

NEW ZEALAND DATA SHEET

1. PRODUCT NAME

KENACORT-A 10 suspension for injection

2. QUALITATIVE AND QUANTITATIVE COMPOSITION

KENACORT-A 10 contains 10 mg/1 mL triamcinolone acetonide.

For the list of excipients, see section 6.1.

3. PHARMACEUTICAL FORM

KENACORT-A 10 is a sterile, aqueous opaque white suspension.

4. CLINICAL PARTICULARS

4.1 Therapeutic indications

Intra-articular:

Kenacort-A 10 Injection is indicated for intra-articular or intra-synovial administration, and for injections into tendon sheaths, as adjunctive therapy for short-term administration (to tide the patient over an acute episode or exacerbation) in: synovitis of osteoarthritis, rheumatoid arthritis, acute and subacute bursitis, acute gouty arthritis, epicondylitis, acute nonspecific tenosynovitis, post-traumatic osteoarthritis.

Intradermal:

Intralesional administration of Kenacort-A 10 Injection is indicated for the treatment of keloids, discoid lupus erythematosus, necrobiosis lipoidica diabetorum, alopecia areata, and localised hypertrophic, infiltrated, inflammatory lesions of: lichen planus, psoriatic plaques, granuloma annulare, and lichen simplex chronicus (neurodermatitis). Kenacort-A 10 Injection also may be useful in cystic tumours of an aponeurosis or tendon (ganglia).

4.2 Dose and method of administration

Kenacort-A 10 is suitable for intradermal, intra-articular and intra-synovial injection and for injection into tendon sheaths. This formulation is not suitable for intravenous, intramuscular or intraocular use.

This preparation contains benzyl alcohol. Not for use in newborn or premature infants (see section 4.4 Special Warnings and precautions for use, Use in children).

General:

The initial dosage should be maintained or adjusted until a satisfactory response is noted. If after a reasonable period of time there is a lack of satisfactory clinical

response, Kenacort-A 10 Injection should be discontinued and the patient transferred to other appropriate therapy.

It should be emphasised that dosage requirements are variable and must be individualised on the basis of the disease under treatment and the response of the patient.

After a favourable response is noted, the proper maintenance dosage should be determined by decreasing the initial dosage in small increments at appropriate time intervals until the lowest dosage which will maintain an adequate clinical response is reached. It should be kept in mind that constant monitoring is needed in regard to medication dosage.

Included in the situations which may make dosage adjustments necessary are changes in clinical status secondary to remissions or exacerbations in the disease process, the patient's individual medication responsiveness, and the effect of patient exposure to stressful situations not directly related to the disease entity under treatment; in this latter situation it may be necessary to increase the dosage of Kenacort-A 10 Injection for a period of time consistent with the patient's condition.

If after long-term therapy the medication is to be stopped, it is recommended that it be withdrawn gradually rather than abruptly.

Dosage:

For intra-articular or intra-synovial administration and for injection into tendon sheaths, the initial dose of Kenacort-A 10 Injection may vary from 5 mg to 10 mg for smaller joints and up to 40 mg for larger joints depending on the specific disease entity being treated. (A more concentrated form of Sterile Triamcinolone Acetonide Suspension USP is available as Kenacort-A 40). Single injections into several joints for multiple locus involvement, up to a total of 80 mg have been given without undue reactions.

For intradermal administration the initial dose of triamcinolone acetonide will vary depending upon the specific disease entity being treated but should be limited to 1.0 mg (0.1 mL) per injection site since larger volumes are more likely to produce cutaneous atrophy. Multiple sites (separated by one centimetre or more) may be so injected, keeping in mind that the greater the total volume employed the more corticosteroid become available for possible systemic absorption and subsequent corticosteroid effects. Such injections may be repeated if necessary at weekly or less frequent intervals.

The lower dosages in the initial dosage range of triamcinolone acetonide may produce the desired effect when the corticosteroid is administered to provide a localised concentration. The site of the injection and the volume of the injection should be carefully considered when triamcinolone acetonide is administered for this purpose.

Administration - general:

STRICT ASEPTIC TECHNIQUE IS MANDATORY.

Shake the vial before use to ensure a uniform suspension. Prior to withdrawal, inspect suspension for clumping or granular appearance (agglomeration). An agglomerated product results from exposure to freezing temperatures and should not

be used. After withdrawal, inject without delay to prevent settling in the syringe. Careful technique should be employed to avoid the possibility of entering a blood vessel or introducing infection.

Routine laboratory studies, such as urinalysis, two-hour post-prandial blood sugar, determination of blood pressure and body weight and a chest x-ray should be made at regular intervals during prolonged therapy. Upper GI x-rays are desirable in patients with an ulcer history or significant dyspepsia.

For treatment of joints:

The usual intra-articular injection technique, as described in standard textbooks, should be followed. If an excessive amount of synovial fluid is present in the joint, some, but not all, should be aspirated to aid in the relief of pain and to prevent undue dilution of the corticosteroid.

With intra-articular or intra-synovial administration, and with injection of the medication into tendon sheaths, the use of a local anaesthetic may often be desirable. When a local anaesthetic is used its package insert should be read with care and all the precautions connected with its use should be observed. It should be injected into the surrounding soft tissues prior to the injection of the corticosteroid. A small amount of the anaesthetic solution may be instilled into the joint.

In treating acute nonspecific tenosynovitis, care should be taken to ensure that the injection of the corticosteroid is made into the tendon sheath rather than the tendon substance. Epicondylitis (tennis elbow) may be treated by infiltrating the preparation into the area of the greatest tenderness.

For treatment of dermal lesions:

Kenacort-A 10 Injection is injected directly into the lesion, i.e. intradermally or sometimes subcutaneously. For accuracy of dosage measurement and ease of administration, it is preferable to employ a tuberculin syringe and a small bore needle (23 to 25 gauge). Ethyl chloride spray may be used to alleviate the discomfort of the injection.

4.3 Contraindications

Corticosteroids are generally contraindicated in patients with systemic infections. Live virus immunisation. Kenacort-A 10 is also contraindicated in patients with sensitivity to the active or inactive ingredients.

4.4 Special warnings and precautions for use

General precautions:

Because it is a suspension, the preparation should not be administered intravenously. Strict aseptic technique is mandatory. This preparation is not recommended for children under six years of age.

Because rare instances of anaphylactoid reactions have occurred in patients receiving parenteral corticosteroid therapy, appropriate precautionary measures should be taken prior to administration, especially when the patient has a history of allergy to any medication.

This product contains benzyl alcohol as a preservative. Benzyl alcohol has been associated with serious adverse events and death, particularly in paediatric patients. The "gasping syndrome" has been associated with benzyl alcohol. Although normal therapeutic doses of this product deliver amounts of benzyl alcohol that are substantially lower than those reported in association with the "gasping syndrome", the minimum amount of benzyl alcohol at which toxicity may occur is not known. Premature and low-birth-weight infants, as well as patients receiving high dosages, may be more likely to develop toxicity.

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

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

Corticosteroids should be used with caution in patients with nonspecific ulcerative colitis if there is a probability of impending perforation, abscess, or other pyogenic infection. Corticosteroids should also be used cautiously in patients with diverticulitis, fresh intestinal anastomoses, active or latent peptic ulcer, renal insufficiency, hypertension, osteoporosis, acute glomerulonephritis, vaccinia, varicella, exanthema, Cushing's syndrome, antibiotic-resistant infections, diabetes mellitus or in those with a family history of diabetes, congestive heart failure, chronic nephritis, thromboembolic tendencies, thrombophlebitis, convulsive disorders, metastatic carcinoma and myasthenia gravis.

Although therapy with Kenacort-A 10 will ameliorate symptoms, it is in no sense a cure and the hormone has no effect on the cause of the inflammation. Therefore, this method of treatment does not obviate the need for the conventional measures usually employed.

Intra-articular injection of the suspension into the soft tissues surrounding a joint is not harmful but may lead to the occurrence of systemic effects, and is the most common cause of failure to achieve the desired local results.

Following intra-articular steroid therapy, patients should be specifically warned to avoid overuse of joints in which symptomatic benefit has been obtained. Negligence in this matter may permit an increase in joint deterioration that will more than offset the beneficial effects of the steroid. To detect deterioration, follow-up x-ray examination is suggested in selected cases.

Overdistention of the joint capsule and deposition of steroid along the needle track should be avoided in intra-articular injection since this may lead to subcutaneous atrophy.

Corticosteroids should not be injected into unstable joints. Repeated intra-articular injection may in some cases result in instability of the joint. In selected cases, particularly when repeated injections are given, x-ray follow-up is suggested.

An increase in joint discomfort has seldom occurred. A marked increase in pain accompanied by local swelling, further restriction of joint motion, fever, and malaise are suggestive of a septic arthritis. If these complications should appear, and the diagnosis of septic arthritis is confirmed, administration of triamcinolone acetonide should be stopped and antimicrobial therapy should be instituted immediately and

continued for 7 to 10 days after all evidence of infection has disappeared. Appropriate examination of any joint fluid present is necessary to exclude a septic process.

Local injection of a steroid into a previously infected joint is to be avoided. Repeated injection into inflamed tendons has been followed by tendon rupture. Therefore it should also be avoided.

Kenacort-A 10 should be administered only with full knowledge of characteristic activity of, and varied responses to, adrenocortical hormones. Like other potent corticosteroids, triamcinolone acetonide should be used under close clinical supervision.

During prolonged therapy a liberal protein intake is essential for counteracting the tendency to gradual weight loss sometimes associated with negative nitrogen balance, wasting and weakness of skeletal muscles.

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

In peptic ulcer, recurrence may be asymptomatic until perforation or haemorrhage occurs. Long-term adrenocorticoid therapy may evoke hyperacidity or peptic ulcer: therefore, as a prophylactic measure, an ulcer regimen and the administration of an antacid are highly recommended. X-rays should be taken in peptic ulcer patients complaining of gastric distress or when therapy is prolonged. Whether or not changes are observed, an ulcer regimen is recommended.

As with other corticosteroids the possibility of other severe reactions should be considered. If such reactions should occur appropriate corrective measures should be instituted and use of the medication discontinued.

Continued supervision of the patient after termination of triamcinolone acetonide therapy is essential since there may be a sudden reappearance of severe manifestations of the disease for which the patient was treated.

Adrenocortical insufficiency:

Average and large 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 they are used in large doses: dietary salt restriction and potassium supplementation may be necessary. All corticosteroids increase calcium excretion.

Medication-induced secondary adrenocortical insufficiency may be minimised by a gradual reduction of dosage. This type of relative insufficiency may persist for months after discontinuation of therapy: therefore, in any situation of stress (such as trauma, surgery or severe illness) occurring during that period, hormone therapy should be reinstated. Since mineralocorticoid secretion may be impaired, salt and/or a mineralocorticoid should be administered concurrently.

When patients who are receiving corticosteroid therapy are subjected to unusual stress, increased dosage of rapidly acting corticosteroids is indicated before, during, and after the stressful situation. Kenacort A-10 is a long-acting preparation and is not suitable for use in acute stress situations.

Symptoms of adrenal insufficiency include: malaise, muscle weakness, mental changes, muscle and joint pain, desquamation of the skin, dyspnoea, anorexia, nausea and vomiting, fever, hypoglycaemia, hypotension and dehydration.

The lowest possible dose of corticosteroid should be used to control the condition being treated. A gradual reduction in dosage should be made when possible.

Anti-inflammatory/immunosuppressive effects and infection:

Corticosteroids may mask some signs of infection, and new infections may appear during their use. There may be decreased resistance and inability to localise infection when corticosteroids are used. In addition, patients who are on immunosuppressant drugs including corticosteroids are more susceptible to infections than those not taking these drugs.

Moreover, chickenpox and measles can have a more serious or even fatal course in patients on corticosteroids. In such children, or adults receiving corticosteroids who have not had these diseases, particular care should be taken to avoid exposure. If exposed, therapy with varicella zoster immune globulin (VZIG) or pooled intravenous immunoglobulin (IVIG), as appropriate, may be indicated. If chickenpox or herpes zoster develops, treatment with antiviral agents may be considered.

Similarly, corticosteroids should be used with great caution in patients with *Strongyloides* (threadworm) infestation because corticosteroid-induced immunosuppression may lead to *Strongyloides* hyperinfection and dissemination with widespread larval migration, often accompanied by severe enterocolitis and potentially fatal gram-negative septicaemia.

Patients should not be vaccinated against smallpox while on corticosteroid therapy. Other immunisation procedures should not be undertaken in patients who are on corticosteroids, especially on high dose because of possible hazards of neurological complications and a lack of antibody response.

The use of triamcinolone acetonide in patients with active tuberculosis should be restricted to those cases of fulminating or disseminated tuberculosis in which corticosteroid is used for the management of the disease in conjunction with an appropriate antituberculous regimen. If corticosteroids are indicated in patients with latent tuberculosis or tuberculin reactivity, close observation is necessary since reactivation of the disease may occur. During prolonged corticosteroid therapy these patients should receive chemoprophylaxis.

When local or systemic microbial infections are present, therapy with triamcinolone acetonide is not recommended, but may be employed with caution and only in conjunction with appropriate antibiotic or chemotherapeutic medication. Triamcinolone acetonide may mask signs of infection and enhance dissemination of the infecting organism. Hence, all patients receiving triamcinolone acetonide should be watched for evidence of inter-current infection. Should infection occur, vigorous appropriate anti-infective therapy should be initiated. If possible, abrupt cessation of steroids should be avoided because of the danger of superimposing adrenocortical insufficiency on the infectious process.

Live vaccines are contraindicated in individuals on high doses of corticosteroids and should be postponed until at least 3 months after stopping corticosteroid therapy.

Ocular effects:

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.

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.

Adequate studies to demonstrate the safe use of Kenacort-A 10 Injection by intratubinal, subconjunctival, subtenons, retrobulbar and intraocular (intravitreal) injection have not been performed. Endophthalmitis, eye inflammation, increased intraocular pressure and visual disturbances including vision loss have been reported with intravitreal administration. Several instances of blindness have been reported following injection of corticosteroid suspensions into the nasal turbinates and intralesional injection about the head. Administration of Kenacort-A 10 by any of these routes is not recommended.

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

Psychiatric effects:

Patients and/or carers should be warned that potentially severe psychiatric derangements may appear when corticosteroids are used. Symptoms typically emerge within a few days or weeks of starting treatment. These may range from euphoria, insomnia, mood swings, personality changes and severe depression to frank psychotic manifestations. Most patients recover after either dose reduction or withdrawal. Patients and/or carers should be encouraged to seek medical advice if worrying psychological symptoms develop, especially if depressed mood or suicidal ideation is suspected.

Particular care is required when considering the use of corticosteroids in patients with existing or previous history of psychotic instability. The use of antidepressant medications does not relieve and may exacerbate adrenocorticoid-induced mental disturbances.

Use in children:

Because corticosteroids can suppress growth, the development of infants, children and adolescents on prolonged corticosteroid therapy should be carefully observed.

Caution should be used in the event of exposure to chicken pox, measles or other communicable diseases. Children should not be vaccinated or immunised while on corticosteroid therapy. Corticosteroids may also affect endogenous steroid production.

Exposure to excessive amounts of benzyl alcohol has been associated with toxicity (hypotension, metabolic acidosis), particularly in neonates, and an increased incidence of kernicterus, particularly in small preterm infants. There have been rare

reports of deaths, primarily in preterm infants, associated with exposure to excessive amounts of benzyl alcohol.

Children are at specially risk from raised intracranial pressure.

Use in the elderly:

The common adverse effects of systemic corticosteroids such as osteoporosis, diabetes, hypertension, hypokalaemia, susceptibility to infection and thinning of the skin may have more serious consequences in the elderly. Close clinical supervision is recommended.

Other conditions:

Caution is necessary when oral corticosteroids are used in patients with the following other conditions and frequent monitoring is necessary. This includes patients with:

- Glaucoma
- A history of severe affective disorders particularly of steroid induced psychoses
- Previous steroid myopathy
- Duchenne's muscular dystrophy since transient rhabdomyolysis and myoglobinuria have been reported following strenuous physical activity.

4.5 Interactions with other medicines and other forms of interaction

Amphotericin B injection and potassium-depleting agents:

Patients should be observed for hypokalaemia

Anticholinesterases:

Effects of the anticholinesterase agent may be antagonised.

Anticoagulants, oral:

Corticosteroids may potentiate or decrease anticoagulant action. Patients receiving oral anticoagulants and corticosteroids should be closely monitored.

Antidiabetics:

Corticosteroids may increase blood glucose; diabetic control should be monitored, especially when corticosteroids are initiated, discontinued, or changed in dosage.

Antitubercular drugs:

Isoniazid serum concentrations may be decreased.

Digitalis glycosides:

Co-administration may enhance the possibility of digitalis toxicity.

Oestrogens, including oral contraceptives:

Corticosteroid half-life and concentration may be increased and clearance decreased.

Hepatic enzyme inducers (e.g. barbiturates, phenytoin, carbamazepine, rifampin):

There may be increased metabolic clearance of triamcinolone acetonide. Patients should be carefully observed for possible diminished effect of steroid, and the dosage of Kenacort A should be adjusted accordingly.

Human growth hormone (e.g. somatrem):

The growth-promoting effect of somatrem may be inhibited.

Hepatic microsomal enzyme inhibitors (e.g. ketoconazole, cyclosporine, ritonavir):

Corticosteroid clearance may be decreased, resulting in increased effects. Monitor for evidence of increased toxicity of cyclosporine when the two are used concurrently.

Nondepolarising muscle relaxants:

Corticosteroids may decrease or enhance the neuromuscular blocking action.

Non-steroidal anti-inflammatory agents (NSAIDs):

Corticosteroids may increase the incidence and/or severity of GI bleeding and ulceration associated with NSAIDs. Also, corticosteroids can reduce serum salicylate levels and therefore decrease their effectiveness. Conversely, discontinuing corticosteroids during high-dose salicylate therapy may result in salicylate toxicity. Patients should be observed closely for adverse effects of either medicine. Aspirin should be used cautiously in conjunction with corticosteroids in patients with hypoprothrombinaemia.

Thyroid drugs:

Metabolic clearance of adrenocorticoids is increased in hyperthyroid patients. Changes in thyroid status of the patient may necessitate adjustment in adrenocorticoid drugs.

Vaccines:

Neurological complications and lack of antibody response may occur when patients taking corticosteroids are vaccinated.

Other interactions for oral and systemic corticosteroids:

Mifepristone:	The effect of corticosteroids may be reduced for 3-4 days after mifepristone.
Vaccines:	Live vaccines should not be given to individuals with impaired immune responsiveness.
Sympathomimetics:	There is an increased risk of hypokalaemia if high doses of corticosteroids are given with high doses of salbutamol, salmeterol, terbutaline or formoterol.
Diuretics:	Excessive potassium loss may be experienced if glucocorticoids and potassium-depleting diuretics (such as frusemide and thiazides) or carbonic anhydrase inhibitors (such as acetazolamide) are given together.
Antacids:	Concurrent use of antacids may decrease absorption of corticosteroids – efficacy may be decreased sufficiently to require dosage adjustments in patients receiving small doses of corticosteroids.

4.6 Fertility, pregnancy and lactation

Use in pregnancy:

Many corticosteroids have been shown to be teratogenic in laboratory animals at low doses. Since adequate human reproduction studies have not been done with corticosteroids, the use of these medications in pregnancy, nursing mothers, or

women of child-bearing potential requires that the possible benefits of the medication be weighed against the potential hazards to the mother and the embryo, foetus, or nursing infant. Infants born of mothers who have received substantial doses of corticosteroids during pregnancy should be carefully observed for signs of hypoadrenalism.

4.7 Effects on ability to drive and use machines

Not relevant.

4.8 Undesirable effects

Reporting of suspected adverse reactions:

Reporting suspected adverse reactions after authorisation of the medicine is important. It allows continued monitoring of the benefit/risk balance of the medicine. Healthcare professionals are asked to report any suspected adverse reactions to <https://nzphvc.otago.ac.nz/reporting/>.

Following administration by any route:

Since systemic absorption may occasionally occur with intra-articular or other local administration, patients should be watched closely for the following adverse reactions which may be associated with any corticosteroid therapy:

Body as a whole:

Anaphylactoid reactions, thromboembolism, fatigue, necrotising angiitis

Cardiovascular:

Hypertension, syncope, congestive heart failure, arrhythmias, necrotising angiitis, thrombophlebitis.

Metabolic/nutritional:

Hyperglycaemia, glycosuria, sodium retention, fluid retention, associated with hypertension or congestive heart failure in susceptible patients, potassium loss, cardiac arrhythmias or ECG changes due to potassium deficiency and hypokalaemic alkalosis.

Musculoskeletal:

Muscle weakness, steroid myopathy, loss of muscle mass, osteoporosis, vertebral compression fractures, delayed healing of fractures, aseptic necrosis of femoral and humeral heads, pathologic fractures of long bones and spontaneous fractures.

Gastro-intestinal:

Peptic ulcer with possible subsequent perforation and haemorrhage, pancreatitis, abdominal distension and ulcerative oesophagitis.

Skin:

Impaired wound healing, thin fragile skin, petechiae and ecchymoses, facial erythema, increased sweating, purpura, striae, hirsutism, acneiform eruptions, lupus erythematosus-like lesions, hives, rash and suppressed reactions to skin tests.

Nervous system:

Convulsions, increased intracranial pressure with papilloedema (pseudo-tumour cerebri) usually after treatment, vertigo, headache, neuritis or paraesthesias,

aggravation of pre-existing psychiatric conditions, depression (sometimes severe), euphoria, mood swings, psychotic symptoms and personality changes.

Endocrine:

Menstrual irregularities, development of the cushingoid state, suppression of growth in children, secondary adrenocortical and pituitary unresponsiveness particularly in times of stress (e.g. trauma, surgery or illness), decreased carbohydrate tolerance, manifestations of latent diabetes mellitus and increased requirements for insulin or oral hypoglycaemic agents in diabetics.

Eye disorders:

Posterior subcapsular cataracts, increased intraocular pressure, glaucoma, exophthalmos and central serous chorioretinopathy (CSCR).

Anti-inflammatory and immunosuppressive effects:

Aggravation or masking of infections.

Following intra-articular administration:

Undesirable reactions have included post injection flare, transient pain, occasional irritation at the injection site, sterile abscess formation, hyper- and hypopigmentation, charcot-like arthropathy and occasional brief increase in joint discomfort.

Following intradermal administration:

Rare instances of blindness associated with intralesional therapy around the face and head, transient local discomfort, sterile abscesses, hyper- and hypopigmentation, and subcutaneous and cutaneous atrophy (which usually disappears, unless the basic disease process is itself atrophic) have occurred.

Withdrawal symptoms:

Too rapid a reduction of corticosteroids following prolonged treatment can lead to acute adrenal insufficiency, hypotension and death. A steroid withdrawal syndrome seemingly unrelated to adrenocortical insufficiency may also occur and include symptoms such as anorexia, nausea, vomiting, lethargy, headache, fever, weight loss and/or hypotension.

Other adverse effects for oral and systemic corticosteroids:

Body as a whole:	Leucocytosis
Gastro-intestinal:	Dyspepsia, nausea, abdominal pain, increased appetite which may result in weight gain, diarrhoea, oesophageal candidiasis.
Musculoskeletal:	Tendon rupture, myalgia
Skin:	Bruising, telangiectasia, pruritis
Endocrine:	Weight gain, increased appetite
Nervous system:	Psychological dependence, dizziness, aggravation of suicidal ideation, irritability, anxiety and cognitive dysfunction.
Eye disorders:	Corneal or sclera thinning, exacerbation of ophthalmic viral or fungal disease
Anti-inflammatory and immunosuppressive	Opportunistic infections, recurrence of dormant tuberculosis

effects:	
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4.9 Overdose

Chronic:

The symptoms of glucocorticoid overdose may include confusion, anxiety, depression, gastrointestinal cramping or bleeding, ecchymosis, moon face and hypertension. After long-term use, rapid withdrawal can result in acute adrenal insufficiency (which also may occur in times of stress). Cushingoid changes can result from continued use of large doses.

Acute:

There is no specific treatment for overdose, but supportive therapy should be instituted and, if gastrointestinal bleeding occurs, it should be managed.

For advice on the management of overdose please contact the National Poisons Centre on 0800 POISON (0800 764766).

5. PHARMACOLOGICAL PROPERTIES

Actions:

Naturally occurring glucocorticoids (hydrocortisone), which also have salt-retaining properties, are used as replacement therapy in adrenocortical deficiency states. Their synthetic analogues are primarily used for their potent anti-inflammatory effect 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.

Kenacort-A 10 injection provides a synthetic glucocorticoid corticosteroid with marked anti-inflammatory action.

6. PHARMACEUTICAL PARTICULARS

6.1 List of excipients

Each mL of the sterile, aqueous suspension provides 6.6 mg sodium chloride for isotonicity, 15 mg benzyl alcohol as a preservative, 6.4 mg carmellose sodium, and 0.4 mg polysorbate 80 in water for injections q.s. to 1 mL.

At the time of manufacture, the air in the container is replaced by nitrogen.

6.2 Incompatibilities

Not relevant.

6.3 Shelf life

36 months

6.4 Special precautions for storage

Store below 30°C, avoid freezing. Store upright. Protect from light.

6.5 Nature and contents of container

5 x 1 mL ampoules.

6.6 Special precautions for disposal and other handling

No special requirements for disposal.

7. MEDICINE SCHEDULE

Prescription medicine.

8. SPONSOR

Pharmacy Retailing (NZ) Limited trading as Healthcare Logistics
58 Richard Pearse Drive
Airport Oaks
Auckland, New Zealand

Telephone: (09) 918 5100
Email: aspen@aspenpharma.co.nz

9. DATE OF REVISION OF THE TEXT

28 May 2018

SUMMARY TABLE OF CHANGES

Section changed	Summary of new information
	Update to the SPC-style.
4.4 & 4.8	4.4: Addition of 'visual disturbance' safety text at the request of Medsafe. 4.8: Addition of central serous chorioretinopathy (CSCR) as an adverse event.