REVIEW ARTICLE

Comparative safety and efficacy of topical mometasone furoate with other topical corticosteroids

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ABSTRACT

Derivatives of hydrocortisone, such as mometasone furoate, a (2') furoate-17 ester with chlorine substitutions at positions 9 and 21, have been designed to improve efficacy and reduce the incidence of adverse effects. An extensive literature search of MEDLINE, Embase and other databases was conducted to review the safety and efficacy of various formulations of topical mometasone furoate. Mometasone furoate exhibits high potency with greater anti-inflammatory activity and a longer duration of action than betamethasone. In clinical trials, mometasone furoate shows comparable or significantly better efficacy, depending on the comparator, in all indications studied in both adults and children. It is well tolerated with only transient, mild to moderate local adverse effects. It is characterised by low systemic availability due to its high lipophilicity, low percutaneous absorption and rapid hepatic biotransformation, and consequently has no significant effect on the hypothalamic-pituitary-adrenal axis. The molecular biotransformation of mometasone furoate in the skin results in a lower affinity with dermal cells than epidermal cells, which contributes to its low atrophogenicity. Sensitisation to mometasone furoate is low. Overall, mometasone furoate is a highly efficacious potent corticosteroid with a low risk of both local and systemic adverse effects.

Key words: corticosteroid, eczema, mometasone furoate, psoriasis, seborrhoeic dermatitis.

INTRODUCTION

Since the introduction of hydrocortisone in 1952, topical corticosteroids have become the cornerstone of treatment for many inflammatory skin conditions due to their ability to reduce inflammation. Initial success with hydrocortisone spurred the development of new topical corticosteroids by modifying both the ring structure and side chains of the hydrocortisone molecule, leading to compounds with variable anti-inflammatory potency and side-effect profiles. These topical corticosteroids are classified in order of decreasing potency into four classes in Australia and the UK and seven classes in the USA, as per the Stoughton–Cornell classification (Table 1).

Topical corticosteroids have been associated with both local (more frequent) and systemic (infrequent) adverse effects including cutaneous atrophy, telangiectasia, striae, steroid rosacea and perioral dermatitis, hypothalamic-pituitary-adrenal axis suppression and skin infections. The potential for side-effects is often associated with the prolonged or widespread use of topical corticosteroids and usually correlates with increased clinical potency. The risk is much less when topical corticosteroids are used appropriately as per guidelines.

Topical corticosteroids are available in a variety of vehicles such as ointments, creams, lotions and gels. Recent advances in formulation technology have resulted in the development of hydrogel vehicles that are water-based, alcohol-free, non-irritating, non-greasy and moisturising. The pharmaceutical formulation of topical corticosteroids has a great influence on whether or not it penetrates the stratum corneum, and consequently on the local bioavailability and efficacy of the steroid. Further, the cosmetic aspect of treatment has been found to have a major impact on patients’ adherence, and therefore on the efficacy of the corticosteroid.

Topical mometasone furoate (0.1% w/w) is classified as a high potency corticosteroid and has been available in Australia since 1987 for S4 (prescription-only medicine in Australia) topical use as a cream, an ointment and a lotion (Table 1). An extensive literature search of MEDLINE, Embase, the Cochrane Library and Dialog databases was conducted to March 2017. Only articles of high quality directly pertaining to the safety and efficacy of topical mometasone

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Moderate potency (class 4/5 USA, class III UK)
Betamethasone dipropionate 0.05% in optimised vehicle
Clobetasol propionate 0.05%
High potency (class 2/3 USA, class II UK)
Betamethasone dipropionate 0.05%
Betamethasone valerate 0.1%
Mometasone furoate 0.1%
Moderate potency (class 4/5 USA, class III UK)
Betamethasone dipropionate 0.05%
Betamethasone valerate 0.02–0.05%
Triamcinolone acetonide 0.02%
Methylprednisolone aceponate 0.1%
Clobetasol 0.05%
Desonide 0.05%
Low potency (class 6/7 USA, class IV UK)
Hydrocortisone 0.5–1%
Hydrocortisone acetate 0.5–1%

Table 1 Classification of the potency of commonly used topical corticosteroid preparations available in Australia. Adapted from Carlos and colleagues4

| Formulations available | Ointment | Cream | Lotion | Other |
|------------------------|----------|-------|--------|-------|
| Betamethasone dipropionate 0.05% in optimised vehicle | x        |       |        | Shampoo |
| Clobetasol propionate 0.05% |         |       |        |        |
| Betamethasone dipropionate 0.05% | x        |       |        |        |
| Betamethasone valerate 0.1% | x        | x     |        |        |
| Mometasone furoate 0.1% | x        |       | x      | Hydrogel |
| Betamethasone dipropionate 0.05% | x        |       | x      |        |
| Betamethasone valerate 0.02–0.05% | x        | x     |        |        |
| Triamcinolone acetonide 0.02% | x        | x     |        |        |
| Methylprednisolone aceponate 0.1% | x        | x     | x      |        |
| Clobetasol 0.05% | x        | x     |        |        |
| Desonide 0.05% |          |       | x      |        |
| Hydrocortisone 0.5–1% | x        |       |        | Spray |
| Hydrocortisone acetate 0.5–1% | x        | x     |        |        |

Mometasone furoate of various formulations in relation to other corticosteroids in the treatment of psoriasis, eczema, atopic dermatitis and seborrhoeic dermatitis were included. Abstracts and studies relating to allergic contact dermatitis, dermatitis and seborrhoeic dermatitis were included. Activity and a longer duration of action than both topical betamethasone dipropionate 0.05% and betamethasone valerate 0.1% ointments, and its activity is similar to methylprednisolone aceponate 0.1% ointment in suppressing erythema induced by UV light in healthy volunteers.5 The precise mechanism by which corticosteroids exert their anti-inflammatory effect is unknown. In general, corticosteroids bind to specific corticosteroid receptors present in the cytoplasm.4,5 The newly formed corticosteroid-corticosteroid receptor complex translocates into the cell nucleus where it binds to corticosteroid response elements in the promoter region of the target genes, resulting in the regulation of gene expression.5 This results in the synthesis of certain anti-inflammatory proteins, while inhibiting the synthesis of certain inflammatory mediators.5 Specifically, mometasone furoate is thought to act by inhibiting the arachidonic acid pathway, significantly reducing leukotriene production, inhibiting the production of inflammatory cytokines and growth factors, and decreasing the expression of adhesion molecules.5

PHARMACOKINETICS
The extent of percutaneous absorption of topical corticosteroids depends on the vehicle, the condition of the epidermal barrier and the use of occlusive dressings.7 The high lipophilicity of mometasone furoate ensures that it binds very strongly with its receptor in the skin, thereby limiting its potential for systemic effects.5,7 Only very minimal amounts of mometasone furoate have been shown to reach the systemic circulation following topical administration.5,7 For example, following a single application of radiolabelled mometasone furoate 0.1% cream or ointment to the skin of healthy volunteers for 8 h, approximately 0.4% or 0.7%, respectively, of the applied dose was found to be absorbed systemically, with 94% of the total dose...
remaining unabsorbed on the skin and approximately 1.6% diffusing into the skin over the 5-day study period.\(^5,7\)

When 10 g/day of mometasone furoate 0.1% ointment was applied under occlusion for 20 h/day for 5 days in healthy volunteers, plasma concentrations of mometasone furoate peaked at 150 ng/L and declined rapidly to 15 ng/L after 72 h. Only 0.00076% of the entire dose was excreted in the urine, and no metabolites were detected in the plasma.\(^5,7\)

Any mometasone furoate that does reach systemic circulation has a low resorption rate and undergoes rapid biotransformation in the liver.\(^5,7\)

**CLINICAL SAFETY**

In clinical studies designed to investigate the effect of topical mometasone furoate on the hypothalamic-pituitary-adrenal axis in healthy adult volunteers (Table 2), no clinically significant decrease in serum cortisol levels were observed when 10 g/day mometasone furoate 0.1% ointment was applied with occlusion for up to 20 h/day for 5 days.\(^8\) A decrease in serum cortisol levels was found when 50 g/day mometasone furoate 0.1% ointment was applied to 60% of the body surface with occlusion for 22 h/day for 5 days. However, this decrease was found to be equivalent to that observed for methylprednisolone aceponate 0.1% ointment.\(^9\)

In another study, 16 g/day mometasone furoate 0.1% ointment applied with occlusion for 11 h/day for 5 days was found to produce a significant decrease in plasma cortisol levels, which was greater than that observed for hydrocortisone butyrate 0.1%.\(^10\) Although some decreases in serum cortisol levels were observed in these studies, no symptoms of hypothalamic-pituitary-adrenal axis suppression such as fatigue, anorexia, nausea, vomiting or weight loss, was observed in any of the volunteers.\(^5,8\)–\(^10\)

In further clinical studies in patients with psoriasis,\(^11\) adult patients with atopic dermatitis\(^12\) children with atopic dermatitis,\(^13,14\) and in adults with various other dermatoses,\(^2\) mometasone furoate 0.1% cream was applied once daily for up to 12 weeks and was not associated with any significant change in mean cortisol levels from baseline.

In clinical studies designed to investigate the atrophogenic potential of topical mometasone furoate 0.1% (Table 2), no clinical or histological signs of skin atrophy were observed in six volunteers after 12 months of a once-daily application of mometasone furoate 0.1% cream.\(^5\) Further clinical trials of up to 6 weeks in healthy adult volunteers\(^15–17\) and patients with psoriasis\(^18\) have found the atrophogenic potential of mometasone furoate 0.1% ointment or cream to be low. However, in two studies, mometasone furoate 0.1% ointment used for 6 weeks with occlusion in healthy adult volunteers was associated with a significantly greater incidence and severity of skin atrophy and telangiectasia than methylprednisolone aceponate 0.1% ointment\(^9\) and prednicarbate 0.1% ointment,\(^1\) but it was not as pronounced as it was for betamethasone valerate 0.1% ointment.\(^1\)

In clinical trials (presented in Supplementary Table S1, S2, and S3), a once-daily application of various topical
### Table 2  Effect of topical mometasone furoate 0.1% ointment and cream on serum cortisol levels and its potential to cause skin atrophy

| Reference                        | Trial design | Treatment                                      | Duration | Patients treated (evaluated) (n) | Comparator potency | Safety (n of patients) |
|----------------------------------|--------------|------------------------------------------------|----------|----------------------------------|--------------------|------------------------|
| **Effect on serum cortisol levels** |              |                                                 |          |                                  |                    |                        |
| Higashi and colleagues\(^8\)     | nb           | MF 0.1% ung 10 g/day (20 h/day) oc              | 5 days   | 5 (5)                            | NA                 | No effect on serum cortisol levels |
| Bressinck and colleagues\(^11\)  | r, db, pg    | MF 0.1% ung 15 g od HYD 1.0% ung 15 g od        | 5 weeks  | 24 (24)                          | Low                | Slight change in plasma cortisol level from baseline for both MF and HYD, which was NS from each other; MF  ̸=  HYD |
| Kecskés and colleagues\(^9\)     | r, db        | MF 0.1% ung 50 g od (22 h/day), 60% body oc     | 5 days   | 11 (11)                          |                    | Both MF and MPA decreased serum cortisol levels to a similar extent; MF  ̸=  MPA |
|                                   |              | MPA 0.1% ung 50 g od (22 h/day), 60% body oc    |          |                                  |                    |                        |
| Visscher and colleagues\(^10\)  | r, o, co     | MF 0.1% cr 16 g/day (11 h/day) oc               | 5 days   | 12 (12)                          | Moderate           | Both MF and HYDB produced significant suppression of plasma cortisol concentrations during treatment, however complete recovery of the adrenal function took place once treatment ceased; MF  >  HYDB |
|                                   |              | HYDB 0.1% cr 16 g/day (11 h/day) oc             |          |                                  |                    |                        |
| **Effect on the skin**           |              |                                                 |          |                                  |                    |                        |
| Brasch in Prakash\(^5\)          | o            | MF 0.1% cr od                                  | 52 weeks | 6                                | NA                 | No clinical or histological signs of skin atrophy seen |
| Katz and colleagues\(^18\)      | bpc          | MF 0.1% ung od                                 | 6 weeks  | 51 (51)                          | Low                | MF: mild skin thinning (1), moderate telangiectasia (1) |
|                                  |              | HYD 1.0% ung od                                 |          |                                  |                    | HYD: mild skin thinning (1); MF  ̸=  HYD |
| Kerscher and colleagues\(^16\)   | r, db        | MF 0.1% ung od                                 | 6 weeks  | 12                               | Low                | All treatments reduced skin thickness over 6 weeks, however this reduction was NS compared to baseline; PRD  ̸=  MF  ̸=  HYD  ̸=  V |
|                                  |              | HYD 0.25% ung bid                               |          |                                  | Moderate           | NS changes in skin thickness as determined by ultrasound for all groups |
|                                  |              | PRD 0.25% ung bid                              |          |                                  |                    | No signs of skin atrophy for all groups; MF  ̸=  MPA  ̸=  HYD  ̸=  V |
| Hoffmann and colleagues\(^15\)   | r, db, ic    | MF cr 200 mg od oc HYD 0.1% cr 200 mg od oc MPA cr 200 mg od oc V (MPA, MPA concentration unknown) | 5 weeks  | 10 (10)                          | Low                | No clinical or histological signs of skin atrophy seen |
|                                  |              |                                                  |          |                                  |                    |                        |
| Kecskés and colleagues\(^9\)     | r, db, ic    | MF 0.1% ung 5 x /week oc                       | 6 weeks  | 20 (20)                          | Moderate           | MF: pronounced skin atrophy (10), moderate skin atrophy (8), slight skin atrophy (2), very pronounced telangiectasia (5), pronounced telangiectasia (12), moderate telangiectasia (2), slight telangiectasia (1) |
|                                  |              | MPA 0.1% ung 5x/week oc                         |          |                                  |                    | MPA: slight skin atrophy (15), no skin atrophy (5), moderate telangiectasia (5), slight telangiectasia (15), no telangiectasia (4) |
|                                  |              | V                                                |          |                                  |                    | V: slight skin atrophy (5), telangiectasia (0); MF  >  MPA |
| Korting and colleagues\(^1\)     | r, db        | MF 0.1% ung bid                                 | 6 weeks  | 24 (22)                          | High               | MF: skin thickness reduced by 17%; skin atrophy (2); telangiectasia (2) |
|                                  |              | BMV 0.1% ung bid                                |          |                                  |                    | BMV: skin thickness reduced by 24%; skin atrophy (2); telangiectasia (2) |
|                                  |              | PRD 0.25% ung bid                               |          |                                  | Moderate           | PRD: skin thickness reduced by 15%; skin atrophy (0); telangiectasia (0) |
|                                  |              | V                                                |          |                                  |                    | V: skin atrophy (0); telangiectasia (0); BMV  >  MF  >  PRD  >  V |

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formulations of mometasone furoate 0.1% applied without occlusion was found to be generally well tolerated, regardless of the patient’s age or dermatological condition. Adverse reactions reported in <5% of patients include transient and mild to moderate pruritus, burning, stinging, folliculitis, dryness, acneiform eruptions, and signs of mild skin atrophy and telangiectasia. Less common adverse reactions found in <1% of patients include erythema, oedema, fissures, urticaria, disease exacerbation, pimples, papular and pustular formations. These adverse events were no more pronounced than those observed for other corticosteroids, even those of low potency.5,18 A few cases have been reported of severe side-effects. However, these are very rare and have been associated with patient abuse or the long-term use of topical mometasone furoate.19

Patch test studies have shown that topical mometasone furoate is associated with a negligible risk of primary contact sensitisation and allergic cross-reaction, even in patients known to be hypersensitive to corticosteroids.5

**SAFETY IN PREGNANCY AND BREASTFEEDING**

Topical mometasone furoate 0.1% has been classified as a category B3 drug in Australia. There are no adequate and well-controlled studies of the teratogenic effects of mometasone furoate in pregnant women.4 It should therefore be used with caution during pregnancy and only if the potential benefit to the patient outweighs the potential risk to the foetus.4 Further, high-potency corticosteroids should not be used on pregnant patients in large amounts or for prolonged periods of time.

Systemically administered corticosteroids are secreted into breast milk but the quantities are too low to have a deleterious effect on the infant. It is not known whether topicaly applied mometasone furoate is absorbed in sufficient quantities to produce detectable levels in breast milk.4 Therefore, mometasone furoate should be used with caution during breastfeeding.4 Temporary cessation of breastfeeding during treatment should also be considered.

**CLINICAL EFFICACY**

Supplementary Table S1 shows the efficacy of mometasone furoate 0.1% ointment compared with other corticosteroid ointments observed in clinical trials, as determined by the percentage improvement from baseline in total disease sign or symptom scores. The efficacy of mometasone furoate 0.1% ointment in patients with moderate to severe psoriasis vulgaris (n = 48-245) in comparative 2–8 week trials was significantly greater than that of the vehicle,3 mildly potent hydrocortisone 1.0% ointment applied once daily,11,18 moderately potent fluocinolone acetonide 0.025% ointment applied thrice daily20 and several other highly potent topical corticosteroids ointments applied twice daily, including triamcinolone acetonide 0.1%,20 fluticasone propionate 0.005%,21 and betamethasone valerate 0.1%,5,22,23 in patients aged 12 years or older. However, there was no significant difference in patients with psoriasis24 or atopic dermatitis25 treated with either mometasone furoate 0.1% ointment or highly potent betamethasone dipropionate 0.05% ointment for up to 4 weeks. In addition, there was no significant difference in treatment outcomes when topical mometasone furoate 0.1% ointment was applied either once or twice daily in patients with psoriasis for up to 15 days.26

**Table 2 Continued**

| Reference | Trial design | Treatment | Duration | Patients treated (evaluated) (n) | Comparator potency | Safety (n of patients) |
|-----------|-------------|-----------|----------|--------------------------------|-------------------|-----------------------|
| Koivukangas and colleagues17 | o, db | MF 0.1% cr od BMV 0.1% cr hid | 1 weeks | 15 (15) | High | No detectable effect on skin thickness was seen; MF = BMV |
|           |             |           |          | NA | No difference between MF and BMV in their ability to reduce collagen synthesis |

1Patients with psoriasis. bid, twice daily; BMV, betamethasone valerate; bpc, bilateral paired comparison; co, cross-over; cr, cream; db, double blind; HYD, hydrocortisone; HYDB, hydrocortisone butyrate; ic, intra-individual comparison; MF, mometasone furoate; MPA, methylprednisolone aceponate; NA, not applicable; nb, nonblind; NS, not significant; o, open label; oc, with occlusion; od, once daily; pg, parallel group; PRD, prednicarbate; r, randomised; ung, ointment; V, vehicle.

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more efficacious than corticosteroids of moderate potency applied twice daily for up to 5 weeks, such as hydrocortisone butyrate 0.1% cream, but it was significantly inferior to super-potent clobetasol propionate 0.05% cream in patients with eczema. In children aged between 6 months and 12 years with atopic dermatitis, mometasone furoate 0.1% cream applied once daily was found to be significantly superior to less potent corticosteroids applied twice daily, such as hydrocortisone 1.0%, clobetasone valerate 0.05% and hydrocortisone valerate 0.2% creams. However, mometasone furoate 0.1% cream was found to have similar efficacy to clobetasol butyrate 0.05% in children with mixed dermatoses. In further studies it was shown that a regimen of mometasone furoate 0.1% cream applied 2 or 5 days/week for up to 56 weeks, and 2 days/week for 26 weeks was prophylactic in both adults with eczema and children with atopic dermatitis. Recently, new cream formulations of mometasone furoate have become available, which have been shown to be bioequivalent to the older preparations.

The efficacy of mometasone furoate 0.1% lotion applied once daily to patients with moderate to severe scalp psoriasis aged ≥12 years (n = 192–205) in comparative 5-week trials was significantly greater than that of other highly potent topical corticosteroids lotions including triamcinolone acetonide 0.1% and betamethasone valerate 0.1% applied twice daily (Supplementary Table S5). A formulation of mometasone furoate 0.1% using a novel water-based, alcohol-free, non-irritating, non-greasy and moisturising hydrogel as a vehicle has been developed for use in Australia. Clinical trials have not been conducted with mometasone furoate 0.1% hydrogel; however, mometasone furoate 0.1% hydrogel formulation has been shown to be bioequivalent to mometasone furoate 0.1% lotion.

CONCLUSION

The effect of topical mometasone furoate 0.1% in various topical preparations has been well studied over many years. In clinical trials the efficacy of mometasone furoate 0.1% ointment, cream and lotion applied once daily to patients with a variety of inflammatory skin conditions including psoriasis, eczema, atopic dermatitis and seborrheic dermatitis for between 2–12 weeks, was found to be significantly superior to twice-daily applications of less potent corticosteroids of similar formulations, and it was comparable to or significantly superior to that of several other highly potent corticosteroids of a similar formulation that required application twice or thrice daily, regardless of the patients’ age.

Although mometasone furoate 0.1% demonstrates greater anti-inflammatory activity and a longer duration of action than betamethasone relative to other topical corticosteroids with a similar or weaker potency, topical formulations of mometasone furoate 0.1% have been shown to be associated with a low risk of corticosteroid-related adverse events, such as skin atrophy and other local events, and to have a very limited potential to induce systemic adverse effects, including hypothalamic-pituitary-adrenal axis suppression.

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Supporting Information

Additional Supporting Information may be found online in Supporting Information:

Table S1 Clinical trials examining the comparative safety and efficacy of mometasone furoate 0.1% ointment versus other corticosteroids in the management of patients with psoriasis vulgaris and atopic dermatitis.

Table S2 Clinical trials examining the comparative safety and efficacy of mometasone furoate 0.1% cream versus other corticosteroids in the management of patients with psoriasis vulgaris, atopic dermatitis, seborrhoeic dermatitis, eczema and other corticosteroid-responsive dermatoses.

Table S3 Clinical trials examining the comparative safety and efficacy of mometasone furoate 0.1% lotion versus other corticosteroids in the management of patients with scalp psoriasis.