For the use of only a Registered Medical Practitioner or a Hospital or a Laboratory

Not to be sold by retail without the prescription of a Registered Medical Practitioner

Prescribing Information

1. Generic Name

Amlodipine and Hydrochlorothiazide Tablets
(Brand Name: ANGICAM®-H Tablets)

2. Qualitative and Quantitative Composition

Each uncoated tablet contains:
Amlodipine Besylate IP equivalent to Amlodipine ……..… 5 mg.
Hydrochlorothiazide IP ……...….. 12.5 mg.
Excipients …….. q.s.

3. Dosage Form and Strength

Dosage Form: Tablets.
Dosage Strength: Amlodipine 5 mg and hydrochlorothiazide 12.5 mg per tablet.

4. Clinical Particulars

4.1 Therapeutic Indication

ANGICAM-H Tablets are indicated in the treatment of mild to moderate hypertension in adult patients whose blood pressure is not adequately controlled by monotherapy.

4.2Posology and Method of Administration

For oral administration.
Adults: 1 tablet to be administered once daily. If blood pressure goal is not achieved within 2 to 4 weeks of therapy, the dose may be increased to 2 tablets once daily. The dosage, however, should be individualized.
Amlodipine is effective in doses between 2.5 mg to 10 mg once daily.
Hydrochlorothiazide is effective in doses between 12.5 mg to 50 mg once daily.
ANGICAM-H Tablets can be administered with or without food.
Or, as directed by the physician.

4.3Contraindications

ANGICAM-H Tablets are contraindicated in patients with:

- Hypersensitivity to amlodipine or to hydrochlorothiazide/other sulfonamide-derived drugs or to any component of the formulation.

- Cardiogenic shock.
- Unstable angina.
- Significant aortic stenosis.
- Severe hypotension.
- Heart failure after acute myocardial infarction.
- Acute porphyria.
- In patients with anuria.

4.4Special Warnings and Precautions for Use

Amlodipine

Hypotension: Symptomatic hypotension is possible, particularly in patients with severe aortic stenosis. Because of the gradual onset of action, acute hypotension is unlikely.

Increased Angina or Myocardial Infarction: Worsening angina and acute myocardial infarction can develop after starting or increasing the dose of amlodipine, particularly in patients with severe obstructive coronary artery disease.

Patients with Hepatic Failure: Amlodipine is extensively metabolized by the liver and the plasma elimination half-life (t½) is 56 hours in patients with impaired hepatic function. Thus, caution should be exercised and dose should be titrated slowly in patients with severe hepatic impairment.

Hydrochlorothiazide

Acute Myopia and Secondary Angle-Closure Glaucoma: Hydrochlorothiazide, a sulfonamide, can cause an idiosyncratic reaction, resulting in acute transient myopia and acute angle closure glaucoma. Symptoms include acute onset of decreased visual acuity or ocular pain and typically occur within hours to weeks of drug initiation. Untreated acute angle-closure glaucoma can lead to permanent vision loss. The primary treatment is to discontinue hydrochlorothiazide as rapidly as possible. Seek immediate medical attention if patients experience any symptoms. Prompt medical or surgical treatments may need to be considered if the intraocular pressure remains uncontrolled. Risk factors for developing acute angle-closure glaucoma may include a history of sulfonamide or penicillin allergy.

Diabetes and Hypoglycemia: Latent diabetes mellitus may become manifest and diabetic patients given thiazides may require adjustment of their insulin dose.

Renal Disease: Cumulative effects of the thiazides may develop in patients with impaired renal function. In such patients, thiazides may precipitate azotemia.

Fluid and Electrolyte Imbalance: In published studies, clinically significant hypokalemia has been consistently less common in patients who received 12.5 mg of hydrochlorothiazide than in patients who received higher doses. Nevertheless, periodic determination of serum electrolytes should be performed in patients who may be at risk for the development of hypokalemia. Patients should be observed for signs of fluid or electrolyte disturbances, i.e., hyponatremia, hypochloremic alkalosis, and hypokalemia and hypomagnesemia.

1. **Hypokalemia:** Hypokalemia may develop, especially with brisk diuresis when severe cirrhosis is present, during concomitant use of corticosteroid or adrenocorticotropic hormone (ACTH) or after prolonged therapy. Interference with adequate oral electrolyte intake will also contribute to hypokalemia. Hypokalemia and hypomagnesemia can provoke ventricular arrhythmias or sensitize or exaggerate the response of the heart to the toxic effects of digitalis. Hypokalemia may be avoided or treated by potassium supplementation or increased intake of potassium rich foods.
2. **Hyponatremia:** Dilutional hyponatremia is life-threatening and may occur in edematous patients in hot weather; appropriate therapy is water restriction rather than salt administration, except in rare instances when the hyponatremia is life-threatening. In actual salt depletion, appropriate replacement is the therapy of choice.
3. **Hyperuricemia:** Hyperuricemia or acute gout may be precipitated in certain patients receiving thiazide diuretics.

Warning signs or symptoms of fluid and electrolyte imbalance include dryness of mouth, thirst, weakness, lethargy, drowsiness, restlessness, muscle pains or cramps, muscular fatigue, hypotension, oliguria, tachycardia, and gastrointestinal disturbances such as nausea and vomiting.
Impaired Hepatic Function: Thiazides should be used with caution in patients with impaired hepatic function. They can precipitate hepatic coma in patients with severe liver disease.
Parathyroid Disease: Calcium excretion is decreased by thiazides. Also, pathologic changes in the parathyroid glands with hypercalcemia and hypophosphatemia have been observed in a few patients on prolonged thiazide therapy.

4.5Drug Interactions

Amlodipine

1. Impact of Other Drugs on Amlodipine

Sildenafil: Hypotensive effect is enhanced when amlodipine is given with sildenafil. Monitor for hypotension when these drugs are given together.
CYP3A Inhibitors (e.g., Ketoconazole, Itraconazole, Diltiazem, Erythromycin, Fluconazole, etc.): Co-administration with CYP3A inhibitors results in increased systemic exposure to amlodipine and may require dose reduction. Monitor for symptoms of hypotension and edema when amlodipine is co-administered with CYP3A inhibitors to determine the need for dose adjustment.
CYP3A Inducers (e.g., Carbamazepine, Phenytoin, Rifampin, etc.): No information is available on the quantitative effects of CYP3A inducers on amlodipine. Blood pressure and edema should be closely monitored when amlodipine is co-administered with CYP3A inducers.

2. Impact of Amlodipine on Other Drugs

Simvastatin: Co-administration of simvastatin with amlodipine increases the systemic exposure of simvastatin. Limit the dose of simvastatin in patients on amlodipine to 20 mg daily.

Immunosuppressants: Amlodipine may increase the systemic exposure of cyclosporine or tacrolimus when co-administered. Frequent monitoring of blood levels of cyclosporine and tacrolimus and dosage adjustment (whenever appropriate) is recommended.

Hydrochlorothiazide

When administered concurrently, the following drugs may interact with thiazide diuretics:

Alcohol, Barbiturates, or Narcotics: Potentiation of orthostatic hypotension may occur.

Antidiabetic Drugs (oral agents and insulin): Dosage adjustment of the antidiabetic drug may be required.

Other Antihypertensive Drugs: Additive antihypertensive effect may occur, thus, reduction in dosage is required.

Cholestyramine and Colestipol Resins: Absorption of hydrochlorothiazide is impaired in the presence of anionic exchange resins. Single doses of either cholestyramine or colestipol resins bind hydrochlorothiazide and reduce its absorption from the gastrointestinal tract by up to 85 and 43 %, respectively.

Corticosteroids, ACTH: Intensified electrolyte depletion, particularly hypokalemia.

Pressor Amines (e.g., norepinephrine): Possible decreased response to pressor amines, but not sufficient to preclude their use.

Skeletal Muscle Relaxants (e.g., tubocurarine): Possible increased responsiveness to the muscle relaxants such as curare derivatives.

Non-Steroidal Anti-Inflammatory Drugs (NSAIDs): In some patients, administration of NSAID can reduce the diuretic, natriuretic, and antihypertensive effects of loop, potassium-sparing and thiazide diuretics.

Digoxin: Thiazide-induced hypokalemia or hypomagnesemia may predispose the patient to digoxin toxicity.

Lithium: Lithium generally should not be given with diuretics. Diuretic agents reduce the renal clearance of lithium and greatly increase the risk of lithium toxicity.

Laboratory Test Interactions: Thiazides should be discontinued before carrying out tests for parathyroid function.

4.6Use in Special Populations

Pregnant Women

Amlodipine: Pregnancy Category C; Hydrochlorothiazide: Pregnancy Category B. There are no adequate and well-controlled studies in pregnant women. In animal studies, reproductive toxicity was observed at high doses of amlodipine. Reports of animal studies provided no evidence of harm to the fetus. However, the routine use of diuretics exposes mother and fetus to unnecessary hazard. Thus, use of ANGIAM-H Tablets in pregnancy is only recommended when there is no safer alternative available and when the disease itself carries greater risk for the mother and foetus.

Lactating Women

Limited available data from a published clinical study reports that amlodipine is present in human milk. However, no adverse effects of amlodipine on the breastfed infant have been observed. Thiazides appear in human milk. Because of the potential for adverse effects on the nursing infant, a decision should be made whether to discontinue nursing or discontinue the drug, taking into account the importance of the drug to the mother.

Paediatric Patients
Safety and effectiveness in paediatric patients have not been established. Thus, ANGICAM-H Tablets are not recommended for use in children.

Geriatric Patients
A greater blood pressure reduction and an increase in side effects have been reported in the elderly (>65 years) patients treated with hydrochlorothiazide. Elderly patients have decreased clearance of amlodipine and hydrochlorothiazide. Thus, in elderly patients, dose selection should be cautious and usually a lower initial dose is recommended.

Renal Impairment Patients
The pharmacokinetics of amlodipine is not significantly influenced by renal impairment. Patients with renal failure, therefore, may receive the usual initial dose.
In patients with renal disease, thiazides may precipitate azotemia. Cumulative effects of the drug may develop in patients with impaired renal function.
Thus, ANGICAM-H Tablets should be used with caution. Safety and effectiveness of ANGICAM-H Tablets in patients with severe renal impairment [creatinine clearance (CrCl) < 30 ml/min] have not been established.

Hepatic Impairment Patients
Amlodipine is extensively metabolized by the liver. Patients with hepatic insufficiency have decreased clearance of amlodipine with a resulting increase in area under the plasma drug concentration-time curve (AUC) to approximately 40 to 60%. A similar increase in AUC has been observed in patients with moderate to severe heart failure. Thiazides should be used with caution in patients with impaired hepatic function.
Thus, caution should be exercised and lower initial dose to be used in patients with hepatic impairment.

4.7Effect on Ability to Drive and Use Machines
Both, amlodipine and hydrochlorothiazide can have minor or moderate influence on the ability to drive and use machines, particularly at the ignition of the therapy. If patients taking this medicine suffer from dizziness, headache, fatigue or nausea the ability to react may be impaired. Caution is recommended especially at the start of treatment.

4.8Undesirable Effects

Amlodipine

Most common adverse reactions reported with amlodipine are headache and pedal edema. Adverse effects with an incidence >1% in clinical studies included abdominal pain, nausea, dizziness, somnolence, flushing, palpitations, and fatigue.

Rare adverse events observed in controlled clinical trials or open trials or post-marketing surveillance studies (causal relationship is uncertain) included:

Cardiovascular: Arrthythmia (including ventricular tachycardia and atrial fibrillation), bradycardia, chest pain, hypotension, peripheral ischemia, syncope, tachycardia, postural hypotension, vasculitis, cardiac failure, pulse irregularity, extrasystoles.

Central and Peripheral Nervous System: Hypoesthesia, peripheral neuropathy, paresthesia, tremor, vertigo, ataxia, hypertonia, migraine, apathy, agitation, amnesia.

Gastrointestinal: Anorexia, constipation, dysphagia, diarrhea, flatulence, gastritis, increased appetite, loose stools, pancreatitis, vomiting, gingival hyperplasia/hypertrophy.

Musculoskeletal System: Arthralgia, arthrosis, muscle cramps, myalgia, muscle weakness, twitching.

Psychiatric: Sexual dysfunction, insomnia, nervousness, depression, abnormal dreams, anxiety, depersonalization.

Respiratory System: Dyspnea, epistaxis, cough, rhinitis.

Skin and Appendages: Rash, pruritus, urticaria, angioedema, skin dryness, alopecia, skin discoloration.

Special Senses: Abnormal vision, conjunctivitis, diplopia, eye pain, abnormal visual accommodation, xerophthalmia, tinnitus, parosmia, taste perversion.

Urinary System: Increased micturition frequency, nocturia, dysuria, polyuria.

Autonomic Nervous System: Dry mouth, increased sweating, cold and clammy skin.

Metabolic and Nutritional: Hyperglycemia, thirst.

Hemopoietic: Leukopenia, purpura, thrombocytopenia.

General: Allergic reaction, back pain, hot flushes, malaise, pain, rigors, weight gain, weight loss.

Hydrochlorothiazide

Adverse events that have been reported with hydrochlorothiazide, without regard to causality, are listed below:

Body as a Whole: Weakness.

Digestive: Pancreatitis, jaundice (intrahepatic cholestatic jaundice), sialadenitis, cramping, gastric irritation.

Hematologic: Aplastic anemia, agranulocytosis, leukopenia, hemolytic anemia, thrombocytopenia.

Hypersensitivity: Purpura, photosensitivity, urticaria, necrotizing angiitis (vasculitis and cutaneous vasculitis), fever, respiratory distress including pneumonitis and pulmonary edema, anaphylactic reactions.

Metabolic: Electrolyte imbalance, hyperglycemia, glycosuria, hyperuricemia.
Musculoskeletal: Muscle spasm.
Nervous System/Psychiatric: Restlessness.
Renal: Renal dysfunction, interstitial nephritis, renal failure.
Skin: Erythema multiforme including Stevens-Johnson syndrome, exfoliative dermatitis including toxic epidermal necrolysis.
Special Senses: Transient blurred vision, xanthopsia.
Urogenital: Impotence.

4.9Overdose

Amlodipine

Overdose might be expected to cause excessive peripheral vasodilation with marked hypotension and possibly reflex tachycardia. In humans, experience with intentional overdose of amlodipine is limited.

If massive overdose should occur, initiate active cardiac and respiratory monitoring. Frequent blood pressure measurements are essential. Should hypotension occur, provide cardiovascular support including elevation of the extremities and the judicious administration of fluids. If hypotension remains unresponsive to these conservative measures, consider administration of vasopressors (such as phenylephrine) with attention to circulating volume and urine output. As amlodipine is highly protein bound, hemodialysis is not likely to be of benefit.

Hydrochlorothiazide

The most common signs and symptoms observed are those caused by electrolyte depletion (hypokalemia, hypochloremia, hyponatremia) and dehydration resulting from excessive diuresis. If digitalis has also been administered, hypokalemia may accentuate cardiac arrhythmias.

In the event of overdose, symptomatic and supportive measures should be employed. Emesis should be induced or gastric lavage performed. Correct dehydration, electrolyte imbalance, hepatic coma and hypotension by established procedures. If required, give oxygen or artificial respiration for respiratory impairment. The degree to which hydrochlorothiazide is removed by hemodialysis has not been established.

5. Pharmacological Properties

5.1 Mechanism of Action

Amlodipine

Amlodipine is a dihydropyridine class of long-acting calcium channel blocker – CCB (calcium antagonist). Amlodipine inhibits the transmembrane influx of calcium ions (Ca^{++}) into vascular smooth muscle and cardiac muscle.

Experimental data suggest that amlodipine binds to both dihydropyridine and nondihydropyridine binding sites. The contractile processes of cardiac muscle and vascular smooth muscle are dependent upon the movement of extracellular calcium ions into these cells through specific ion

channels. Amlodipine inhibits calcium ion influx across cell membranes selectively, with a greater effect on vascular smooth muscle cells than on cardiac muscle cells. Serum calcium concentration is not affected by amlodipine.
Amlodipine is a peripheral arterial vasodilator that acts directly on vascular smooth muscle to cause a reduction in peripheral vascular resistance and reduction in blood pressure.

Hydrochlorothiazide

Hydrochlorothiazide (HCTZ) is a thiazide class of diuretic drugs which blocks the reabsorption of sodium (Na^+) and chloride (Cl^-) ions, and it thereby increases the quantity of sodium traversing the distal tubule of nephron. By this mechanism, hydrochlorothiazide increases excretion of water (diuretic effect), reduces blood volume, and thereby decreases cardiac output. These effects helps to reduce increased blood pressure. As hydrochlorothiazide produces weak antihypertensive effect it is usually combined with other antihypertensive drugs such as amlodipine.
A portion of the additional sodium presented to the distal tubule of nephron is exchanged there for potassium (K^+) and hydrogen (H^+) ions. With continued use of hydrochlorothiazide and depletion of sodium, compensatory mechanisms tend to increase this exchange and may produce excessive loss of potassium, hydrogen, and chloride ions (electrolyte imbalance). Hydrochlorothiazide also decreases the excretion of calcium (Ca^{++}) and uric acid, may increase the excretion of iodide and may reduce glomerular filtration rate.

5.2Pharmacodynamic Properties

Amlodipine

Following administration of therapeutic doses to patients with hypertension, amlodipine produces vasodilation resulting in a reduction of supine and standing blood pressures. With chronic once daily oral administration, antihypertensive effectiveness is maintained for at least 24 hours. These decreases in blood pressure are not accompanied by a significant change in heart rate or plasma catecholamine levels with chronic dosing.

Hydrochlorothiazide

Hydrochlorothiazide is a thiazide class of diuretic agent. Hydrochlorothiazide is widely used to treat hypertension and edema. Acute antihypertensive effects of thiazides are thought to result from a reduction in blood volume and cardiac output, secondary to a natriuretic effect, although a direct vasodilatory mechanism has also been proposed. With chronic administration, plasma volume returns toward normal, but peripheral vascular resistance is decreased.
Thiazides do not affect normal blood pressure. Peak effect of hydrochlorothiazide is observed at about 4 hours of dosing and activity persists for up to 24 hours.

5.3Pharmacokinetic Properties

Amlodipine

Absorption: After oral administration of therapeutic doses, amlodipine produces peak plasma concentrations between 6 to 12 hours. Absolute bioavailability has been estimated to be between 64 to 90%. The bioavailability is not altered by the presence of food.
Distribution: The volume of distribution is approximately 21 l/kg. *Ex vivo* studies have shown that approximately 93% of the circulating drug is bound to plasma proteins in hypertensive patients.
Metabolism: Amlodipine is extensively (about 90%) converted to inactive metabolites via hepatic metabolism with 10% of the parent compound and 60% of the metabolites excreted in the urine.
Excretion: Excretion from the plasma is biphasic with a terminal elimination half-life of about 30 to 50 hours. Steady-state plasma levels of amlodipine are reached after 7 to 8 days of consecutive daily dosing.

Hydrochlorothiazide
Absorption: Hydrochlorothiazide is well absorbed (65% to 75%) following oral administration. Absorption of hydrochlorothiazide is reduced in patients with congestive heart failure. Peak plasma concentrations are observed within 1 to 5 hours of dosing, and range from 70 to 490 ng/ml following oral doses of 12.5 to 100 mg.
Distribution: Plasma concentrations are linearly related to the administered dose. Concentrations of hydrochlorothiazide are 1.6 to 1.8 times higher in whole blood than in plasma. Plasma protein binding is approximately 40% to 68%.
Metabolism and Excretion: The plasma elimination half-life is 6 to 15 hours. Hydrochlorothiazide is eliminated primarily by renal pathways. Following oral doses of 12.5 to 100 mg, 55% to 77% of the administered dose appears in urine and greater than 95% of the absorbed dose is excreted in urine as unchanged drug. In patients with renal disease, plasma concentration of hydrochlorothiazide is increased and the elimination half-life is prolonged.

6. Nonclinical Properties

6.1 Animal Toxicology

Amlodipine
Carcinogenesis: Rats and mice treated with amlodipine in the diet for up to two years, at concentrations calculated to provide daily dosage levels of 0.5, 1.25, and 2.5 amlodipine mg/kg/day, showed no evidence of a carcinogenic effect of the drug.
Mutagenesis: Mutagenicity studies conducted with amlodipine revealed no drug related effects at either the gene or chromosome level.
Impairment of Fertility: There was no effect on the fertility of rats treated orally with amlodipine (males for 64 days and females for 14 days prior to mating) at doses up to 10 mg amlodipine/kg/day (8 times [based on body weight of 50 kg] the maximum recommended human dose of 10 mg/day on a mg/m^2 basis).
Teratogenicity: No evidence of teratogenicity or other embryo/fetal toxicity was found when pregnant rats and rabbits were treated orally with amlodipine at doses up to 10 mg

amlodipine/kg/day (approximately 10 and 20 times the maximum recommended human dose based on body surface area, respectively) during their respective periods of major organogenesis. However for rats, litter size was significantly decreased (by about 50%) and the number of intrauterine deaths was significantly increased (about 5-fold) in rats receiving amlodipine at a dose equivalent to 10 mg amlodipine/kg/day for 14 days before mating and throughout mating and gestation. Amlodipine has been shown to prolong both the gestation period and the duration of labor in rats at this dose.

Hydrochlorothiazide

Carcinogenesis: Two-year feeding studies in mice and rats conducted under the auspices of the National Toxicology Program (NTP) uncovered no evidence of a carcinogenic potential of hydrochlorothiazide in female mice (at doses of up to approximately 600 mg/kg/day) or in male and female rats (at doses of approximately 100 mg/kg/day). The NTP, however, found equivocal evidence for hepatocarcinogenicity in male mice.

Mutagenesis: Hydrochlorothiazide was not genotoxic *in vitro* in the Ames mutagenicity assay of Salmonella typhimurium strains TA 98, TA 100, TA 1535, TA 1537, and TA 1538 and in the Chinese Hamster Ovary (CHO) test for chromosomal aberrations, or *in vivo* in assays using mouse germinal cell chromosomes, Chinese hamster bone marrow chromosomes, and the Drosophila sex-linked recessive lethal trait gene. Positive test results were obtained only in the *in vitro* CHO Sister Chromatid Exchange (clastogenicity) and in the Mouse Lymphoma Cell (mutagenicity) assays, using concentrations of hydrochlorothiazide from 43 to 1300 mcg/mL, and in the Aspergillus nidulans non-disjunction assay at an unspecified concentration.

Impairment of Fertility: Hydrochlorothiazide had no adverse effects on the fertility of mice and rats of either sex in studies wherein these species were exposed, via their diet, to doses of up to 100 and 4 mg/kg, respectively, prior to conception and throughout gestation.

Teratogenicity: Studies in which hydrochlorothiazide was orally administered to pregnant mice and rats during their respective periods of major organogenesis at doses up to 3000 and 1000 mg hydrochlorothiazide/kg, respectively, provided no evidence of harm to the fetus.

7. Description

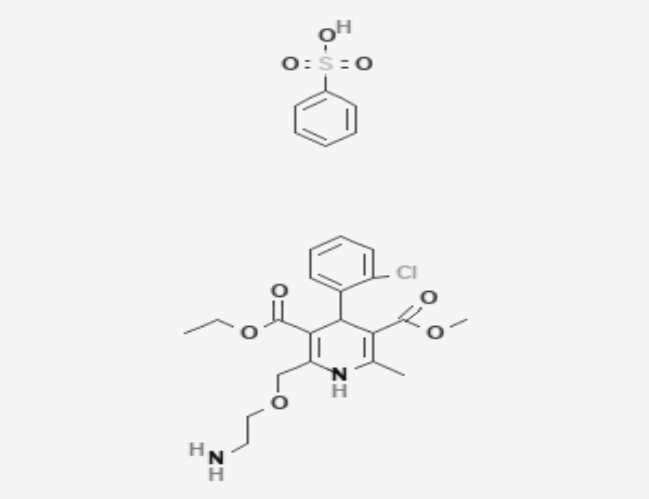
ANGICAM-H Tablets are white to off-white, round, flat faced, beveled edge, breakline on one side and plain on other side, and uncoated tablet.

ANGICAM-H Tablets contains 5 mg of amlodipine and 12.5 mg of hydrochlorothiazide for oral administration in adults.

Amlodipine Besylate

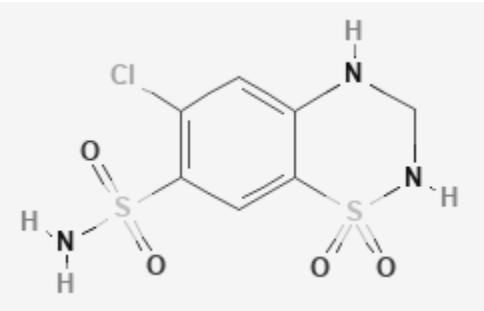
Amlodipine Besylate is the besylate salt of amlodipine. Amlodipine is a long-acting calcium channel blocker (CCB) of a synthetic dihydropyridine class with antihypertensive and antianginal effects.

Amlodipine besylate is a white crystalline powder.
Molecular Weight: 567.1 g/mol.
Molecular Formula: C20H25ClN2O5•C6H6O3S.
Chemical Name: 3-Ethyl-5-methyl (±)-2-[(2-aminoethoxy)methyl]-4-(2- chlorophenyl)-1,4-dihydro-6-methyl-3,5-pyridinedicarboxylate, monobenzenesulphonate.
Structural Formula:



Hydrochlorothiazide

Hydrochlorothiazide is short acting thiazide class of diuretic used for the treatment of hypertension and congestive heart failure.
Hydrochlorothiazide is a white or practically white crystalline powder.
Molecular Weight: 297.7 g/mol.
Molecular Formula: C7H8ClN3O4S2.
Chemical Name: 6-chloro-1,1-dioxo-3,4-dihydro-2H-1λ6,2,4-benzothiadiazine-7-sulfonamide.
Structural Formula:



Inactive ingredients (excipients) of ANGICAM-H Tablets contain Dibasic Calcium Phosphate, Microcrystalline Cellulose, Polyvinyl Pyrrolidone, Isopropyl Alcohol, Sodium Starch Glycolate, and Magnesium Stearate.

8. Pharmaceutical Particulars

8.1 Incompatibilities

None known.

8.2Shelf-life

24 months.

8.3Packaging Information

Strip of 15 tablets.

8.4Storage and Handling Instructions

Store protected from light and moisture at a temperature not exceeding 30°C.
Keep out of reach of children.

9. Patient Counseling Information

Instructions to Patients

- Instruct patients to take this medicine exactly as prescribed by your doctor. Do not change the dose or stop therapy without consulting doctor.
- Instruct patients to take ANGICAM-H Tablets once a day, with or without food. It may be easier to take your dose if you do it at the same time every day, such as with breakfast or dinner, or at bedtime. Do not take more than one dose at a time.
- If patients miss a dose, they can take it as soon as they remember. Do not take ANGICAM-H Tablets if it has been more than 12 hours since the last missed dose. Wait and take the next dose at regular scheduled time.
- Pregnant women and breastfeeding mothers should avoid use of this medicine.
- Use of this medicine is not recommended in children.
- Patients should be informed that while taking ANGICAM-H Tablets do not stop taking other prescription medicines, including any other blood pressure medicines, without consulting their doctor.

10.Details of Manufacturer

Akums Drugs & Pharmaceuticals Ltd.
Plot No. 26A, 27-30, Sector-8A, I.I.E., SIDCUL,
Ranipur, Haridwar – 249403, Uttarakhand.

11. Details of Permission or License Number with Date

Mfg. Lic. No. : 4/UA/LL/2014; Date of FDA Product Permission: 27/01/2015.

12. Date of Revision

April 2021.

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BLUE CROSS LABORATORIES PVT LTD.
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