

Hydrocortisone 20mg Tablets

Summary of Product Characteristics Updated 15-Jul-2021 | Accord-UK Ltd

1. Name of the medicinal product

Hydrocortisone 20mg Tablets

Hydrocortone 20mg Tablets

Hydrocortisone Accord 20mg Tablets

Hitoden 20mg Tablets

2. Qualitative and quantitative composition

20mg tablet: 20mg hydrocortisone

Excipient with known effect

Each tablet contains 182.7mg of lactose.

For the full list of excipients, see section 6.1.

3. Pharmaceutical form

Tablets

20mg tablets: white, half-scored tablets, marked 'HYD 20'. The tablet can be divided into equal doses.

4. Clinical particulars

4.1 Therapeutic indications

Corticosteroid

For use as replacement therapy in primary, secondary, or acute adrenocortical insufficiency.

Pre-operatively, and during serious trauma or illness in patients 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. The lowest possible dosage should be used. Doses should be multiples of 10 (i.e. 10mg, 20mg, 30mg, etc.).

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.

To avoid hypoadrenalism and/or a relapse of the underlying disease, it may be necessary to withdraw the drug gradually (see section 4.4).

Replacement therapy

In chronic adrenocortical insufficiency, a dosage of 20 to 30mg a day is usually recommended, sometimes together with 4-6 g of sodium chloride or 50-300 micrograms of fludrocortisone daily.

When immediate support is mandatory, one of the soluble adrenocortical hormone preparations (e.g. dexamethasone sodium phosphate), which may be effective within minutes after parenteral administration, can be life-saving.

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.

Elderly:

Treatment of elderly patients, particularly if long term, should be planned bearing in mind the more serious consequences of the common side effects of corticosteroids in old age, especially osteoporosis, diabetes, hypertension, susceptibility to infection and thinning of the skin.

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).

Method of administration

For oral administration.

4.3 Contraindications

Hypersensitivity to hydrocortisone 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

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

The lowest possible dosage of corticosteroids should be used and when reduction in dosage is possible, the reduction should be gradual.

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.), although dose levels do not allow prediction of the onset, type, severity or duration of reactions. Most reactions recover after either dose reduction or withdrawal, 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 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.

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.

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 not be stopped and the dose may need to be increased. Corticosteroids may exacerbate systemic fungal infections and therefore should not be used in the presence of such infections unless they are needed to control life-threatening drug reactions due to amphotericin. Moreover, there have been cases reported in which concomitant use of amphotericin and hydrocortisone was followed by cardiac enlargement and congestive failure.

Literature reports suggest an apparent association between use of corticosteroids and left ventricular free wall rupture after a recent myocardial infarction; therefore, therapy with corticosteroids should be used with great caution in these patients.

Average and large dosages of hydrocortisone or cortisone can cause elevation of blood pressure, salt and water retention, and increase 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.

A report shows that the use of corticosteroids in cerebral malaria is associated with a prolonged coma and an increased incidence of pneumonia and gastro-intestinal bleeding.

If corticosteroids are indicated in patients with latent tuberculosis or tuberculin reactivity, close observation is necessary as reactivation may occur. During prolonged corticosteroid therapy, these patients should receive prophylactic chemotherapy.

The use of hydrocortisone tablets in active tuberculosis should be restricted to those cases of fulminating or disseminated tuberculosis.

Corticosteroids should be used with caution in renal insufficiency, hypertension, diabetes mellitus or in those with a family history of diabetes, congestive heart failure, thrombophlebitis, exanthematous disease, chronic nephritis, acute glomerulonephritis, metastatic carcinoma, osteoporosis (postmenopausal patients are at special risk), severe affective disorders (particularly if there is a history of steroid-induced psychosis), epilepsy, previous steroid myopathy, liver failure, glaucoma (or family history of glaucoma), myasthenia gravis, non-specific ulcerative colitis if there is a probability of impending perforation, diverticulitis, fresh intestinal anastomoses, active or latent peptic ulcer. Signs of peritoneal irritation following gastro-intestinal perforation in patients receiving large doses of corticosteroids may be minimal or absent.

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

Fat embolism has been reported as a possible complication of hypercortisonism.

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

Prolonged courses of corticosteroids increase susceptibility to infections and their severity. The clinical presentation of infections may also be atypical.

Corticosteroids may mask some signs of infection and some serious infection such as septicaemia and tuberculosis may reach an advanced stage before being recognised. There may be an inability to localise infection in patients on corticosteroids. Corticosteroids may affect the nitrobluetetrazolium test for bacterial infection and produce false negative results.

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

Prolonged use of corticosteroids may produce posterior subcapsular cataracts, glaucoma with possible damage to the optic nerves, and may enhance the establishment of secondary ocular infections due to fungi or viruses.

Corticosteroids should be used cautiously in patients with ocular herpes simplex because of possible corneal perforation.

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.

Corticosteroids may increase or decrease motility and number of spermatozoa. Diabetes may be aggravated, necessitating a higher insulin dosage. Latent diabetes mellitus may be precipitated.

Menstrual irregularities may occur, and this possibility should be mentioned to female patients.

Rare instances of anaphylactoid reactions have occurred in patients receiving corticosteroids, especially when a patient has a history of drug allergies.

Aspirin should be used cautiously in conjunction with corticosteroids in patients with hypoprothrombinaemia.

Withdrawal

Drug-induced secondary adrenocortical insufficiency may result from too rapid a withdrawal of corticosteroids and may 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, corticosteroid therapy should be reinstated. If the patient is receiving steroids already, the dosage may have to be increased. Since mineralocorticoid secretion may be impaired, salt and/or a mineralocorticoid should be administered concurrently (see section 4.5).

Stopping corticosteroid after prolonged therapy may cause withdrawal symptoms, including fever, myalgia, arthralgia and malaise. In patients who have received more than physiological doses of systemic corticosteroids (approximately 30mg hydrocortisone) 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 of 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 160mg 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 hydrocortisone
- patients repeatedly taking doses in the evening.

Paediatric population

Corticosteroids cause growth retardation in infancy, childhood and adolescence. Treatment should be limited to the minimum dosage in order to minimise suppression of the hypothalamo-pituitary-adrenal axis and growth retardation. Growth and development of infants and children on prolonged corticosteroid therapy should be carefully monitored.

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.

This medicine contains lactose

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

4.5 Interaction with other medicinal products and other forms of interaction

Drug interactions listed below have been reported in pharmacological doses of corticosteroids and may not occur at replacement therapy doses of corticosteroids.

Aspirin should be used cautiously in conjunction with corticosteroids in hypoprothrombinaemia. There is an increased risk of gastro-intestinal bleeding and ulceration when corticosteroids are given with aspirin and NSAIDs, although topical NSAIDs do not generally interact with corticosteroids. The renal clearance of salicylates is increased by corticosteroids and steroid withdrawal may result in salicylate intoxication.

Corticosteroids reduce plasma concentrations of salicylate and such an interaction may occur with pharmacological doses of glucocorticoids.

Phenytoin, ephedrine, rifabutin, carbamazepine, barbiturates, rifampicin, primidone, sympathomimetics and aminoglutethimide may enhance the metabolic clearance of corticosteroids, resulting in decreased blood levels and lessened physiological activity, thus requiring adjustment in corticosteroid dosage.

The INR or prothrombin time should be checked frequently in patients who are receiving corticosteroids and coumarin anticoagulants at the same time to avoid spontaneous bleeding because of reports of altered response to these anticoagulants. Studies have shown that the usual effect produced by adding corticosteroids is inhibition of response to coumarins, although there have been some conflicting reports of potentiation not substantiated by studies.

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

Corticosteroids antagonise the effects of diuretics. Glucocorticosteroids are necessary for free water clearance by the kidneys. When corticosteroids are administered concomitantly with potassium-depleting diuretics (e.g. acetazolamide, loop diuretics, thiazides, carbenoxolone), patients should be observed closely for development of hypokalaemia.

Moreover, corticosteroids may affect the nitroblue tetrazolium test for bacterial infection and produce false negative results.

Corticosteroids antagonise the hypotensive effects of beta-blockers, alpha- blockers, calcium channel blockers, clonidine, diazoxide, methyldopa, moxonidine, nitrates, nitroprusside, hydralazine, minoxidil, adrenergic neurone blockers, ACE inhibitors and angiotensin II receptor antagonists.

Corticosteroids increase risk of hypokalaemia when given with cardiac glycosides, e.g. digoxin, theophylline and beta2 sympathomimetics, e.g. bambuterol, fenoterol, formoterol, ritodrine, salbutamol, salmeterol and terbutaline.

There is an increased risk of hypokalaemia when corticosteroids are given with amphotericin. Concomitant use of amphotericin with corticosteroids should be avoided unless amphotericin is needed to control reactions.

The effect of corticosteroids may be reduced for 3-4 days after interaction with mifepristone.

The plasma concentration of corticosteroids is increased by oral contraceptives containing oestrogens dosage adjustments may be required if oral contraceptives are added to or withdrawn from a stable dosage regimen. Interactions of combined oral contraceptives may also apply to combined contraceptive patches. In the case of hormone replacement therapy, low doses are unlikely to induce interactions. The plasma concentration of corticosteroids may possibly be increased by ritonavir.

Corticosteroids reduce absorption of calcium salts.

The metabolism of corticosteroids can be inhibited by erythromycin, although not when small amounts of erythromycin are used topically.

Corticosteroids antagonise hypoglycaemic effect of antidiabetics.

There is an increased risk of haematological toxicity when corticosteroids are given with methotrexate.

Corticosteroids may inhibit the growth promoting effect of somatropin.

High doses of corticosteroids impair immune response to vaccines, avoid concomitant use with live vaccines.

Corticosteroids possibly reduce the effects of sodium benzoate and sodium phenyl butyrate.

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.

4.6 Fertility, pregnancy and lactation

Pregnancy

The ability of corticosteroids to cross the placenta varies between individual drugs, however, hydrocortisone 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. Pregnant patients should be monitored closely if they develop fluid retention or pre-eclampsia. 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 state.

Breastfeeding

Corticosteroids are excreted in breast milk, although no data are available for hydrocortisone. Infants of mothers taking high doses 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. 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.

Hydrocortisone may cause fatigue, vertigo, visual field loss and muscle wasting and weakness. If affected, patients should not drive or operate machinery (see section 4.8).

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	Not known	Leucocytosis.

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

4.9 Overdose

Reports of acute toxicity and/or deaths following overdosage with glucocorticoids are rare. No antidote is available.

Symptoms

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

Management

Treatment is probably not indicated for reactions due to chronic poisoning unless the patient has a condition that would render him unusually susceptible to ill effects from corticosteroids. In this case, symptomatic treatment should be instituted as necessary 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.

Anaphylactic and hypersensitivity reactions may be treated with adrenaline, positive-pressure artificial respiration and aminophylline. The patient should be kept warm and quiet.

The biological half-life of hydrocortisone is about 100 minutes.

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: H02AB02

Hydrocortisone is a glucocorticoid. Glucocorticoids are adrenocortical steroids, both naturally-occurring and synthetic, which are readily absorbed from the gastro-intestinal 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 gastro-intestinal tract and 90% or more of the drug is reversibly bound to protein.

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

Biotransformation

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 foetal development including cleft palate, intra-uterine growth retardation and effects on brain growth and development.

6. Pharmaceutical particulars

6.1 List of excipients

Lactose

Magnesium stearate

Maize starch

6.2 Incompatibilities

None known

6.3 Shelf life

3 years

6.4 Special precautions for storage

Do not store above 25°C

Store in original package

6.5 Nature and contents of container

PVC/aluminium blister containing 30 tablets

6.6 Special precautions for disposal and other handling

None

7. Marketing authorisation holder

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8. Marketing authorisation number(s)

PL 20075/1250

9. Date of first authorisation/renewal of the authorisation

23/02/1989

10. Date of revision of the text

13/07/2021

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