PRANDIAL M Tablets (Voglibose + Metformin hydrochloride)

Composition

PRANDIAL M 0.2

Each uncoated bilayer tablet contains

Metformin hydrochloride IP……500 mg

Voglibose……0.2 mg

PRANDIAL M 0.3

Each uncoated bilayer tablet contains

Metformin hydrochloride IP……500 mg

Voglibose……0.3 mg

Dosage Form

Tablets

Description

PRANDIAL M contains two oral antihyperglycaemic drugs, voglibose and metformin hydrochloride, used in the management of type 2 diabetes mellitus. Vogliboseinhibits the activity of alpha glucosidases in the intestine, thereby delaying the digestion and absorption of carbohydrates, resulting in improvement of postprandial hyperglycaemia. The primary mechanism of action of metformin hydrochloride is to reduce fasting plasma glucose by decreasing hepatic glucose production and intestinal absorption of glucose, and improving insulin sensitivity by increasing peripheral glucose uptake and utilization.

Hence, the combination of voglibose and metformin hydrochloride complement each other and provide better glycaemic control in management of type 2 diabetes and may help prevent its associated macrovascular and microvascular complications.

Pharmacology

Pharmacodynamics

Voglibose

Voglibose is an alpha glucosidase inhibitor which inhibits the activity of alpha glucosidases that

catalyse the decomposition of disaccharides into monosaccharides in the intestine, thereby delaying the digestion and absorption of carbohydrates, resulting in improvement of postprandial hyperglycaemia.

Metformin Hydrochloride

Metformin improves glucose tolerance in patients with type 2 diabetes (NIDDM), lowering both basal and postprandial plasma glucose. Metformin is not chemically or pharmacologically related to sulphonylureas, thiazolidinediones, or alpha-glucosidase inhibitors. Metformin decreases hepatic glucose production, decreases intestinal absorption of glucose, and improves insulin sensitivity by increasing peripheral glucose uptake and utilization. Unlike sulphonylureas, metformin does not produce hypoglycaemia in either patients with type 2 diabetes or normal subjects and does not cause hyperinsulinaemia. With metformin therapy, insulin secretion remains unchanged while fasting insulin levels and day-long plasma insulin response may actually decrease.

Pharmacokinetics

Absorption& Distribution

Voglibose

Voglibose is poorly absorbed after oral doses. Plasma concentrations after oral doses have usually been undetectable. After an 80 mg dose (substantially higher than recommended dose), peak plasma levels of about 20 ng/mL were observed in 1-1.5 hours.

Metformin Hydrochloride

The absolute bioavailability of a metformin 500-mg tablet given under fasting conditions is approximately 50-60%. Studies using single oral doses of metformin 500 mg to 1500 mg, and 850 mg to 2550 mg, indicate that there is a lack of dose proportionality with increasing doses, which is due to decreased absorption rather than an alteration in elimination. Food decreases the extent of and slightly delays the absorption of metformin, as shown by approximately a 40% lower mean peak plasma concentration (C_{max}), a 25% lower area under the plasma concentration versus time curve (AUC), and a 35 minute prolongation of time to peak plasma concentration (T_{max}) following administration of a single 850-mg tablet of metformin with food, compared to the same tablet strength administered fasting. The clinical relevance of these decreases is unknown.

The apparent volume of distribution (V/F) of metformin following single oral doses of metformin immediate-release 850 mg averaged 654 ± 358 L. Metformin is negligibly bound to plasma proteins, in contrast to sulphonylureas, which are more than 90% protein bound. Metformin partitions into erythrocytes, most likely as a function of time. At usual clinical doses and dosing schedules of metforminimmediate-release, steady state plasma concentrations of metformin are reached within 24-48 hours and are generally <1 µg/mL. During controlled clinical trials of immediate-release metformin, maximum metformin plasma levels did not exceed 5 µg/mL, even at maximum doses.

Metabolism & Excretion

Voglibose

Voglibose is metabolized negligibly and rapidly excreted. When voglibose tablets were repeatedly administered to healthy male adults in a single dose of 0.2 mg, three times a day, for 7 consecutive days, voglibose was not detected in plasma or urine. Also, on administration of voglibose to 10 healthy male subjects in a single dose of 2 mg, voglibose was not detected in plasma or urine.

Metformin Hydrochloride

Intravenous single-dose studies in normal subjects demonstrate that metformin is excreted unchanged in the urine and does not undergo hepatic metabolism (no metabolites have been identified in humans) nor biliary excretion. Renal clearance of metformin is approximately 3.5 times greater than creatinine clearance, which indicates that tubular secretion is the major route of metformin elimination. Following oral administration, approximately 90% of the absorbed drug is eliminated via the renal route within the first 24 hours, with a plasma elimination half-life of approximately 6.2 hours. In blood, the elimination half-life is approximately 17.6 hours, suggesting that the erythrocyte mass may be a compartment of distribution.

Special Populations

Renal Impairment

No pharmacokinetic studies for voglibose have been conducted in subjects with renal insufficiency.In patients with decreased renal function (based on creatinine clearance), the plasma and blood half-life of metformin is prolonged and the renal clearance is decreased in proportion to the decrease in creatinine clearance.

Hepatic Impairment

Rises in liver enzymes have been observed in up to 20% of patients during voglibose therapy. No pharmacokinetic studies for metformin have been conducted in subjects with hepatic insufficiency.

Geriatric

No pharmacokinetic data of voglibose in elderly population is available.Pharmacokinetic studies of metformin hydrochloride in healthy elderly subjects suggest that total plasma clearance is decreased, the half-life is prolonged, and C_{max} is increased, compared to healthy young subjects. From these data, it appears that the change in metformin pharmacokinetics with aging is primarily accounted for by a change in renal function.

Paediatric

No pharmacokinetic data ofvoglibose in paediatric population is available.After administration of a single oral metformin hydrochloride 500 mg tablet with food, geometric mean metformin C_{max} and AUC differed less than 5% between paediatric type 2 diabetes patients (12 to 16 years of age) and gender- and weight-matched healthy adults (20 to 45 years of age), all with normal renal function.

Indications

PRANDIAL M is indicated as an adjunct to diet and exercise to improve glycaemic control in patients with type 2 diabetes who are already treated with a combination of voglibose and metformin given separately or whose diabetes is not adequately controlled with metformin alone, or for those patients who have initially responded to voglibose alone and require additional glycaemic control.

Dosage and Administration

Dosage should be individualised on the basis of both effectiveness and tolerance. The initial recommended dose is one tablet of **PRANDIAL M 0.2**three times daily just before each meal.In case of inadequate effect, the dose may be increased up to 1 tablet of **PRANDIAL M 0.3**, three times

daily just before each meal under close observation of the course of the disease.

Patients Switching from Combination Therapy of Voglibose Plus Metformin Administered as Separate Tablets

PRANDIAL M 0.2 or **PRANDIAL M 0.3** may be initiated based on the dose of voglibose and metformin already being taken.

During administration of this combination, disease progression should be closely observed with monitoring of blood glucose levels at regular intervals.

Contraindications

- Renal disease or renal dysfunction (e.g., as suggested by serum creatinine levels ³ 1.5 mg/dL [males], ³ 1.4 mg/dL [females] or abnormal creatinine clearance), which may also result from conditions such as cardiovascular collapse (shock), acute myocardial infarction, and septicaemia.
- Known hypersensitivity to metformin, voglibose or any of the components of this product.
- Acute or chronic metabolic acidosis, including diabetic ketoacidosis, with or without coma. Diabetic ketoacidosis should be treated with insulin.
- Patients with severe infection, before and after operation or with serious trauma.
- Patients with gastrointestinal obstruction or predisposed to it.

Patients undergoing radiologic studies involving intravascular administration of iodinated contrast materials should temporarily discontinue taking this combination, because use may result in acute alteration of renal function.

Warnings and Precautions

Loss of Control of Blood Glucose

When a patient stabilised on any diabetic regimen is exposed to stress such as fever, trauma, infection, or surgery, a temporary loss of glycaemic control may occur. At such times, it may be necessary to withhold oral antidiabetic agents and temporarily administer insulin. This combinationmay be reinstituted after the acute episode is resolved.

Hypoxic States

Cardiovascular collapse (shock) from whatever cause, acute congestive heart failure, acute myocardial infarction and other conditions characterised by hypoxaemia have been associated with lactic acidosis and may also cause prerenalazotaemia. When such events occur in patients on **PRANDIAL M**, the drug should be promptly discontinued.

Surgical Procedures

PRANDIAL M should be temporarily suspended for any surgical procedure (except minor procedures not associated with restricted intake of food and fluids) and should not be restarted until the patient’s oral intake has resumed and renal function has been evaluated as normal.

Alcohol Intake

Alcohol is known to potentiate the effect of metformin on lactate metabolism. Patients, therefore, should be warned against excessive alcohol intake, acute or chronic, while receiving **PRANDIAL M**.

Change in Clinical Status of Patients with Previously Controlled Type 2 Diabetes

A patient with type 2 diabetes with previously well controlled type 2 diabetes who develops laboratory abnormalities or clinical illness (especially vague and poorly defined illness) should be evaluated promptly for evidence of ketoacidosis or lactic acidosis. Evaluation should include serum electrolytes and ketones, blood glucose and, if indicated, blood pH, lactate, pyruvate, and metformin levels. If acidosis of either form occurs, **PRANDIAL M** must be stopped immediately and other appropriate corrective measures initiated.

Hypoglycaemia

Hypoglycaemia does not occur in patients receiving **PRANDIAL M** alone under usual circumstances of use, but could occur when caloric intake is deficient, when strenuous exercise is not compensated by caloric supplementation, or during concomitant use with other glucose-lowering agents (such as sulphonylureas and insulin) or ethanol. Elderly, debilitated, or malnourished patients and those with adrenal or pituitary insufficiency or alcohol intoxication are particularly susceptible to hypoglycaemic effects. Hypoglycaemia may be difficult to recognize in the elderly, and in people who are taking beta-adrenergic blocking drugs. Patients should be instructed and explained to recognize hypoglycaemic symptoms and its management.

Vitamin B_{12}

In controlled clinical trials of metformin of 29 weeks duration, a decrease to subnormal levels of previously normal serum vitamin B_{12} levels, without clinical manifestations, was observed in approximately 7% of patients. Such decrease, possibly due to interference with B_{12} absorption from the B_{12} intrinsic factor complex, however is very rarely associated with anaemia and appears to be rapidly reversible with discontinuation of metformin or vitamin B_{12} supplementation. Measurement of haematologic parameters on an annual basis is advised in patients on metformin and any apparent abnormalities should be appropriately investigated and managed.

Certain individuals (those with inadequate vitamin B_{12} or calcium intake or absorption) appear to be predisposed to developing subnormal vitamin B_{12} levels. In these patients, routine serum vitamin B_{12} measurements at 2- or 3- year intervals may be useful.

Macrovascular Outcomes

There have been no clinical studies establishing conclusive evidence of macrovascular risk reduction with voglibose, metformin or any other anti-diabetic drug.

Lactic Acidosis

Lactic acidosis is a rare, but serious, metabolic complication that can occur due to metformin accumulation during treatment with metformin; when it occurs, it is fatal in approximately 50% of cases. Lactic acidosis may also occur in association with a number of pathophysiologic conditions, including diabetes mellitus, and whenever there is significant tissue hypo perfusion and hypoxaemia. Lactic acidosis is characterised by elevated blood lactate levels (>5 mmol/L), decreased blood pH, electrolyte disturbances with an increased anion gap, and an increased lactate/pyruvate ratio. When metformin is implicated as the cause of lactic acidosis, metformin plasma levels >5 µg/mL are generally found.

The reported incidence of lactic acidosis in patients receiving metformin hydrochloride is very low

(approximately 0.03 cases/1000 patient-years, with approximately 0.015 fatal cases/1000 patient-years). In more than 20,000 patient-years exposure to metformin in clinical trials, there were no reports of lactic acidosis. Reported cases have occurred primarily in diabetes patients with significant renal insufficiency, including both intrinsic renal disease and renal hypo perfusion, often in the setting of multiple concomitant medical/surgical problems and multiple concomitant medications. Patients with congestive heart failure requiring pharmacologic management, in particular those with unstable or acute congestive heart failure who are at risk of hypo perfusion and hypoxaemia, are at increased risk of lactic acidosis. The risk of lactic acidosis increases with the degree of renal dysfunction and the patient's age. The risk of lactic acidosis may, therefore, be significantly decreased by regular monitoring of renal function in patients taking metformin and by use of the minimum effective dose of metformin. In particular, treatment of the elderly should be accompanied by careful monitoring of renal function. Metformin treatment should not be initiated in patients ≥80 years of age unless measurement of creatinine clearance demonstrates that renal function is not reduced, as these patients are more susceptible to developing lactic acidosis. In addition, metformin should be promptly withheld in the presence of any condition associated with hypoxaemia, dehydration, or sepsis. Because impaired hepatic function may significantly limit the ability to clear lactate, metformin should generally be avoided in patients with clinical or laboratory evidence of hepatic disease. Patients should be cautioned against excessive alcohol intake, either acute or chronic, when taking metformin, since alcohol potentiates the effects of metformin hydrochloride on lactate metabolism. In addition, metformin should be temporarily discontinued prior to any intravascular radiocontrast study and for any surgical procedure.

The onset of lactic acidosis often is subtle, and accompanied only by nonspecific symptoms such as malaise, myalgias, respiratory distress, increasing somnolence, and nonspecific abdominal distress. There may be associated hypothermia, hypotension, and resistant bradyarrhythmias with more marked acidosis. The patient and the patient's physician must be aware of the possible importance of such symptoms and the patient should be instructed to notify the physician immediately if they occur. Metformin should be withdrawn until the situation is clarified. Serum electrolytes, ketones, blood glucose, and if indicated, blood pH, lactate levels, and even blood metformin levels may be useful. Once a patient is stabilised on any dose level of metformin, gastrointestinal symptoms, which are common during initiation of therapy, are unlikely to be drug related. Later occurrence of gastrointestinal symptoms could be due to lactic acidosis or other serious disease.

Levels of fasting venous plasma lactate above the upper limit of normal but less than 5 mmol/L in patients taking metformin do not necessarily indicate impending lactic acidosis and may be explainable by other mechanisms, such as poorly controlled diabetes or obesity, vigorous physical activity, or technical problems in sample handling.

Lactic acidosis should be suspected in any diabetes patient with metabolic acidosis lacking evidence of ketoacidosis (ketonuria and ketonemia).

Lactic acidosis is a medical emergency that must be treated in a hospital setting. In a patient with lactic acidosis who is taking metformin, the drug should be discontinued immediately and general supportive measures promptly instituted. Because metformin hydrochloride is dialyzable (with a clearance of up to 170 mL/min under good hemodynamic conditions), prompt haemodialysis is recommended to correct the acidosis and remove the accumulated metformin. Such management often results in prompt reversal of symptoms and recovery.

Drug Interactions

Cationic Drugs

Certain medications used concomitantly with metformin may increase the risk of lactic acidosis. Cationic drugs that are eliminated by renal tubular secretions (e.g: amiloride, cimetidine, digoxin, morphine, procainamide, quinidine, quinine, ranitidine, triamterene, trimethoprim or vancomycin) may decrease metformin elimination by competing for common renal tubular transport systems. Hence, careful patient monitoring and dose adjustment of **PRANDIAL M** and/or interfering drug is recommended in patients who are taking cationic medications that are excreted via the proximal renal tubular secretory system.

Furosemide

A single-dose, metformin-furosemide drug interaction study in healthy subjects demonstrated that pharmacokinetic parameters of both compounds were affected by co-administration. Furosemide increased the metformin plasma and blood C_{max} by 22% and blood AUC by 15%, without any significant change in metformin renal clearance. When administered with metformin, the C_{max} and AUC of furosemide were 31% and 12% smaller, respectively, than when administered alone, and the terminal half-life was decreased by 32%, without any significant change in furosemide renal clearance. No information is available about the interaction of metformin and furosemide when co-administered chronically.

Nifedipine

Nifedipine appears to enhance the absorption of metformin, it increases plasma metformin C_{max} and AUC by 20% and 9% respectively and increases the amount of metformin excreted in the urine. Metformin has minimal effects on nifedipine.

Danazol

If the use of this active substance cannot be avoided, warn the patients and emphasise the importance of urine and blood glucose monitoring. It may be necessary to adjust the dose of voglibose and metformin during and after treatment with danazol.

Salicylates

If salicylates are administered or discontinued in patients receiving oral antidiabetic agents, patients should be monitored for hypoglycaemia or loss of blood glucose control.

Thiazide

Interactions between thiazide diuretics and oral antidiabetic agents decreases insulin sensitivity thereby leading to glucose intolerance and hyperglycaemia, thus leading to a loss of diabetic control. Hence diabetic patients should be monitored closely.

Other

Concomitant administration of angiotensin enzyme inhibitors (captopril, enalapril), other antidiabetic drugs (insulin, acarbose) beta-blockers, fluconazole, histamine (H_2) receptor antagonist, monoamine oxidase inhibitors (MAOIs), sulphonamides and non-steroidal anti-inflammatory agents increase sensitivity to insulin and potentiation of blood glucose lowering effect and thus, in some instances, hypoglycaemia may occur . Certain drugs tend to produce hyperglycaemia and may lead to loss of glycaemic control. These drugs include corticosteroids, phenothiazines, thyroid products, estrogens, oral contraceptives, phenytoin, nicotinic acid, sympathomimetics, calcium channel blocking drugs, quinolones and isoniazid. Patients receiving these drugs should be closely monitored for loss of diabetic control when therapy is instituted or discontinued. Dosage of the oral antidiabetic

agents may need to be reduced.

Drugs Enhancing** (beta-blockers, salicylic acid preparations, MAOs, fibrate derivatives) **or Diminishing** (epinephrine, adrenocortical hormone, thyroid hormone etc.) **the Hypoglycaemic Action of Anti-diabetic Drugs

When **PRANDIAL M** is administered concomitantly with drugs that enhance or diminish the hypoglycaemic action of anti-diabetic drugs, caution should be taken as this might additionally delay the action of voglibose on the absorption of carbohydrates.

Warfarin

Metformin and voglibose do not affect the pharmacokinetics of warfarin, hence **PRANDIAL M**can be safely administered along with warfarin.

Renal Impairment

Metformin is known to be substantially excreted by the kidney and the risk of metformin accumulation and lactic acidosis increases with the degree of impairment of renal function. Thus, patients with serum creatinine levels above the upper limit of normal for their age should not receive **PRANDIAL M**. In patients with advanced age, **PRANDIAL M** should be carefully titrated to establish the minimum dose for adequate glycaemic effect, because aging is associated with reduced renal function. In elderly patients, particularly those ≥80 years of age, renal function should be monitored regularly and, generally, **PRANDIAL M** should not be titrated to the maximum dose. Before initiation of **PRANDIAL M** and at least annually thereafter, renal function should be assessed and verified as normal. In patients in whom development of renal dysfunction is anticipated, renal function should be assessed more frequently and **PRANDIAL M** discontinued if evidence of renal impairment is present.

Hepatic Impairment

Since impaired hepatic function has been associated with some cases of lactic acidosis, metformin should generally be avoided in patients with clinical or laboratory evidence of hepatic disease.Hence, **PRANDIAL M** should be avoided in patients with hepatic impairment.

Pregnancy

Recent information suggests that abnormal blood glucose levels during pregnancy are associated with the higher incidence of congenital abnormalities. There are no adequate and well-controlled studies in pregnant women with metformin. Metformin was not teratogenic in rats and rabbits at doses up to 600 mg/kg/day.The safety and effectiveness of voglibose in pregnant women has not been established. Animal studies have shown that voglibose is transferred to the foetus. **PRANDIAL M** should be used during pregnancy only if the potential benefit justifies the potential risk to the foetus. Most experts suggest insulin be used to maintain the blood glucose levels as close to normal as possible.

Lactation

Studies in lactating rats show that metformin is excreted into milk and reaches levels comparable to those in plasma. Similar studies have not been conducted on nursing mothers. Animal studies have shown a suppressive action of voglibose on body weight increase in newborns, mainly due to suppression of milk production resulting from inhibition of carbohydrate absorption in mother

animals. Nursing should be discontinued if **PRANDIAL M** has to be administered. If the use of this combination is discontinued, and if the diet alone is inadequate for controlling blood glucose, insulin therapy should be considered.

Paediatric Use

The safety and effectiveness of metformin for the treatment of type 2 diabetes have been established in paediatricpatients ages 10 to 16 years (studies have not been conducted in paediatric patients below the age of 10 years). The safety and effectiveness of voglibose in children has not been established. Hence **PRANDIAL M** should not be considered in this population.

Geriatric Use

Metformin is known to be excreted through the kidneys and because risk of serious adverse reactions to the drug is greater in patients with impaired renal function, **PRANDIAL M** should be used only in patients with normal renal function. Because aging is associated with reduced renal function, the use of **PRANDIAL M** should be with caution as age increases. Care should be taken in the dose selection and regular renal function be monitored. The administration of **PRANDIAL M** should be initiated at a lower dose in elderly patients and generally, should not be titrated to the maximum dose. The combination should be carefully administered under close observation of the course of disease conditions, with careful attention to the blood sugar level and the onset of gastrointestinal symptoms.

Effects on Ability to Drive and Use Machines

This combination containing voglibose and metformin immediate release does not cause hypoglycaemia and therefore has no effect on the ability to drive or to use machines. However, patients should be alerted to the risk of hypoglycaemia when **PRANDIAL M** is used in combination with other antidiabetic agents (e.g. sulphonylureas, insulin, or meglinitides).

Undesirable Effects

In a US double-blind clinical study of metformin in patients with type 2 diabetes, a total of 141 patients received metformin therapy (up to 2550 mg per day) and 145 patients received placebo. Adverse reactions reported in greater than 5% of the metformin patients, and those were more common in metformin- than placebo-treated patients, are listed in table 1.

Table 1: Most common adverse reactions (>5.0 percent) in placebo-controlled studies of metformin hydrochloride

Adverse Reaction	Metformin hydrochloride (n=141)	Placebo (n=145)
	% of Patients	
Diarrhoea	53.2	11.7
Nausea/Vomiting	25.5	8.3
Flatulence	12.1	5.5
Asthenia	9.2	5.5
Indigestion	7.1	4.1
Abdominal Discomfort	6.4	4.8

Headache	5.7	4.8

Diarrhoea led to discontinuation of study medication in 6% of patients treated with metformin. Additionally, the following adverse reactions were reported in ≥1% to ≤5% of metformin patients and were more commonly reported with metformin than placebo: abnormal stools, hypoglycaemia, myalgia, lightheaded, dyspnea, nail disorder, rash, sweating increased, taste disorder, chest discomfort, chills, flu syndrome, flushing, palpitation.

Very Rare Effects

- Isolated reports of liver function tests abnormalities or hepatitis resolving upon metformin discontinuation
- Skin reactions such as erythema, pruritis, urticaria

Paediatric Patients

In clinical trials with metformin hydrochloride in paediatric patients with type 2 diabetes, the profile of adverse reactions was similar to that observed in adults.

The gastrointestinal adverse effects like diarrhoea, loose stools, abdominal pain, constipation, anorexia, nausea, vomiting or heartburn may occur with the use of voglibose. Abdominal swelling, increased flatus, and intestinal obstruction like symptoms due to an increase in intestinal gas, etc. may occur. Serious hepatic dysfunction accompanied with jaundice, increased aspartate aminotransferase (AST) or alanine aminotransferase (ALT), etc. may also occur. One case of hepatitis with severe cholestasis attributed to voglibose hypersensitivity has been reported; a causal relationship appears likely. When voglibose is administered to the patients with serious liver cirrhosis, hyperammonia may worsen with the development of constipation, etc., followed by disturbance of consciousness.

When voglibose is used in combination with other antidiabetic drugs, hypoglycaemia may occur (0.1% - < 5%). Furthermore, hypoglycaemia has been reported to occur (<0.1%) even when other antidiabetic drugs were not concomitantly used with this drug.

Overdosage

Overdose of metformin hydrochloride has occurred, including ingestion of amounts greater than 50 grams. Hypoglycaemia was reported in approximately 10% of cases, but no causal association with metformin hydrochloride has been established. Lactic acidosis has been reported in approximately 32% of metformin overdose cases. Metformin is dialysable with a clearance of up to 170 mL/min under good hemodynamic conditions. Therefore, haemodialysis may be useful for removal of accumulated drug from patients in whom metformin overdosage is suspected.

Voglibose competitively and reversibly inhibits the alpha-glucosidase enzymes in the brush border in the small intestine, which delays the hydrolysis of complex carbohydrates. It appears unlikely to produce hypoglycaemia in overdose, but abdominal discomfort and diarrhea may occur.

Incompatibility

None.

Shelf Life

2 years.

Storage and Handling Instructions

Store in a cool, dry place. Protect from light.

Packaging Information

PRANDIAL M 0.2: Blister of 10 tablets

PRANDIAL M 0.3: Blister of 10 tablets

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