30XXXXX

CARVEDILOL PHOSPHATE

CARVETA XR

10mg Extended Release Capsule
20mg Extended Release Capsule
40mg Extended Release Capsule

ALPHA/BETA-ADRENERGIC BLOCKING AGENTS

FORMULATION

Each extended release capsule contains:
Carvedilol phosphate……..10mg
Carvedilol phosphate …….. 20mg
Carvedilol phosphate …….. 40mg

DESCRIPTION

10mg - Orange/ White, hard gelatin capsule of size '0' containing white to off - white colored, circular, biconvex uncoated tablet plain on both sides and white to off-white colored powder.

20mg - Purple/ White, hard gelatin capsule of size '0' containing two white to off - white colored, circular, biconvex uncoated tablet plain on both sides and white to off-white colored powder.

40mg - Bluish gray/ Bluish gray hard gelatin capsule of size '0' containing one white to off - white colored, circular, biconvex uncoated tablet plain on both sides and white to off-white free flowing colored powder.

80mg - Maroon/ Maroon hard gelatin capsule of size '0' containing two white to off - white colored, circular, biconvex uncoated tablet plain on both sides and white to off-white colored free flowing powder.

PHARMACOLOGICAL PROPERTIES

Mechanism of Action

Carvedilol is a racemic mixture in which nonselective β-adrenoreceptor blocking activity is present in the S(-) enantiomer and α-adrenergic blocking activity is present in both R(+) and S(-) enantiomers at equal potency. Carvedilol has no intrinsic sympathomimetic activity.

Pharmacokinetics

Absorption

Carvedilol ER has approximately 85% of the bioavailability of Carvedilol immediate–release. For corresponding dosages, the exposure (AUC, C_{trough} concentration) of Carvedilol as Carvedilol ER is equivalent to those of immediate-release Carvedilol when both are administered with food. The absorption of Carvedilol from Carvedilol ER is slower or more prolonged compared to the Carvedilol immediate-release with peak concentrations achieved approximately 5 hours after administration. Plasma concentrations of Carvedilol increase in a dose-proportional manner over the dosage range of Carvedilol ER 10-80mg.

Effect of Food: Administration of Carvedilol with a high-fat meal resulted in increases (~20%) in AUC and C_{max} compared to Carvedilol administered with a standard meal. Decreases in AUC (27%) and C_{max} (43%) were observed when Carvedilol was administered in the fasted state compared to administration after a standard meal. Carvedilol should be taken with food.

Distribution

Carvedilol is more than 98% bound to plasma proteins, primarily with albumin. The plasma-protein binding is independent of concentration over the therapeutic range. Carvedilol is a basic, lipophilic compound with a steady-state volume of distribution of approximately 115L, indicating substantial distribution into extravascular tissues.

Metabolism and Excretion

Carvedilol is extensively metabolized. Following oral administration of Carvedilol, it is accounted for only about 7% of the total radioactivity in plasma as measured by AUC. Less than 2% of the dose was excreted unchanged in the urine. Carvedilol is metabolized primarily by aromatic ring oxidation and glucuronidation. The oxidative metabolites are further metabolized by conjugation via glucuronidation and sulfation. The metabolites of Carvedilol are excreted primarily via the bile into the feces. Demethylation and hydroxylation at the phenol ring produce 3 active metabolites with β-receptor blocking activity.

Compared to Carvedilol, the 3 active metabolites exhibit weak vasodilating activity. Plasma concentrations of the active metabolites are about one-tenth of those observed for Carvedilol and have pharmacokinetics similar to the parent.

Carvedilol undergoes stereoselective first-pass metabolism with plasma levels of R(+)-Carvedilol approximately 2 to 3 times higher than S(-)-Carvedilol following oral administration of Carvedilol ER. Apparent clearance is 90 L/h and 213 L/h for R(+)- and S(-)-Carvedilol, respectively.

The primary P450 enzymes responsible for the metabolism of both R(+) and S(-)-Carvedilol in human liver microsomes were CYP2D6 and CYP2C9 and to a lesser extent CYP3A4, 2C19, 1A2, and 2E1. CYP2D6 is thought to be the major enzyme in the 4'- and 5'-hydroxylation of Carvedilol, with a potential contribution from 3A4. CYP2C9 is thought to be of primary importance in the O-methylation pathway of S(-)-Carvedilol.

Carvedilol is subject to the effects of genetic polymorphism with poor metabolizers of debrisoquin (a marker for cytochrome P450 2D6) exhibiting 2- to 3-fold higher plasma concentrations of R(+)-Carvedilol compared to extensive metabolizers. In contrast, plasma levels of S(-)-Carvedilol are increased only about 20% to 25% in poor metabolizers, indicating this enantiomer is metabolized to a lesser extent by cytochrome P450 2D6 than R(+)-Carvedilol. The pharmacokinetics of Carvedilol do not appear to be different in poor metabolizers of S-mephenytoin (patients deficient in cytochrome P450 2C19).

Heart Failure: Steady-state plasma concentrations of Carvedilol and its enantiomers increased proportionally over the dose range in patients with heart failure.

Geriatric: Plasma levels of Carvedilol average about 50% higher in the elderly compared to young subjects.

Hepatic Impairment: Patients with severe liver impairment (cirrhosis) exhibit a 4- to 7-fold increase in Carvedilol levels. Carvedilol is contraindicated in patients with severe liver impairment.

Renal Impairment: No studies have been performed with Carvedilol ER in patients with renal impairment. Although Carvedilol is metabolized primarily by the liver, plasma concentrations of Carvedilol have been reported to be increased in patients with renal impairment. Consistent with its high degree of plasma protein-binding, Carvedilol does not appear to be cleared significantly by hemodialysis.

INDICATION

Indicated for the treatment of mild to severe chronic heart failure, left ventricular dysfunction following myocardial infarction in clinically stable patients, and hypertension.

CONTRAINDICATION

Carvedilol ER is contraindicated in the following conditions:

Bronchial asthma or related bronchospastic conditions. Deaths from status asthmaticus have been reported following single doses of immediate-release Carvedilol.

Second- or third-degree AV block

Sick sinus syndrome

Severe bradycardia (unless a permanent pacemaker is in place)

Patients with cardiogenic shock or have decompensated heart failure requiring the use of intravenous inotropic therapy. Such patients should first be weaned from intravenous therapy before initiating Carvedilol.

Patients with severe hepatic impairment

Patients with a history of serious hypersensitivity reaction (e.g., Stevens-Johnson syndrome, anaphylactic reaction, angioedema) to Carvedilol or any of the components of the drug product.

WARNINGS AND PRECAUTIONS

Cessation of therapy

Patients with coronary artery disease, who are being treated with Carvedilol, should be advised against abrupt discontinuation of therapy. Severe exacerbation of angina and the occurrence of myocardial infarction and ventricular arrhythmias have been reported in angina patients following the abrupt discontinuation of therapy with β-blockers. The last two complications may occur with or without preceding exacerbation of the angina pectoris. As with other β-blockers, when discontinuation of Carvedilol is planned, the patients should be carefully observed and advised to limit physical activity to a minimum. Carvedilol should be discontinued over 1 to 2 weeks whenever possible. If the angina worsens or acute coronary insufficiency develops, it is recommended that Carvedilol be promptly reinstituted, at least temporarily. Because coronary artery disease is common and may be unrecognized, it may be prudent not to discontinue therapy with Carvedilol abruptly even in patients treated only for hypertension or heart failure.

Bradycardia

Immediate-release Carvedilol causes bradycardia in about 2% of hypertensive patients, 9% of heart failure patients, and 6.5% of myocardial infarction patients with left ventricular dysfunction. There were no reports of bradycardia in clinical trial of Carvedilol ER in hypertension. However, if pulse rate drops below 55 beats/minute, the dosage should be reduced.

Hypotension

Heart failure patients switched to Carvedilol ER or maintained on immediate-release Carvedilol have a 2-fold increase in the combined incidence of hypotension, syncope or dizziness in elderly patients (> 65 years) switched from the highest dose of Carvedilol (25mg twice daily) to Carvedilol ER 80mg once daily. Carvedilol ER in hypertensive patients, syncope was reported. There were no reports of postural hypotension. Starting with a low dose, administration with food, and gradual up-tiration should decrease the likelihood of syncope or excessive hypotension. During initiation of therapy, the patient should be cautioned to avoid situations such as driving or hazardous tasks, where injury could result should syncope occur.

Heart Failure / Fluid Retention

Worsening heart failure or fluid retention may occur during up-titration of Carvedilol. If such symptoms occur, diuretics should be increased and the Carvedilol dose should not be advanced until clinical stability resumes. Occasionally it is necessary to lower the Carvedilol dose or temporarily discontinue it.

Such episodes do not preclude subsequent successful titration of, or a favorable response to Carvedilol. Patients with severe heart failure during the first 3 months were reported to a similar degree with immediate-release Carvedilol and with placebo. When treatment was maintained beyond 3 months, worsening heart failure was reported less frequently in patients treated with Carvedilol. Worsening heart failure observed during long-term therapy is more likely to be related to the patients' underlying disease than to treatment with Carvedilol.

Non-allergic Bronchospasm

Patients with bronchospastic disease (e.g., chronic bronchitis and emphysema) should, in general, not receive β-blockers. Carvedilol may be used with caution, however, in patients who do not respond to, or cannot tolerate, other antihypertensive agents. It is prudent, if Carvedilol is used, to use the smallest effective dose, so that inhibition of endogenous or exogenous β-agonists is minimized.

Patients with heart failure, patients with bronchospastic disease were enrolled if they did not require oral or inhaled medication to treat their bronchospastic disease. In such patients, it is recommended that Carvedilol be used with caution. The dosing recommendations should be followed closely and the dose should be lowered if any evidence of bronchospasm is observed during up-titration.

Glycemic Control in Type 2 Diabetes

In general, β-blockers may mask some of the manifestations of hypoglycemia, particularly tachycardia. Nonselective β-blockers may potentiate insulin-induced hypoglycemia and delay recovery of serum glucose levels. Patients subject to spontaneous hypoglycemia, or diabetic patients receiving insulin or oral hypoglycemic agents, should be cautioned about these possibilities.

In heart failure patients with diabetes, Carvedilol therapy may lead to worsening hyperglycemia, which responds to intensification of hypoglycemic therapy. It is recommended that blood glucose be monitored when Carvedilol dosing is initiated, adjusted, or discontinued. Studies designed to examine the effects of Carvedilol on glycemic control in patients with diabetes and heart failure have not been conducted.

Peripheral Vascular Disease

β-blockers can precipitate or aggravate symptoms of arterial insufficiency in patients with peripheral vascular disease. Caution should be exercised in such individuals.

Deterioration of Renal Function

Rarely, use of Carvedilol in patients with heart failure has resulted in deterioration of renal function. Patients at risk appear to be those with low blood pressure (systolic blood pressure <100 mmHg), ischemic heart disease and diffuse vascular disease, and/or underlying renal insufficiency. Renal function has returned to baseline when Carvedilol was stopped. In patients with these risk factors it is recommended that renal function be monitored during up-titration of Carvedilol and the drug discontinued or dosage reduced if worsening of renal function occurs.

Major Surgery

Chronically administered beta-blocking therapy should not be routinely withdrawn prior to major surgery; however, the impaired ability of the heart to respond to reflex adrenergic stimuli may augment the risks of general anesthesia and surgical procedures.

Thyrotoxicosis

β-adrenergic blockade may mask clinical signs of hyperthyroidism, such as tachycardia. Abrupt withdrawal of β-blockade may be followed by an exacerbation of the symptoms of hyperthyroidism or may precipitate thyroid storm.

Pheochromocytoma

In patients with pheochromocytoma, a α-blocking agent should be initiated prior to the use of any β-blocking agent. Although Carvedilol has both α- and β-blocking pharmacologic activities, there has been no experience with its use in this condition. Therefore, caution should be taken in the administration of Carvedilol to patients suspected of having pheochromocytoma.

Prinzmetal's Variant Angina

Agents with non-selective β-blocking activity may provoke chest pain in patients with Prinzmetal's variant angina. There has been no clinical experience with Carvedilol in these patients although the α-blocking activity may prevent such symptoms. However, caution should be taken in the administration of Carvedilol to patients suspected of having Prinzmetal's variant angina.

Risk of Anaphylactic Reaction

While taking β-blockers, patients with a history of severe anaphylactic reaction to a variety of allergens may be more reactive to repeated challenge; accidental, diagnostic, or therapeutic. Such patients may be unresponsive to the usual doses of epinephrine used to treat allergic reaction.

Intraoperative Floppy Iris Syndrome

Intraoperative Floppy Iris Syndrome (IFIS) has been observed during cataract surgery in some patients treated with alpha-1 blockers (Carvedilol is an alpha/beta blocker). This variant of small pupil syndrome is characterized by the combination of a flaccid iris that

billows in response to intraoperative irrigation currents, progressive intraoperative miosis despite preoperative dilation with standard mydriatic drugs, and potential prolapsed of the iris toward the phacoemulsification incisions. The patient's ophthalmologist should be prepared for possible modifications to the surgical technique, such as utilization of iris hooks, iris dilator rings, or viscoelastic substances. There does not appear to be a benefit of stopping alpha-1 blocker therapy prior to cataract surgery.

DOSAGE AND ADMINISTRATION

Carvedilol is an extended release preparation intended for once-daily administration in the morning with food. Its contents should not be crushed, chewed, or taken in divided doses. Patients controlled with immediate-release Carvedilol alone or in combination with other medications may be switched to Carvedilol ER based on the total daily doses shown below.

Dosing Conversion

Daily Dose of Immediate-Release Carvedilol Tablets	Daily Dose of Carvedilol ER Tablets/Capsules*
6.25 mg (3.125 mg twice daily)	10 mg Tablet once daily
12.5 mg (6.25 mg twice daily)	20 mg Tablet once daily
25 mg (12.5 mg twice daily)	40 mg Capsule once daily
50mg (25mg twice daily)	80 mg Capsule once daily

* When switching from Carvedilol 12.5 mg or 25mg twice daily, a starting dose of Carvedilol ER 20mg or 40mg once daily, respectively, may be warranted for elderly patients or those at increased risk of hypotension, dizziness, or syncope. Subsequent titration to higher doses should, as appropriate, be made after an interval of at least 2 weeks. Or as prescribed by the physician.

Heart Failure

DOSAGE MUST BE INDIVIDUALIZED AND CLOSELY MONITORED BY A PHYSICIAN DURING UP-TITRATION. Prior to initiation Carvedilol, it is recommended that fluid retention be minimized. The recommended starting dose of Carvedilol is 10mg once daily for 2 weeks. Patients who tolerate a dose of 10mg once daily may have their dose increased to 20, 40, and 80 mg over successive intervals of at least 2 weeks. Patients should be maintained on lower doses if higher doses are not tolerated. The dose of Carvedilol should not be increased until symptoms of worsening heart failure or vasodilation have been stabilized.

Fluid retention (with or without transient worsening heart failure symptoms) should be treated by an increase in the dose of diuretics.

The dose of Carvedilol should be reduced if patients experience bradycardia (heart rate <55 beats/minute).

Episodes of dizziness or fluid retention during initiation of Carvedilol can generally be managed without discontinuation of treatment and do not preclude subsequent successful titration of, or a favorable response to, Carvedilol.

Left Ventricular Dysfunction Following Myocardial Infarction

DOSAGE MUST BE INDIVIDUALIZED AND CLOSELY MONITORED BY A PHYSICIAN DURING UP-TITRATION. Treatment with Carvedilol may be started as an inpatient or outpatient and should be started after the patient is hemodynamically stable and fluid retention has been minimized. It is recommended that Carvedilol be started at 20mg once daily and increased after 3 to 10 days, based on tolerability, to 40mg once daily, then again to the target dose of 80mg once daily. A lower starting dose may be used (10mg once daily) and/or the rate of up-titration may be slowed if clinically indicated (e.g., due to low blood pressure or heart rate, or fluid retention).

Patients should be maintained on lower doses if higher doses are not tolerated. The recommended dosing regimen need not be altered in patients who received treatment with an IV or oral β-blocker during the acute phase of the myocardial infarction.

Hypertension

DOSAGE MUST BE INDIVIDUALIZED. The recommended starting dose of Carvedilol is 20mg once daily. If this dose is tolerated, using standing systolic pressure measured about one hour after dosing as a guide, the dose should be maintained for 7 to 14 days, and then increased to 40mg once daily if needed, based on trough blood pressure, again using standing systolic pressure one hour after dosing as a guide for tolerance. This dose should also be maintained for 7 to 14 days and then can be adjusted upward to 80mg once daily if tolerated and needed. Total daily dose should not exceed 80mg.

Geriatric use

When switching elderly patients (65 years of age or older) who are taking the higher doses of immediate-release Carvedilol (25mg twice daily) to Carvedilol ER, a lower starting dose (40mg) of Carvedilol ER is recommended to minimize the potential for dizziness, syncope, or hypotension. Patients who have switched and who tolerate Carvedilol ER should, as appropriate, have their dose increased after an interval of at least 2 weeks.

Hepatic Impairment

Carvedilol ER should not be given with severe hepatic impairment.

DRUG INTERACTIONS

CYP2D6 Inhibitors and Poor Metabolizers

Interactions of Carvedilol with potent inhibitors of CYP2D6 isoenzyme (such as quinidine, fluoxetine, paroxetine, and propafenone) have not been studied, but these drugs would be expected to increase blood levels of the R(+) enantiomer of Carvedilol. Retrospective analysis of side effects showed that poor 2D6 metabolizers had a higher rate of dizziness during up-titration, presumably resulting from vasodilating effects of the higher concentrations of the α-blocking R(+) enantiomer.

Hypotensive agents

Patients taking both agents with β-blocking properties and a drug that can deplete catecholamines (e.g., reserpine and monoamine oxidase inhibitors) should be observed closely for signs of hypotension and/or severe bradycardia.

Concomitant administration of clonidine with agents with β-blocking properties may potentiate blood-pressure- and heart-rate-lowering effects. When concomitant treatment with agents with β-blocking properties and clonidine is to be terminated, the β-blocking agent should be discontinued first. Clonidine therapy can then be discontinued several days later by gradually decreasing the dosage.

Cyclosporine

Modest increases in mean trough cyclosporine concentrations were observed following initiation of Carvedilol treatment in renal transplant patients suffering from chronic vascular rejection. It is recommended that cyclosporine concentrations be monitored closely after initiation of Carvedilol therapy and that the dose of cyclosporine be adjusted as appropriate.

Digitalis Glycosides

Both digitalis glycosides and β-blockers slow atrioventricular conduction and decrease heart rate. Concomitant use can increase the risk of bradycardia. Digoxin concentrations are increased by about 15% when digoxin and Carvedilol are administered concomitantly. Therefore, increased monitoring of digoxin is recommended when initiating, adjusting, or discontinuing Carvedilol.

Inducers/inhibitors of Hepatic Metabolism

Rifampicin reduced plasma concentration of Carvedilol by about 70%. Cimetidine increased AUC by about 30% but caused no change in C_{max}.

Amiodarone

The concomitant administration of amiodarone or other CYP2C9 inhibitors such as fluconazole with Carvedilol may enhance the β-blocking properties of Carvedilol resulting in further slowing of the heart rate or cardiac conduction. Patients should be observed for signs of bradycardia or heart block, particularly when one agent is added to pre-existing treatment with the other.

Calcium Channel Blockers

Conduction disturbance (rarely with hemodynamic compromise) has been observed when Carvedilol is co-administered with diltiazem. As with other agents with β-blocking properties, if Carvedilol is to be administered with calcium channel blockers of the verapamil or diltiazem type, it is recommended that ECG and blood pressure be monitored.

Insulin or Oral Hypoglycemics

Agents with β-blocking properties may enhance the blood-sugar-reducing effect of insulin and oral hypoglycemics. Therefore, in patients taking insulin or oral hypoglycemics, regular monitoring of blood glucose is recommended.

Proton Pump Inhibitors

There is no clinically meaningful increase in AUC and C_{max} with concomitant administration of Carvedilol ER with pantoprazole.

Anesthesia

If treatment with Carvedilol is to be continued perioperatively, particular care should be taken when anesthetic agents which depress myocardial infarction, such as ether, cyclopropane, and trichloroethylene, are used.

Pregnancy

There are no adequate and well-controlled studies in pregnant women. Carvedilol should be used during pregnancy only if the potential benefit justifies the potential risk to the fetus.

Nursing Mothers

It is not known whether this drug is excreted in human milk but because many drugs are excreted in human milk and the potential for serious adverse reactions in nursing infants from β-blockers, especially bradycardia, a decision should be made whether to discontinue nursing or to discontinue the drug, taking into account the importance of the drug to the mother. The effects of other α- and β-blocking agents have included perinatal and neonatal distress.

Pediatric Use

Effectiveness of Carvedilol in patients younger than 18 years of age has not been established.

Geriatric Use

Other reported clinical experience has not identified differences in responses between the elderly and younger patients, but greater sensitivity of some older individuals cannot be ruled out.

ADVERSE EFFECTS

Adverse events occurring more frequently with Carvedilol ER in patients with hypertension are nasopharyngitis, dizziness, nausea, edema, peripheral, nasal congestion, paresthesia, sinus congestion, diarrhea, and insomnia.

Liver function abnormalities, reversible on stopping treatment with Carvedilol, have been reported rarely. Carvedilol is extensively metabolized in the liver and is not recommended in patients with hepatic impairment. Acute renal failure and renal abnormalities have been reported in patients with heart failure who also suffered from diffuse vascular disease and/or renal impairment. The risk of hypotension may be reduced by taking Carvedilol with food to decrease the rate of absorption.

Effects on the liver

Pruritus and elevated serum transaminase concentrations occurred in a man who had been taking Carvedilol for 6 months. Carvedilol was discontinued and the liver function tests returned to normal within 3 weeks. However, pruritus recurred when the patient was started on metoprolol approximately 1 year later.

Laboratory Abnormalities

Reversible elevations in serum transaminases (ALT or AST) have been observed during treatment with Carvedilol. Rates of transaminase elevations (2- to 3-times the upper limit of normal) observed in patients treated with Carvedilol.

Carvedilol therapy has not been associated with clinically significant changes in serum potassium, total triglycerides, total cholesterol, HDL cholesterol, uric acid, blood urea nitrogen, or creatinine.

OVERDOSE AND TREATMENT

Signs and Symptoms: Overdosage may cause severe hypotension, bradycardia, cardiac insufficiency, cardiogenic shock, and cardiac arrest. Respiratory problems, bronchospasms, vomiting, lapses of consciousness, and generalized seizures may also occur.

Treatment: The patient should be placed in a supine position and, where necessary, kept under observation and treated under intensive-care conditions. Gastric lavage or pharmacologically induced emesis may be used shortly after ingestion. The following agents may be administered:

For excessive tachycardia: Atropine, 2mg IV.

To support cardiovascular function: Glucagon, 5 to 10mg IV rapidly over 30 seconds, followed by a continuous infusion of 5mg/hour; sympathomimetics (dobutamine, isoprenaline, and adrenaline) at doses according to body weight and effect.

If peripheral vasodilation dominates, it may be necessary to administer adrenaline or noradrenaline with continuous monitoring of circulatory conditions. For therapy-resistant bradycardia, pacemaker therapy should be performed. For bronchospasm, β-sympathomimetics (as aerosol or IV) or aminophylline IV should be given. In the event of seizures, slow IV injection or diazepam or clonazepam is recommended.

In the event of severe intoxication where there are symptoms of shock, treatment with antidotes must be continued for a sufficiently long period of time consistent with the 7 to 10-hour half-life of Carvedilol.

There is no experience of overdosage with Carvedilol ER. Cases of overdosage with Carvedilol alone or in combination with other drugs have been reported. Quantities ingested in some cases exceeded 1,000 milligrams. Symptoms experienced included low blood pressure and heart rate. Standard supportive treatment was provided and individuals recovered.

STORE AT TEMPERATURES NOT EXCEEDING 30°C. PROTECT FROM LIGHT.

CAUTION

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

AVAILABILITY

Carvedilol (Carveta XR) 10mg Alu-alu Blister pack x 10's, Box of 30 Extended-Release Capsules

Carvedilol (Carveta XR) 20mg Alu-alu Blister pack x 10's, Box of 30 Extended-Release Capsules

Carvedilol (Carveta XR) 40mg Alu-alu Blister pack x 10's, Box of 30 Extended-Release Capsules

FOR SUSPECTED ADVERSE DRUG REACTION, REPORT TO THE FDA: www.fda.gov.ph *Seek medical attention immediately at the first sign of any adverse drug reaction shall appear.*

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Registration No.: 40mg – DR-XY44156
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