SUMMARY OF PRODUCT CHARACTERISTICS

1. NAME OF THE MEDICINAL PRODUCT

Prednisolon G.L. Pharma 5 mg tablets

2. QUALITATIVE AND QUANTITATIVE COMPOSITION

Each tablet contains 5 mg of prednisolone.

Excipient with known effect:
Each tablet contains 121 mg lactose monohydrate.

For the full list of excipients, see section 6.1.

3. PHARMACEUTICAL FORM

Tablet

Round, flat, bevelled, white tablets, about 7 mm in diameter and with a break line on one side and debossed with “5” on the other side.

The tablet can be divided into equal doses.

4. CLINICAL PARTICULARS

4.1 Therapeutic indications

Unspecified therapy in conditions where the anti-inflammatory and immunosuppressive effects of prednisolone are desired. Examples include rheumatoid arthritis, SLE, certain kinds of vasculitis, such as temporal arteritis and periarteritis nodosa, sarcoidosis, bronchial asthma, ulcerative colitis, hemolytic anaemia and granulocytopenia, as well as serious allergic conditions.

Treatment of tumours, in certain cases of acute leukaemia, lymphoma, breast cancer and prostate cancer.

4.2 Posology and method of administration

Posology

Individual The dosage generally depends on the nature and severity of the disease. Dosing should begin with a dose that provides the desired control of the disease. The dosage should be reduced to the lowest dose that provides adequate control of the disease. Withdrawal should always be considered depending on the indication and disease activity.

After long-term treatment (for adults usually longer than 3 weeks), withdrawal should be done gradually over weeks or months, depending on the dose and duration of treatment. Gradual discontinuation should also be considered after short-term treatment with higher doses or in patients with risk factors for adrenal insufficiency. The gradual discontinuation should be adapted individually, but the majority of adult patients can tolerate dose reductions of 2.5 mg every three to seven days until a dose corresponding to 5–10 mg/day.

Adults
Inflammatory diseases:
The daily oral dose is usually between 5 and 60 mg depending on the nature and the severity of the disease.

Replacement treatment:
The recommended starting dose is 5 mg in the morning and half the morning dose in the evening.

Pediatric population
It is important that the maintenance dose in children is gradually reduced to the lowest possible dose that provides adequate disease control and a minimum of side effects due to the risk of growth retardation (see sections 4.4 and 4.8).

Elderly
No special dose adjustment is required for elderly patients. Long-term use of corticosteroids in the elderly people may lead to an increase in overt and latent diseases (see sections 4.4 and 4.8).

Impaired liver function
Dose adjustment may be required, as patients with impaired liver function may be at greater risk of adverse events due to reduced plasma protein binding in hypoalbuminemia.

Renal insufficiency
No special dose adjustment is necessary

Method of administration
Oral use.

4.3 Contraindications

- Hypersensitivity to the active substance or to any of the excipients listed in section 6.1.
- Systemic fungal infections

Administration of live vaccines is contraindicated in patients receiving immunosuppressive doses of corticosteroids.

In general, none of the contraindications apply in conditions when treatment with prednisolone may be lifesaving.

4.4 Special warnings and precautions for use

As the complications of glucocorticoid treatment depend on the dose and length of treatment, a risk/benefit assessment must be made in each case concerning dose and treatment length, as well as if daily or intermittent treatment must be used.

The lowest possible corticosteroid dose must be used to control the disease being treated. When dose reduction is possible, this must occur gradually.

Immunosuppressive effects/increased susceptibility to infection
Glucocorticoids, including prednisolone, may increase susceptibility to infection, mask some symptoms of infection, and new infections may appear during treatment. Infections caused by viruses, bacteria, fungi, protozoa or helminths may be associated with the use of corticosteroids

alone or in combination with other immunosuppressive agents that affect cellular immunity, humoral immunity, or neutrophil function. These infections may be mild but can be severe and at times, fatal. The risk of infectious complications increases with increased doses.

Glucocorticoids should not be given in case of infections without concomitant causal treatment.

Chicken pox and measles can be more serious or even fatal in non-immunized children and adults treated with corticosteroids. Children or adults who have not had these diseases and take immunosuppressant doses of corticosteroids should be warned to avoid exposure to chicken pox and measles and, if exposed, to obtain medical advice.

The use of prednisolone in active tuberculosis should be restricted to those cases of fulminating or disseminated tuberculosis in which the corticosteroid is used to treat the disease in combination with an appropriate tuberculosis treatment. If corticosteroids are indicated in patients with latent tuberculosis or tuberculin reactivity, close observation is necessary as reactivation of the disease may occur. During prolonged corticosteroid therapy, these patients should receive tuberculosis prophylaxis.

High doses of corticosteroids may interfere with active immunisation. Vaccination with live vaccine should be performed under close monitoring and not to patients on long-term treatment with immunosuppressive doses of corticosteroids.

Immune system disorders
Because rare skin reactions and anaphylactic/anaphylactoid reactions have occurred in patients being treated with corticosteroids, appropriate precautionary measures should be taken before administration, especially when the patient has previously had an allergic reaction to any drug.

Endocrine disorders
Long-term treatment with pharmacological doses of corticosteroids may lead to secondary adrenocortical insufficiency. The risk can be reduced by administering the treatment every second day (see section 4.2).

Patients on corticosteroid maintenance therapy who are subjected to unusual stress (e.g. infection, surgery, trauma) require increased corticosteroid dosage before, during, and after the stressful situation.

Abrupt withdrawal of the treatment may lead to acute adrenal insufficiency, which can be fatal. The risk of secondary adrenocortical insufficiency can be minimised by gradual reduction of dosage. This type of relative insufficiency may persist for months after discontinuation of therapy; therefore, in any situation of stress occurring during that period, hormone therapy should be reinstituted. Since mineralocorticoid secretion may be impaired, salt and/or a mineralocorticoid should be administered concurrently.

A “steroid withdrawal syndrome,” seemingly unrelated to adrenocortical insufficiency, may also occur following abrupt discontinuation of glucocorticoids. This syndrome includes symptoms such as anorexia, nausea, vomiting, lethargy, headache, fever, joint pain, desquamation, myalgia, weight loss, and/or hypotension. These effects are thought to be due to the sudden change in glucocorticoid concentration rather than low corticosteroid levels.

There is an enhanced effect of corticosteroids in patients with hypothyroidism and those with liver cirrhosis.

Pheochromocytoma-related crisis, which can be fatal, have been reported after administration of systemic corticosteroids. Corticosteroids should only be administered to patients with suspected

or identified pheochromocytoma after consideration of the individual risk/benefit.

Metabolism and nutrition disorders
Corticosteroids, including prednisolone, can increase blood glucose, worsen pre-existing diabetes, and increase the risk of developing diabetes in patients who are on long-term treatment with corticosteroids.

Psychiatric disorders
Potentially serious psychiatric disorders may occur during treatment with corticosteroids, including prednisolone. This can range from euphoria, sleep disorders, mood swings, personality changes, and severe depression to psychotic manifestations. Also, existing emotional instability or psychotic tendencies may be aggravated by corticosteroids (see section 4.8). Symptoms typically emerge within a few days or weeks of starting treatment. Most reactions resolve after dose reduction or withdrawal, although specific treatment may be necessary.

Psychiatric effects have been reported upon withdrawal of corticosteroids; the frequency is unknown. Patients/caregivers should be encouraged to seek medical attention if psychiatric symptoms develop in the patient, especially if depressed mood or suicidal ideation is suspected. Patients/caregivers should be alert to possible psychiatric disturbances that may occur either during or immediately after dose tapering/withdrawal of systemic steroids.

Nervous system disorders
Corticosteroids should be used with caution in patients with seizure disorders.

Cardiac disorders
Adverse effects of glucocorticoids on the cardiovascular system, such as dyslipidaemia and hypertension, may predispose treated patients with existing cardiovascular risk factors to additional cardiovascular events at high doses and prolonged treatment time. Accordingly, corticosteroids should be employed judiciously in such patients, and attention should be paid to risk modification and additional cardiac monitoring if needed. Low-dose and alternate-day therapy may reduce the incidence of complications in corticosteroid therapy.

Vascular disorders
Because cortisone has been reported in rare cases to increase blood coagulability and thus could precipitate intravascular thrombosis, thromboembolism, and thrombophlebitis, corticosteroids should be used cautiously in patients with thromboembolic disorders.

Gastrointestinal disorders
High doses of corticosteroids may cause acute pancreatitis.

There is no universal data that establishes whether corticosteroids cause gastric ulcers. Glucocorticoid therapy may mask peritonitis or other signs or symptoms associated with gastrointestinal disorders such as perforation, obstruction or pancreatitis. In combination with NSAIDs, the risk of developing gastrointestinal ulcers is increased.

Corticosteroids should be used with caution in nonspecific ulcerative colitis if there is a probability of impending perforation, abscess or other pyogenic infection, diverticulitis, fresh intestinal anastomoses, or active or latent peptic ulcer.

Hepatobiliary disorders
Hepatobiliary disorders have been reported in rare cases, and in the majority of these cases, the condition was reversible after discontinuation of therapy. Therefore, appropriate monitoring is required.

Musculoskeletal and connective tissue disorders
Acute myopathy has been reported with the use of high doses of corticosteroids, most often occurring in patients with disorders of neuromuscular transmission (e.g., myasthenia gravis) or in patients receiving concomitant therapy with anticholinergics, such as neuromuscular blocking drugs (e.g., pancuronium) (see section 4.5). This acute myopathy is generalised, may involve ocular and respiratory muscles, and may result in quadriparesis. Elevations of creatine kinase may occur. Clinical improvement or recovery after stopping corticosteroids may require weeks to years.

Corticosteroids should be used with caution in patients with osteoporosis.

Renal and urinary disorders
Corticosteroids should be used with caution in patients with kidney insufficiency.

Acute kidney crisis (scleroderma kidney crisis)
Caution is required in patients with systemic sclerosis because of an increased incidence of (possibly fatal) scleroderma kidney crisis with hypertension and decreased urinary output observed with a daily dose of 15 mg or more prednisolone. Blood pressure and kidney functions (S-creatinine) should, therefore, be routinely checked. Blood pressure should be carefully controlled When a kidney crisis is suspected.

Effect on electrolytes and fluid balance
Systemic corticosteroids should be used with caution in patients with heart failure or hypertension. High doses of hydrocortisone or cortisone can cause elevation of blood pressure, salt and water retention, and increased excretion of potassium. These effects are less likely to occur with the synthetic derivatives except when used in large doses. Dietary salt restriction and potassium supplementation may be necessary.

All corticosteroids increase calcium excretion.

Eye disorders
Visual disturbance may be reported with systemic and topical corticosteroid use. If a patient presents with symptoms such as blurred vision or other visual disturbances, the patient should be considered for referral to an ophthalmologist for evaluation of possible causes, which may include cataract, glaucoma or rare diseases such as central serous chorioretinopathy (CSCR) which have been reported after use of systemic and topical corticosteroids.

Tumour lysis syndrome (TLS)
Tumour lysis syndrome (TLS) was reported in patients with malignancies, including haematological malignancies and solid tumours, following the use of systemic corticosteroids alone or in combination with other cytostatics. Patients at a high risk of TLS, for example patients with tumours with a high rate of cell division, high tumour burden and high sensitivity to cytotoxic agents, must be closely monitored and suitable precautions must be taken.

Paediatric population
Corticosteroids cause growth retardation in infants, children and adolescents and, therefore, long-term administration of pharmacological doses should be avoided. If prolonged therapy is necessary, the growth and development of infants and children should be closely monitored (see section 4.2).

Infants and children on prolonged corticosteroid therapy are at special risk from elevated intracranial pressure.

Lactose
This medicinal product contains lactose, as an excipient, and patients with rare hereditary problems of galactose intolerance, total lactase deficiency or glucose-galactose malabsorption should not take this medicine.

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

4.5 Interaction with other medicinal products and other forms of interaction

The following combinations with Prednisolon G.L. Pharma may require dose adjustment.

Phenobarbital, phenytoin and carbamazepine
Phenobarbital (which is also a metabolite of primidone), phenytoin and carbamazepine, either as monotherapy or in combination, induce the metabolism of hydrocortisone, prednisolone and methylprednisolone (shown in children with asthma), resulting in a need for a dose increase. This interaction probably concerns the whole group of glucocorticoids.

Non-steroidal anti-inflammatory drugs
- The incidence of gastrointestinal bleeding and ulceration may increase if corticosteroids are given together with an NSAID.
- Corticosteroids may increase the clearance of high doses of acetylsalicylic acid, which can lead to lower salicylate levels in serum. When withdrawing corticosteroid treatment, the salicylate levels in serum can increase, which will probably lead to an increased risk of toxic effects from salicylate.

Anti-diabetic agents
Glucocorticoids increase blood sugar content. Patients with diabetes mellitus who simultaneously receive insulin and/or oral hypoglycaemic products may need the doses of such treatment adjusted.

Oestrogens (including oral contraceptives containing oestrogens)
Oestrogens increase the concentration of transcortin. The effect of glucocorticoids that bind to transcortin may be potentiated, and dose adjustment may be needed if oestrogens are added to or removed from a stable treatment regimen.

Potassium-reducing agents
Potassium-reducing diuretics (e.g. thiazides, furosemide, ethacrynic acid) and other medicinal products that reduce the quantity of potassium, such as amphotericin B, xanthine's and beta-2 agonists, may potentiate the potassium-reducing effect of glucocorticoids. Serum-potassium should be monitored closely in patients who receive glucocorticoids and potassium-reducing products.

Rifampicin
Rifampicin induces the microsomal oxidation of glucocorticoids (hydrocortisone, prednisolone, methylprednisolone). This leads to increased steroid requirements during treatment with rifampicin and reduced steroid requirements after such treatment.

Isoniazid
Prednisolone also has a potential effect that leads to increased acetylation rate and clearance of isoniazid.

Oral anticoagulants
Changed effects from anticoagulants have been reported when given in combination with prednisolone. The prothrombin time (INR) should be monitored during the treatment.

CYP3A inhibitors, including cobicistat-containing products
These are expected to increase the risk of systemic side effects. The combination should be avoided unless the benefit outweighs the increased risk of systemic corticosteroid side effects, in which case patients should be monitored for systemic corticosteroid side effects.

Anticholinergic, neuromuscular blockers
Corticosteroids can affect the effect of anticholinergics.
- Acute myopathy has been reported in concomitant use of high doses of corticosteroids and anticholinergics, such as neuromuscular blockers (see section 4.4).
- Antagonism with the neuromuscular blocking effect of pancuronium and vecuronium has been reported in patients who take glucocorticosteroids. This interaction may be expected with all competitive neuromuscular blockers.

Anticholinesterases
Interaction between glucocorticoids and anticholinesterases such as ambenonium, neostigmine and pyridostigmine may lead to significant weakness in persons with myasthenia gravis. If possible, treatment with anticholinesterase should be discontinued at least 24 hours before administering glucocorticoid treatment.

4.6 Fertility, pregnancy and lactation

Pregnancy
Animal studies have shown that corticosteroids may cause various types of malformations (cleft palate, skeletal malformation, see section 5.3). The relevance to humans is unknown. Reduced placental and birth weight have been established following long-term treatment in humans and animals. There is also a risk of long-term treatment of adrenocortical failure in the newborn child. During pregnancy, corticosteroids should, therefore, be given after special consideration.

Breastfeeding
Prednisolone passes into breast milk, but the risk of effect on the child is considered unlikely at therapeutic doses.

Fertility
Animal studies have shown that corticosteroids impair fertility (see section 5.3).

4.7 Effects on the ability to drive and use machines

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

4.8 Undesirable effects

Apart from substitution therapy, treatment with corticosteroids always constitutes an overdose compared with the physiological state. Side effects occur predominantly during long-term treatment but also depend on dose size and individual sensitivity.

The following undesirable effects have been observed and reported during treatment with prednisolone with the following frequencies: 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), unknown (cannot be estimated from the available data).

System Organ Class	Very common	Common	Uncommon	Rare	Not known
Infections and infestations		Opportunistic infection Activation of infection (e.g. tuberculosis)			
Blood and lymphatic system disorders					Leukocytosis (due to redistribution of intravascular granulocytes)
Immune system disorders					Drug hypersensitivity Anaphylactic reaction Anaphylactoid reaction
Endocrine disorders		Inhibition of endogenous ACTH and cortisol secretion, Cushingoid. Growth inhibition (in children)			Steroid withdrawal syndrome (see section 4.4) Pheochromocytosis-related crisis (see section 4.4)
Metabolism and nutrition disorders		Hypokalaemia Sodium retention Increased gluconeogenesis catabolic effects Osteoporosis			Metabolic acidosis Fluid retention Alkalosis hypokalaemic Dyslipidaemia Glucose tolerance impaired (diabetes mellitus may be exacerbated, and latent diabetes may manifest) Lipomatosis Increased appetite (which may result in weight increase)
Psychiatric disorders			Activation of previous psychological disturbances (high dose)	Depression and mania in patients with no prior history of mental illness	Affective disorder (including euphoric mood, affect lability, drug dependence, suicidal ideation) Psychotic disorder (including delusion, hallucination, and schizophrenia) Mental disorder Personality change

System Organ Class	Very common	Common	Uncommon	Rare	Not known
					Confusional state Anxiety Mood swings Abnormal behaviour Insomnia Irritability
Nervous system disorders				Benign intracranial hypertension	Epidural lipomatosis Seizure Amnesia Cognitive disorder Dizziness Headache
Eye disorders			Cataract Glaucoma		Central serous chorioretinopathy (see section 4.4) Exophthalmos Blurred vision (see section 4.4)
Cardiac disorders					Cardiac failure (in susceptible patients) Bradycardia*
Vascular disorders		Oedema Hypertension			Thromboembolic events
Respiratory, thoracic and mediastinal disorders					Hiccups
Gastrointestinal disorders					Peptic ulcer (with possible perforation and haemorrhage) Intestinal perforation Pancreatitis Oesophagitis ulcerative Abdominal distension Abdominal pain Diarrhoea Dyspepsia Nausea
Skin and subcutaneous tissue disorders		Skin atrophy Poor wound healing			Angioedema Hirsutism Petechiae Ecchymosis Erythema Hyperhidrosis Skin striae

System Organ Class	Very common	Common	Uncommon	Rare	Not known
					Pruritus Urticaria Acne
Musculoskeletal and connective tissue disorders		Muscle atrophy		Aseptic bone necrosis Tendon rupture	Muscular weakness Myalgia Myopathy Pathological fracture Neuropathic arthropathy Arthralgia
Renal and urinary disorders					Acute renal crisis (renal crisis in scleroderma)**
Reproductive system and breast disorders					Menstruation irregular
General disorders and administration site conditions					Fatigue Malaise
Investigations					Urine calcium increased Alanine aminotransferase increased Aspartate aminotransferase increased Blood alkaline phosphatase increased Blood urea increased Suppression of reactions to skin tests[1]

[1] not a MedDRA term.

* Following high doses

** Acute renal crisis (scleroderma renal crisis)

Amongst the different subpopulations, the occurrence of scleroderma renal crisis varies. The highest risk has been reported in patients with diffuse systemic sclerosis. The lowest risk has been reported in patients with limited systemic sclerosis (2%) and juvenile-onset systemic sclerosis (1%)

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 via [to be completed nationally]

4.9 Overdose

Reports of acute toxicity and/or death following an overdose of glucocorticoids are rare. Acute overdose may aggravate pre-existing disease states, e.g. ulcer, electrolyte imbalance, infections and oedema.

Treatment
Generally, it is not required. If justified, gastric lavage, charcoal. In the event of an overdose, no specific antidote is available; treatment is supportive and symptomatic.

5. PHARMACOLOGICAL PROPERTIES

5.1 Pharmacodynamic properties

Pharmacotherapeutic group: glucocorticoids
ATC code: H02AB06

Synthetic glucocorticoid has anti-inflammatory, immunosuppressive, and anti-allergenic effects. Prednisolone has a per unit of weight anti-inflammatory effect 4-5 times higher than cortisone but affects electrolyte metabolism to a lesser extent. The mechanism of action has not yet been fully verified.

5.2 Pharmacokinetic properties

Absorption
Prednisolone is rapidly absorbed from the gastrointestinal tract when administered orally. Peak plasma concentrations are reached 1 to 2 hours after oral administration. The usual plasma half-life is 2 to 4 hours. Its initial absorption, but not its overall bioavailability, is affected by food.

Distribution
Prednisolone is extensively bound to plasma proteins and has a high affinity to transcortin. The volume of distribution and clearance is reported to increase from low to moderate doses.

Biotransformation
Prednisolone is metabolised primarily in the liver to a biologically inactive compound. Prednisolone can be reversibly converted to prednisone by 11β-hydroxysteroid dehydrogenase. The absolute bioavailability of prednisolone is, on average, 82% compared with intravenously administered prednisolone after a single dose of 10 mg. With usual dosage, the effect duration is 12-36 hours.

Elimination
Prednisolone is excreted in the urine as free and conjugated metabolites, with small amounts of unchanged prednisolone. More than 90% of the administered amount is excreted in the urine. 7-15% is excreted in unchanged form.

5.3 Preclinical safety data

In animal trials, corticosteroids have been shown to cause various types of malformation (cleft palate, skeletal malformations). After long-term treatment in animals, reduced placenta and birth weights have been observed.

Corticosteroids have been shown to reduce fertility when administered to rats.

6. PHARMACEUTICAL PARTICULARS

6.1 List of excipients

Lactose monohydrate
Cellulose powdered
Sodium starch glycolate (Type A)
Silica, colloidal anhydrous
Magnesium stearate

6.2 Incompatibilities

Not applicable.

6.3 Shelf life

2 years

6.4 Special precautions for storage

Do not store above 25°C. Store in the original package in order to protect from light.

6.5 Nature and contents of container

PVC/PVDC-Aluminium blister packs with 25 or 100 tablets.

Not all pack sizes may be marketed.

6.6 Special precautions for disposal

Any unused medicinal product or waste material should be disposed of in accordance with local requirements.

7. MARKETING AUTHORISATION HOLDER

[To be completed nationally]

8. MARKETING AUTHORISATION NUMBER(S)

[To be completed nationally]

9. DATE OF FIRST AUTHORISATION/RENEWAL OF THE AUTHORISATION

[To be completed nationally]

10. DATE OF REVISION OF THE TEXT

2025-10-21