



Moexipril

Updated: February 11, 2018.

OVERVIEW

Introduction

Moexipril is an angiotensin-converting enzyme (ACE) inhibitor which is used in the therapy of hypertension. Moexipril is associated with a low rate of transient serum aminotransferase elevations, but has yet to be linked to instances of acute liver injury.

Background

Moexipril (moe ex' i pril) is an ACE inhibitor which is approved for use alone and in combination with other agents in the therapy of hypertension. Like other ACE inhibitors, moexipril inhibits the conversion of angiotensin I, a relatively inactive molecule, to angiotensin II which is the major mediator of vasoconstriction and volume expansion induced by the renin-angiotensin system. Other host enzymes besides that which converts angiotensin I to II may be inhibited as well, which may account for some of the side effects of the ACE inhibitors. Moexipril was approved for use in the United States in 1991 and is available in 7.5 and 15 mg tablets in several generic forms and under the trade name Univasc. The typical daily dose in adults is 7.5 to 30 mg in one or two divided doses which is administered long term. Moexipril is also available in fixed dose combinations with hydrochlorothiazide (Uniretic and generics). Common side effects include dizziness, fatigue, headache, cough, gastrointestinal upset and skin rash.

Hepatotoxicity

Moexipril, like other ACE inhibitors, is associated with a low rate of serum aminotransferase elevations (<2%) that in controlled trials was no higher than with placebo therapy. These elevations are transient and rarely require dose modification. Clinically apparent cases of acute liver injury due to moexipril have yet to be published. Most ACE inhibitors have been associated with rare instances of clinically apparent liver injury, which typically arises 2 to 12 weeks after starting therapy and is marked by a cholestatic pattern of injury which can be severe and prolonged. Immunoallergic manifestations (rash, fever, eosinophilia) are infrequent and most patients do not develop autoantibodies. Rare instances of severe acute hepatocellular injury, sometimes arising 1 to 4 years after starting ACE inhibitors, have been described, but not in association with moexipril. Nevertheless, the product label mentions the possibility of drug induced liver injury.

Likelihood score: E* (unlikely but suspected rare cause of clinically apparent liver injury).

Mechanism of Injury

The cause of the minor serum aminotransferase elevations associated ACE inhibitors including moexipril is not known. Moexipril is hydrolyzed in the liver to the active metabolite moexiprilat, and idiosyncratic liver injury might be induced by a minor metabolite.

Outcome and Management

There have been too few instances of moexipril associated liver injury described to provide an overall description of its course and outcome. Most instances of acute liver injury reported with ACE inhibitors have been self limited, but there have been rare reports of acute liver failure due to captopril, enalapril, lisinopril and benazepril and several reports of cholestatic hepatitis due to ACE inhibitors leading to prolonged jaundice and vanishing bile duct syndrome. Although cross sensitivity to liver injury has not always been shown, patients with severe acute liver injury due to an ACE inhibitor should avoid use of other drugs belonging to this class of agents.

References to the safety and potential hepatotoxicity of moexipril are given in the Overview section on the Angiotensin-Converting Enzyme (ACE) Inhibitors.

Drug Class: [Antihypertensive Agents, Angiotensin-Converting Enzyme Inhibitors](#)

PRODUCT INFORMATION

REPRESENTATIVE TRADE NAMES

Moexipril – Generic, Univasc®

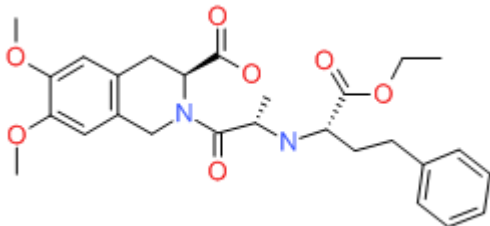
DRUG CLASS

Angiotensin-Converting Enzyme Inhibitors

COMPLETE LABELING

Product labeling at [DailyMed](#), National Library of Medicine, NIH

CHEMICAL FORMULA AND STRUCTURE

DRUG	CAS REGISTRY NUMBER	MOLECULAR FORMULA	STRUCTURE
Moexipril	103775-10-6	C ₂₇ -H ₃₄ -N ₂ -O ₇	 <p>The chemical structure of Moexipril is a complex molecule. It features a central piperazine ring system. One nitrogen atom of the piperazine is substituted with a 3,4-dimethoxyphenyl group. The other nitrogen atom is substituted with a propyl chain that is further substituted with a benzyl group and a propyl ester group. The piperazine ring is also substituted with a propyl chain that is further substituted with a propyl ester group.</p>