Summary of Product Characteristics
SOLUGOOD
(Methylprednisolone Sodium Succinate for Injection USP 500 mg)

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1. Name of the medicinal product

SOLUGOOD

Methylprednisolone Sodium Succinate for Injection USP 500 mg

2. Qualitative and quantitative composition

SOLUGOOD is presented as a combipack comprising a powder vial and a solvent ampoule.

Powder vial
Each vial contains methylprednisolone sodium succinate USP (sterile) equivalent to 500 mg of methylprednisolone.

Solvent ampoule
Each 10 ml ampoule contains Sterile Water for Injections USP.

Excipient with known effect
This medicinal product contains sodium (approximately 58 mg per vial, arising from the methylprednisolone sodium succinate active substance), equivalent to about 2.9% of the WHO recommended maximum daily intake of 2 g of sodium for an adult.

For the full list of excipients, see section 6.1.

3. Pharmaceutical form

Powder and solvent for solution for injection.

The powder is white to almost white; the solvent is a clear, colourless solution.

4. Clinical particulars

4.1 Therapeutic indications

Methylprednisolone is indicated to treat any condition in which a rapid and intense corticosteroid effect is required, such as:

Allergic states
Bronchial asthma, severe seasonal and perennial allergic rhinitis, angioneurotic oedema and anaphylaxis.

Dermatological disease
Severe erythema multiforme (Stevens-Johnson syndrome).

Gastrointestinal diseases
Crohn's disease and ulcerative colitis.

Neurological disorders

Acute exacerbations of multiple sclerosis superimposed on a relapsing-remitting background, and cerebral oedema secondary to a cerebral tumour.

Respiratory diseases
Aspiration of gastric contents, and fulminant or disseminated pulmonary tuberculosis (with appropriate anti-tuberculosis chemotherapy).

4.2 Posology and method of administration

Methylprednisolone may be administered intravenously or intramuscularly, the preferred method for emergency use being intravenous injection given over a suitable time interval. When administered in high doses intravenously, it should be given over a period of at least 30 minutes; doses up to 250 mg should be given intravenously over a period of at least 5 minutes. Undesirable effects may be minimised by using the lowest effective dose for the minimum period. Parenteral products should be inspected visually for particulate matter and discolouration before administration.

Posology

Adults
Dosage should be varied according to the severity of the condition; the initial dose is usually between 10 and 500 mg. In the treatment of graft rejection reactions following transplantation, a dose of up to 1 g/day may be required, with 500 mg to 1 g most commonly used for acute rejection. Treatment at these doses should be limited to a 48–72 hour period until the patient's condition has stabilised, as prolonged high-dose corticosteroid therapy can cause serious corticosteroid-induced side-effects (see sections 4.4 and 4.8).

Paediatric population
For high-dose indications (such as haematological, rheumatic, renal and dermatological conditions), 30 mg/kg/day up to a maximum of 1 g/day is recommended, which may be repeated for up to three consecutive cycles, daily or on alternate days. In graft rejection following transplantation, 10 to 20 mg/kg/day for up to 3 days (maximum 1 g/day) is recommended. In status asthmaticus, 1 to 4 mg/kg/day for 1 to 3 days is recommended.

Elderly
Methylprednisolone is primarily used in acute short-term conditions, and there is no information to suggest that a change in dosage is warranted in the elderly. Treatment should, however, be planned bearing in mind the more serious consequences of the common side-effects of corticosteroids in old age, and close clinical supervision is required.

Recommendations for specific indications
In anaphylactic reactions, adrenaline or noradrenaline should be administered first for an immediate haemodynamic effect, followed by intravenous methylprednisolone with other accepted procedures. In status asthmaticus, methylprednisolone may be given at 40 mg intravenously, repeated according to patient response, or by slow intravenous infusion over several hours. In graft

rejection following transplantation, doses of 500 mg to 1 g per day have been used, usually not beyond 48–72 hours. In cerebral oedema associated with brain tumours, tapering of the dose is important to avoid a rebound rise in intracranial pressure, and it is generally recommended to aim to discontinue therapy after a total of about 10 days. In acute exacerbations of multiple sclerosis in adults, the recommended dose is 500 mg to 1 g daily for 3 days, given as an intravenous infusion over at least 30 minutes. In other indications, the initial dose (10 to 500 mg) depends on the condition being treated. Corticosteroid therapy is an adjunct to, and not a replacement for, conventional therapy.

Method of administration

For intravenous or intramuscular use after reconstitution. For instructions on reconstitution and dilution, see section 6.6. This medicinal product is not recommended for intrathecal or epidural administration.

4.3 Contraindications

- Hypersensitivity to the active substance or to any of the excipients listed in section 6.1.
- Systemic fungal infection, and systemic infection unless specific anti-infective therapy is employed.
- Cerebral oedema associated with malaria.
- Administration of live or live, attenuated vaccines in patients receiving immunosuppressive doses of corticosteroids.
- Intramuscular corticosteroid preparations are contraindicated in idiopathic thrombocytopenic purpura.

Severe medical events have been associated with the intrathecal and epidural routes of administration; these routes are not recommended (see section 4.4).

4.4 Special warnings and precautions for use

Undesirable effects may be minimised by using the lowest effective dose for the minimum period, with frequent patient review to titrate the dose against disease activity (see section 4.2).

Adrenal suppression and withdrawal

Adrenal cortical atrophy develops during prolonged therapy and may persist for months after stopping treatment. Withdrawal after prolonged therapy must therefore always be gradual, tapered over weeks or months according to dose and duration, to avoid acute adrenal insufficiency, rebound exacerbation of disease or a steroid withdrawal syndrome. During prolonged therapy, any intercurrent illness, trauma, anaesthesia or surgical procedure requires a temporary increase in dosage. Abrupt withdrawal of doses up to 32 mg daily of methylprednisolone for up to 3 weeks is unlikely to cause clinically relevant HPA-axis suppression in most patients; gradual withdrawal should nevertheless be considered in patients who have received repeated or prolonged courses, doses above 32 mg daily, repeated evening doses, or who may have other reasons for adrenocortical

insufficiency. Patients should carry a 'Steroid Treatment' card. Since mineralocorticoid secretion may be impaired, salt and/or a mineralocorticoid should be administered concurrently where appropriate.

Infections

Corticosteroids may mask signs of infection, reduce resistance and impair the localisation of infection, and new infections (viral, bacterial, fungal, protozoal or helminthic) may appear; the risk increases with dose. Anaphylactic reactions have occurred rarely with parenteral corticosteroid therapy, and appropriate precautions should be taken, especially in patients with a history of drug allergy. Chickenpox is of serious concern, as it may be fatal in immunosuppressed patients; non-immune patients exposed while receiving systemic corticosteroids (or within the previous 3 months) require passive immunisation with varicella/zoster immunoglobulin within 10 days of exposure. Measles may follow a more serious or fatal course; exposed patients should seek medical advice promptly. Live vaccines are contraindicated at immunosuppressive doses; the response to inactivated vaccines may be diminished. In active tuberculosis, use should be restricted to fulminating or disseminated disease managed together with appropriate anti-tuberculous therapy; patients with latent tuberculosis or tuberculin reactivity require close observation and chemoprophylaxis during prolonged therapy.

Psychiatric effects

Potentially severe psychiatric reactions may occur, ranging from euphoria, insomnia, mood swings and personality change to severe depression and frank psychosis; symptoms typically emerge within a few days or weeks. Patients and carers should seek medical advice if worrying psychological symptoms develop, especially depressed mood or suicidal ideation, and should be alert to disturbances occurring during or after dose tapering. Particular care is required in patients with existing or previous severe affective disorders, in themselves or their first-degree relatives.

Cardiac and vascular effects

Care is required in patients receiving cardioactive drugs such as digoxin because of steroid-induced electrolyte disturbance and potassium loss. Cardiac arrhythmias, circulatory collapse and cardiac arrest have been reported following rapid administration of large intravenous doses (more than 0.5 g over less than 10 minutes); bradycardia has been reported during or after large doses and may be unrelated to the speed or duration of infusion. Systemic corticosteroids should be used with caution in congestive heart failure and hypertension, and in patients predisposed to thromboembolic disorders.

Ocular effects

Prolonged use may produce posterior subcapsular and nuclear cataracts (particularly in children) and glaucoma with possible optic nerve damage; frequent ophthalmic monitoring is necessary, and secondary fungal and viral eye infections may be enhanced. Visual disturbance may be reported; patients

presenting with blurred vision or other visual symptoms should be considered for ophthalmological referral for possible causes including cataract, glaucoma or central serous chorioretinopathy.

Hepatobiliary effects

Drug-induced liver injury, including acute hepatitis and liver enzyme increases, can result from cyclical pulsed intravenous methylprednisolone (usually at an initial dose ≥ 1 g/day); onset may be several weeks or longer, and resolution has generally been observed after discontinuation. Appropriate monitoring is required.

Scleroderma renal crisis

Caution is required in patients with systemic sclerosis, as an increased incidence of (possibly fatal) scleroderma renal crisis, with hypertension and decreased urinary output, has been observed. Blood pressure and renal function should be checked routinely.

Other precautions

A higher mortality was observed in septic-shock patients with elevated serum creatinine or secondary infection in a clinical study, and methylprednisolone should not be used to treat septic shock. Systemic corticosteroids should not be used routinely to treat traumatic head injury, as an increased mortality has been demonstrated. Intramuscular injection should avoid the deltoid area because of the risk of tissue atrophy. Co-treatment with CYP3A inhibitors, including cobicistat-containing products, is expected to increase the risk of systemic side-effects and should be avoided unless the benefit outweighs the risk. An acute myopathy may occur with high doses, particularly in patients with disorders of neuromuscular transmission or receiving neuromuscular blocking drugs. Particular care and frequent monitoring are required in patients with osteoporosis, hypertension or heart failure, diabetes mellitus (or a family history), previous corticosteroid-induced myopathy, hepatic or renal impairment, epilepsy, peptic ulceration, fresh intestinal anastomoses, ulcerative colitis, diverticulitis, ocular herpes simplex, Cushing's syndrome or exanthematous infectious disease.

Paediatric population

Corticosteroids cause growth retardation in infancy, childhood and adolescence, which may be irreversible; treatment should be limited to the minimum dose for the shortest possible time and, where possible, administered as a single dose on alternate days to minimise HPA-axis suppression and growth retardation. Infants and children on prolonged therapy are at special risk from raised intracranial pressure.

Elderly

The common adverse effects of systemic corticosteroids may have more serious consequences in old age, especially osteoporosis, hypertension, hypokalaemia, diabetes, susceptibility to infection and thinning of the skin; close clinical supervision is required.

Excipient information

This medicinal product contains sodium (approximately 58 mg per vial); this should be taken into consideration in patients on a controlled sodium diet.

4.5 Interaction with other medicinal products and other forms of interaction

Methylprednisolone is a cytochrome P450 substrate metabolised mainly by CYP3A4.

- CYP3A4 inhibitors (e.g. erythromycin, clarithromycin, ketoconazole, itraconazole, diltiazem, ciclosporin, HIV protease inhibitors and cobicistat-containing products) may decrease clearance and increase plasma concentrations of methylprednisolone; the dose should be titrated to avoid steroid toxicity, and cobicistat combinations avoided where possible.
- CYP3A4 inducers (e.g. rifampicin, rifabutin, carbamazepine, phenobarbital, phenytoin, primidone and aminoglutethimide) enhance metabolism and may reduce the effect of methylprednisolone; an increased dose may be required.
- Ciclosporin: mutual inhibition of metabolism occurs, and convulsions have been reported with concurrent use.
- Anticholinesterases: steroids may reduce their effect in myasthenia gravis.
- Neuromuscular blocking agents (e.g. pancuronium): partial reversal of the block, and an increased risk of acute myopathy with high corticosteroid doses.
- Hypoglycaemic agents (including insulin), antihypertensives and diuretics: their effects are antagonised by corticosteroids.
- Potassium-depleting agents (loop and thiazide diuretics, acetazolamide, carbenoxolone, amphotericin B, xanthines, beta-2 agonists): enhanced hypokalaemic effect; monitor potassium.
- Coumarin anticoagulants: the anticoagulant effect may be enhanced; monitor INR/prothrombin time.
- Salicylates and NSAIDs: corticosteroids increase the renal clearance of salicylates (withdrawal may cause salicylate intoxication), and there is an increased risk of gastrointestinal bleeding; use with caution.
- Antipsychotics and sympathomimetics (e.g. salbutamol): dose adjustment may be required, with a potential for altered CNS control or increased response.

4.6 Fertility, pregnancy and lactation

Pregnancy

Animal studies have shown that high corticosteroid doses in pregnant females may cause foetal malformations; however, corticosteroids do not appear to cause congenital malformations in humans. Methylprednisolone sodium succinate should be used during pregnancy only in critical cases, as human studies cannot establish its safety. Some corticosteroids cross the placenta readily. An increased

frequency of low birth weight has been observed in children of mothers treated with corticosteroids, and children exposed to high doses in utero should be monitored for adrenal insufficiency. Cataract has been observed in neonates whose mothers received long-term corticosteroid treatment during pregnancy.

Breast-feeding

Corticosteroids are excreted in breast milk and may suppress growth and endogenous glucocorticoid production in the breast-fed infant. Corticosteroids should be used in lactating mothers only if the expected benefit outweighs the possible risk to the child.

Fertility

There is no evidence that corticosteroids impair fertility, although corticosteroid treatment can lead to menstrual irregularities in women.

4.7 Effects on ability to drive and use machines

The effect of methylprednisolone on the ability to drive or use machinery has not been systematically evaluated. Undesirable effects such as dizziness, vertigo, visual disturbances and fatigue are possible after treatment with corticosteroids; if affected, patients should not drive or operate machinery.

4.8 Undesirable effects

Summary of the safety profile

Methylprednisolone therapy is normally short-term; nevertheless, side-effects attributable to corticosteroid therapy should be recognised, particularly with high-dose therapy (see section 4.4). Severe medical events have been reported in association with the intrathecal and epidural routes of administration, including arachnoiditis, gastrointestinal and bladder dysfunction, headache, meningitis, paraparesis or paraplegia, seizure and sensory disturbances.

Tabulated list of adverse reactions

Frequency categories are defined as: common (≥ 1/100 to < 1/10); uncommon (≥ 1/1,000 to < 1/100); rare (≥ 1/10,000 to < 1/1,000); and not known (cannot be estimated from the available data).

System Organ Class	Frequency	Undesirable effects
Infections and infestations	Not known	Infection (including increased susceptibility to and severity of infections, with suppression of clinical signs and symptoms); opportunistic infection; recurrence of dormant tuberculosis; peritonitis
Neoplasms benign, malignant and unspecified	Not known	Kaposi's sarcoma
Blood and lymphatic system disorders	Not known	Leukocytosis
Immune system disorders	Not known	Drug hypersensitivity (including anaphylactic and anaphylactoid reactions)

System Organ Class	Frequency	Undesirable effects
Endocrine disorders	Not known	Cushingoid; hypothalamic-pituitary-adrenal axis suppression; steroid withdrawal syndrome
Metabolism and nutrition disorders	Not known	Metabolic acidosis; sodium retention; fluid retention; impaired glucose tolerance; hypokalaemic alkalosis; dyslipidaemia; increased insulin or oral hypoglycaemic requirements in diabetics; lipomatosis; epidural lipomatosis; increased appetite (which may result in weight increase)
Psychiatric disorders	Not known	A wide range of psychiatric reactions, including affective disorders (irritable, euphoric, depressed or labile mood, drug dependence, suicidal ideation), psychotic reactions (mania, delusions, hallucinations, aggravation of schizophrenia), behavioural disturbances, irritability, anxiety, sleep disturbance and cognitive dysfunction (including confusion and amnesia)
Nervous system disorders	Not known	Increased intracranial pressure with papilloedema (benign intracranial hypertension); seizure; amnesia; cognitive disorder; dizziness; headache
Eye disorders	Rare	Vision blurred
	Not known	Posterior subcapsular cataracts; exophthalmos; glaucoma; papilloedema with possible optic nerve damage; corneal or scleral thinning; exacerbation of ophthalmic viral or fungal disease; chorioretinopathy
Ear and labyrinth disorders	Not known	Vertigo
Cardiac disorders	Not known	Congestive heart failure in susceptible patients; arrhythmia
Vascular disorders	Not known	Hypertension; hypotension; thrombotic events; flushing
Respiratory, thoracic and mediastinal disorders	Not known	Hiccups; pulmonary embolism
Gastrointestinal disorders	Not known	Peptic ulcer (with possible perforation and haemorrhage); gastric haemorrhage; intestinal perforation; pancreatitis; ulcerative oesophagitis; oesophagitis; oesophageal candidiasis; abdominal pain; abdominal distension; diarrhoea; dyspepsia; nausea; vomiting
Hepatobiliary disorders	Not known	Hepatitis; increased liver enzymes (alanine aminotransferase, aspartate aminotransferase)
Skin and subcutaneous tissue disorders	Not known	Ecchymosis; skin atrophy; acne; angioedema; petechiae; striae; telangiectasia; skin hypo- or hyperpigmentation; hirsutism; rash; erythema; pruritus; urticaria; hyperhidrosis; panniculitis

System Organ Class	Frequency	Undesirable effects
Musculoskeletal and connective tissue disorders	Not known	Growth retardation; osteoporosis; muscular weakness; osteonecrosis; pathological fracture; muscle atrophy; myopathy; rhabdomyolysis; neuropathic arthropathy; arthralgia; myalgia
Reproductive system and breast disorders	Not known	Irregular menstruation; amenorrhoea
General disorders and administration site conditions	Not known	Impaired wound healing; peripheral oedema; injection site reaction; fatigue; malaise; withdrawal symptoms
Investigations	Not known	Increased intraocular pressure; decreased carbohydrate tolerance; decreased blood potassium; increased urine calcium; increased blood alkaline phosphatase; increased blood urea; suppression of reactions to skin tests
Injury, poisoning and procedural complications	Not known	Tendon rupture (particularly of the Achilles tendon); spinal (vertebral) compression fracture

Peritonitis may be the primary presenting sign or symptom of a gastrointestinal disorder such as perforation, obstruction or pancreatitis. Panniculitis may occur following dose reduction or discontinuation and has been reported more frequently in the paediatric population. The occurrence of scleroderma renal crisis varies among subpopulations, the highest risk being in patients with diffuse systemic sclerosis and the lowest in limited (2%) and juvenile-onset (1%) systemic sclerosis.

Reporting of suspected adverse reactions

Reporting suspected adverse reactions after authorisation of the medicinal product is important. It allows continued monitoring of the benefit/risk balance of the medicinal product. Healthcare professionals are asked to report any suspected adverse reactions to the National Regulatory Authority.

4.9 Overdose

There is no clinical syndrome of acute overdose with methylprednisolone; reports of acute toxicity or death are rare. Acute overdose may aggravate pre-existing disease states such as gastrointestinal ulceration, electrolyte disturbances, infections, diabetes and oedema, and repeated high doses have caused hepatic necrosis and increased amylase. Bradyarrhythmias, ventricular arrhythmias and cardiac arrest have been observed following intravenous administration of high doses. Repeated frequent doses over a protracted period may result in a Cushingoid state, and the possibility of adrenal suppression should be guarded against by gradual reduction of the dose. There is no specific antidote; treatment is symptomatic and supportive, with close monitoring of fluids and electrolytes in chronic toxicity. Methylprednisolone is dialysable.

5. Pharmacological properties

5.1 Pharmacodynamic properties

Pharmacotherapeutic group: corticosteroids for systemic use, glucocorticoids. ATC code: H02AB04.

Methylprednisolone is a potent anti-inflammatory steroid, with greater anti-inflammatory potency than prednisolone and even less tendency to induce sodium and water retention; its anti-inflammatory activity is at least five times that of hydrocortisone. Methylprednisolone sodium succinate has the same metabolic and anti-inflammatory actions as methylprednisolone and, when given parenterally in equimolar quantities, the two compounds are equivalent in biological activity. An enhanced separation of glucocorticoid and mineralocorticoid effect results in a reduced incidence of sodium and water retention.

5.2 Pharmacokinetic properties

The pharmacokinetics of methylprednisolone are linear and independent of the route of administration.

Absorption

After intravenous infusion of 30 mg/kg over 20 minutes or 1 g over 30 to 60 minutes, peak plasma concentrations of approximately 20 micrograms/mL were achieved. With intramuscular injection, lower peak levels are obtained than with intravenous injection but persist longer, so that the extent of absorption is equivalent by either route.

Distribution

Methylprednisolone is widely distributed throughout the body, with an apparent volume of distribution of approximately 1.4 L/kg, and readily crosses the blood-brain barrier (peak cerebrospinal fluid levels being about 5 to 6% of plasma levels). It is approximately 77% bound to plasma proteins (mainly globulin) and crosses the placental barrier. It may also be a substrate for P-glycoprotein.

Biotransformation

Methylprednisolone sodium succinate is rapidly and extensively hydrolysed in vivo by cholinesterases to free methylprednisolone, which is metabolised in the liver, primarily via CYP3A4, to inactive metabolites (mainly 20α- and 20β-hydroxymethylprednisolone).

Elimination

The mean elimination half-life of total methylprednisolone is in the range of 1.8 to 5.2 hours, and total body clearance is approximately 15 to 16 L/hour. Metabolites are excreted mainly in the urine; following an intravenous radiolabelled dose, about 75% of radioactivity was recovered in the urine within 96 hours.

5.3 Preclinical safety data

Non-clinical data reveal no unexpected hazards based on conventional studies of safety pharmacology and repeated-dose toxicity; the toxicities observed were those expected with continued exposure to exogenous adrenocortical steroids

(including effects on carbohydrate, electrolyte, fluid and protein metabolism, blood cells and lymphatic tissue, and atrophy of the adrenal cortex). No evidence of genetic or chromosomal mutation was obtained in the tests performed, and there is no evidence of carcinogenicity of corticosteroids; no long-term carcinogenicity studies have been conducted, as the substance is intended for short-term use. In reproductive studies, teratogenic effects were seen in rats at subcutaneous doses, whereas no teratogenic effects were detected in mice or rats at the intraperitoneal doses studied; animal data on fertility are insufficient.

6. Pharmaceutical particulars

6.1 List of excipients

Powder vial

The sterile powder contains the active substance (methylprednisolone sodium succinate) and no other listed excipients.

Solvent ampoule

- Water for Injections

6.2 Incompatibilities

To avoid compatibility problems, methylprednisolone should be administered separately from other medicinal products, and only in the diluents mentioned in section 6.6.

6.3 Shelf life

36 months.

6.4 Special precautions for storage

Store below 30°C. Protect from light. Do not freeze. Keep out of the reach of children.

6.5 Nature and contents of container

A combipack containing one 500 mg clear glass vial of powder and one 10 ml ampoule of Sterile Water for Injections USP.

6.6 Special precautions for disposal and other handling

Reconstitute the powder with the accompanying 10 ml of Sterile Water for Injections. For intravenous infusion, the reconstituted solution may be further diluted with 5% dextrose in water, isotonic (0.9%) saline, or 5% dextrose in isotonic saline. The solution should be inspected visually for particulate matter and discolouration before administration and used immediately after reconstitution. Any unused medicinal product or waste material should be disposed of in accordance with local requirements.

7. Marketing Authorisation Holder

GOODMED HEALTHCARE LTD

P.O. Box 76337, Nairobi, Kenya.

Manufacturer
OM BIOMEDIC PVT. LTD.

Plot No. 7 & 8, National Highway No. 8, Vasai Phata, Vasai (East), Thane 401208, Maharashtra State, India.

8. Marketing Authorisation Number

CTD12045

9. Date of first authorisation / renewal of the authorisation

03.07.2026

10. Date of revision of the text

02.07.2026