MedChoice
Endocrine

Propranolol Hydrochloride

Indirin

10 mg • 40 mg Film-Coated Tablet
Beta Blocking Agent, Non-Selective

FORMULATION

Each film-coated tablet contains:
Propranolol Hydrochloride, USP..... 10 mg
Propranolol Hydrochloride,USP..... 40 mg

PRODUCT DESCRIPTIONS

Propranolol Hydrochloride (Indirin) 10 mg Film-Coated Tablet: Purple-red, round, biconvex, film-coated tablet, scored on one side and plain on the other side.

Propranolol Hydrochloride (Indirin) 40 mg Film-Coated Tablet: Purple-red, oval-shaped, biconvex, film-coated tablet, scored on one side and plain on the other side.

PHARMACOLOGICAL PROPERTIES

Pharmacodynamic Properties

Propranolol Hydrochloride (Indirin) is a competitive antagonist at both the β-1 and β-2 adrenoceptors. It has no agonist activity at the beta-adrenoceptor, but has membrane stabilizing activity at concentrations exceeding 1-3 mg/liter, though such concentrations are rarely achieved during oral therapy. Competitive beta-adrenoceptor blockade has been demonstrated in man by a parallel shift to the right in the dose-heart rate response curve to beta agonists such as Isoprenaline.
Propranolol Hydrochloride (Indirin), as with other beta-blockers, has negative inotropic effects, and is therefore contraindicated in uncontrolled heart failure (See Warnings and Precautions).
Propranolol is a racemic mixture and the active form is the S (-) isomer of propranolol. With the exception of inhibition of the conversion of thyroxine to triiodothyronine, it is unlikely that any additional ancillary properties possessed by R (+) propranolol in comparison with racemic mixture will give rise to different therapeutic effects.
Propranolol Hydrochloride (Indirin) is effective and well-tolerated in most ethnic populations, although the response may be less in black patients.

Pharmacokinetic Properties

Propranolol is completely absorbed after oral administration and peak plasma concentrations occur 1-2 hours after dosing in fasting patients. The liver removes up to 90% of an oral dose with an elimination half-life of 3 to 6 hours. Propranolol is widely and rapidly distributed throughout the body with the highest levels occurring in the lungs, liver, kidney, brain and heart. Propranolol is highly protein bound (80-95%).

Preclinical Safety Data

Non-clinical data reveal no special hazard for humans based on conventional studies of safety pharmacology, repeated dose toxicity, local tolerance, genotoxicity, carcinogenic potential and toxicity to reproduction.

INDICATIONS

- Control of hypertension
- Management of phaeochromocytoma (should only be started in the presence of effective alpha blocker)
- Management of angina pectoris
- Adjunctive management of thyrotoxicosis and thyrotoxic crisis
- Long term prophylaxis after recovery from acute myocardial infarction
- Control of cardiac arrhythmias
- Prophylaxis of migraine
- Management of essential tremor
- Control of anxiety and anxiety tachycardia
- Management of hypertrophic obstructive cardiomyopathy
- Prophylaxis of upper gastrointestinal bleeding in patients with portal hypertension and oesophageal varices

MODE OF ADMINISTRATION

Since the half-life may be increased in patients with significant hepatic or renal impairment, caution must be exercised when starting treatment and selecting the initial dose.

For Oral Administration

Adults

Hypertension: Initially 40 mg two to three times daily, which may be increased by 80 mg per day at weekly intervals according to response. The usual dose range is 160 to 320 mg per day. With concurrent diuretic or other antihypertensive drugs, a further reduction of blood pressure is obtained.
Angina, migraine and essential tremor: A starting dose of 40 mg two or three times daily may be increased by the same amount at weekly intervals according to patient response. An adequate response in migraine is usually seen in the range 80 to 160 mg/day and in angina and essential tremor in the range 120 to 240 mg/day.
Arrhythmias, hypertrophic obstructive cardiomyopathy and thyrotoxicosis: A dosage range of 10 to 40 mg three or four times a day usually achieves the required response.
Post-myocardial infarction: Treatment should start between days 5 and 21 after myocardial infarction, with an initial dose of 40 mg four times a day for 2 or 3 days. In order to improve compliance, the total daily dosage may thereafter be given as 80 mg twice a day.
Hyperthyroidism: The dose is adjusted according to clinical response.
Portal Hypertension: Dosage should be titrated to achieve approximately 25% reduction in heart rate at rest. Dosing should begin with 40 mg twice daily, increasing to 80 mg twice daily depending on heart rate response. If necessary, the dose may be increased incrementally to a maximum of 160 mg twice daily.
Phaeochromocytoma: Propranolol Hydrochloride (Indirin) is used only in the presence of effective alpha-blocker. Pre-operative: 60 mg daily for 3 days is recommended. Non-operable malignant cases: 30 mg daily.
Hepatic Impairment: The bioavailability of propranolol may be increased in patients with hepatic impairment and dose adjustments may be required. In patients with severe liver disease (e.g. cirrhosis), a low initial dose is recommended (not exceeding 20 mg three times a day) with close monitoring of the response to treatment (such as the effect on heart rate).
Renal Impairment: Concentrations of propranolol may increase in patients with significant renal impairment and hemodialysis. Caution should be exercised when starting treatment and selecting the initial dose. As with the other beta-adrenoreceptor blocking agents, treatment should not be discontinued abruptly. The dosage should be withdrawn gradually over a period of 7 to 14 days. Either the equivalent dosage of another beta-adrenoceptor blocker may be substituted or the withdrawal of propranolol should be gradual. Patients should be followed during withdrawal especially those with ischaemic heart disease. The risk/benefit of stopping beta blockade should be made for each patient.

Elderly

Evidence concerning the relationship between blood level and age is conflicting. Propranolol should be used to treat older people with caution. It is suggested that treatment should start with the lowest dose. The optimum dose should be individually determined according to clinical response.

Pediatrics

Arrhythmias: Dosage should be determined according to the cardiac status of the patient and the circumstances necessitating treatment. The dose should be adjusted individually and the following is a guide: Children and adolescents: 0.25-0.5 mg/kg three to four times daily, adjusted according to clinical response.
Migraine:
Under the age of 12: 20 mg two or three times daily.
Over the age of 12: the adult dose

CONTRAINDICATIONS

- Patients with known hypersensitivity to propranolol
- Patients with history of bronchospasm, bronchial asthma, or chronic obstructive pulmonary disease
- Cardiac decompensation which is not adequately treated
- Sick sinus syndrome/SA-block
- Metabolic acidosis
- Second and third-degree heart block
- Patients prone to hypoglycemia, e.g. due to prolonged fasting or restricted counter regulatory reserve
- Cardiogenic shock
- Untreated phaeochromocytoma
- Severe bradycardia
- Severe hypotension
- Severe peripheral arterial disturbances
- Prinzmetal's angina

WARNINGS AND PRECAUTIONS

Propranolol Hydrochloride (Indirin) as with other beta-blockers:

- Although contraindicated in uncontrolled heart failure, it may be used in patients whose signs of heart failure have been controlled. Caution must be exercised in patients whose cardiac reserve is poor.
- Should not be used in combination with calcium channel blockers with negative inotropic effects (e.g. verapamil, diltiazem), as it can lead to an exaggeration of these effects particularly in patients with impaired ventricular function and/or SA or AV conduction abnormalities. This may result in severe hypotension, bradycardia and cardiac failure. Neither the beta-blocker nor the calcium channel blocker should be administered intravenously within 48 hours of discontinuing the other.
- Although contraindicated in severe peripheral arterial circulatory disturbances, it may also aggravate less severe peripheral arterial circulatory disturbances.
- Due to its negative effect on conduction time, caution must be exercised if it is given to patients with first degree heart block.
- May block/modify the signs and symptoms of the hypoglycemia (especially tachycardia). Propranolol occasionally causes hypoglycemia, even in non-diabetic patients, e.g. neonates, infants, children, elderly patients, patients on hemodialysis or patients suffering from chronic liver disease and patients suffering from overdose. Severe hypoglycemia associated with propranolol has rarely presented with seizures and/or coma in isolated patients. Caution must be exercised in the concurrent use of propranolol and hypoglycemic therapy in diabetic patients. Propranolol may prolong hypoglycemic response to insulin.
- May mask the signs of thyrotoxicosis.
- Should not be used in untreated phaeochromocytoma. However, in patients with phaeochromocytoma, an alpha-blocker may be given concomitantly.
- Will reduce heart rate as a result of its pharmacological action. In the rare instances when a treated patient develops symptoms which may be attributable to a slow heart rate, the dose may be reduced.
- May cause a more severe reaction to a variety of allergens, when given to patients with a history of anaphylactic reaction to such allergens. Such patients may be unresponsive to the usual doses of adrenaline used to treat the allergic reactions.
- Avoid abrupt withdrawal of beta-blockers. The dosage should be withdrawn gradually over a period of 7-14 days. Patients should be followed during withdrawal especially those with ischaemic heart disease.
- When a patient is scheduled for surgery and a decision is made to discontinue beta-blocker therapy, this should be done at least 48 hours prior to the procedure. The risk/benefit of stopping beta blockade should be made for each patient.
- Since the half-life may be increased in patients with significant hepatic or renal impairment, caution must be exercised when starting treatment and selecting the initial dose.
- Propranolol must be used with caution in patients with decompensated cirrhosis. In patients with portal hypertension, liver function may deteriorate and hepatic encephalopathy may develop. There have been reports suggesting that treatment with propranolol may increase the risk of developing hepatic encephalopathy.
- In patients with chronic obstructive pulmonary disease, non-selective beta-blockers such as propranolol may aggravate the obstructive condition. Therefore propranolol should not be used in this condition.
- Bronchospasms can usually be reversed by beta2 agonist bronchodilators such as salbutamol. Large doses of beta bronchodilators may be required to overcome the beta blockade produced by propranolol and the dose should be titrated according to the clinical response; both intravenous and inhalational administration should be considered. The use of intravenous aminophylline and/or the use of ipratropium (given by nebuliser) may also be considered. Glucagon (1 to 2 mg given intravenously) has also been reported to produce a bronchodilator effect in asthmatic patients. Oxygen or artificial ventilation may be required in severe cases.
- Isolated reports of myasthenia gravis like syndrome or exacerbation of myasthenia gravis have been reported in patients administered with propranolol.

Interference with laboratory tests: Propranolol has been reported to interfere with the estimation of serum bilirubin by the diazo method and with the determination of catecholamines by methods using fluorescence.

Lactose: This medicine contains lactose. Patients with rare hereditary problems of galactose intolerance, the Lapp lactase deficiency or glucose-galactose malabsorption should not take this medicine.

INTERACTION WITH OTHER MEDICINAL PRODUCTS AND OTHER FORMS OF INTERACTION

(Combination not recommended)

- Combined use of beta-blockers and calcium channel blockers with negative inotropic effects (e.g., verapamil, diltiazem) can lead to an exaggeration of the negative AV conduction and sinus node function particularly in patients with impaired ventricular function and/or SA or AV conduction abnormalities. This may result in severe hypotension and bradycardia. The combination with propranolol should be avoided, especially in patients with cardiac decompensation.
- Concomitant use of sympathomimetic agents e.g., adrenaline, may counteract the effect of beta-blockers. Caution must be exercised in the parenteral administration of preparations containing adrenaline to patients taking beta-blockers as in, rare cases, vasoconstriction, hypertension and bradycardia may result.

(Combination not recommended)

- *Beta-agonist bronchodilators:* Non-cardio selective beta-blockers oppose the bronchodilator effects of beta-agonist bronchodilators. Propranolol is contraindicated in patients with asthma.
- *Fingolimod:* Potentiation of bradycardia effects with possible fatal outcomes. Treatment with Fingolimod should not be initiated in patients receiving beta-blockers. In case of combination, at least overnight monitoring is appropriate for treatment initiation.
- *Barbiturates:* The plasma levels and the effects of beta-blockers are reduced by the barbiturates. Barbiturates are potent liver enzyme inducers which may increase the metabolism of propranolol.
- *Propafenone*: Plasma propranolol levels can be raised up to 100% by propafenone. This probably was because propranolol is partially metabolized by the same enzyme like propafenone (CYP2D6). This combination is also not advisable because propafenone has negative inotropic effects.
- *Warfarin:* Propranolol may cause a reduction in clearance and an increase in plasma concentrations of warfarin.
- *MAO inhibitors:* Concomitant use of MAO inhibitors (except MAO-B inhibitors) with antihypertensive agents may diminish the antihypertensive effect and lead to hypertensive reactions.
- *Glycosides:* Digitalis glycosides, in association with beta-blockers, may increase atrio-ventricular conduction time.

(Combination to be used with caution, dose adjustment may be required.)

- *Amiodarone:* A few cases suggest that patients treated with amiodarone can have severe sinus bradycardia when treated concomitantly with propranolol. Amiodarone has an extremely long half-life (about 50 days), which means that interactions may occur long after discontinuation of therapy.
- *Class I Antiarrhythmic drugs (Disopyramide, Quinidine):* Class I antiarrythmic drugs and beta-blockers have additive negative inotropic effects which may result in hypotension and severe hemodynamic side effects in patients with impaired left ventricular function.
- *Non-steroidal anti-inflammatory/anti-rheumatic drugs (NSAIDS):* Anti-inflammatory drugs of NSAID-type counter the antihypertensive effect of beta-blockers. It has been studied mainly in indomethacin. In a study on diclofenac, no such interaction could be detected. Data for COX-2 inhibitors are missing.
- *Cimetidine:* Cimetidine increases levels of propranolol in plasma, probably by inhibiting its first pass metabolism. There may be a risk of bradycardia with oral dosing.
- *Alcohol:* Concomitant use of alcohol may increase the plasma levels of propranolol.
- *Anaesthetics:* Concomitant use of beta-adrenergic antagonists and anaesthetics may attenuate reflex tachycardia and increase the risk of hypotension. As a general rule, avoid sudden withdrawal of beta-blocker treatment. The anaesthesiologist should be informed when the patient is receiving beta-adrenergic antagonists. Anaesthetics agents causing myocardial depression are best avoided.
- *Epinephrine (Adrenaline):* A number of reports are available for severe hypertension and bradycardia in patients treated with propranolol and epinephrine. These clinical observations have been confirmed by studies in healthy volunteers. It has also been suggested that the intravascular administration of epinephrine may trigger these reactions.
- *Fluvoxamine:* Fluvoxamine inhibits oxidative metabolism and increases plasma concentrations of propranolol. This may result in severe bradycardia.
- *Centrally-acting antihypertensives (Clonidine, Moxonidine, Methyldopa):* Concomitant use of centrally acting antihypertensive drugs may worsen heart failure by a decrease in the central sympathetic tonus (reduction of heart rate and cardiac output, vasodilation). Abrupt withdrawal, particularly if prior to beta-blocker discontinuation, may increase risk of "rebound hypertension". If the two drugs are co-administered, the beta-blocker should be withdrawn several days before discontinuing clonidine. If replacing clonidine by beta blocker therapy, the introduction of beta-blockers should be delayed for several days after clonidine administration has stopped.
- *Rifampicin:* The metabolism of propranolol may be increased by potent liver enzyme inducer rifampicin.
- *Alpha blockers:* Concomitant use with alpha-blockers increases the risk of hypotension, especially orthostatic hypotension, and tachycardia and palpitations.
- *Dihydropyridine calcium channel blockers (Nifedipine):* Concomitant use may increase the risk of hypotension, and cardiac failure may occur with latent cardiac insufficiency.
- *Chlorpromazine:* The concurrent use of chlorpromazine with propranolol can result in a marked rise in plasma levels of both drugs, and thereby enhance its effects on heart rate and blood pressure as well as an enhanced antipsychotic effect for chlorpromazine and an increased antihypertensive effect for propranolol.
- *Lidocaine:* Administration of propranolol during infusion of lidocaine may increase the plasma concentration of lidocaine by approximately 30%. Patients already receiving propranolol tend to have higher lidocaine levels than controls. The combination should be avoided.
- *Anti-migraine drugs:* During concomitant treatment with propranolol, it inhibited the first-pass metabolism of rizatriptan whose AUC increases by 70-80%. A dose of 5 mg of rizatriptan is recommended for combination therapy. Ergotamine with propranolol has resulted in reports of vasospastic reactions in some patients.
- *Theophylline:* Propranolol reduces the metabolic clearance of theophylline by about 30% at a dosage of 120 mg/day and 50% at doses of 720 mg/day.
- *Insulin and oral antidiabetic drugs:* Concomitant use may mask certain symptoms of hypoglycemia (palpitations, tachycardia). Propranolol may prolong the hypoglycemic response to insulin.
- *Tobacco:* Tobacco smoking can reduce the beneficial effects of the beta-blockers on heart rate and blood pressure.
- *Laboratory tests:* Interference with laboratory tests - propranolol has been reported to interfere with the estimation of serum bilirubin by the diazo method and with the determination of catecholamines by methods using fluorescence.

PREGNANCY AND LACTATION

Pregnancy: As with all drugs propranolol should not be given during pregnancy unless its use is essential. There is no evidence of teratogenicity with propranolol. However, beta-blockers reduce placental perfusion, which may result in intra-uterine fetal death, immature and premature deliveries. In addition, adverse effects (especially hypoglycemia and bradycardia in the neonate and bradycardia in the fetus) may occur. There is an increased risk of cardiac and pulmonary complications in the neonate in the post-natal period.

Lactation: Most beta-adrenoceptor blocking drugs, particularly lipophilic compounds, will pass into breast milk although to a variable extent. Breast feeding is therefore not recommended following administration of these compounds.

Fertility: No relevant data on effect of fertility in humans is available.

EFFECT ON ABILITY TO DRIVE AND USE MACHINES

Propranolol has no or negligible influence on the ability to drive and use machines. It should be taken into account that occasionally, dizziness or fatigue may occur.

POSSIBLE ADVERSE REACTIONS

Propranolol Hydrochloride (Indirin) is usually well tolerated. In clinical studies, the possible adverse reactions reported are usually attributable to the pharmacological actions of propranolol. The following possible adverse reactions, listed by body system, have been reported.

- Blood and Lymphatic System Disorders: thrombocytopaenia, agranulocytosis
- Immune System Disorder: angioedema
- Metabolism and Nutrition Disorders: hypoglycemia in neonates, infants, children, elderly patients, patients on hemodialysis, patients on concomitant antidiabetic therapy, patients with prolonged fasting and patients with chronic liver disease has been reported; changes in lipid metabolism (changes in blood concentrations of triglycerides and cholesterol); severe hypoglycemia may rarely lead to seizures or coma.
- Psychiatric Disorders: sleep disturbances, nightmares, hallucinations, psychosis, mood changes, depression
- Nervous System Disorders: confusion, memory loss, paraesthesia, dizziness, isolated reports of myasthenia gravis like syndrome or exacerbation of myasthenia gravis have been reported, headache, seizure linked to hypoglycemia
- Eye Disorders: dry eyes, visual disturbance, conjunctivitis
- Cardiac Disorders: bradycardia, cold extremities, heart failure deterioration, precipitation of heart block, postural hypotension which may be associated with syncope, worsening of attacks of angina pectoris.
- Vascular Disorders: Raynaud's phenomenon, exacerbation of intermittent claudication
- Respiratory, Thoracic and Mediastinal Disorders: breathlessness, bronchospasm may occur in patients with bronchial asthma or a history of asthmatic complaints, sometimes with fatal outcome; dyspnea
- Gastrointestinal Disorders: gastrointestinal disturbances such as nausea, vomiting, diarrhea; constipation; dry mouth
- Skin and Subcutaneous Tissue Disorders: purpura, alopecia, psoriasiform skin reactions, exacerbation of psoriasis, skin rashes; isolated cases of hyperhidrosis have been reported.
- Musculoskeletal and Connective Tissue Disorder: arthralgia
- Renal and Urinary Disorders: reduced renal blood flow and GFR
- Reproductive System and Breast Disorder: impotence
- General Disorders and Administration Site Conditions: fatigue and/or lassitude (often transient), dizziness

Discontinuance of the drug should be considered if, according to clinical judgement, the well-being of the patient is adversely affected by any of the reactions. Cessation of therapy with a beta-blocker should be gradual. In the rare event of intolerance manifested as bradycardia and hypotension, the drug should be withdrawn and, if necessary, treatment for overdosage is instituted.

"For suspected adverse drug reaction, report to FDA: www.fda.gov.ph. Seek medical attention immediately at first sign of any adverse drug reaction"

OVERDOSE AND TREATMENT

Toxicity: Individual response varies greatly, death in adult has followed ingestion of about 2 g, and ingestion of more than 40 mg may cause serious problem in children.

Symptoms:

- ***Cardiac:*** Bradycardia, hypotension, pulmonary edema, syncope and cardiogenic shock may develop. Conduction abnormalities such as first or second degree AV block may occur. Rarely arrythmias may occur. Development of cardiovascular complications is more likely if other cardioactive drugs, especially calcium channel blockers, digoxin cyclic antidepressants or neuroleptics have also been ingested. The elderly and those with underlying ischaemic heart disease are risk of developing severe cardiovascular compromise.
- ***CNS:*** Drowsiness, confusion, seizures, hallucinations, dilated pupils and in severe cases coma may occur. Neurological sign such as coma or absence of pupil reactivity are unreliable prognostic indicators during resuscitation.
- ***Other features:*** bronchospasm, vomiting and occasionally CNS-mediated respiratory depression may occur. The concept of cardioselectivity is much less applicable in the overdose situation and systemic effects of beta-blockade include bronchospasm and cyanosis. Particularly in those with pre-existing airways disease. Hypoglycemia and hypocalcemia are rare and occasionally generalized spasm may also be present.

Treatments:

In cases of overdose or extreme falls in the heart rate or blood pressure, treatment with propranolol must be stopped. In addition to primary poison elimination measures, vital parameters must be monitored and corrected accordingly in intensive care. In case of cardiac arrest, the resuscitation of several hours may be indicated. This should include general symptomatic and supportive measures including a clear airway and monitoring of vital sign until stable. Consider activated charcoal (50 g for adults, 1 g/kg for children) if an adult presents within 1 hour of ingestion of more than a therapeutic dose or a child for any amount. Atropine should be administered before gastric lavage, when required as there is a risk of vagal stimulation. Alternatively consider gastric lavage in adults within 1 hour of a potentially life-threatening overdose. Excessive bradycardia may respond to large dose of atropine (3 mg intravenously for an adult and 0.04 mg/kg for a child) and/or a cardiac pacemaker.

For severe hypotension, heart failure or cardiogenic shock in adults a 5-10 mg IV bolus of glucagon (50-150 micrograms/kg in a child) should be administered over 10 minutes to reduce the likelihood of vomiting, followed by an infusion of 1-5 mg/hour (50 micrograms/kg/hour), titrated to clinical response. If glucagon is not available or if there is severe bradycardia and hypotension, which is not improved by glucagon, the beta blocking effect can be counteracted by slow intravenous administration of isoprenaline hydrochloride, dopamine or noradrenalin. In severe hypotension additional inotropic support may be necessary with a beta agonist such as dobutamine 2.5 – 40 micrograms/kg/min (adults and children). It is likely that these doses would be inadequate to reverse the cardiac effects of beta blockade if a large overdose has been taken. The dose of dobutamine should therefore be increased if necessary to achieve the required response according to the clinical condition of the patient.

Nebulised salbutamol 2.5-5 mg should be given for bronchospasm. Intravenous aminophylline may be of benefit in severe cases (5 mg/kg over 30 mins followed by an infusion of 0.5-1 mg/kg/hour). Do not give the initial loading dose of 5 mg/kg if the patient is taking oral theophylline or aminophylline. Cardiac pacing may also be effective at increasing heart rate but does not always correct hypotension secondary to myocardial depression.

In cases of generalized spasm, a slow intravenous dose of diazepam may be used (0.1-0.3 mg/kg body weight).

CAUTION

Foods, Drugs, Devices, and Cosmetics Act prohibits dispensing without prescription.

STORAGE CONDITION

Propranolol Hydrochloride (Indirin) should be stored at temperatures not exceeding 30°C.

Protect from light and moisture. Keep out of reach of children.

DOSAGE FORMS AND PACKAGING AVAILABLE

Propranolol Hydrochloride (Indirin) 10 mg Film-Coated Tablet:
Alu/WhiteOpaque PVC Blister pack x 10's (Box of 100 Tablets)
Registration No.: DR-XY46602
Date of First Authorization: May 2019

Propranolol Hydrochloride (Indirin) 40 mg Film-Coated Tablet:
Alu/White Opaque PVC Blister pack x 10's (Box of 100 Tablets)
Registration No.: DR-XY46106
Date of First Authorization: October 2017

Date of Revision of Package Insert: July 2019

Manufactured for:
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