Eyelid dermatitis to red face syndrome to cure: Clinical experience in 100 cases

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A retrospective review of all eyelid dermatitis patients seen over an 18-year period revealed a large subgroup of patients who had, as the basis for their ongoing problem, an addiction to the use of topical or systemic corticosteroids. This group of 100 patients often sought many consultations with various physicians. Unrelenting eyelid or facial dermatitis often resulted in the use of increasing amounts of corticosteroids for longer periods of time. Soon the skin became addicted. Once the work-up ruled out other causes, the remedy for the problem was absolute total cessation of corticosteroid usage. This article describes the typical history of the problem, the evaluation of these patients, and the distinctive pattern of flaring erythema that ensued when the corticosteroids were ceased. We stress the absolute necessity of total cessation of corticosteroid use as the only treatment for corticosteroid addiction. We also demonstrate that no additional therapy or further consultations were necessary once remission was obtained after topical corticosteroid abuse was halted. (J Am Acad Dermatol 1999;41:435-42.)

Since the inception of the Contact Dermatitis clinic at UCLA in the mid 1970s, many referred patients had eyelid rashes. The etiology of most of the cases was atopic or seborrheic dermatitis. A much smaller number of patients had allergic or irritant contact dermatitis.

Retrospective reviews cite larger numbers of "contact" dermatitis as the cause of eyelid dermatitis.[1-3] Our patient population appeared to be different.

When allergy was demonstrated it was often to ophthalmic medications, lens cleaning solutions, or preservatives in cosmetics or topical medications. "Anti-aging" cosmetics with varying amounts of alpha-hydroxy acids and retinoids were the most common source of the irritant contact dermatitis cases. Not one case of isolated eyelid dermatitis was seen resulting from nail or hair products.

Many of our patients had a complex dermatitis that began with atopic or seborrheic dermatitis; they had invariably been using topical corticosteroids chronically around the eyes. Increasing the strength or frequency of topical corticosteroids or even systemic corticosteroids had not solved the problem before referral. Indeed, attempts at lowering the dose or strength of the topical corticosteroid or even stopping the medication met with only failure and a more severe recurrence of the rash.

We suspected that the problem was substantially one of recurrent vasodilation and vasoconstriction along with other effects of topical corticosteroid abuse. Therefore, after full evaluation, the main treatment of 100 patients described herein was absolute cessation of topical and oral corticosteroids.

PATIENTS AND METHODS

A total of 100 patients were identified and included in this analysis. All patients with eyelid dermatitis were evaluated with a detailed contact dermatitis history including cosmetic and occupational chemical exposure. Patch testing, photo-patch testing, blood studies, and, in a few instances, skin biopsies were performed when indicated. Patients with specific allergic contact dermatitis proven by patch testing and cessation of the offending chemical resulting in cure are not included in this analysis. The most frequent primary diagnoses among the 100 patients we discuss here were atopic dermatitis and seborrheic dermatitis. A few cases of dry skin, post-laser dermatitis, and post-phenol peel dermatitis were also seen. Patients defined as atopic had a childhood or early adult history of asthma, hay fever, or eczema. They had evidence of irritation from soaps, pruritus from wearing wool, hand eczema, and some had histories of short-term eyelid rashes. Patients defined as seborrheic had a typical malar rash with some scaliness in the scalp, behind the ears, or near the lower eyelids. The patients who had undergone surgical peel had been given topical or systemic corticosteroids immediately after or within days of the cosmetic proce-

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Table I. Therapy

1. Total corticosteroid cessation
2. Ice or cool water compresses, 4 times a day for 15 minutes
3. Multiple reassuring phone calls and visits
4. Hydroxyzine 10 mg twice daily if pruritic
5. Mild lubrication when exfoliating
6. UVB therapy once or twice a week when flare subsides

dure. None of the patients exhibited acneiform perioricial dermatitis.

An expanded tray of 45 patch test chemicals was used for testing. For the last 10 years various corticosteroid preparations have also been tested. Initially, creams of finished products were used, but in the last 5 years, 4 standardized preparations, tixocortol, budesonide, clobetasol, and hydrocortisone-17-butyrate have been used. Readings were done at 48 and 72 hours with, on occasion, more delayed readings. Photo-patch testing was performed on 2 patients.

When no culprit allergic chemical was detected by patch test, no systemic problem was demonstrated, and no other etiology for the rash was suggested by the workup, the patients were given the diagnosis of steroid addiction and erythema resulting from the corticosteroids. After total cessation of topical and systemic corticosteroid usage, they were followed through total remission and for several months to years later.

RESULTS

Initial pattern of erythema and rash

The typical history began with the use of an over-the-counter (OTC) weak topical corticosteroid when patients were left to their own devices or with the use of a mid strength to super potent topical corticosteroid prescribed by a physician. The story proceeded the same way in almost all patients. The initial dermatitis improved or appeared to clear but then a few days to a few weeks later, it relapsed. The corticosteroid cream was used again. Ensuing flares came closer together and reapplications of medication became more frequent. The dermatitis free periods became shorter and shorter. As more physicians were seen, the strength of the topical corticosteroid preparation was increased and then, as frustration mounted, systemic intramuscular or oral corticosteroids were used. At times, pharmacies renewed prescriptions without physician approval or patients were able to obtain topical corticosteroids from friends or family members.

As the dermatitis began to spread or became more chronic, more erythema occurred in a larger area around the eyes. This was usually accompanied by a burning sensation. New consultations were sought and, on occasion, patch testing was performed; it usually revealed no clinically helpful data. Patients were often instructed to avoid cosmetics, chemicals in the workplace, clothing, bedding, and a large array of suspected, but unproven, allergens.

As the number of corticosteroid preparations and physicians mounted, and as the days of freedom from dermatitis decreased, burning sensations grew significantly and became disabling. It was at this point that most of the patients were referred to us (see Figs 1 and 4).

Clinical response to cessation of topical corticosteroids

When all corticosteroids were discontinued, a flare of the dermatitis around the eyes usually occurred in approximately 5 to 7 days and was accompanied by marked erythema and a severe burning sensation. Sometimes the entire face blossomed with redness. Treatment consisted of cool ice compresses, time, and many navigated "panic" phone calls (Table I). This initial flare lasted anywhere from 2 to 10 days with resultant peeling and normalization of the skin. The skin did not tolerate any lubricating or medicinal creams. Only more irritation and itching would occur. All cosmetics were avoided. There was no need for analgesia even though the burning sensation was very severe at times.

Within 2 to 3 weeks the patients experienced repeated flares of erythema in the exact same locations or with progression down the face onto the neck or upper chest. On occasion, marked edema of the eyelids occurred during flares. The same therapy and the same supportive care were offered and this episode also cleared, often in less time than before. During typical flares, the central portion of the face, along with the periorbital skin and forehead, became very red but the outer cheeks back toward the ears retained normal skin color. A sharp cutoff line between red and normal-looking skin often ran longitudinally down the mid to outer cheeks. The nose and upper lip remained clear; this has been referred to as the "headlight"[4] or "neon sign" (see Figs 2 and 5).

The duration of previous corticosteroid use and the potency of these agents determined the subsequent length and frequency of flares. The length of the "normal time" between flares usually increased and the redness became more short lived. Many of the patients required 6 to 18 months to clear totally, experiencing 3 to 12 flares. The flares appeared to occur randomly with no antecedent event.

On occasion, if the length of previous corticosteroid use had been very long, a distant or "metastatic" rash occurred elsewhere on the body when the face flared. The atopic patient typically flared in the

Table II. Summary of results

	Atopic dermatitis	Seborrheic dermatitis	Other*
1. Number	67†	24†	9
Male	15	12	1
Female	52	12	8
2. Age range (y)	22-86	22-80	37-78
3. Topical corticosteroid range (mo)	3-480	2-156	3-120
4. Systemic corticosteroid range (mo)	0-240	0-12	0-6
5. Burning	43	10	4
6. Telangiectases	31	5	5
7. Atrophy	26	4	2
8. Patch test done	57	12	5
9. Distant rash when corticosteroid stopped	20	1	1
10. Clearing time range (mo)	2-30	1-19	2-7
11. Follow-up	50	21	6
12. Follow-up range (mo)	3-120	4-156	24-48
13. Resumed using corticosteroids or lost to follow-up	13‡	1	1

*Dry skin = 3; laser/phenol = 3; psoriasis = 1; urticaria = 1; flat warts = 1.
†Three of the 67 atopic patients and 1 of the 24 patients with seborrheic dermatitis also had phenol/laser procedures.
‡Two patients are being followed (included in follow-up numbers) while they continue to use topical steroids, mid-strength, 2 to 3 times/week. The other 13 patients sought other medical advice after several episodes of rebound erythema. Ten patients were not seen in long-term follow-up, only until clear for 6 to 8 weeks.

Table III. Estimated clearing times*

	Duration of steroid use				
Class of steroid	2 mo	3-6 mo	7-12 mo	12-24 mo	> 24 mo
Weak					
Class I	<1 mo	1-2 mo	2-3 mo	3-6 mo	3-6 mo
Class II					
Moderate					
Class III					
Class IV	1 mo	2-3 mo	2-6 mo	6-12 mo	12 mo
Class V					
Potent					
Class VI	1 mo	2-3 mo	2-6 mo	>12 mo	18-36 mo
Class VII					

*This is only a guide. Some patients used a steroid 3 times a day for 6 months whereas others used it only once a day for 6 months. Their subsequent clearing times differed on the basis of the amount of steroid used and not on duration. The use of systemic steroids increased the clearing times in any category.

antecubital areas or the legs or upper chest, often having never experienced a rash in those locations before. On some occasions when pruritus or mild eczema occurred in a distant site, the judicious minimal use of a topical low potency corticosteroid to the nonfacial site for 1 to 3 days was helpful and did not exacerbate the facial problem. Systemic corticosteroids were not used. Topical corticosteroid application was not allowed anywhere within the vicinity of the face, neck, or chest for many months. UVB phototherapy and natural sunshine were used as therapy on occasion.[5] Table II summarizes the results.

There appeared to be a relationship between the intensity of previous topical corticosteroid use (duration, frequency of application, amount, potency) and the amount of time needed for total clearing of the problem. For example, those patients who used only weak steroids for moderate periods of time had a shorter recovery phase than those who used super potent topical corticosteroids for longer periods of time. Table III gives a time framework for recovery (see Figs 3 and 6).

Atrophy of the eyelid was noted in several patients. The skin was telangiectatic and shiny. These

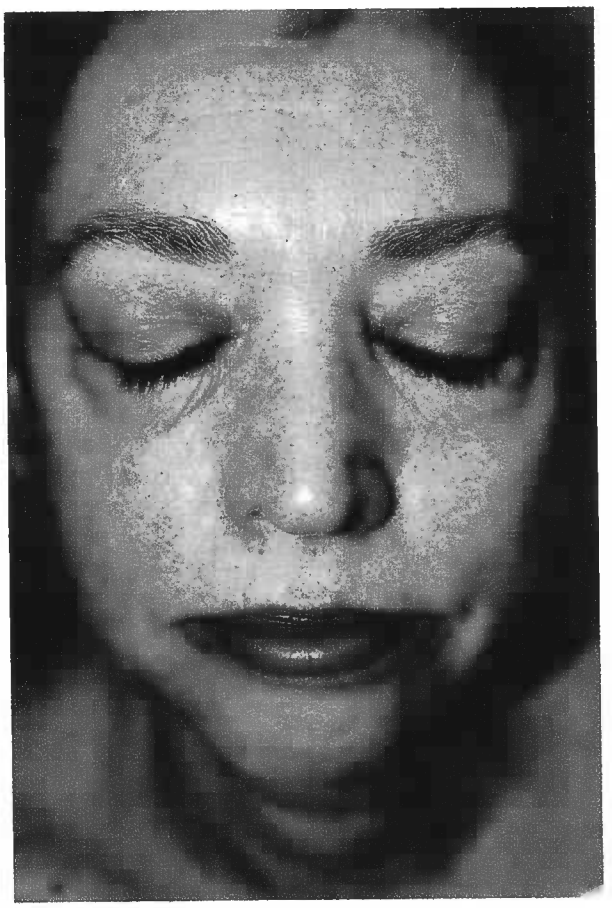

Fig 1. Patient 23. Eyelid dermatitis after 36 months of super potent steroid application.

Fig 2. Patient 23, 4 months later. Third flare of erythema.

patients, having used corticosteroids for a long period of time, required about a year for the atrophy to clear. Topical retinoic acid was tried in a few patients but irritation usually occurred and the drug had to be stopped. As long as the telangiectasia and atrophy were present, mild recurring erythema continued. Ultimately, atrophy did repair in all patients and subsequently the erythema ceased.

Patch test results

Twelve patients demonstrated between 4 and 10 positive patch test results. There appeared to be no specificity. Retesting was not done. Several patients demonstrated strong positives but continued to use these chemicals with no intolerance (rubber, fragrance, nickel, corticosteroids elsewhere). Irritability to taping was common. Because of the observation of distant rashes occurring when corticosteroids were discontinued, we believe that the total skin of these patients is "excitable" and that interpretation of positive patch tests is suspect. Photo-patch tests in 2 patients were negative.

Summary of results

Women made up 72% of the patients in this series; 67% of all patients were atopic. The duration of previous corticosteroid use ranged from 3 to 480 months with the vast majority between and 1 and 2 years. The atopic patients had used corticosteroids for the longest periods of time. It was evident that addiction could occur with as little as 2 months of continuous usage. Systemic corticosteroids had been prescribed in 47% of patients for varying periods of time; 57% of all patients experienced burning, 41% had telangiectasia, and 32% had some signs of atrophy of the eyelid skin. Patch testing was performed in 74 of the patients, but positive reactions did not appear to be relevant.

After the corticosteroid use was stopped, 85% of the patients had a rocky course of flaring and rebounding. Fifteen patients would not agree with the diagnosis, could not tolerate the cessation of the corticosteroids, or sought other medical care. A distant or "metastatic" rash was seen when the corticosteroids were stopped in 22% of the patients. This

was typically eczematous and often involved the neck, arms, or trunk. The rash usually cleared within 2 weeks, but on rare occasions some topical corticosteroids were very judiciously used for 1 to 3 days on distant sites. UVB therapy was given to several of these patients for about 3 or 4 treatments over approximately 10 days. Clearing, with no flares for at least 4 to 6 weeks, occurred between 2 and 30 months after corticosteroid cessation for the atopic patients and 1 to 19 months for the seborrheic dermatitis patients. The "other" category of 9 patients took 2 to 7 months to clear. Follow-up from 3 to 156 months was accomplished in 77% of the patients. No recurrences of the rash occurred in 75 of these individuals (Table III).

DISCUSSION

The problems associated with systemic corticosteroids are well known.[6] Side effects of long-term topical corticosteroid application to the face such as perioral dermatitis,[7] steroid rosacea,[8-14] periocular dermatitis,[15] and glaucoma[16,17] are also well known.

When treatment is stopped after long-term topical corticosteroid usage, a rebound phenomenon occurs that takes the form of a dermatitis with intense redness, scaling, crusting, and sometimes pustulation.[10] Even though this phenomenon was observed 20 years ago, the cure of this addiction has not been sufficiently addressed in the literature.

Allergic contact dermatitis to corticosteroids has also been described.[18-25] Positive patch tests to various forms of corticosteroids have been offered as evidence for their involvement in many chronic rashes. At times patients are said to be allergic to only some of the corticosteroid creams but not ointments of the same corticosteroid, to preparations that they had never used, and to the chemical tixocortol, which is not available, and has never been used clinically, in the United States. Papers discuss patch tests, not the outcome of the patient's rashes. Patient histories in many of the papers reveal chronic erythema around leg ulcers, on the face, and in the anal area. We believe that the vast majority of these patients have corticosteroid erythema, not an allergy to corticosteroids. Atopic patients can have unusual patch test reactions. When their skin is in an "excited state" they can exhibit many positive reactions that we feel are not clinically significant. In over 500 patch test patients seen in our facilities, we have only observed 3 positive reactions to tixocortol or to other corticosteroid preparations. In all 3 patients, application of corticosteroids to unaffected areas failed to elicit an allergic response. This 0.6% positivity (even though clinically insignificant) is well below the rate of 4.5% reported in other studies.[24,25] It is also rare that an atopic individual demonstrates allergic contact dermatitis.[26,27]

Fig 3. Patient 23, 6 months after last flare of erythema. No steroids, no medications, and using all cosmetics.

Multiple factors in vascular physiology affect dilation of blood vessels, one of which is nitric oxide. This chemical, which is released by the endothelium of blood vessels as endothelium-derived relaxing factor,[28] is a natural dilator. It is profoundly inhibited by cortisol and all synthetic glucocorticoids.[29] When a vessel is constricted either endogenously or exogenously (ie, with the use of topical corticosteroids), various metabolites, including nitric oxide, build up to counteract this constriction. Because of this build-up, when the corticosteroid has worn off and the vessels are allowed to return to their normal size, they actually dilate to a size larger than their original diameter. With the daily use of topical corticosteroids of mid or moderate strength, the vessels are constantly being constricted and a continual build-up of natural dilators occurs. Instead of returning to their normal size after corticosteroid cessation, the vessels begin to remain dilated for longer amounts of

Fig 4. Patient 65. Eyelid dermatitis after 7 years of topical and systemic steroids.

Fig 5. Patient 65, 27 months after steroid cessation. Last significant flare of dermatitis involving parts of face.

time. This potentiates the erythema, burning, and itching.

The vasoconstrictor effect of corticosteroids might not be confined just to the site of application but can spread locally.[30] This effect could occur by direct spreading of the medicament on the skin or by a physiologically mediated vasoconstriction adjacent to the area of application without any spread of the medication. A patient was described who had been applying betamethasone valerate lotion to the scalp for 16 years. She experienced distant telangiectases on the cheek, and it was felt that percutaneous

Fig 6. Patient 65, 4 years after last flare. No steroid usage, no other drugs taken, and using all cosmetics.

absorption of the steroid allowed for a local spread from the scalp to the face.[31] The rebound phenomenon after topical corticosteroid discontinuation is also associated with dermal hyperplasia.[32]

Atopic individuals have vasomotor instability or hypermobility of their vessels, which might account for a peculiar or exaggerated response to continuous corticosteroid usage. Their vasomotor instability might account for the prolonged erythema that is associated with a corticosteroid rebound. In our patients, the atopic individuals had a much more stormy course than the patients with seborrheic dermatitis.

Uehara, Mitsuyoshi, and Sugiura[33] reported on 135 patients with the "red face syndrome." They were all using mild and "safe" topical corticosteroids and had been applying these preparations to their face for a year or more. To reduce the frequency of topical corticosteroid application, most patients also had been using topical nonsteroidal antiinflammatory agents. Many patients confessed that they had often tried to discontinue the topical corticosteroid application but cessation was always followed by withdrawal symptoms of severely pruritic, oozing dermatitis. They then reinstated the use of mild or moderately potent corticosteroids. Biopsy specimens from these patients revealed mixtures of steroid rosacea, chronic eczema, and chronic eczema with granuloma formation. Patch testing was performed and 17% or 23 patients demonstrated positive patch test reactions to 1 or more topical corticosteroids. Positive photo-patch test reactions occurred in 4 cases. Also, these patients had positive reactions to topical nonsteroidal antiinflammatory agents, moisturizers, and cosmetics. The authors treated these patients by keeping the face absolutely clean, using nonsteroidal antiinflammatory agents, and applying topical corticosteroids in a decreasing fashion. The use of oral corticosteroids, PUVA, topical immunosuppressive agents, cyclosporine ointment, and tacrolimus ointment were also suggested. We believe that these patients are probably similar to ours and would clear with the approach we have described.

Other similar syndromes have been described. Prolonged post peel erythema or PPE[34] is characterized by increased or prolonged erythema, pruritus, burning, and stinging after phenol peels around the eyes. Typically, hydrocortisone valerate ointment has been used 3 times/day immediately after the peel procedure. If erythema persisted, a different corticosteroid was given and systemic corticosteroids were also prescribed. Again, we believe that this kind of erythema would have cleared quickly if no corticosteroids had been used.

"Status cosmeticus"[35,36] is a syndrome in which women tolerate no eye makeup preparations at all and complain of a continual burning sensation after anything is applied to the face. All of these patients had used corticosteroids for long periods of time.

The burning sensation described in these patients is typical of our patients. Other entities such as the red scrotum syndrome[37] and perianal atrophoderma[38] probably represent the same mechanisms in a different body location. Some cases of vulvodynia[39] probably have the same explanation.

Even though it is often recognized that use and overuse of corticosteroids cause or contribute to these problems, low strength corticosteroids on a chronic basis to allay the redness and to help keep patients comfortable are still advocated by some. We believe strongly that this just perpetuates the problem indefinitely and does not allow for a full recovery.

With topical corticosteroids in wide usage, easily obtained over the counter from drug stores, hidden away in drawers from previous prescriptions, borrowed from friends and family, and often over-prescribed by physicians, problems of corticosteroid addiction occur frequently. In our analysis of 100 patients, excellent results were obtained in almost all the patients who were able to adhere to a program of total cessation of any and all corticosteroid preparations. The longer and stronger the corticosteroid, the rockier the course and the longer it took to recover.

REFERENCES

1. Svensson A, Moeller H. Eyelid dermatitis: the role of atopy and contact allergy. Contact Dermatitis 1986;15:178-82.
2. Valsecchi R, Imberti G, Martino D, Cainelli T. Eyelid dermatitis: an evaluation of 150 patients. Contact Dermatitis 1992;27:143-7.
3. Nethercott JR, Nield G, Holness DL. A review of 79 cases of eyelid dermatitis. J Am Acad Dermatol 1989;21:223-30.
4. Bender B, Prestia AE, Lynfield YL. The head light sign in neurodermatitis. Cutis 1969;5:1406-8.
5. Prystowsky JH, Keen MS, Rabinowitz AD, Stevens AW, DeLeo VA. Present status of eyelid phototherapy. J Am Acad Dermatol 1992;26:607-13.
6. Storrs FJ. Therapy. Use and abuse of systemic corticosteroid therapy. J Am Acad Dermatol 1979;1:95-105.
7. Webber G. Perioral dermatitis, an important side effect of corticosteroids. Dermatologica 1976;152:161-4.
8. Burry JN. Topical drug addiction. Adverse effects of fluorinated corticosteroid creams and ointments. Med J Aust 1973;I:393-6.
9. Kligman AM, Leyden JJ. Adverse effects of fluorinated steroids applied to the face. JAMA 1974;229:60-2.
10. Kligman AM, Frosch PJ. Steroid addiction. Int J Dermatol 1979; 18:23-7.
11. Leyden JJ, Thew M, Kligman AM. Steroid rosacea. Arch Dermatol 1974;110:619-22.
12. Calnan CD. Use and abuse of topical steroids. Dermatologica 1976;152:247-50.
13. Sneddon I. Adverse effect of topical corticosteroids in rosacea. Br Med J 1969;I:671-3.
14. Dykes PJ, Marks R. Review article: an appraisal of the methods used in the assessment of atrophy from topical corticosteroids. Br J Dermatol 1979;101:599-609.
15. Fisher AA. Periocular dermatitis akin to the perioral variety. J Am Acad Dermatol 1986;15:642-4.
16. Brubaker RF, Hapin JA. Open-angle glaucoma associated with topical administration of flurandrenolide to the eye. Mayo Clin Proc 1975;50:322-6.
17. Eisenlohr JE. Glaucoma following the prolonged use of topical steroid medication to the eyelids. J Am Acad Dermatol 1983;8: 878-81.
18. Tomoyuki H, Katoh J, Yoshioka K, Taniguchi S, Mochida K, Nishimura T, et al. Contact allergies to topical corticosteroids. Contact Dermatitis 1993;28:174-9.
19. Kalish RS. Studies in patients with corticosteroid contact allergy: a flawed analysis of structure-function relationships [letter]. Arch Dermatol 1995;131:825.
20. Lepoittevin JP, Drieghe J, Dooms-Goossens A. Studies in patients with corticosteroid contact allergy. Arch Dermatol 1995;131:31-5.
21. Lutz ME, El-Azhary RA. Allergic contact dermatitis due to topical application of corticosteroids: review and clinical implications. Mayo Clin Proc 1997;72:1141-4.
22. Morton CE, Dohil MA. A survey of patch test results with tixocortol pivalate in Vancouver. Am J Contact Dermat 1995;6:17-8.
23. Dunkel FG, Elsner P, Burg G. Contact allergies to topical corticosteroids: 10 cases of contact dermatitis. Contact Dermatitis 1991;25:97-103.
24. Lutz ME, el-Azhary RA, Gibson LE, Fransway AF. Contact hypersensitivity to tixocortol pivalate. J Am Acad Dermatol 1998;38: 691-5.
25. Isaksson M, Persson LM. Contact allergy to hydrocortisone and systemic contact dermatitis from prednisone with tolerance of betamethasone. Am J Contact Dermatitis 1998;9:136-8.
26. Samahy MH, El-Kerdani T. Value of patch testing in atopic dermatitis. Am J Contact Dermat 1997;8:154-7.
27. Halbert AR, Weston WL, Morelli JG. Atopic dermatitis: is it an allergic disease? J Am Acad Dermatol 1995;33:1008-18.
28. Ganong WF. Cardiovascular regulatory mechanisms. In: Holmes SA, editor. Review of medical physiology. Norwalk (CT): Appleton & Lange; 1991. p. 550-1.
29. Berne RM, Levy MN. The microcirculation and lymphatics. In: Underdown E, editor. Physiology 4th ed. St. Louis (MO): Mosby; 1998. p. 429-32.
30. Hessel A, Zyniewicz K. Facial blanching caused by superpotent corticosteroids. Cutis 1991;48:395.
31. Hogan DJ, Rooney ME. Facial telangiectasia associated with long-term application of a topical corticosteroid to the scalp. J Am Acad Dermatol 1989;20:1129-30.
32. Lavker RM, Schechter NM, Lazarus GS. Effects of topical corticosteroids on human dermis. Br J Dermatol 1986;115(suppl 31):101-7.
33. Uehara M, Mitsuyoshi O, Sugiura H. Diagnosis and management of the red face syndrome. Dermatol Ther 1996;1:19-23.
34. Maloney BP, Millman B, Monheit G, McCollough EG. The etiology of prolonged erythema after chemical peel. Dermatol Surg 1998;24:337-41.
35. Fisher AA. Part I: "Status Cosmeticus": a cosmetic intolerance syndrome. Cutis 1990;46:109.
36. Fisher AA. Part II: The management of eyelid dermatitis in patients with "status cosmeticus": the cosmetic intolerance syndrome. Cutis 1990;46:199.
37. Fisher BK. The red scrotum syndrome. Cutis 1997;60:139-41.
38. Goldman L, Kitzmiller KW. Perianal atrophoderma from topical steroids. Arch Dermatol 1973;107:611-3.
39. McKay M. Vulvodynia. A multifactorial clinical problem. Arch Dermatol 1989;125:256-62.