 1)$^{-}$	Based on data from 10 trials	Based on readily available clinical data	Expresses the GFR against a standard BSA of 1.73m^2, as weight is not used in

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(CKD-EPI)	$^{1.209} \times (0.993)^{age}$			the calculation
	Max is the higher of (serum creatinine/*k*) or 1	5504 of the 8254 patients' data used for development	Ease of use	Validated for IDMS serum creatinine only
	Min is the lower of (serum creatinine/*k*) or 1.	The remaining for validation	Based on measured GFR as the standard comparator	Small number of patients older than 70 years
	k = 0.7 for females and 0.9 for males, *a* = –0.329 for females and –0.411 for males	Externally validated using 3896 patients from 16 further studies	Most accurate formula developed to date for estimation of renal function	Only minor variations in ethnicity
	Multiply by a factor of 1.159 if black	Greater accuracy than the MDRD Study equation, particularly at higher GFR	Well validated in renal patients	Not validated for drug dosing or in other patient groups outside renal failure
		It reported 84.1% of calculated GFR within 30% of measured values versus 80.6% with MDRD		
		^{125}I-iothalamatewas used as the reference		

BSA, body surface area.[14]

EFFECTS OF UNDERDOSING AND OVERDOSING OF CARBOPLATIN IN AUC GUIDED THERAPY AND THEIR CORRELATION WITH THE CALVERT FORMULA:

Suboptimal carboplatin dosing results in insufficient drug exposure and reduced therapeutic efficacy. Carboplatin's mechanism involves forming platinum-DNA cross-links that inhibit cancer cell proliferation. Inadequate drug levels lead to fewer cross-links, thereby diminishing the drug's ability to destroy cancer cells, potentially leading to disease progression and treatment failure. A strong correlation exists between achieved AUC and treatment response, highlighting the detrimental impact of underdosing.

Factors contributing to a low AUC include impaired renal function, inaccuracies in serum creatinine measurement, or dose reductions made in anticipation of heightened toxicity. If the Calvert formula

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underestimates renal function, the calculated carboplatin dose will be insufficient, leading to lower drug levels. Furthermore, rapid drug clearance by hyperfunctioning kidneys can further reduce AUC. Inadequate dosing compromises treatment effectiveness and can accelerate disease advancement.
Conversely, supra-therapeutic doses of carboplatin result in an elevated AUC and an increased risk of severe adverse events. The severity of toxicity is dose-dependent. The most significant side effect of carboplatin is myelosuppression, characterized by a profound decrease in platelet count, owing to the drug's impact on rapidly dividing bone marrow cells. Higher AUC values are also associated with an increased risk of severe neutropenia, anemia, and other hematologic abnormalities.[9][14]

Clinical studies have demonstrated a direct relationship between higher carboplatin AUC and an increased incidence of severe hematologic toxicity. Patients receiving higher AUC doses are at greater risk of profound thrombocytopenia and neutropenia, escalating the susceptibility to infection and bleeding, and potentially necessitating delays in subsequent chemotherapy cycles. In some instances, dose adjustments or treatment cessation may be required to manage these toxicities. Other potential adverse effects of excessive carboplatin exposure include nephrotoxicity, neurotoxicity, and ototoxicity, although hematologic effects are the most prevalent serious concerns.[8]

Therefore, precise estimation of renal function using the Calvert formula is essential for accurate carboplatin dosing. Overestimation of GFR leads to an excessively high calculated dose, resulting in an elevated AUC and increased toxicity risk. Conversely, underestimation of GFR results in a sub-therapeutic dose and diminished treatment efficacy. To enhance dosing accuracy, clinicians often employ validated methods for estimating renal function, such as the Cockcroft-Gault equation.[15]

In clinical oncology, carboplatin is typically administered within AUC ranges of 4 to 6 mg·min/mL, tailored to the specific cancer type and treatment regimen. Maintaining this range is crucial for balancing therapeutic efficacy with patient safety. Deviations below the target AUC may compromise treatment effectiveness, while exceeding the target increases the risk of severe adverse events. The application of the Calvert formula for AUC-guided dosing remains a cornerstone strategy for maximizing the benefits of carboplatin therapy and improving patient outcomes.[11]

ADVERSE REACTIONS:

- Dermatologic: Alopecia, rash.
- Endocrine and metabolic: Hypocalcemia, hypokalemia, hypomagnesemia, hyponatremia.
- Gastrointestinal: Severe nausea and vomiting, abdominal pain.
- Hematologic: Anemia, dose-related bone marrow suppression (including leukopenia, neutropenia, and thrombocytopenia). Bone marrow suppression is typically more pronounced in patients with impaired renal function.
- Hepatic: Elevated alkaline phosphatase and aspartate aminotransferase levels.
- Nephrotoxicity: Reduced estimated GFR and renal plasma flow, elevated blood urea nitrogen.
- Neurotoxicity: Peripheral neuropathy.
- Ophthalmic: Complete loss of vision, including color and light perception, has been reported with high-dose carboplatin. Vision typically recovers within weeks of discontinuing high-dose treatment.
- Constitutional: Fatigue, pain.
- Infusion-related: Infusion reaction or anaphylactic reaction.
- Local: Carboplatin is an irritant and can cause tissue damage upon extravasation.[16]

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CONCLUSION:

Carboplatin is a pivotal platinum-based chemotherapy agent widely used in the management of various solid tumors. Compared to cisplatin, carboplatin offers a more favorable toxicity profile while retaining significant anticancer activity through DNA cross-linking. Its pharmacokinetic behavior is intimately linked to renal function, making accurate GFR assessment critical for safe and effective dosing. The adoption of AUC-guided dosing via the Calvert formula has simplified dose individualization and mitigated the risk of severe adverse events. While carboplatin can induce myelosuppression and gastrointestinal disturbances, these effects can often be managed through vigilant monitoring and appropriate dose adjustments. Carboplatin remains a cornerstone of modern chemotherapy regimens, particularly in combination therapies. However, continuous patient monitoring is indispensable for ensuring safety and optimizing treatment outcomes.

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