SUMMARY OF PRODUCT CHARACTERISTICS

1 NAME OF THE MEDICINAL PRODUCT

Hydrocortisone 10 mg Tablets

2 QUALITATIVE AND QUANTITATIVE COMPOSITION

Each tablet contains 10 mg hydrocortisone.

Excipient with known effect

Each tablet contains 191 mg lactose monohydrate.

For the full list of excipients, see section 6.1.

3 PHARMACEUTICAL FORM

Tablet.

A white, 10.8 mm x 7.0 mm, oval, tablet, engraved with ‘HC 10’ on one side and break-marked on both sides.

The tablet can be divided into equal doses

4 CLINICAL PARTICULARS

4.1 Therapeutic indications

Corticosteroid

- For use as replacement therapy in congenital adrenal hyperplasia in children.

- Pre-operatively, and during serious trauma or illness in children with known
  adrenal insufficiency or doubtful adrenocortical reserve.

4.2 Posology and method of administration

Posology

Dosage must be individualised according to the response of the individual patient.

Replacement therapy

Paediatric population: In chronic adrenocortical insufficiency, the dosage should be approximately 0.4 to 0.8mg/kg/day in two or three divided doses, adjusted to the needs of the individual child.

In patients requiring replacement therapy, the daily dose should be given when practicable, in two doses. The first dose in the morning should be larger than the second dose in the evening, thus simulating the normal diurnal rhythm of cortisol secretion.

Use in serious trauma or illness with known adrenal insufficiency or doubtful adrenocortical reserve

Paediatric population: Doses are generally higher than that used for chronic adrenocortical insufficiency and should be selected as appropriate for the clinical situation. Patients should be observed closely for signs that might require dosage adjustment, including changes in clinical status resulting from remissions or exacerbations of the disease, individual drug responsiveness and the effect of stress (e.g. surgery, infection, trauma). During stress it may be necessary to increase the dosage temporarily.

Pre-operative use

Anaesthetists must be informed if the patient is taking corticosteroids or has previously taken corticosteroids.

When long term treatment is to be discontinued, the dose should be gradually reduced over a period of weeks or months, depending on dosage and duration of therapy (see section 4.4).

Undesirable effects may be minimised by using the lowest effective dose for the minimum period and by administering the daily requirement as a single morning dose or whenever possible, as a single morning dose on alternate days. Frequent patient review is required to titrate the dose against disease activity.

Method of administration

For oral administration.

4.3 Contraindications

Hypersensitivity to the active substance or to any of the excipients listed in section 6.1.

Contraindicated in infections including systemic infections where anti-infective therapy has not been started.

High doses of corticosteroids impair the immune response to vaccines. Therefore the concomitant administration of live vaccines with corticosteroids should be avoided.

4.4 Special warnings and precautions for use

Adrenal suppression

Adrenal cortical atrophy develops during prolonged therapy and may persist for years after stopping treatment. Withdrawal of corticosteroids after prolonged therapy must therefore always be gradual to avoid acute adrenal insufficiency, being tapered off over weeks or months according to the dose and duration of treatment. During prolonged therapy, any intercurrent illness, trauma or surgical procedure will require a temporary increase in dosage. If corticosteroids have been stopped following prolonged therapy, they may need to be temporarily re-introduced.

Patients should carry 'steroid treatment' cards, which give clear guidance on the precautions to be taken to minimise risk and which provide details of the prescriber, drug, dosage and the duration of treatment.

Anti-inflammatory / immunosuppressive effects and infection

Suppression of inflammatory response and immune function increases the susceptibility to infections and their severity. The clinical presentation can often be atypical and serious infections such as septicaemia and tuberculosis may be masked and may reach an advanced stage before being recognized. New infections may appear during their use.

Corticosteroids may activate latent amoebiasis or strongyloidiasis or exacerbate active disease. Therefore it is recommended that latent or active amoebiasis and strongyloidiasis be ruled out before initiating corticosteroid therapy in any patient at risk of or with symptoms suggestive of either condition.

Caution should be exercised in immunocompromised patients.

Chickenpox is of particular concern since this normally minor illness may be fatal in immunosuppressed patients. Patients (or parents of children receiving hydrocortisone tablets) without a definite history of chickenpox should be advised to avoid close personal contact with chickenpox or herpes zoster. If exposed they should seek urgent medical attention. Passive immunisation with *Varicella zoster* immunoglobulin (VZIG) is needed by exposed non-immune patients who are receiving systemic corticosteroids or who have used them within the previous 3 months; this should be given within 10 days of exposure to chickenpox. If a diagnosis of chickenpox is confirmed, the illness warrants specialist care and urgent treatment. Corticosteroids should not be stopped and the dose may need to be increased.

Patients should be advised to take particular care to avoid exposure to measles and to seek immediate medical advice if exposure occurs. Prophylaxis with intramuscular normal immunoglobulin may be needed.

Live vaccines should not be given to individuals with impaired immune responsiveness caused by high doses of corticosteroids. Killed vaccines or toxoids may be given though their effects may be attenuated.

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

Particular care is required when prescribing systemic corticosteroids in patients with the following conditions and frequent patient monitoring is necessary:

a) osteoporosis (postmenopausal females are particularly at risk);
b) hypertension or congestive heart failure;
c) existing or previous history of severe affective disorders (especially previous history of steroid psychosis);
d) diabetes mellitus (or a family history of diabetes);
e) previous history of tuberculosis or characteristic appearance on a chest x-ray. The emergence of active tuberculosis can, however, be prevented by the prophylactic use of anti-tuberculous therapy;
f) glaucoma (or family history of glaucoma);
g) previous corticosteroid-induced myopathy;
h) liver failure;
i) renal insufficiency;
j) epilepsy;
k) peptic ulceration;
l) recent myocardial infarction.

During treatment, the patient should be observed for psychotic reactions, weakness, electrocardiographic changes, hypertension and untoward hormonal effects.

Corticosteroids should be used with caution in patients with hypothyroidism.

Thyrotoxic Periodic Paralysis (TPP) can occur in patients with hyperthyroidism and with hydrocortisone-induced hypokalaemia. TPP must be suspected in patients treated with hydrocortisone presenting signs or symptoms of muscle weakness, especially in patients with hyperthyroidism.

If TPP is suspected, levels of blood potassium must be immediately monitored and adequately managed to ensure the restoration of normal levels of blood potassium.

Paediatric population: Corticosteroids cause growth retardation in infancy, childhood and adolescence; this may be irreversible. Treatment should be limited to the minimum dosage for the shortest possible time (see section 4.2).

Hypertrophic cardiomyopathy was reported after administration of hydrocortisone to prematurely born infants, therefore appropriate diagnostic evaluation and monitoring of cardiac function and structure should be performed.

Withdrawal symptoms:

In patients who have received more than physiological doses of systemic corticosteroids (approximately 40 mg cortisone or equivalent) for greater than three weeks, withdrawal should not be abrupt. How dose reduction should be carried out depends largely on whether the disease is likely to relapse as the dose of systemic corticosteroids is reduced. Clinical assessment of disease activity may be needed during withdrawal. If the disease is unlikely to relapse on withdrawal of systemic corticosteroids but there is uncertainty about hypothalamic-pituitary adrenal (HPA) suppression, the dose of systemic corticosteroid may be reduced rapidly to physiological doses. Once a daily dose equivalent to 30 mg hydrocortisone is reached, dose reduction should be slower to allow the HPA-axis to recover.

Abrupt withdrawal of systemic corticosteroid treatment, which has continued up to three weeks, is appropriate if it is considered that the disease is unlikely to relapse. Abrupt withdrawal of doses of up to 160 mg daily hydrocortisone for three weeks is unlikely to lead to clinically relevant HPA-axis suppression, in the majority of patients. In the following patient groups, gradual withdrawal of systemic corticosteroid therapy should be considered even after courses lasting three weeks or less:

• patients who have had repeated courses of systemic corticosteroids, particularly if taken for greater than three weeks;

• when a short course has been prescribed within one year of cessation of long term therapy (months or years);

• patients who may have reasons for adrenocortical insufficiency other than exogenous corticosteroid therapy;

• patients receiving doses of systemic corticosteroid greater than 160 mg daily of hydrocortisone;

• patients repeatedly taking doses in the evening.

Patients and/or carers should be warned that potentially severe psychiatric adverse reactions may occur with systemic steroids (see Section 4.8). Symptoms typically emerge within a few days or weeks of starting the treatment. Risks may be higher with high doses/systemic exposure (see also Section 4.5), although dose levels do not allow prediction of the onset, type, severity or duration of reactions. Most adverse reactions resolve after either dose reduction or withdrawal of the medicine, although specific treatment may be necessary. Patients/carers should be encouraged to seek medical advice if worrying psychological symptoms develop, especially if depressed mood or suicidal ideation is suspected. Patients/carers should also be alert to possible psychiatric disturbances that may occur either during or immediately after dose tapering/withdrawal of systemic steroids, although such reactions have been reported infrequently.

Particular care is required when considering the use of systemic corticosteroids in patients with existing or a previous history of severe affective disorders in themselves or in their first degree relatives. These would include depressive or manic-depressive illness and previous steroid psychosis.

This medicine contains lactose monohydrate. Patients with rare hereditary problems of galactose intolerance, total lactase deficiency or glucose-galactose malabsorption should not take this medicine.

Visual disturbance
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.

4.5 Interaction with other medicinal products and other forms of interaction

The metabolism of corticosteroids may be enhanced and the therapeutic effects reduced by certain barbiturates (e.g. phenobarbital) and by phenytoin, rifampicin, rifabutin, primidone, carbamazepine and aminoglutethimide.

Co-treatment with CYP3A inhibitors, including cobicistat-containing products, is 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.

Mifepristone may reduce the effect of corticosteroids for 3-4 days.

Erythromycin and ketoconazole may inhibit the metabolism of corticosteroids.

Ketoconazole alone can inhibit adrenal corticosteroid synthesis and may cause adrenal insufficiency during corticosteroid withdrawal (see section 4.4).

Ritonavir may increase the plasma concentration of hydrocortisone.

Oestrogens and other oral contraceptives increase the plasma concentration of corticosteroids and dosage adjustments may be required if oral contraceptives are added to or withdrawn from a stable dosage regimen.

The growth promoting effect of somatropin may be inhibited by the concomitant use of corticosteroids.

The desired actions of hypoglycemic drugs (including insulin), antihypertensives and diuretics are antagonised by corticosteroids.

The effectiveness of coumarin anticoagulants may be affected by concurrent corticosteroid therapy and close monitoring of the INR or prothrombin time is required to avoid spontaneous bleeding.

Serum levels of salicylates, such as aspirin and benorilate, may increase considerably if corticosteroid therapy is withdrawn, possibly causing intoxication. Concomitant use of salicylates or of non-steroidal anti-inflammatory drugs (NSAIDs) with corticosteroids increases the risk of gastrointestinal bleeding and ulceration.

The potassium-depleting effects of acetazolamide, loop diuretics, thiazide diuretics and carbenoxolone are enhanced by corticosteroids and signs of hypokalaemia should be looked for during their concurrent use. The risk of hypokalaemia is increased with theophylline and amphotericin.

Corticosteroids should not be given concomitantly with amphotericin, unless required to control reactions.

The risk of hypokalaemia also increases if high doses of corticosteroids are given with high doses of sympathomimetics e.g. bambuterol, fenoterol, formoterol, ritodrine, salbutamol, salmeterol and terbutaline. The toxicity of cardiac glycosides, e.g. digoxin, is increased if hypokalaemia occurs.

Concomitant use with methotrexate may increase the risk of haematological toxicity.

High doses of corticosteroids impair the immune response and so live vaccines should be avoided (see also section 4.4).

4.6 Pregnancy and lactation

Pregnancy

The ability of corticosteroids to cross placenta varies between individual drugs; however, cortisone readily crosses the placenta.

Administration of corticosteroids to pregnant animals can cause abnormalities of foetal development including cleft palate, intra-uterine growth retardation and effects on brain growth and development. There is no evidence that corticosteroids result in an increased incidence of congenital abnormalities, such as cleft palate / lip in man. However, when administered for prolonged periods or repeatedly during pregnancy, corticosteroids may increase the risk of intra-uterine growth retardation. Hypoadrenalism may, in theory, occur in the neonate following prenatal exposure to corticosteroids but usually resolves spontaneously following birth and is rarely clinically important. As with all drugs, corticosteroids should only be prescribed when the benefits to the mother and child outweigh the risks. When corticosteroids are essential however, patients with normal pregnancies may be treated as though they were in the non-gravid states.

Breast-feeding

Corticosteroids are excreted in breast milk, although no data are available for hydrocortisone. Infants of mothers taking highdoses of systemic corticosteroids for prolonged periods may have a degree of adrenal suppression. Mothers taking pharmacological doses of corticosteroids should be advised not to breast-feed. Any maternal treatment should be carefully documented in the infant's medical records to assist in follow up.

Fertility

Patients with adrenal insufficiency have been shown to have reduced parity, which is most likely due to the underlying disease, but there is no indication that hydrocortisone in doses for replacement therapy will affect fertility.

4.7 Effects on ability to drive and use machines

Hydrocortisone has minor influence on the ability to drive and use machines. Fatigue and episodes of short lasting vertigo have been reported.

Untreated and poorly replaced adrenal insufficiency may affect the ability to drive and use machines.

4.8 Undesirable effects

The incidence of predictable undesirable effects, including hypothalamic-pituitary-adrenal suppression correlates with the relative potency of the drug, dosage, timing of administration and the duration of treatment (see section 4.4).

The following side effects may be associated with the long-term systemic use of corticosteroids with the following frequency:

Not known (cannot be estimated from available data)

System organ class	Frequency	Undesirable effects
Infections and infestations	Not known	Infection[a], candidiasis
Blood and lymphatic system disorders	Not known	Leucocytosis
Immune system disorders	Not known	Hypersensitivity, including anaphylaxis has been reported
Endocrine disorders	Not known	Suppression of the hypothalamo-pituitary-adrenal axis, cushingoid facies
Metabolism and nutrition disorders	Not known	Sodium and water retention, hypokalaemia, hypokalaemic alkalosis, impaired carbohydrate tolerance with increased requirement for antidiabetic therapy, negative protein and calcium balance and increased appetite
Psychiatric disorders	Not known	Euphoria, psychological dependence, depression, insomnia and aggravation of schizophrenia. Aggravation of epilepsy, depressed and labile mood and suicidal thoughts, mania, delusions, hallucinations, behavioural disturbances, irritability, anxiety, sleep disturbances, confusion and amnesia[b]

Eye disorders	Not known	Increased intra-ocular pressure, glaucoma, papilloedema, posterior subcapsular cataracts, corneal or scleral thinning, exacerbation of ophthalmic viral or fungal diseases and vision, blurred (see also section 4.4)
Cardiac disorders	Not known	Myocardial rupture following recent myocardial infarction, hypertrophic cardiomyopathy in prematurely born infants
Vascular disorders	Not known	Hypertension, thromboembolism
Gastrointestinal disorders	Not known	Dyspepsia, peptic ulceration with perforation and haemorrhage, abnormal distension, oesophageal ulceration, acute pancreatitis, nausea
Skin and subcutaneous tissue disorders	Not known	Skin atrophy, striae, acne, telangiectasia, hirsutism
Musculoskeletal and connective tissue disorder	Not known	Proximal myopathy, osteoporosis, vertebral and long bone fractures, avascular osteonecrosis, tendon rupture
Reproductive system disorders	Not known	menstrual irregularity, amenorrhoea
General disorders and administration site conditions	Not known	Impaired healing, malaise
Injury, poisoning and procedural complications	Not known	Tendon rupture, bruising
Investigations	Not known	Weight increased

a. Increased susceptibility and severity of infections with suppression of clinical symptoms and signs, opportunistic infections and recurrence of dormant tuberculosis (see section 4.4).

b. Reactions are common and may occur in both adults and children. In adults, the frequency of severe reactions has been estimated to be 5-6%. Psychological effects have been reported on withdrawal of corticosteroids.

Paediatric population

Growth suppression in infancy, childhood and adolescence, increased intracranial pressure with papilloedema in children (pseudotumour cerebri), usually after treatment withdrawal.

Withdrawal symptoms:

Too rapid a reduction of corticosteroid dosage following prolonged treatment can lead to acute renal insufficiency, hypotension and death (see section 4.4). A withdrawal syndrome may also occur including fever, myalgia, arthralgia, rhinitis, conjunctivitis, painful itchy skin nodules and weight loss.

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 the Yellow Card Scheme at: www.mhra.gov.uk/yellowcard or search for MHRA Yellow Card in the Google Play or Apple App Store.

4.9 Overdose

Symptoms

Overdosage may cause nausea and vomiting, sodium and water retention, hyperglycemia and occasional gastrointestinal bleeding.

Management

Treatment need only be symptomatic although cimetidine (200-400 mg by slow intravenous injection every 6 hours) or ranitidine (50 mg by slow intravenous injection every 6 hours) may be administered to prevent gastrointestinal bleeding.

5 PHARMACOLOGICAL PROPERTIES

5.1 Pharmacodynamic properties

Pharmacotherapeutic group: Systemic Hormonal Preparations (excluding sex hormones and insulins); Corticosteroids for Systemic Use; Plain; Hydrocortisone.

ATC code: H02A B09.

Pharmacodynamic effects

Hydrocortisone is a glucocorticoid. Glucocorticoids are adrenocortical steroids, both naturally-occurring and synthetic, which are readily absorbed from the gastrointestinal tract.

Hydrocortisone is believed to be the principal corticosteroid secreted by the adrenal cortex. Naturally-occurring glucocorticosteroids (hydrocortisone and cortisone), which also have salt-retaining properties, are used as replacement therapy in adrenocortical deficiency states. They are also used for their potent anti-inflammatory effects in disorders of many organ systems. Glucocorticoids cause profound and varied metabolic effects. In addition they modify the body’s immune responses to diverse stimuli.

5.2 Pharmacokinetic properties

Absorption

Hydrocortisone is readily absorbed from the gastrointestinal tract and 90% or more of the drug is reversibly bound to protein.

Distribution

The binding is accounted for by two protein fractions. One, corticosteroid-binding globulin which is a glycoprotein; the other is albumin.

Biotransformation and Elimination

Hydrocortisone is metabolised in the liver and most body tissues to hydrogenated and degraded forms such as tetrahydrocortisone and tetrahydrocortisol which are excreted in the urine, mainly conjugated as glucuronides, together with a very small proportion of unchanged hydrocortisone.

Half-life of hydrocortisone is about 1.5 hours.

5.3 Preclinical safety data

Administration of corticosteroids to pregnant animals can cause abnormalities of fetal development including cleft palate, intra-uterine growth retardation and effects on brain growth and development.

6 PHARMACEUTICAL PARTICULARS

6.1 List of excipients

Lactose monohydrate

Maize starch

Magnesium stearate

6.2 Incompatibilities

Not applicable

6.3 Shelf life

30 months

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 blisters lidded with aluminium foil containing 30 tablets.

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

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8 MARKETING AUTHORISATION NUMBER(S)

PL 20046/0302

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