

For the Use of a Registered Medical Practitioner Only

1. GENERIC NAME AND BRAND NAME

Sitagliptin Tablets I.P. 50mg
SIAGLIDE™ 50

Sitagliptin Tablets I.P.100mg
SIAGLIDE™ 100

2. QUALITATIVE AND QUANTITATIVE COMPOSITION:

SIAGLIDE 50

Each film coated tablet contains:

Sitagliptin Phosphate Monohydrate I.P.

Equivalent to Sitagliptin..... 50mg

Excipients..... q.s.

Colours: Red oxide of Iron, Yellow oxide of Iron and Titanium Dioxide I.P.

SIAGLIDE 100

Each film coated tablet contains:

Sitagliptin Phosphate Monohydrate I.P.

Equivalent to Sitagliptin..... 100mg

Excipients..... q.s.

Colours: Red oxide of Iron, Yellow oxide of Iron and Titanium Dioxide I.P.

3. DOSAGE FORM AND STRENGTH:

Refer section 1 & 2

4. CLINICAL PARTICULARS:

4.1. THERAPEUTIC INDICATION

(i) As adjunct to diet and exercise to improve glycemic control in patients with type-II diabetes.

(ii) Use of Sitagliptin Phosphate in combination with Metformin and a PPAR γ agonist as an adjunct to diet & exercise in adult patients with type-2 Diabetes mellitus who are inadequately controlled on combination therapy with Metformin and a PPAR γ agonist.

4.2. POSOLOGY AND METHOD OF ADMINISTRATION:

Posology

The dose is 100 mg sitagliptin once daily. When used in combination with metformin and/or a PPAR γ agonist, the dose of metformin and/or PPAR γ agonist should be maintained, and Siaglide administered concomitantly.

When Siaglide is used in combination with a sulphonylurea or with insulin, a lower dose of the sulphonylurea or insulin may be considered to reduce the risk of hypoglycaemia (see section 4.4).

If a dose of Siaglide is missed, it should be taken as soon as the patient remembers. A double dose should not be taken on the same day.

Special populations

Renal impairment

When considering the use of sitagliptin in combination with another anti-diabetic medicinal product, its conditions for use in patients with renal impairment should be checked.

For patients with mild renal impairment (glomerular filtration rate [GFR] ≥ 60 to < 90 mL/min), no dose adjustment is required.

For patients with moderate renal impairment (GFR ≥ 45 to < 60 mL/min), no dosage adjustment is required.

For patients with moderate renal impairment (GFR ≥ 30 to < 45 mL/min), the dose of sitagliptin is 50 mg once daily.

For patients with severe renal impairment (GFR ≥ 15 to < 30 mL/min) or with end-stage renal disease (ESRD) (GFR < 15 mL/min), including those requiring haemodialysis or peritoneal dialysis, the dose of sitagliptin is 25 mg once daily. Treatment may be administered without regard to the timing of dialysis.

Because there is a dosage adjustment based upon renal function, assessment of renal function is recommended prior to initiation of sitagliptin and periodically thereafter.

Hepatic impairment

No dose adjustment is necessary for patients with mild to moderate hepatic impairment.

Sitagliptin has not been studied in patients with severe hepatic impairment and care should be exercised (see section 5.2).

However, because sitagliptin is primarily renally eliminated, severe hepatic impairment is not expected to affect the pharmacokinetics of sitagliptin.

Elderly

No dose adjustment is necessary based on age.

Pediatric population

Sitagliptin should not be used in children and adolescents 10 to 17 years of age because of insufficient efficacy. Sitagliptin has not been studied in paediatric patients under 10 years of age.

Method of administration

Siaglide can be taken with or without food.

4.3. CONTRAINDICATIONS

Hypersensitivity to the active substance or to any of the excipients listed in section 6.1 (see sections 4.4 and 4.8).

4.4. SPECIAL WARNINGS AND PRECAUTIONS FOR USE

General

Sitagliptin should not be used in patients with type 1 diabetes or for the treatment of diabetic ketoacidosis.

Acute pancreatitis

Use of DPP-4 inhibitors has been associated with a risk of developing acute pancreatitis. Patients should be informed of the characteristic symptom of acute pancreatitis: persistent, severe abdominal pain. Resolution of pancreatitis has been observed after discontinuation of sitagliptin (with or without supportive treatment), but very rare cases of necrotising or haemorrhagic pancreatitis and/or death have been reported. If pancreatitis is suspected, sitagliptin and other potentially suspect medicinal products should be discontinued; if acute pancreatitis is confirmed, sitagliptin should not be restarted.

Caution should be exercised in patients with a history of pancreatitis. Hypoglycaemia when used in combination with other anti-hyperglycaemic medicinal products.

In clinical trials of sitagliptin as monotherapy and as part of combination therapy with medicinal products not known to cause hypoglycaemia (i.e. metformin and/or a PPAR γ agonist), rates of hypoglycaemia reported with sitagliptin were similar to rates in patients taking placebo. Hypoglycaemia has been observed when sitagliptin was used in combination with insulin or a sulphonylurea. Therefore, to reduce the risk of hypoglycaemia, a lower dose of sulphonylurea or insulin may be considered (see section 4.2).

Renal impairment

Sitagliptin is renally excreted. To achieve plasma concentrations of sitagliptin similar to those in patients with normal renal function, lower dosages are recommended in patients with GFR < 45 mL/min, as well as in ESRD patients requiring haemodialysis or peritoneal dialysis (see sections 4.2 and 5.2).

When considering the use of sitagliptin in combination with another anti-diabetic medicinal product, its conditions for use in patients with renal impairment should be checked.

Hypersensitivity reactions

Post-marketing reports of serious hypersensitivity reactions in patients treated with sitagliptin have been reported. These reactions include anaphylaxis, angioedema, and exfoliative skin conditions including Stevens-Johnson syndrome. Onset of these reactions occurred within the first 3 months after initiation of treatment, with some reports occurring after the first dose. If a hypersensitivity reaction is suspected, sitagliptin should be discontinued. Other potential causes for the event should be assessed, and alternative treatment for diabetes initiated.

Bullous pemphigoid

There have been post-marketing reports of bullous pemphigoid in patients taking DPP-4 inhibitors including sitagliptin. If bullous pemphigoid is suspected, sitagliptin should be discontinued.

Sodium

This medicinal product contains less than 1 mmol sodium (23 mg) per tablet, that is to say essentially 'sodium free'.

4.5. DRUGS INTERACTIONS

Effects of other medicinal products on sitagliptin

Clinical data described below suggest that the risk for clinically meaningful interactions by co-administered medicinal products is low.

In vitro studies indicated that the primary enzyme responsible for the limited metabolism of sitagliptin is CYP3A4, with contribution from CYP2C8. In patients with normal renal function, metabolism, including via CYP3A4, plays only a small role in the clearance of sitagliptin. Metabolism may play a more significant role in the elimination of sitagliptin in the setting of severe renal impairment or end-stage renal disease (ESRD). For this reason, it is possible that potent CYP3A4 inhibitors (i.e. ketoconazole, itraconazole, ritonavir, clarithromycin) could alter the pharmacokinetics of sitagliptin in patients with severe renal impairment or ESRD. The effect of potent CYP3A4 inhibitors in the setting of renal impairment has not been assessed in a clinical study.

In vitro transport studies showed that sitagliptin is a substrate for p-glycoprotein and organic anion transporter-3 (OAT3). OAT3 mediated transport of sitagliptin was inhibited in vitro by probenecid, although the risk of clinically meaningful interactions is considered to be low. Concomitant administration of OAT3 inhibitors has not been evaluated in vivo.

Metformin: Co-administration of multiple twice-daily doses of 1,000 mg metformin with 50 mg sitagliptin did not meaningfully alter the pharmacokinetics of sitagliptin in patients with type 2 diabetes.

In vitro data suggest that sitagliptin does not inhibit nor induce CYP450 isoenzymes. In clinical studies, sitagliptin did not meaningfully alter the pharmacokinetics of metformin, glyburide, simvastatin, rosiglitazone, warfarin, or oral contraceptives, providing in vivo evidence of a low propensity for causing interactions with substrates of CYP3A4, CYP2C8, CYP2C9, and organic cationic transporter (OCT). Sitagliptin may be a mild inhibitor of p-glycoprotein in vivo.

4.6. USE IN SPECIAL POPULATIONS (SUCH AS PREGNANT WOMEN, LACTATING WOMEN, PAEDIATRIC PATIENTS, GERIATRIC PATIENTS ETC.)

Pregnancy

There are no adequate data from the use of sitagliptin in pregnant women. Studies in animals have shown reproductive toxicity at high doses (see section 5.3). The potential risk for humans is unknown. Due to lack of human data, sitagliptin should not be used during pregnancy.

Breast-feeding

It is unknown whether sitagliptin is excreted in human breast milk. Animal studies have shown excretion of sitagliptin in breast milk. sitagliptin should not be used during breast-feeding.

Fertility

Animal data do not suggest an effect of treatment with sitagliptin on male and female fertility. Human data are lacking.

4.7. EFFECTS ON ABILITY TO DRIVE AND USE MACHINES

Sitagliptin has no or negligible influence on the ability to drive and use machines.

However, when driving or using machines, it should be taken into account that dizziness and somnolence have been reported.

In addition, patients should be alerted to the risk of hypoglycemia when sitagliptin is used in combination with a sulphonyl urea or with insulin.

4.8. UNDESIRABLE EFFECTS

Summary of the safety profile

Serious adverse reactions including pancreatitis and hypersensitivity reactions have been reported. Hypoglycemia has been reported in combination with sulphonyl urea (4.7 %-13.8 %) and insulin (9.6 %) (see section 4.4).

Tabulated list of adverse reactions

Adverse reactions are listed below (Table 1) by system organ class and frequency.

Frequencies are defined as: very common ($\geq 1/10$); common ($\geq 1/100$ to $< 1/10$); uncommon ($\geq 1/1,000$ to $< 1/100$); rare ($\geq 1/10,000$ to $< 1/1,000$); very rare ($< 1/10,000$) and not known (cannot be estimated from the available data).

Table 1. The frequency of adverse reactions identified from placebo-controlled clinical studies of sitagliptin monotherapy and post-marketing experience

Adverse reaction	Frequency of adverse reaction
Blood and lymphatic system disorders	
thrombocytopenia	Rare
Immune system disorders	
hypersensitivity reactions including anaphylactic responses ^{*,†}	Frequency not known
Metabolism and nutrition disorders	
hypoglycaemia [†]	Common
Nervous system disorders	
Headache	Common
Dizziness	Uncommon

Respiratory, thoracic and mediastinal disorders	
interstitial lung disease*	Frequency not known
Gastrointestinal disorders	
Constipation	Uncommon
vomiting*	Frequency not known
acute pancreatitis*,†,‡	Frequency not known
fatal and non-fatal hemorrhagic and necrotizing pancreatitis*,†	Frequency not known
Skin and subcutaneous tissue disorders	
pruritus*	Uncommon
angioedema*,†	Frequency not known
rash*,†	Frequency not known
urticaria*,†	Frequency not known
cutaneous vasculitis*,†	Frequency not known
exfoliative skin conditions including Stevens-Johnson syndrome*,†	Frequency not known
bullous pemphigoid*	Frequency not known
Musculoskeletal and connective tissue disorders	
arthralgia*	Frequency not known
myalgia*	Frequency not known
back pain*	Frequency not known
arthropathy*	Frequency not known
Renal and urinary disorders	
impaired renal function*	Frequency not known
acute renal failure*	Frequency not known

* Adverse reactions were identified through post-marketing surveillance.

† See section 4.4.

‡ See TECOS Cardiovascular Safety Study below.

TECOS Cardiovascular Safety Study

The Trial Evaluating Cardiovascular Outcomes with Sitagliptin (TECOS) included 7,332 patients treated with sitagliptin, 100 mg daily (or 50 mg daily if the baseline eGFR was ≥ 30 and < 50 mL/min/1.73 m²), and 7,339 patients treated with placebo in the intention-to-treat population. Both treatments were added to usual care targeting regional standards for HbA_{1c} and CV risk factors. The overall incidence of serious adverse events in patients receiving sitagliptin was similar to that in patients receiving placebo.

In the intention-to-treat population, among patients who were using insulin and/or a sulfonylurea at baseline, the incidence of severe hypoglycemia was 2.7 % in sitagliptin-treated patients and 2.5 % in placebo-treated patients; among patients who were not using insulin and/or a sulfonylurea at baseline, the incidence of severe hypoglycemia was 1.0 %

in sitagliptin-treated patients and 0.7 % in placebo-treated patients. The incidence of adjudication-confirmed pancreatitis events was 0.3 % in sitagliptin-treated patients and 0.2 % in placebo-treated patients.

4.9. OVERDOSE

During controlled clinical trials in healthy subjects, single doses of up to 800 mg sitagliptin were administered. Minimal increases in QTc, not considered to be clinically relevant, were observed in one study at a dose of 800 mg sitagliptin. There is no experience with doses above 800 mg in clinical studies. In Phase I multiple-dose studies, there were no dose-related clinical adverse reactions observed with sitagliptin with doses of up to 600 mg per day for periods of up to 10 days and 400 mg per day for periods of up to 28 days.

In the event of an overdose, it is reasonable to employ the usual supportive measures, e.g., remove unabsorbed material from the gastrointestinal tract, employ clinical monitoring (including obtaining an electrocardiogram), and institute supportive therapy if required.

Sitagliptin is modestly dialysable. In clinical studies, approximately 13.5 % of the dose was removed over a 3- to 4-hour hemodialysis session. Prolonged hemodialysis may be considered if clinically appropriate. It is not known if sitagliptin is dialysable by peritoneal dialysis.

5. PHARMACOLOGICAL PROPERTIES:

5.1. MECHANISM OF ACTION

Pharmacotherapeutic group: Drugs used in diabetes, Dipeptidyl peptidase 4 (DPP-4) inhibitors, ATC code: A10BH01.

Sitagliptin is a member of a class of oral anti-hyperglycemic agents called dipeptidyl peptidase 4 (DPP-4) inhibitors. The improvement in glycaemic control observed with this medicinal product may be mediated by enhancing the levels of active incretin hormones. Incretin hormones, including glucagon-like peptide-1 (GLP-1) and glucose-dependent insulinotropic polypeptide (GIP), are released by the intestine throughout the day, and levels are increased in response to a meal. The incretins are part of an endogenous system involved in the physiologic regulation of glucose homeostasis. When blood glucose concentrations are normal or elevated, GLP-1 and GIP increase insulin synthesis and release from pancreatic beta cells by intracellular signaling pathways involving cyclic AMP. Treatment with GLP-1 or with DPP-4 inhibitors in animal models of type 2 diabetes has been demonstrated to improve beta cell responsiveness to glucose and stimulate insulin biosynthesis and release. With higher insulin levels, tissue glucose uptake is enhanced. In addition, GLP-1 lowers glucagon secretion from pancreatic alpha cells.

Decreased glucagon concentrations, along with higher insulin levels, lead to reduced hepatic glucose production, resulting in a decrease in blood glucose levels. The effects of GLP-1 and GIP are glucose-dependent such that when blood glucose concentrations are low, stimulation of insulin release and suppression of glucagon secretion by GLP-1 are not observed. For both GLP-1 and GIP, stimulation of insulin release is enhanced as glucose rises above normal concentrations. Further, GLP-1 does not impair the normal glucagon response to hypoglycemia. The activity of GLP-1 and GIP is limited by the DPP-4 enzyme, which rapidly hydrolyzes the incretin hormones to produce inactive products. Sitagliptin prevents the hydrolysis of incretin hormones by DPP-4, thereby increasing plasma concentrations of the active forms of GLP-1 and GIP. By enhancing active incretin levels, sitagliptin increases insulin release and decreases glucagon levels in a glucose-dependent manner. In patients with type 2 diabetes with hyperglycemia, these changes in insulin and glucagon levels lead to lower haemoglobin A1c (HbA1c) and lower fasting and postprandial glucose concentrations. The glucose-dependent mechanism of sitagliptin is distinct from the mechanism of sulphonyl urea's, which increase insulin secretion even when glucose levels are low and can lead to hypoglycemia in patients with type 2 diabetes and in normal subjects. Sitagliptin is a potent and highly selective inhibitor of the enzyme DPP-4 and does not inhibit the closely related enzymes DPP-8 or DPP-9 at therapeutic concentrations.

5.2. PHARMACODYNAMIC PROPERTIES

Overall, sitagliptin improved glycaemic control when used as monotherapy or in combination treatment in adult patients with type 2 diabetes (see Table 2).

Table 2. HbA1c results in placebo-controlled monotherapy and combination therapy studies*

Study	Mean baseline HbA _{1c} (%)	Mean change from baseline HbA _{1c} (%) [†]	Placebo-corrected mean change in HbA _{1c} (%) [†] (95 % CI)
Monotherapy Studies			
Sitagliptin 100 mg once daily [§] (N=193)	8.0	-0.5	-0.6 [‡] (-0.8, -0.4)
Sitagliptin 100 mg once daily [%] (N=229)	8.0	-0.6	-0.8 [‡] (-1.0, -0.6)
Combination Therapy Studies			
Sitagliptin 100 mg once daily added to ongoing metformin therapy [%] (N=453)	8.0	-0.7	-0.7 [‡] (-0.8, -0.5)
Sitagliptin 100 mg once daily added to ongoing pioglitazone therapy [%]	8.1	-0.9	-0.7 [‡] (-0.9, -0.5)

(N=163)			
Sitagliptin 100 mg once daily added to ongoing glimepiride therapy [%] (N=102)	8.4	-0.3	-0.6 [‡] (-0.8, -0.3)
Sitagliptin 100 mg once daily added to ongoing glimepiride + metformin therapy [%] (N=115)	8.3	-0.6	-0.9 [‡] (-1.1, -0.7)
Sitagliptin 100 mg once daily added to ongoing pioglitazone + metformin therapy [#] (N=152)	8.8	-1.2	-0.7 [‡] (-1.0, -0.5)
Initial therapy (twice daily) [%] : Sitagliptin 50 mg + metformin 500 mg (N=183)	8.8	-1.4	-1.6 [‡] (-1.8, -1.3)
Initial therapy (twice daily) [%] : Sitagliptin 50 mg + metformin 1,000 mg (N=178)	8.8	-1.9	-2.1 [‡] (-2.3, -1.8)
Sitagliptin 100 mg once daily added to ongoing insulin (+/- metformin) therapy [%] (N=305)	8.7	-0.6 [¶]	-0.6 ^{‡,¶} (-0.7, -0.4)

¶ Least squares mean adjusted for metformin use at Visit 1 (yes/no), insulin use at Visit 1 (pre-mixed vs. non-pre-mixed [intermediate- or long-acting]), and baseline value. Treatment by stratum (metformin and insulin use) interactions were not significant (p > 0.10).

5.3. PHARMACOKINETIC PROPERTIES:

Absorption

Following oral administration of a 100-mg dose to healthy subjects, sitagliptin was rapidly absorbed, with peak plasma concentrations (median Tmax) occurring 1 to 4 hours post-dose, mean plasma AUC of sitagliptin was 8.52 µM•hr, Cmax was 950 nM. The absolute bioavailability of sitagliptin is approximately 87 %. Since co-administration of a high-fat meal with sitagliptin had no effect on the pharmacokinetics, sitagliptin may be administered with or without food.

Plasma AUC of sitagliptin increased in a dose-proportional manner. Dose-proportionality was not established for Cmax and C24hr (Cmax increased in a greater than dose-proportional manner and C24hr increased in a less than dose-proportional manner).

Distribution

The mean volume of distribution at steady state following a single 100-mg intravenous dose of sitagliptin to healthy subjects is approximately 198 litres. The fraction of sitagliptin reversibly bound to plasma proteins is low (38 %).

Biotransformation

Sitagliptin is primarily eliminated unchanged in urine, and metabolism is a minor pathway. Approximately 79 % of sitagliptin is excreted unchanged in the urine.

Following a [14C] sitagliptin oral dose, approximately 16 % of the radioactivity was excreted as metabolites of sitagliptin. Six metabolites were detected at trace levels and are not expected to contribute to the plasma DPP-4 inhibitory activity of sitagliptin. In vitro studies indicated that the primary enzyme responsible for the limited metabolism of sitagliptin was CYP3A4, with contribution from CYP2C8.

In vitro data showed that sitagliptin is not an inhibitor of CYP isozymes CYP3A4, 2C8, 2C9, 2D6, 1A2, 2C19 or 2B6, and is not an inducer of CYP3A4 and CYP1A2.

Elimination

Following administration of an oral [14C] sitagliptin dose to healthy subjects, approximately 100 % of the administered radioactivity was eliminated in faeces (13 %) or urine (87 %) within one week of dosing. The apparent terminal t_{1/2} following a 100-mg oral dose of sitagliptin was approximately 12.4 hours. Sitagliptin accumulates only minimally with multiple doses. The renal clearance was approximately 350 mL/min.

Elimination of sitagliptin occurs primarily via renal excretion and involves active tubular secretion. Sitagliptin is a substrate for human organic anion transporter-3 (hOAT-3), which may be involved in the renal elimination of sitagliptin. The clinical relevance of hOAT-3 in sitagliptin transport has not been established. Sitagliptin is also a substrate of p-glycoprotein, which may also be involved in mediating the renal elimination of sitagliptin. However, ciclosporin, a p-glycoprotein inhibitor, did not reduce the renal clearance of sitagliptin. Sitagliptin is not a substrate for OCT2 or OAT1 or PEPT1/2 transporters. In vitro, sitagliptin did not inhibit OAT3 (IC₅₀=160 µM) or p-glycoprotein (up to 250 µM) mediated transport at therapeutically relevant plasma concentrations. In a clinical study sitagliptin had a small effect on plasma digoxin concentrations indicating that sitagliptin may be a mild inhibitor of p-glycoprotein.

Characteristics in patients

The pharmacokinetics of sitagliptin were generally similar in healthy subjects and in patients with type 2 diabetes.

6. NONCLINICAL PROPERTIES:

6.1. ANIMAL TOXICOLOGY OR PHARMACOLOGY

Renal and liver toxicity were observed in rodents at systemic exposure values 58 times the human exposure level, while the no-effect level was found at 19 times the human exposure level. Incisor teeth abnormalities were observed in rats at exposure levels 67 times the clinical exposure level; the no-effect level for this finding was 58-fold based on the 14-week rat study. The relevance of these findings for humans is unknown. Transient treatment-related physical signs, some of which suggest neural toxicity, such as open-mouth breathing, salivation, white foamy emesis, ataxia, trembling, decreased activity, and/or hunched posture were observed in dogs at exposure levels approximately 23 times the clinical exposure level. In addition, very slight to slight skeletal muscle degeneration was also observed histologically at doses resulting in systemic exposure levels of approximately 23 times the human exposure level. A no-effect level for these findings was found at an exposure 6-fold the clinical exposure level.

Sitagliptin has not been demonstrated to be genotoxic in preclinical studies. Sitagliptin was not carcinogenic in mice. In rats, there was an increased incidence of hepatic adenomas and carcinomas at systemic exposure levels 58 times the human exposure level. Since hepatotoxicity has been shown to correlate with induction of hepatic neoplasia in rats, this increased incidence of hepatic tumours in rats was likely secondary to chronic hepatic toxicity at this high dose. Because of the high safety margin (19-fold at this no-effect level), these neoplastic changes are not considered relevant for the situation in humans.

No adverse effects upon fertility were observed in male and female rats given sitagliptin prior to and throughout mating.

In a pre-/postnatal development study performed in rats sitagliptin showed no adverse effects.

Reproductive toxicity studies showed a slight treatment-related increased incidence of foetal rib malformations (absent, hypoplastic and wavy ribs) in the offspring of rats at systemic exposure levels more than 29 times the human exposure levels. Maternal toxicity was seen in rabbits at more than 29 times the human exposure levels. Because of the high safety margins, these findings do not suggest a relevant risk for human reproduction. Sitagliptin is secreted in considerable amounts into the milk of lactating rats (milk/plasma ratio: 4:1)

7. DESCRIPTION:

SIAGLIDE 50 is supplied for oral administration, as a film coated tablet of Sitagliptin. Each film coated tablet of SIAGLIDE contains Sitagliptin Phosphate Monohydrate equivalent to Sitagliptin 50mg.

SIAGLIDE 100 is supplied for oral administration, as a film coated tablet of Sitagliptin. Each film coated tablet of SIAGLIDE contains Sitagliptin Phosphate Monohydrate equivalent to Sitagliptin 100mg.

8. PHARMACEUTICAL PARTICULARS:

8.1. INCOMPATIBILITIES

Not Applicable

8.2. SHELF-LIFE

Refer Pack

8.3. PACKAGING INFORMATION

Refer Pack

8.4. STORAGE CONDITION AND HANDING INSTRUCTIONS

Refer Pack

9. PATIENT COUNSELLING INFORMATION:

What Sitagliptin is and what it is used for?

Sitagliptin which is a member of a class of medicines called DPP-4 inhibitors (dipeptidyl peptidase-4 inhibitors) that lowers blood sugar levels in adult patients with type 2 diabetes mellitus.

This medicine helps to increase the levels of insulin produced after a meal and decreases the amount of sugar made by the body.

Your doctor has prescribed this medicine to help lower your blood sugar, which is too high because of your type 2 diabetes. This medicine can be used alone or in combination with certain other medicines (insulin, metformin, sulphonylureas, or glitazones) that lower blood sugar, which you may already be taking for your diabetes together with a food and exercise plan.

What you need to know before you take Sitagliptin

Do not take Sitagliptin

- if you are allergic to sitagliptin or any of the other ingredients of this medicine

Warnings and precautions

Cases of inflammation of the pancreas (pancreatitis) have been reported in patients receiving Sitagliptin.

If you encounter blistering of the skin it may be a sign for a condition called bullous pemphigoid. Your doctor may ask you to stop Sitagliptin.

Tell your doctor if you have or have had:

- a disease of the pancreas (such as pancreatitis)

- gallstones, alcohol dependence or very high levels of triglycerides (a form of fat) in your blood. These medical conditions can increase your chance of getting pancreatitis.
- type 1 diabetes
- diabetic ketoacidosis (a complication of diabetes with high blood sugar, rapid weight loss, nausea or vomiting)
- any past or present kidney problems
- an allergic reaction to Sitagliptin

This medicine is unlikely to cause low blood sugar because it does not work when your blood sugar is low. However, when this medicine is used in combination with a sulphonylurea medicine or with insulin, low blood sugar (hypoglycaemia) can occur. Your doctor may reduce the dose of your sulphonylurea or insulin medicine.

Children and adolescents

Children and adolescents below 18 years should not use this medicine. It is not effective in children and adolescents between the ages of 10 and 17 years. It is not known if this medicine is safe and effective when used in children younger than 10 years.

Other medicines and Sitagliptin

Tell your doctor or pharmacist if you are taking, have recently taken or might take any other medicines.

In particular, tell your doctor if you are taking digoxin (a medicine used to treat irregular heartbeat and other heart problems). The level of digoxin in your blood may need to be checked if taking with Sitagliptin.

Pregnancy and breast-feeding

If you are pregnant or breast-feeding, think you may be pregnant or are planning to have a baby, ask your doctor or pharmacist for advice before taking this medicine.

You should not take this medicine during pregnancy.

It is not known if this medicine passes into breast milk. You should not take this medicine if you are breast-feeding or plan to breast-feed.

Driving and using machines

This medicine has no or negligible influence on the ability to drive and use machines. However, dizziness and drowsiness have been reported, which may affect your ability to drive or use machines.

Taking this medicine in combination with medicines called sulphonylureas or with insulin can cause hypoglycaemia, which may affect your ability to drive and use machines or work without safe foothold.

How to take Sitagliptin

Always take this medicine exactly as your doctor has told you. Check with your doctor or pharmacist if you are not sure.

You can take this medicine with or without food and drink.

Your doctor may prescribe this medicine alone or with certain other medicines that lower blood sugar.

Diet and exercise can help your body use its blood sugar better. It is important to stay on the diet and exercise recommended by your doctor while taking Sitagliptin.

If you take more Sitagliptin then you should

If you take more than the prescribed dosage of this medicine, contact your doctor immediately.

If you forget to take Sitagliptin

If you miss a dose, take it as soon as you remember. If you do not remember until it is time for your next dose, skip the missed dose and go back to your regular schedule. Do not take a double dose of this medicine.

If you stop taking Sitagliptin

Continue to take this medicine as long as your doctor prescribes it so you can continue to help control your blood sugar. You should not stop taking this medicine without talking to your doctor first.

If you have any further questions on the use of this medicine, ask your doctor or pharmacist.

Possible side effects

Like all medicines, this medicine can cause side effects, although not everybody gets them.

STOP taking Sitagliptin and contact a doctor immediately if you notice any of the following serious side effects:

- Severe and persistent pain in the abdomen (stomach area) which might reach through to your back with or without nausea and vomiting, as these could be signs of an inflamed pancreas (pancreatitis).

If you have a serious allergic reaction (frequency not known), including rash, hives, blisters on the skin/peeling skin and swelling of the face, lips, tongue, and throat that may cause difficulty in breathing or swallowing, stop taking this medicine and call your doctor right away. Your doctor may prescribe a medicine to treat your allergic reaction and a different medicine for your diabetes.

Some patients have experienced the following side effects after adding sitagliptin to metformin:

Common (may affect up to 1 in 10 people): low blood sugar, nausea, flatulence, vomiting

Uncommon (may affect up to 1 in 100 people): stomach ache, diarrhoea, constipation, drowsiness

Some patients have experienced different types of stomach discomfort when starting the combination of sitagliptin and metformin together (frequency is common).

10. DETAILS OF MANUFACTURER:

Refer Pack for manufacturer details.

™ Trade Mark of Abbott Healthcare Pvt. Ltd.

11. DETAILS OF PERMISSION OR LICENCE NUMBER

Refer Pack for Permission/License details

12. DATE OF REVISION

Version 2.0 Dated 10th Oct 2023

For Product Complaints/Adverse events or Queries please write to
webmasterindia@abbott.com