NOVARTIS

Sandostatin®

Composition
Active substances
Octreotide (as octreotide acetate).
Excipients:
Ampoules (1 ml): Lactic acid, mannitol, sodium hydrogen carbonate (for pH adjustment), water for injections.
Vial (5 ml): Lactic acid, mannitol, phenol (5 mg/ml), sodium hydrogen carbonate (for pH adjustment), water for injections.

Pharmaceutical form and quantity of active substance per unit
Solution for injection/infusion for s.c. injection or i.v. infusion
Ampoules (1 ml) containing 0.05 mg/ml, 0.1 mg/ml or 0.5 mg/ml
Vial (1 mg/5 ml) containing 0.2 mg/ml

Indications/Potential uses
- *Acromegaly*
  Symptom control and reduction of plasma levels of growth hormone (GH) and insulin-like growth factor 1 (IGF-1) in acromegalic patients who have failed to respond to surgery or radiotherapy, are unable or unwilling to undergo surgery or are in the latency period before radiotherapy becomes fully effective.
- *Relief of symptoms of functional gastroenteropancreatic (GEP) endocrine tumours*

Efficacy has been adequately documented in these indications:
- *Carcinoid tumours with features of the carcinoid syndrome*
- *VIPoma (VIP: Vasoactive Intestinal Peptide)*
- *Glucagonomas*

Sandostatin shows efficacy in about 50% of cases (limited number of patients treated thus far) in these indications:
- *Gastrinomas/Zollinger-Ellison syndrome (usually in conjunction with proton pump inhibitors or H_2-antagonist therapy)*
- *Insulinomas (for pre-operative prevention of hypoglycaemia and for maintenance therapy)*
- *GRFomas (GRF: growth-hormone-releasing factor)*

In the above-mentioned conditions Sandostatin often leads to improvement in symptoms, but not to a cure.
- *Prevention of complications following pancreatic surgery*
- *Emergency treatment of bleeding gastro-oesophageal varices secondary to cirrhosis in combination with specific therapy such as endoscopic sclerotherapy*

Dosage/Administration
- *Acromegaly*
  Initial dose of 0.05 to 0.1 mg every 8 hours by s.c. injection. The dose should be adjusted based on monthly assessment of its effects on levels of circulating GH and IGF-1 (target: GH < 2.5 ng/ml; IGF-1 in normal range), clinical symptoms and tolerability. In most patients, the optimum daily dose is 0.2 to 0.3 mg. IGF-1 and/or GH should be measured every 6 months in patients who continue to receive the same dose.
  A maximum daily dose of 1.5 mg should not be exceeded. After several months of treatment, with monitoring of plasma GH levels, dose reduction may be considered.
  If there is no appreciable reduction in IGF-1 and/or GH levels and clinical symptoms have not improved by the end of one month of treatment with Sandostatin, discontinuation of treatment should be considered.
  See also Sandostatin LAR (long-term treatment of acromegaly).
- *Gastroenteropancreatic endocrine tumours*
  The starting dose is 0.05 mg once or twice daily s.c. The dose may be gradually increased to 0.2 mg three times daily, with tolerability and therapeutic efficacy (improvement in symptoms, reduction in elevated levels of tumour-produced hormones) being taken into account. Higher doses may be required in exceptional cases. The maintenance dosage requires individual titration.
  It is recommended that treatment be discontinued after one week if there is a lack of therapeutic efficacy.
- *Prevention of complications following pancreatic surgery*
  0.1 mg three times daily s.c. on 7 successive days, the first dose being administered on the day of the operation at least one hour before the start of surgery.
- *Bleeding gastro-oesophageal varices*
  0.025 mg/hour, given as a continuous i.v. infusion for a maximum of 5 days. Sandostatin may be diluted with physiological saline (see also "Instructions for administration by i.v. infusion" under "Other Information").

Special dosage instructions
Elderly patients: Results of a small-scale, single-dose study in elderly subjects show no need for any special dosage in elderly patients at the start of treatment with Sandostatin.

Children and adolescents: Experience with octreotide in children is limited.

Hepatic impairment: The half-life of the product may be longer in patients with liver cirrhosis and may necessitate a change in the maintenance dose. Sandostatin was well tolerated when administered by continuous i.v. infusion at doses up to 0.050 mg/hour over a period of 5 days to cirrhotic patients with bleeding gastro-oesophageal varices.

Renal impairment: Renal impairment had no effect on total exposure (AUC) to subcutaneously administered octreotide and it is therefore not necessary to adjust the dose of Sandostatin.

Use: See "Instructions for use and handling" under "Other information".

Note: Patients who are to self-administer the drug by s.c. injection must receive precise instructions from the physician or healthcare professional (see "Other information").
It is recommended that the solution for injection be allowed to reach room temperature prior to administration so that injection site pain can be avoided as much as possible. Repeated injection at short intervals at the same site should be avoided. To prevent contamination of the vial, the rubber cap should not be punctured more than ten times.
Gastrointestinal adverse effects can be attenuated by allowing as long an interval as possible between administration and mealtimes, i.e. by giving injections between meals or at bedtime.

Contraindications
Known hypersensitivity to octreotide or to any of the excipients.

Warnings and precautions
General
GH-secreting pituitary tumours may expand, causing serious complications (e.g. restriction of the visual field) and patients must therefore be closely monitored. If evidence of tumour expansion is detected, alternative treatment methods should be considered.

Glucose metabolism
Octreotide may exacerbate and prolong hypoglycaemic episodes in patients with insulinoma because it inhibits GH and glucagon secretion more potently than insulin secretion and for a greater length of time. Such patients require particularly close monitoring at the start of Sandostatin treatment and whenever the dose is changed.
Marked fluctuations in blood glucose may sometimes be controlled by more frequent injections with smaller doses.
Sandostatin may reduce the insulin requirements of patients with type I (insulin-dependent) diabetes. Hypoglycaemia was reported.
Sandostatin can cause a postprandial rise in blood sugar in non-diabetics and in type II diabetics with partially intact insulin reserves. It is therefore recommended to monitor blood sugar levels and to adjust anti-diabetic therapy, if required.
Following bleeding episodes from oesophageal varices there is an increased risk of the development of insulin-dependent diabetes or changes in insulin requirements in patients with pre-existing type I diabetes. Appropriate monitoring of blood glucose levels is therefore particularly important in these patients.

Biliary disorders
Somatostatin analogues inhibit gallbladder contractility and decrease bile secretion, which may lead to gallbladder abnormalities, the formation of biliary sludge or gallstones. The incidence of cholelithiasis during Sandostatin treatment is estimated at 15-30% compared to an incidence of 5-20% in the general population. Cholelithiasis is mostly asymptomatic during Sandostatin treatment.

Biliary dilatation has also been reported during use of Sandostatin as well as cases of cholecystitis or cholangitis (as a complication of cholelithiasis). Therefore, a sonographic examination of the gallbladder is recommended before treatment initiation and during treatment with Sandostatin at 6-12-month intervals.

Pancreatitis
In very rare cases, acute pancreatitis has been reported after the first few hours or days of treatment with Sandostatin. It has resolved again on withdrawal of treatment. In addition, cholelithiasis-induced pancreatitis has been reported in patients receiving long-term treatment with Sandostatin.

Cardiovascular adverse effects
Bradycardia is a common adverse effect of somatostatin analogue treatment. Dose adjustment of medicinal products such as beta blockers, calcium channel blockers or medicinal products used to control the electrolyte and fluid balance may be necessary.
Furthermore, cases of AV block (including complete AV block) were reported in patients who received octreotide as an i.v. bolus (50 µg bolus followed by a continuous infusion of 50 µg/hour) or in the form of a high-dose continuous infusion (100 µg/hour). Patients who receive high intravenous octreotide doses should therefore be monitored accordingly. An infusion rate of 50 µg/hour should not be exceeded.
In acromegaly and carcinoid patients ECG changes such as QT prolongation, axis shifts, early repolarisation, low voltage, R/S transition, early R wave progression and non-specific ST-T wave changes have also been observed. The relationship of these events to octreotide has not been definitively established as many of the patients in question had underlying heart disease.

GEP endocrine tumours
There may be rare instances of a sudden loss of symptomatic control with recurrence of severe symptoms in patients with GEP (gastroenteropancreatic) endocrine tumours receiving Sandostatin.

Hypersensitivity reactions
There have been post-marketing reports of hypersensitivity and allergic reactions. These were mainly associated with skin reactions; rarely, the mouth and respiratory tract were affected. Isolated cases of anaphylactic shock have been reported.

Other precautions
Lower vitamin B_{12} blood levels and abnormal Schilling test results have been observed in some patients receiving octreotide therapy. In patients with a history of vitamin B_{12} deficiency monitoring of vitamin B_{12} levels is recommended during treatment with Sandostatin.
Thyroid function should be monitored in patients receiving long-term treatment with octreotide.
Octreotide may alter the absorption of dietary fats in some patients. Faecal fat excretion may be increased in particular. However, there is no evidence of a nutritional deficiency due to malabsorption, even during long-term treatment with octreotide.

The therapeutic benefit of a reduction in growth hormone (GH) levels and normalisation of insulin-like growth factor 1 (IGF-1) concentrations in female acromegalic patients may potentially restore fertility. Where indicated, patients of childbearing potential should be advised to use adequate contraception during treatment with octreotide (see "Pregnancy/Breast-feeding").
This medicinal product contains less than 1 mmol (23 mg) of sodium per dosage volume, making it practically "sodium-free".

Interactions
Pharmacokinetic interactions
Octreotide reduces the intestinal absorption of ciclosporin and delays that of cimetidine.
Co-administration of octreotide and bromocriptine increases the availability of bromocriptine.
A limited amount of published data indicates that somatostatin analogues might reduce the metabolic clearance of substances metabolised by cytochrome P450 enzymes. This is attributed to the suppression of growth hormones. As it cannot be ruled out that octreotide might also have such an effect, caution is indicated when using other drugs that are principally metabolised by CYP3A4 and have a narrow therapeutic index (e.g. quinidine, terfenadine).

Pharmacodynamic interactions
Dose adjustment of medicines such as beta blockers, calcium channel blockers or agents to control fluid and electrolyte balance may be necessary when Sandostatin is co-administered (see "Warnings and precautions").
Dose adjustments of insulin and anti-diabetic medicines may be required when Sandostatin is co-administered (see "Warnings and precautions").

Concomitant use of radiopharmaceuticals coupled to somatostatin analogues (peptide receptor radionuclide therapy (PRRT))
Somatostatin and its analogues (e.g. octreotide) bind competitively to somatostatin receptors and may impair the efficacy of relevant radiopharmaceuticals (e.g. ^{177}Lu) oxodotreotide). Sandostatin should therefore not be administered in the 24 hours prior to administration of a PRRT.

Pregnancy/Breast-feeding
Pregnancy
Animal studies with octreotide have not shown reproductive toxicological effects, apart from transient delayed growth of offspring (see "Preclinical data").
There are no adequate and well-controlled studies in pregnant women. In the post-marketing period, there have been a limited number of reports concerning female acromegaly patients who were pregnant and received octreotide, but pregnancy outcomes are unknown in half of these cases. Most of the patients received octreotide during the first trimester of pregnancy at doses ranging from 100 to 300 µg Sandostatin s.c. daily or 20 to 30 mg Sandostatin LAR per month. In approximately two-thirds of the cases of pregnancies with known outcome, the women chose to continue octreotide therapy during their pregnancies. Normal newborns were reported in most of the cases with known outcome, but some spontaneous abortions during the first trimester were also reported. Congenital abnormalities or malformations were not observed.
Sandostatin should only be prescribed to pregnant woman if absolutely necessary.

Breast-feeding
It is not known whether octreotide is excreted in human milk. Animal studies have shown excretion of octreotide in the breast milk. Women should not breast-feed while undergoing treatment with Sandostatin.

Fertility
It is not known whether octreotide has an effect on human fertility. Octreotide did not impair fertility in male and female rats at doses of up to 1 mg/kg body weight per day (see "Preclinical data").

Effects on the ability to drive and to use machines
No data are available on the effect of Sandostatin on the ability to drive and to use machines.

Adverse effects
In clinical studies, the most commonly reported adverse effects following administration of octreotide were diarrhoea, abdominal pain, nausea, flatulence, headache, cholelithiasis, hyperglycaemia and constipation.

Gastrointestinal disorders and nutrition
In rare instances, gastrointestinal adverse effects may resemble acute intestinal obstruction with progressive abdominal distension, severe epigastric pain and painful abdominal tenderness.

Thrombocytopenia
There have been post-marketing reports of thrombocytopenia, particularly during treatment with Sandostatin (i.v.) in patients with liver cirrhosis. The thrombocytopenia was reversible after discontinuation of treatment.

Administration-site reactions
Local reactions to Sandostatin include paraesthesia, pain, stinging or burning at the site of s.c. injection with redness and swelling. Such symptoms do not normally last more than 15 minutes and can be attenuated by allowing the Sandostatin solution to reach room temperature prior to injection or by injecting a smaller volume in a more concentrated solution.

The adverse effects observed in clinical studies or in the post-marketing period with octreotide are listed below by MedDRA system organ class and frequency. The frequency is ranked using the following convention: *Very common (≥1/10), common (≥1/100 to <1/10), uncommon (≥1/1,000 to <1/100), rare (≥1/10,000 to <1/1,000), very rare (<1/10,000), not known* (primarily based on spontaneous post-marketing reports; precise frequency cannot be estimated).

Blood and lymphatic system disorders
Not known: Thrombocytopenia.

Immune system disorders
Not known: Hypersensitivity reactions (including anaphylactoid reactions).

Endocrine disorders
Common: Hypothyroidism, thyroid dysfunction (e.g. decreased TSH, decreased total T4 and decreased free T4).

Metabolism and nutrition disorders
Very common: Hyperglycaemia (10.8%).
Common: Hypoglycaemia, impaired glucose tolerance, decreased appetite.
Uncommon: Dehydration.

Nervous system disorders
Very common: Headache (12.4%).
Common: Dizziness.

Cardiac disorders
Common: Bradycardia.
Uncommon: Tachycardia.
Not known: Arrhythmias.

Respiratory, thoracic and mediastinal disorders
Common: Dyspnoea.

Gastrointestinal disorders
Very common: Diarrhoea (26.1%), abdominal pain (24.2%), nausea (14.3%), flatulence (14.2%), constipation (12.7%).
Common: Dyspepsia, vomiting, abdominal distension, steatorrhoea, discoloured faeces.
Unknown: acute pancreatitis

Hepatobiliary disorders
Very common: Cholelithiasis (12.0%).
Common: Increased transaminases, hyperbilirubinaemia, cholecystitis.
Not known: Increased blood alkaline phosphatase, increased gamma glutamyl transferase, jaundice, cholestasis, cholestatic jaundice, cholestatic hepatitis, acute hepatitis without cholestasis.

Skin and subcutaneous tissue disorders
Common: Pruritus, skin rash, alopecia.
Not known: Urticaria.

General disorders and administration site conditions
Very common: Injection site reactions (10 to 30% depending on the dose and injection interval, e.g. pain, paraesthesia, erythema).
Common: Asthenia
Reporting suspected adverse effects after authorisation of the medicinal product is very important. It allows continued monitoring of the risk-benefit ratio of the medicinal product.

Overdose
Symptoms
A limited number of accidental overdoses of Sandostatin have been reported in adults and children. In adults the doses ranged from 2,400-6,000 µg/day administered as a continuous infusion (100-250 µg/hour), intravenous bolus (50 µg followed by a continuous infusion) or subcutaneously (1,500 µg 3 times daily). The reported symptoms were AV block (including cases of complete AV block), arrhythmia, hypotension, cardiac arrest, cerebral hypoxia, pancreatitis, steatohepatitis, diarrhoea, weakness, lethargy, weight loss, hepatomegaly and lactic acidosis.
In children, the doses ranged from 50 to 3,000 µg/day, administered by continuous infusion (2.1 to 500 µg/hour) or subcutaneously (50 to 100 µg). Mild hyperglycaemia was the only reported adverse effect.
No unexpected adverse effects have been reported in cancer patients receiving s.c. doses of 3,000 to 30,000 µg Sandostatin per day in divided doses.

Treatment
The management of overdosage is symptomatic. Close ECG monitoring should be performed in the case of intravenous octreotide therapy.

Properties/Actions
ATC code
H01CB02

Mechanism of action
Sandostatin is a synthetic octapeptide derivative of naturally occurring somatostatin, with qualitatively similar pharmacological effects but a considerably longer duration of action. It inhibits the pathologically increased secretion of growth hormone (GH) and of peptide hormones of the gastroenteropancreatic (GEP) endocrine system.
In animals, Sandostatin is a more potent inhibitor of GH, glucagon and insulin release than somatostatin is, with greater selectivity for GH and glucagon suppression.

In healthy volunteers Sandostatin has been shown to inhibit:
- GH release in response to arginine, exercise, or insulin-induced hypoglycaemia.
- postprandial release of insulin, glucagon, gastrin and other peptides of the GEP system and the secretion of insulin and glucagon in response to arginine.
- release of thyroid-stimulating hormone (TSH) in response to thyrotropin-releasing hormone (TRH).

In contrast to somatostatin, octreotide inhibits GH secretion preferentially over insulin and does not cause rebound hypersecretion of hormones (e.g. GH in acromegalic patients).
In acromegalic patients, Sandostatin lowers plasma levels of GH and IGF-1. These levels fall by 50% or more in up to 90% of patients, with a reduction in serum GH to < 5 ng/ml in about half of the cases.
In most patients, there is a marked improvement in clinical manifestations such as headache, skin and soft tissue swelling, hyperhidrosis, arthralgia and paraesthesia. In patients with a large pituitary adenoma, Sandostatin treatment may result in a degree of tumour shrinkage.
In *patients with functional tumours of the GEP endocrine system*, Sandostatin exerts a beneficial effect on various clinical features by virtue of its wide spectrum of endocrine activity. Sandostatin may bring about appreciable improvement in patients who, despite other treatment (surgery, hepatic artery embolisation, chemotherapy [e.g. streptozocin or 5-fluorouracil]), suffer from severe tumour-related symptoms.

Pharmacodynamics
Clinical efficacy
Effects of Sandostatin on different types of tumours
Carcinoid tumours
Use of Sandostatin may bring about an improvement in symptoms, in particular flush and diarrhoea. In some cases, there may also be a fall in plasma serotonin and reduced urinary excretion of 5-hydroxyindole acetic acid.
It is recommended that treatment be discontinued after one week if there is a lack of therapeutic efficacy.

VIPomas
The biochemical feature of these tumours is overproduction of vasoactive intestinal peptide (VIP).
In most cases Sandostatin treatment results in alleviation of the severe secretory diarrhoea typical of the condition, with consequent improvement in quality of life. Fluid and electrolyte disturbances (e.g. hypokalaemia) associated with this diarrhoea also improve, so that enteral and parenteral fluid and electrolyte replacement can be withdrawn. CT scan has indicated slowing or arrest of tumour growth – or even shrinkage – in some patients, particularly those with liver metastases. Clinical improvement is usually accompanied by reduction – or even normalisation – of plasma VIP levels.

Glucagonomas
In most cases, there is substantial improvement in the necrotic migratory rash which is characteristic of this condition. Sandostatin has little effect on the mild diabetes mellitus to which glucagonoma patients are prone and there is normally no reduction in the required dosage of insulin or oral antidiabetics. Diarrhoea, where present, responds, resulting in weight gain. Sandostatin frequently brings about an immediate reduction in plasma glucagon. This effect is not sustained as treatment continues, although symptoms continue to improve.

Gastrinomas / Zollinger-Ellison syndrome
Treatment with proton pump inhibitors or H_2-receptor blockers cannot always prevent the recurrent peptic ulceration which results from chronic gastrin-stimulated hypersecretion of gastric acid and does not alleviate diarrhoea, which may be prominent. In such cases, Sandostatin – either alone or in combination with proton pump inhibitors or H_2-receptor blockers – may reduce increased gastric acid secretion and induce an improvement in the clinical manifestations (including diarrhoea) in 50% of cases. Other manifestations assumed to be due to peptide production by the tumour (e.g. flush) may also respond. Sandostatin produces a fall in plasma gastrin levels in some patients.

Insulinomas
Although Sandostatin causes a reduction in circulating immunoreactive insulin, this effect is normally of short duration (approx. 2 hours). In patients with operable tumours, Sandostatin may be given pre-operatively to help achieve and maintain normoglycaemia. Sandostatin may bring about an improvement in blood-sugar regulation in a limited number of patients with inoperable benign or malignant tumours, even without a sustained reduction in circulating insulin.

GRFomas
This is a rare type of tumour that produces growth-hormone-releasing factor (GRF) alone or in conjunction with other biologically active peptides. In one of two cases studied, Sandostatin treatment resulted in clinical improvement of the resulting symptoms of acromegaly. This effect is probably due to reduced production of GRF and inhibition of GH secretion, possibly accompanied by a reduction in the size of the enlarged pituitary gland.

Complications following pancreatic surgery
In patients undergoing pancreatic surgery, peri- and post-operative Sandostatin reduces the incidence of typical post-operative complications (e.g. pancreatic fistula, abscess followed by sepsis, acute post-operative pancreatitis).

Bleeding gastro-oesophageal varices
A clinical study has shown that use of Sandostatin in combination with sclerotherapy in the management of bleeding gastro-oesophageal varices secondary to cirrhosis resulted in improved control of bleeding and of early rebleeding, a reduction in transfusion requirements and an increase in the rate of survival at day 5. The precise mechanism of action of Sandostatin in this indication remains unclear, although it has been suggested that Sandostatin may inhibit splanchnic blood flow by inhibiting vasoactive hormones such as VIP and glucagon.

Pharmacokinetics
Absorption
Octreotide is rapidly and completely absorbed after s.c. injection. Peak plasma concentrations are reached within 30 minutes.

Distribution
The volume of distribution is 0.27 litres/kg and the total body clearance is 160 ml/minute. Plasma protein binding is 65%. The amount of octreotide bound to blood cells is very small.
Metabolism
Metabolism occurs in line with general protein metabolism.

Elimination
The elimination half-life after s.c. administration is 100 minutes. After i.v. injection, the elimination is biphasic, with half-lives of 10 and 90 minutes. Most of the peptide is eliminated via the faeces, while approximately 32% is excreted unchanged into the urine.

Pharmacokinetics in special populations
Renal impairment: Renal impairment had no effect on total exposure (AUC) to subcutaneously administered octreotide.
Hepatic impairment: Cirrhosis of the liver, but not fatty liver, is associated with a 30% reduction in octreotide elimination.

Preclinical data
Mutagenicity
Octreotide and/or its metabolites did not display any mutagenic potential *in vitro*.
In vivo studies did not show any clastogenic activity in the bone marrow of mice treated with octreotide i.v. (micronucleus test) or any evidence of genotoxicity in male mice (DNA assay).

Carcinogenicity/chronic toxicity
In rats, local tumours were observed at the injection site, a species-specific reaction. They were attributed to disordered fibroplasia produced by sustained irritant effects at the injection sites and exacerbated by the vehicle.
Endometrial adenocarcinomas were reported in a carcinogenicity study in rats. The available data clearly indicate that the findings of endocrine-mediated tumours in rats are species-specific and are not relevant for the use of the drug in humans.

Reproductive toxicity
Reproductive and development toxicity studies have been performed in rats and rabbits at doses of up to 1 mg/kg body weight per day. Octreotide did not impair fertility in male and female rats. There was no evidence of teratogenic, embryofetal or other reproduction effects due to octreotide. Some delay in the physiological growth was noted in the offspring of rats which was transient and most likely attributable to GH inhibition brought about by excessive pharmacodynamic activity. In pre- and post-natal development studies, late testicular descent was observed in male offspring of maternal animals treated during pregnancy and lactation. However, the fertility of the F1 offspring was normal. It is assumed that these observations are attributable to octreotide-mediated growth inhibition .

Other information
Shelf life
Do not use after the expiry date (= EXP) printed on the pack.

Special precautions for storage
Store in a refrigerator (2-8°C). Do not freeze. Protect from light.
For day-to-day use, the ampoules and vials may be stored for up to 2 weeks at temperatures not above 30°C and 25°C, respectively.
Keep out of the reach of children.

Instructions for use and handling
S.c. administration: The doctor or healthcare professional must give precise instructions to patients who will be administering the drug to themselves by s.c. injection.
To reduce injection site pain, it is recommended that the solution for injection be allowed to reach room temperature. Repeated injection at short intervals at the same site should be avoided.
Ampoules should not be opened until immediately prior to use. Any remaining solution which is not needed should be discarded.
To avoid contamination, it is recommended that the cap of multiple-dose containers should not be pierced more than 10 times.

I.v. infusion: Parenteral drugs should be visually examined for discoloration and particulate matter prior to administration.
Sandostatin (octreotide acetate) remains physically and chemically stable for 24 hours in sterile physiological saline or a sterile 5% dextrose solution (glucose). Nevertheless, the use of physiological saline rather than glucose is recommended because Sandostatin can influence glucose homoeostasis. The diluted solutions remain physically and chemically stable for 24 hours at temperatures below 25°C, but they should be used immediately for reasons of microbial purity. The user must store the solution at 2 to 8°C if it is not used immediately. The solution must be allowed to reach room temperature before administration. The total time between reconstitution, dilution with infusion media, storage in a refrigerator and completion of administration must not exceed 24 hours.
In cases where Sandostatin is administered intravenously, the contents of one 0.5 mg ampoule are normally dissolved in 60 ml physiological saline and the resulting solution is infused using an infusion pump. This procedure is maintained until the end of the prescribed duration of treatment. Sandostatin has also been infused at lower concentrations.

Pack sizes
1 ml ampoules: Packs containing five 0.05 mg/ml ampoules.
1 ml ampoules: Packs containing five 0.1 mg/ml ampoules.
1 ml ampoules: Packs containing five 0.5 mg/ml ampoules.
5 ml vials containing 0.2 mg/ml: Packs of 1 vial.
Not All Pack Sizes are Marketed

Manufacturer:
Delpharm Dijon, Quetigny, France for Novartis Pharma AG, Basle, Switzerland

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®= registered trademark

Novartis Pharma AG, Basle, Switzerland

This is a medicament
- A medicament is a product which affects your health, and its consumption contrary to instructions is dangerous for you.
- Follow strictly the doctor's prescription, the method of use and the instructions of the pharmacist who sold the medicament.
- The doctor and the pharmacist are experts in medicine, its benefits and risks.
- Do not by yourself interrupt the period of treatment prescribed for you.
- Do not repeat the same prescription without consulting your doctor.

Keep medicaments out of reach of children

Council of Arab Health Ministers
Union of Arab Pharmacists