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MARKET REVIEW AND ANALYSIS OF ATENOLOL

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ABSTRACT

Atenolol is a cardio selective -epinephrinic receptor antagonist used primarily to treat hypertension, angina pectoris, and acute myocardial infarction by decreasing heart rate and blood pressure. It controlled by blocking catecholamines at (beta 1)-receptors without intrinsic sympathomimetic activity, offering once-daily dosing. Adrenergic receptor blockers (b-blockers) are applied to treat high blood pressure, ischemic heart disease, and heart rhythm disturbances. Due to their spreading use and human metabolism, b-blocker is mostly detected in sludge and waste waters. b-Adrenergic receptors have been identify in fish and other animals, so it can be expected that physiological processes controlled by these receptors in animals may be affected by the presence of b-blockers. starting point for the assessment of potential environmental risks was the European guideline on the environmental risk assessment of medicinal products for human use. In Phase I of the risk assessment, the initial predicted environmental concentration (PEC) of atenolol in surface water (500 ng L1) exceeded the action limit of 10 ng L1 . Thus, a Phase II risk assessment was conducted showing acceptable risks for surface water, for groundwater, and for aquatic microorganisms. Furthermore, atenolol showed a low potential for bioaccumulation as indicated by its low lipophilicity

Literature Review And Market Analysis

Atenolol, a cardioselective beta-blocker primarily used for hypertension, angina, and post-myocardial infarction management, remains a mature generic pharmaceutical with a stable but low-growth market. It's off-patent since the 1990s, leading to widespread availability from multiple manufacturers. Below is a concise review based on recent industry data from sources like IQVIA, Statista, GlobalData, and regulatory filings (e.g., FDA Orange Book).

Note: Market figures are estimates in USD and can vary by region; I've focused on global and key markets (US, Europe, Asia-Pacific).

Key Market Size and Growth

Growth Drivers: Steady demand from aging populations (e.g., hypertension affects ~1.3B adults globally per WHO). Shift to fixed-dose combinations (e.g., atenolol + chlorthalidone) boosts volumes. Emerging markets like India and China see 7-10% annual growth due to rising cardiovascular disease (CVD) prevalence.

Challenges: Declining volumes in developed markets due to competition from newer agents like ARBs (e.g., losartan), calcium channel blockers, and ACE inhibitors. Patent cliffs on branded combos have intensified generic erosion.

Pricing and Competition

Average Wholesale Price (US): $0.05-0.15 per 50mg tablet (generic); branded Tenormin discontinued in most markets.

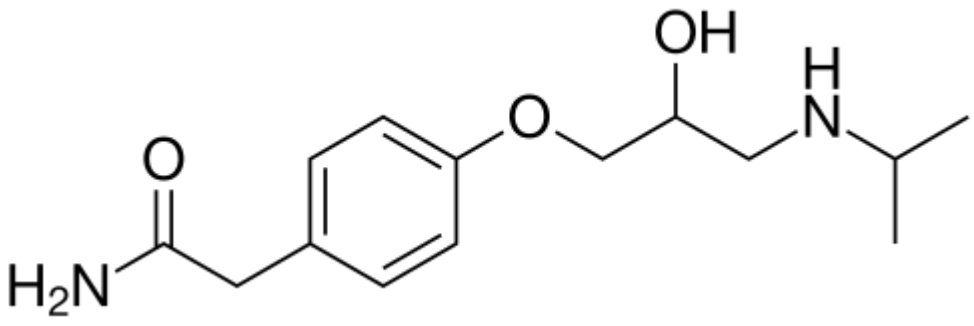
Top Players (by market share, ~2023):

INTRODUCTION

Atenolol, commonly known by the brand name **Tenormin**, is a cardioselective beta -1 adrenergic receptor antagonist (beta-blocker) used to treat high blood pressure (hypertension), angina pectoris, and manage acute myocardial infarction. It reduces heart rate and blood pressure by blocking adrenaline action, mainly improving cardiovascular function.

Atenolol is often used as a first-line treatment for chronic stable angina and is a key medication in reducing the risk of death after a heart attack.

STRUCTURE



ATENOLOL

IUPAC NAME

(RS)-2-{4-[2-hydroxy-3-(propan-2-ylamino)propoxy]phenyl}acetamide

History

Atenolol was patented in 1969 and was approved for medical use in 1975.

Clinical data	
Trade names	Tenormin, others
Other names	ICI-66082; ICI66082
AHFS/Drugs.com	Monograph
MedlinePlus	a684031
License data	US DailyMed: Atenolol
Pregnancy category	AU: C
Routes of administration	Oral, intravenous
Drug class	Beta blocker; β-Adrenergic receptor antagonist; Selective β_1-adrenergic receptor antagonist; Sympatholytic agent; Antihypertensive agent; Anxiolytic

Medical uses

Atenolol is used for a number of conditions including hyperthyroidism, hypertension, angina, long QT syndrome, acute myocardial infarction, supraventricular tachycardia, ventricular tachycardia, essential tremor (ET), and the symptoms of alcohol withdrawal.

The role for β-blockers in general in hypertension was downgraded in June 2006 in the United Kingdom, and later in the United States, as they are less appropriate than other agents such as ACE inhibitors, calcium channel blockers, thiazide diuretics and angiotensin receptor blockers, particularly in the elderly.

Atenolol has been used to treat anxiety disorders, such as simply anxiety disorder and social anxiety disorder. It is thought that beta blockers indirectly treat psychological symptoms of anxiety, but could help manage physical symptoms such as palpitations, and this might interfere with a positive Reply loop to indirectly reduce psychological anxiety. A 2025 systematic review and meta-analysis that included atenolol found widespread prescription of beta blockers for treatment anxiety disorders, but found no evidence of a beneficial effect relative to placebo or benzodiazepines in people with social phobia or panic disorder. However, the quality of evidence, including both numbers of studies and patients as well as quality and risk of bias of those studies, was limited.

Contraindications

Prophilaxis of atenolol include sinus bradycardia, heart block greater than first degree, cardiogenic shock, and overt heart failure. There is no basis for use of atenolol in people with heart rate of lower than 50 bpm or systolic blood pressure of less than 100 mm Hg and hence these can be considered contraindications. It is also contraindicated in people with a history of hypersensitivity to atenolol or any of the drug product's other components.

Side effects

Hypertension treated with a β-blocker such as atenolol, alone or in conjunction with a thiazide diuretic, is associated with a higher incidence of new onset type 2 diabetes mellitus compared to those treated with an ACE inhibitor or angiotensin receptor blocker.

β-blockers, of which atenolol is mainly studied, provides weaker protection against stroke and mortality in patients over 60 years old compared to other antihypertensive medications.Diuretics may be associated with better cardiovascular and cerebrovascular outcomes than β-blockers in the elderly.

Rarely, atenolol has been associated with induction of acute delirium.

Over dose

Symptoms of overdose are due to excessive pharmacodynamic actions on β1 and also β2-receptors. These include bradycardia (slow heartbeat), severe hypotension with shock, acute heart failure, hypoglycemia and bronchospastic reactions. Treatment is largely symptomatic. Hospitalization and intensive monitoring is indicated. Activated charcoal is useful to absorb the drug. Atropine will counteract bradycardia, glucagon helps with hypoglycemia, dobutamine can be given against hypotension and the inhalation of a β2-mimetic such as hexoprenalin or salbutamol will terminate bronchospasms.

Blood or plasma atenolol concentrations may be measured to confirm a diagnosis of poisoning in hospitalized patients or to assist in a medicolegal death investigation. Plasma levels are usually less than 3 mg/L during therapeutic administration, but can range from 3–30 mg/L in overdose victims.

Pharmaco dynamics

Site	***Ki(nm)***	***Species***
5-HT1A	10,000	Rat
5-HT1B	>10,000	Rat
5-HT2A	>10,000	Human
5-HT2C	>10,000	Pig
α1	ND	ND
α2	ND	ND
β1	170–1,500	Human
β2	8,140–9,550	Human
β3	>10,000	Human
D1	ND	ND
D2	>10,000	Rat

Pharmacokinetics

Absorption

The oral bioavailability of atenolol is approximately 50 to 60%.The absorption of atenolol with oral administration is rapid and consistent but is incomplete .About 50% of an oral dose

of atenolol is absorbed from the intestines, with the rest excreted in feces. Maximal concentrations of atenolol occur 2 to 4 hours following an oral dose, whereas peak concentrations occur within 5 minutes with intravenous administration. The pharmacokinetic profile of atenolol results in it having relatively consistent plasma drug levels with about 4-fold variation between individuals.

Distribution

The plasma protein binding of atenolol is 6 to 16%. Atenolol is classified as a hydrophilic beta blocker with low lipophilicity and hence lower potential for crossing the blood–brain barrier and entering the brain. This in turn may result in fewer effects in the central nervous system as well as a lower risk of neuropsychiatric side effects.Only small amounts of atenolol are said to enter the brain.The brain-to-blood ratio of atenolol in humans has been found to be 0.2:1, whereas the ratio for the highly lipophilic propranolol has been found to range from 15:1 to 33:1.

Metabolism

Atenolol undergoes minimal or negligible metabolism by the liver.[3][4] It has been estimated that about 5% of atenolol is metabolized.[5] This is in contrast to other beta blockers like propranolol and metoprolol, but is similar to nadolol.[3] In accordance with its lack of hepatic metabolism, the pharmacokinetics of atenolol are not altered in hepatic impairment, unlike the case of propranolol.[4] Two metabolites of atenolol have been identified: hydroxyatenolol and atenolol glucuronide.It has been said that it is unknown if these metabolites are active. However, another source stated that hydroxyatenolol has one-tenth the beta-blocking activity of atenolol.

Elimination

Instead of by hepatic metabolism, atenolol is eliminated from the blood mainly via renal excretion. Atenolol is excreted about 40 to 50% in urine and 50% in feces with oral administration. Conversely, it is excreted 85 to 100% in urine unchanged and 10% in feces with intravenous administration. Only very small amounts of hydroxyatenolol and atenolol glucuronide are found in urine with atenolol.

The elimination half-life of atenolol is about 6 to 7 hours. The half-life of atenolol does not change with continuous administration. With intravenous administration, atenolol levels

rapidly decline (5- to 10-fold) during the first 7 hours and thereafter decline at a rate similar to that with oral administration.

The elimination of atenolol is slowed in renal impairment, with the elimination rate being closely related to the glomerular filtration rate (GFR) and with

significant accumulation occurring when the creatinine clearance rate is under 35 mL/min/1.73 m2. At a GFR of less than 10 mL/min, the half-life of atenolol increases up to 36 hours.

Chemistry

Atenolol is a substituted phenethylamine derivative. It is specifically β-phenylethylamine with an α-keto substitution and a 4- substitution on the phenyl ring.

The experimental log P of atenolol is 0.16 and its predicted log P ranges from −0.03 to 0.57. Atenolol showed the lowest predicted lipophilicity of 30 clinically relevant beta blockers.

CONCLUSION

In summary, atenolol remains a vital beta-blocker Controlling of cardiovascular conditions, despite facing market challenges from newer agents; its environmental effects and pharmacokinetic properties warrant ongoing assessment to confirm safe usage in both clinical and ecological contexts. As the demand for impact on hypertension treatments persists, understanding atenolol's role and implications will be crucial for healthcare providers and environmentalist regulators alike.

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