U.S. National Library of Medicine
National Center for Biotechnology Information

NLM Citation: LiverTox: Clinical and Research Information on Drug-Induced Liver Injury [Internet]. Bethesda (MD): National Institute of Diabetes and Digestive and Kidney Diseases; 2012-. Betaxolol. [Updated 2017 Jan 15].
Bookshelf URL: https://www.ncbi.nlm.nih.gov/books/

Betaxolol

Updated: January 15, 2017.

OVERVIEW

Introduction

Betaxolol is a cardioselective beta-blocker used in the treatment of hypertension. Betaxolol has not been linked to instances of clinically apparent drug induced liver injury.

Background

Betaxolol (be tax' oh lol) is considered a "selective" beta-adrenergic receptor blocker in that it has potent activity against beta-1 adrenergic receptors which are found in cardiac muscle, but has little or no activity against beta-2 adrenergic receptors found on bronchial and vascular smooth muscle. Betaxolol was approved for use in the United States in 1985 and is currently used mostly in the therapy of hypertension in combination with other agents. Betaxolol is available in 10 and 20 mg tablets in generic forms as well as under the trade name of Kerlone. Liquid ophthalmic solutions of betaxolol and its stereoisomer levobetaxolol are available for use in ocular hypertension and glaucoma. The usual initial oral dose of betaxolol in adults is 10 mg once daily, with subsequent adjustment based upon clinical response and tolerance; the usual maintenance dosage being 10 to 40 mg daily. Common side effects include bradycardia, hypotension, fatigue, dizziness, depression, insomnia, memory loss and impotence. At high doses, betaxolol is less cardioselective and can induce bronchospasm. As with all beta-blockers, sudden withdrawal can trigger rebound hypertension.

Hepatotoxicity

Betaxolol therapy has been associated with a low rate of mild-to-moderate elevations of serum aminotransferase levels which are usually asymptomatic and transient and resolve even with continuation of therapy. There have been no well documented cases of clinically apparent, acute liver injury attributable to betaxolol. Thus, hepatotoxicity due to betaxolol must be very rare, if it occurs at all. Most commonly used beta-blockers have been linked to rare instances of clinically apparent liver injury, typically with onset within 2 to 12 weeks, a hepatocellular pattern of liver enzyme elevations, rapid recovery on withdrawal, and little evidence of hypersensitivity (rash, fever, eosinophilia) or autoantibody formation.

Likelihood score: E (unlikely cause of clinically apparent liver injury).

Mechanism of Injury

The mechanism of drug induced liver injury from beta-blockers such as betaxolol is not known. Betaxolol is extensively metabolized by the liver and excreted as inactive metabolites. The few cases of acute liver injury attributed to beta-blockers were likely idiosyncratic.

Outcome and Management

The severity of liver injury due to beta-blockers ranges from mild serum aminotransferase elevations to acute hepatitis with jaundice. In large case series of drug induced liver injury and acute liver failure due to medications, betaxolol has not been listed as a potential cause. There is little information about cross reactivity among the beta-blockers to hepatic injury. Switching from a beta-blocker that has caused acute liver injury to another should be done with caution and active monitoring.

References to the safety and potential hepatotoxicity of betaxolol are provided in the overview on Beta-Adrenergic Receptor Antagonists, last updated in June 2019.

Drug Class: Beta-Adrenergic Receptor Antagonists

PRODUCT INFORMATION

REPRESENTATIVE TRADE NAMES

Betaxolol – Generic, Kerlone®

DRUG CLASS

Beta-Adrenergic Receptor Antagonists

COMPLETE LABELING

Product labeling at DailyMed, National Library of Medicine, NIH

CHEMICAL FORMULA AND STRUCTURE

DRUG	CAS REGISTRY NUMBER	MOLECULAR FORMULA	STRUCTURE
Betaxolol	63659-18-7	C18-H29-N-O3