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Carboplatin

Updated: January 20, 2014.

OVERVIEW

Introduction

Carboplatin is an intravenously administered platinum coordination complex and alkylating agent which is used as a chemotherapeutic agent for the treatment of various cancers, mainly ovarian, head and neck and lung cancers. Carboplatin therapy is associated with a low rate of transient serum aminotransferase elevations and with rare instances of clinically apparent liver injury.

Background

Carboplatin (kar" boe pla' tin) is a cisplatin analog with a carboxy-cyclobutane moiety instead of the chloride atoms which makes it more stable and perhaps less toxic than cisplatin. Carboplatin and cisplatin act as alkylating agents causing cross linking between and within DNA strands, leading to inhibition of DNA, RNA and protein synthesis and the triggering of programmed cell death, mostly in rapidly dividing cells. Carboplatin was approved for use in cancer chemotherapy in the United States in 1989. It is currently indicated for advanced ovarian carcinoma, but is also used in other solid tumors including lung and head and neck cancer. Carboplatin is available in a powder or aqueous solution for injection in 50, 150 and 450 mg amounts generically and under the brand name Paraplatin. The platinum coordinating complexes have similar toxicities, including nausea and vomiting, diarrhea, bone marrow suppression, as well as neuro-, oto- and nephrotoxicity. They are also mutagenic, teratogenic and carcinogenic. Carboplatin is somewhat better tolerated than cisplatin.

Hepatotoxicity

Mild and transient elevations in serum aminotransferase levels are found in up to one-third of patients taking carboplatin. However, clinically apparent acute liver injury from carboplatin is extremely rare and the characteristics of such injury have not been well defined. In addition, carboplatin has been used in combination with other alkylating agents in high doses in conditioning regimens in preparation of hematopoietic cell transplantation which may be associated with instances of sinusoidal obstruction syndrome, which can be severe and lead to acute liver failure. Onset of sinusoidal obstruction syndrome is generally within 10 to 20 days of transplantation and presents with right upper quadrant pain, hepatic tenderness, weight gain, edema and ascites, followed by jaundice. The role of carboplatin in causing these cases of sinusoidal obstruction syndrome has not been well defined.

Likelihood score: D (possible cause of clinically apparent liver injury).

Mechanism of Injury

The cause of idiosyncratic hepatotoxicity from carboplatin is not known, but is possibly due to an intermediate in its metabolism. Sinusoidal obstruction syndrome is probably due to direct toxic effects of the myeloablative regimen on sinusoidal lining cells.

Outcome and Management

The severity of liver injury from carboplatin ranges from mild, reversible enzyme elevation to sinusoidal obstruction syndrome with acute liver failure and death. Liver injury from carboplatin is extremely rare. There is likely to be cross sensitivity to liver toxicities of the various platinum coordination complexes and rechallenge after clinically apparent liver injury should be avoided.

References to the hepatotoxicity of carboplatin, cisplatin and oxaliplatin are given after the Overview section on Platinum Coordination Complexes.

Drug Class: Antineoplastic Agents, Alkylating Agents, Platinum Coordination Complexes: Cisplatin, Oxaliplatin

PRODUCT INFORMATION

REPRESENTATIVE TRADE NAMES

Carboplatin – Generic, Paraplatin®

DRUG CLASS

Antineoplastic Agents, Alkylating Agents

COMPLETE LABELING

Product labeling at DailyMed, National Library of Medicine, NIH

CHEMICAL FORMULA AND STRUCTURE

DRUG	CAS REGISTRY NUMBER	MOLECULAR FORMULA	STRUCTURE
Carboplatin	41575-94-4	C6-H12-N2-O4-Pt