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The SINGAPORE FAMILY PHYSICIAN

FAMILY PRACTICE DERMATOLOGY

- Hair Loss
- Tinea Pedis
- Atopic Dermatitis
- Nail Problems
- Minimizing Adverse Effects of Corticosteroids

ISSN 0377-5305

Vol. XIX NO. 4
OCT/DEC 1993

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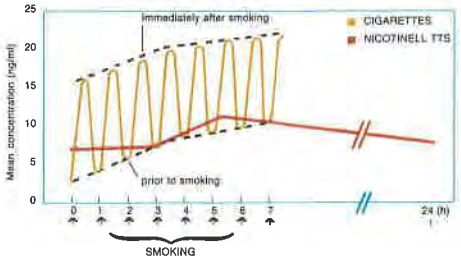

Nicotine plasma mean concentrations after cigarette smoking and after application of NICOTINELL TTS (day 10) (3)

- ***Impressive*** *abstinence rates both with and without a specialist psychological support programme.*[1,2]
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- ***Supportive*** NICOTINELL® *Stop Smoking Programme offering extra support for smokers who want to kick the habit.*

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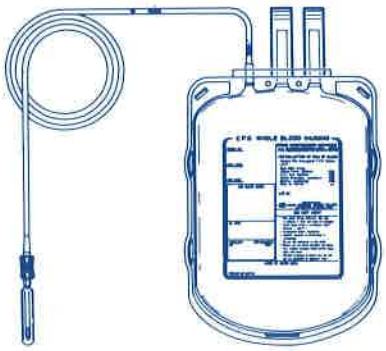

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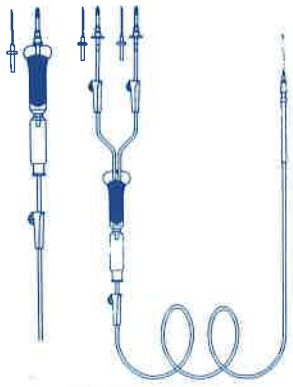

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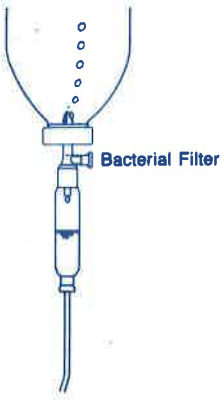

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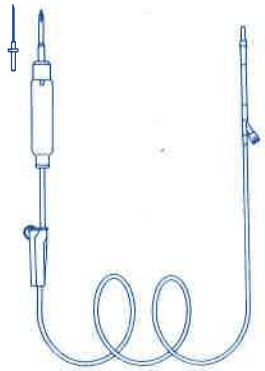

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Now, a key to effective control of PANIC

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- significant improvement in work and social functioning
- sustained effectiveness without escalation of dosage
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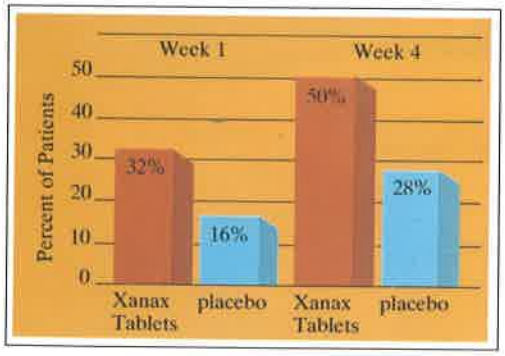
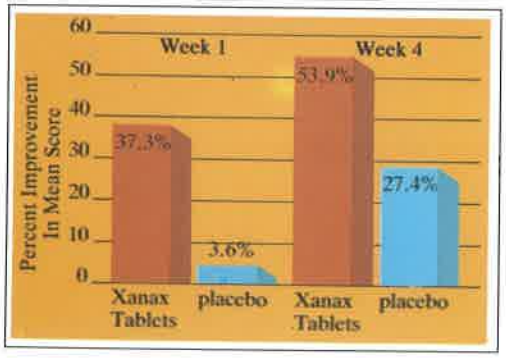
32% of patients free of panic attacks during first week[1]

Patients Reporting Zero Panic Attacks



Significant improvement on Physician's Global Evaluation from first week[2]

Improvement in Physician's Global Evaluation



Availability: Xanax Tablets are available as 0.25mg (White), 0.5mg (peach), and 1mg (lavender) scored, ovoid-shaped tablets in bottles of 100, 500.

1. Sheehan DV, Ballenger J, Jacobsen G: Treatment of endogenous anxiety with phobic, hysterical, and hypochondriacal symptoms. Archives of General Psychiatry 1980; 37: 51–59.

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PREVENTIVE MEDICINE AND PRIMARY CARE

Preventive interventions by primary care providers can dramatically reduce morbidity and premature morbidity. The primary care doctor has more opportunities than his hospital specialist colleague to practice preventive medicine. Ian McWhinney[1] has described preventive skill as a skill specific to family medicine.

PLACE

The place of preventive medicine depends on different groups of patients that the doctor comes into contact with, namely the well person, the person with early disease and the person with established disease. The strategy in these different groups of patients corresponds to primary, secondary and tertiary prevention[2].

In primary prevention, we are dealing with well people and the task is disease prevention and health promotion. Examples are immunisation and counselling on reducing the risk factors of major killer diseases like heart disease, cancer and strokes.

In secondary prevention, we are dealing with asymptomatic diseased individuals and the aim is early diagnosis and early treatment. Opportunistic blood pressure checks for both sexes and routine Pap smear testing in women for carcinoma cervix uteri are examples.

In tertiary prevention, we are dealing with patients with clinical disease and the aim is to maximise function and reduce complications. Rehabilitation for stroke, blood sugar control in diabetes or routine foot care also in diabetes are examples.

PROBLEMS

The typical medical setting presents several barriers to the incorporation of activities that prevent disease and promote health. These have been mentioned by Hayward et al[3]: lack of reimbursement, time and dedicated counsellers, patient non-compliance, lack of tangible short-term benefits from preventive interventions, discordance between the traditional focus of immediate complaints versus an anticipatory approach required for preventive care, perceived inconsistency among recommendations from different professional groups and skepticism of the quality of medical evidence underlying preventive care recommendations.

A study by 10 general practices in Nottinghamshire on whether extending appointment length from seven and a half minutes or less to 10 minutes per patient would increase health promotion in general practice consultations showed interesting results (Wilson et al, 1992)[4]. Mean consultation times were 8.25 minutes in the study sessions and 7.04 minutes in the control sessions. Recording of blood pressure, smoking, alcohol consumption and advice about immunisation was significantly more frequent in the study sessions compared to control sessions. It was concluded that shortage of time was a major factor in general practitioners' failure to realise their potential in health promotion: one minute more will make a difference.

POTENTIAL

Attention to risk factors can reduce morbidity and mortality from the top killers[5]. For cardiovascular disease the risk factors are tobacco use, elevated

serum cholesterol, high blood pressure, obesity and a sedentary lifestyle. For cancer, the risk factors of importance are tobacco use, improper diet and alcohol abuse. For cerebrovascular disease the risk factors are high blood pressure, tobacco use and elevated serum cholesterol. Unintentional injuries and travel related infections, particularly malaria and AIDS, are eminently preventable killers. Health education on seat belt use, frequent reminders not to drive under the influence of alcohol, reduction of occupational hazards and avoidance of stress and fatigue will all contribute to preventing unnecessary incapacity and death.

PRINCIPLES

Preventive medicine has a cost to the patient in terms of time, anxiety and sacrifice. Five general principles are to be remembered in selecting appropriate preventive services: focus on personal health behaviours; be selective in ordering screening tests; emphasise preventive measures of proven value; help patients adopt a new role, and incorporate preventive services into every encounter.

There is compelling evidence that efforts by doctors to influence a few personal health behaviours, such as tobacco use, sexual behaviour, diet, exercise, and injury prevention, are more likely to reduce the risk of future illness and injury than virtually any other type of preventive intervention[2].

There is a need to be selective in ordering screening tests. Screening tests need to be accurate, effective and stratified by risk. There is a need to know which are universally useful and which are suitable to only selective population groups. Many tests are not proven in prolonging life or preventing disease; routine chest Xray is one such example.

There is a need to emphasise preventive measures of proven value. Many activities are useful but not emphasised enough such as adequate exercise and a prudent diet. Another example is the exhortation to wear car seat belts as they have an unrealised potential to save lives.

There is also a need to help patients adopt a new role. The traditional patient role has been a passive one. This relationship is epitomised by the treatment of infectious disease where the patient's role is to be passively compliant. In preventive medicine, the doctor-patient relationship needs to be redefined as a partnership, with the locus of control passing to the patient. Many of the important behavioural changes that need to be made can be made only by the patient, with the advice and guidance of the provider.

There is a need to incorporate preventive services into every encounter. An acute illness visit may present a teachable moment. For example, a smoker presenting with acute bronchitis presents also an opportunity to enquire whether he has ever considered stopping smoking and the explanation that smoking will predispose the person to chronic obstructive lung disease. Many patients visit their doctors only when symptomatic; many opportunities will be lost if preventive services are only offered to patients visiting for routine checkups.

CONCLUSION

Preventive interventions by primary care providers have a place and potential to dramatically reduce morbidity and premature mortality. There are however many problems of providing such care that need to be overcome. For a start we can opportunistically try to incorporate preventive care in our daily patient care.

DR GOH LEE GAN

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ONLY SKIN – DEEP?

Skin disease has been estimated to account for 5.4% of consultations by general practitioners and primary health care doctors in Singapore. Although we no longer see cases of severe bacterial and other chronic infections like tuberculosis and leprosy, the practitioner of today is faced with problems of acne vulgaris, eczemas, contact dermatitis (including occupational disease) and other skin infections e.g. scabies, viral warts and fungal infections. He must be able to diagnose and treat these common conditions appropriately and adequately. More importantly, as the patient's personal / family doctor, he must understand and address the psychosocial factors that influence the pathogenesis of these conditions — the work, community and family factors influencing the patient, his fears (often unexpressed) and the 'social disability' the condition causes in the patient — and tailor the treatment to the individual. Teaching the patient with incurable psoriasis or atopic eczema to live with his disease and helping him cope with its manifestations — in all helping him with his "dis-ease" — is surely the realm of the family physician.

Diagnosis of most skin complaints requires more than just looking at the blemish or rash or eruption. A systemic approach is required, as in any other branch of medicine. The patient's medical and dermatological background, his general health (remembering that many systemic conditions have cutaneous manifestations), the family history (especially of asthma or hay fever) and the social and occupational history are important steps in reaching an initial differential diagnosis. Physical examination must extend beyond the site of the lesion — to the scalp, nails and inside of the mouth, and occasionally the whole body skin — and, if relevant, to other organ systems. The competent family physician will arm himself with a magnifying glass, glass slides for diascopy and a Wood's UV lamp, and will be able to take skin scrapings for microscopic examination. Photopatch testing and skin bioipsies can be left to the dermatologist.

Remedies prescribed should be convenient and cosmetically acceptable. Short-term gains from the use of powerful medications must be weighed against possible side-effects, and the simplest (and maybe oldest) preparations e.g. Calamine lotion used if possible so as to reduce the cost and possible side-effects. Oral medications may include antihistamines, antibiotics or imidazole antifungal agents. Systemic steroids should be prescribed only in chronic conditions when all other measures have failed and the patient is physically and emotionally incapacitated, the dosage being constantly reviewed to use the lowest dose possible to contain the problem, and long-term therapy requires regular checking for unwanted effects like hypertension and glycosuria.

Topical corticosteroids are the drugs most widely used in dermatology, and the practitioner prescribing these must be fully aware and cognisant of the different potencies — ranging from weak through moderate and strong to very strong — and the differences in the vehicle (e.g. cream, ointment or gel) by which the therapeutic agent is delivered to be able to choose the appropriate preparation; the frequency of application influences the response, as does the manner of application (occlusive techniques increase penetration); the age of the patient and the site of the lesion have also to be taken into account — so much so that the prescribing of topical steroids has been described as much as an art as a science. Inappropriate use,

e.g. in dermatophytic or scabietic infections, may result in exacerbation of the condition or even adverse events, and use of large quantities, especially of the very strong fluorinated corticosteroids, can lead to localised or systemic side-effects. Dr Lawrence Leong's paper in this issue will help to serve as a very useful guide.

DR VASWANI MOTI

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APPROACH TO HAIR LOSS

Y C Giam MBBS, M Med (Paed), FAMS

Most hair problems in presenting patients are that of alopecia. The three major points in this challenging problem are to:

a) Make an accurate diagnosis based on sound knowledge of anatomy and pathophysiology of hair, and its affected phase of growth.

b) Exclude systemic disease.

c) Investigate and effectively manage.

ANATOMY: OVERVIEW

There are about 100,000 to 150, 000 hair follicles on the scalp. About 90% are in active growth (anagen). 10% go into an inactive retiring phase (telogen). Thus the anagen: telogen ratio is 90%:10%. Hair grows at about 0.4 mm a day. The follicular density (number of hair follicles / cm^2) decreases from 1135 / cm^2 at birth to 435 / cm^2 by 70-80 years of age. Thus older people have less hair.

The growing area is the bulb which shows a bulbous club and with a firm hair sheath, compared to a telogen hair with a small bulb.

PRACTICAL GUIDELINES TO HAIRLOSS

On average 50-100 hairs will be shed each day. Loss of more then the daily average of 100 hairs is abnormal.

Basically, check the hair and scalp and categorize into two groups:

- If the hair is affected, but the scalp is normal and non-scarred.
- If the hair and scalp is affected and scarred.

I. Non-scarring localised alopecia
For non scarring alopecia, check to see if the hair loss is patchy or generally diffuse.

For patchy alopecia:

- If the scalp shows "exclamation mark hair" at the edge of a bald patch, it is alopecia areata.
- If the scalp is scaly and with broken shafts, think of fungal infection, – tinea capitis (not common).
- If it looks "moth eaten" (small scattered irregular bald spots) think of secondary syphilis.
- If the patient is anxious, think of thyrotoxicosis (check for pulse, exophthalmos and serum thyroxine).
- If the hair loss is of irregular shape, with strong shafts, think of trichotillomania; also check for hairstyle, for traction alopecia.

Non-scarring generalised or diffuse alopecia:

- If the normal hairline is lost, and shows bitemporal "V" recession with vertex hairloss in a male, it is androgenetic alopecia; in women, it is on the vertex.
- If it is diffuse, and hair casts are seen, it is telogen effluvium from stress, acute

illness, flu, crash dieting, sometimes thyroid disease, or chronic illness, even anemia. Ask for history of these events.

– If all the hair falls out early, it is anagen effluvium from cytotoxic drugs,.

– check for hair shaft abnormalities.

II Scarring alopecia: scar seen.

– common : discoid or systemic lupus erythematosus (hair follicular plugs, telangiectasia, with active red edges seen and lesions on the face and body) and morphea (ivory yellow plaque).

– uncommon : fungal abscess as in kerion, with short broken shafts.

: folliculitis decalvans with discharging sinus; lichen planus, (to check for lesions on the body).

: pseudopelade - no apparent cause, but scarred.

GUIDELINE TO HAIR LOSS

HAIR LOSS

Non scarring

Patchy / Localised
- Alopecia areata
- tinea capitis
- thyroid disease
- syphilis
- trichotillomania
- traction
- others - systemic illness

Diffuse
- Androgenetic alopecia
- Telogen effluvium from acute illness, stress, crash diet
- chronic illness
- thyroid disease
- Anagen effluvium - cytotoxic drugs
- hair shaft abnormalities

Scarring
- Infection - fungal, bacterial abscess: e.g. folliculitis decalvans
- Discoid or Systemic lupus erythematosus
- Morphoea
- Lichen planus
- Pseudopelade
- Developmental

INVESTIGATION

After deciding on the possible diagnosis, a few useful tests are

1. *Hairpull test :* to check on hair actually coming out, and see the bulb, to check on anagen or telogen.

2. *Hairpluck test :* to check on anagen to telogen ratio, check about 50 hairs. In telogen effluvium, telogen ratio is increased to 30-40%.

3. *Hair shaft :* to see any exclamation mark hair; and to test with KOH to see if any spores or mycelium on or in the hair shaft. A hair culture for fungus may be taken.

4. *Microscopic examination* to see exclamation mark hairs or hair shaft abnormalities - twists, hair breaks.

5. *Polarisation* to see trichohexis nodosa (break) and trichoschism (tiger tail picture due to low sulphur content).

6. *Scalp biopsy :* useful for androgenetic alopecia, alopecia areata and scarring alopecia (lupus erythematosus, lichen planus etc.).

SOME COMMON SCALP CONDITIONS

Non-Scarring Alopecia

Androgenetic Alopecia

Androgenetic alopecia or male pattern baldness commonly can affect about 40% of men and women, over the age of 40. It is genetically inherited but the expression is variable.

In severe cases, the family history is pronounced and the hair fall begins early, at the late teens.

In males, there is a bitemporal recession and also vertex hair loss. The hair miniaturises and becomes small vellus hair, which will also fall and baldness sets in. The degree of loss is classified by Hamilton's grades I to VII. In women, the hair loss is over the vertex and is classified by Ludwigs I to III. At menopause, the hair fall is accelerated, but they do not bald completely.

Treatment presently is with topical minoxidil (piperidinopyrimidine) which may have an effect on the proliferation of the hair bulb and matrix of the hair. Used daily, results are seen in 30-40% only after 4 to 6 months, with a maximum possibly 2.5 times of regrowth but not to full density. It plateaus out after a year. Continued use is necessary. An uncommon side effect is allergic dermatitis. Cost is a major factor, as a bottle / month costs about $60-$80.

Alopecia Areata

This appears in 50% of persons before the age of 20. This may be localised but can be widespread. In 80% of cases, type I alopecia areata, they appear as patches. They have no history of atopy and endocrine (e.g. thyroid) problems and have a good prognosis.

In type II alopecia areata, about 10% there is a history of asthma, allergic rhinitis and atopic dermatitis and this hairloss lasts longer and recurs.

In type III alopecia areata, there is a family history of hypertension, the hairfall is chronic and can go on to alopecia universalis. The last group, Type IV alopecia areata, have endocrine (thyroid) problems, are present over the age of 40, and often have it over the occiput (ophiasis pattern).

Treatment:

Often treatment is better in children, and when the disease is present for a shorter period. It may last up to 6-8 months.

Topical steroids: Synalar gel may be used with occlusion. Intralesional triamcinolone is to be given just below the epidermis, and not beyond mid-dermis. Not preferred in large areas of alopecia.

Oral steroids: between 20-40 mg for children to adults and later taper. It may be advantageous to combine with intralesional triamcinolone. The side-effects of steroids have to be monitored.

Isoprinosine potentiates T-lymphocyte and phagocyte function. Doses of 50 mg/kg tds for 6 months may be tried. Others tried are Minoxidil 2%, topical chemical sensitisers like DNCB, squaric acid ester and diphencyprone.

Telogen effluvium

The hairloss occurs about 2 months after the event. This could be hormonal, nutritional factors, iron deficiency, psychological stress, drugs, illnesses, major surgery hair grooming procedures, thyroid diseases (hyper or hypothyroidism), pregnancy, infections with high fever, chronic illness like leukemia, carcinoma, ulcerative colitis, syphilis, and a number of drugs like antihypertensives, allopurinol and anti-cholesterolaemic drugs. The prognosis is good — complete regrowth of hair is the rule.

Some hair shaft problems in Congenital Hairloss

Hair shaft defects are related to

a) fractures of hair leading to hair fragility and breakage

b) twisting and curling of the hair shafts

c) indentation and grooves

d) irregularities

Some more common and simple examples are:

a) Hair shaft fractures as in trichorrhexis nodosa, trichoschism as in trichothiodystrophy (deficiency of sulphur content in hair)

b) Twisting and curling, e.g. bamboo hair with invagination e.g. Nehterton's syndrome, woolly hair naevus

c) indentation and grooves in the hair shaft, e.g. uncombable hair syndrome

d) irregularities, beading, e.g. Monilethrix

Scarring Alopecia

This is uncommon, but permanent and frustrating. Often a biopsy of the scalp is required to exclude lupus erythematosus, lichen planus, tinea capitis and infections. The above are the inflammatory causes; non-inflammatory causes include developmental, hereditary and neoplastic conditions.

Lupus Erythematosus : This accounts for about 40% of scarring alopecia, often in females, in adults. Systemic features may not be present. The lesions are discoid, scarred, with keratin plugs. A biopsy is confirmatory.

Lichen Planus : This accounts for another 30-40% of scarring alopecia. Features are like lupus erythematosus and a biopsy is mandatory.

Folliculitis : may be scarred and flat, from staph aureus, or bulging due to dissecting cellulitis, often associated with acne conglobate and hidradenitis suppurativa. These are boggy plaques and the sides may exude pus. Surgical intervention gives a permanent cure.

SURGICAL TECHNIQUES OF HAIR TREATMENT

This is mainly for advanced stages of androgenetic alopecia. Older techniques include punch grafting and scalp reduction. Newer techniques give improved cosmetic features and include strip harvesting of hair follicles, use of minigrafts, use of Ultraplus CO_2, hair implants, tissue expansion preceding scalp reduction. Scars of cicatrical alopecia are excised by a combination of techniques.

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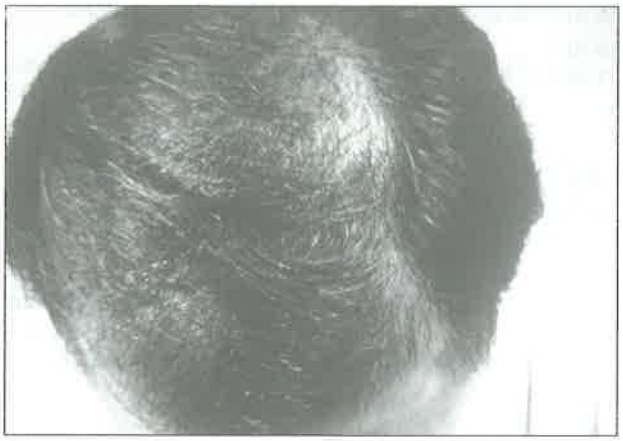
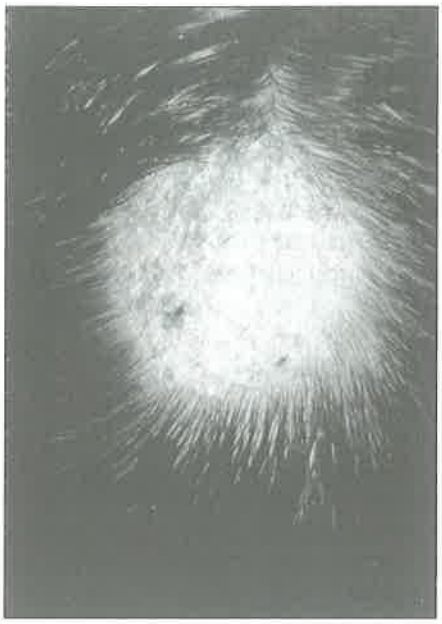
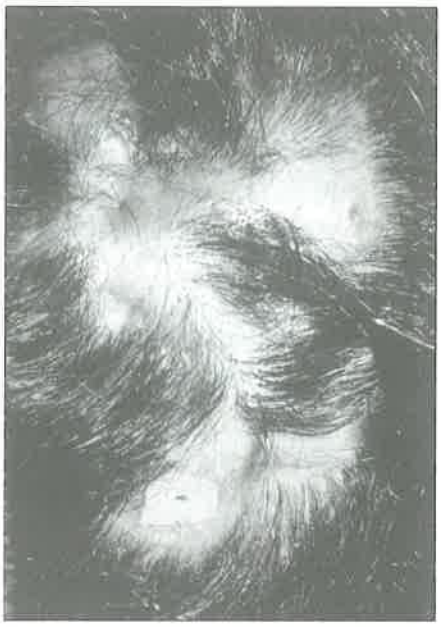


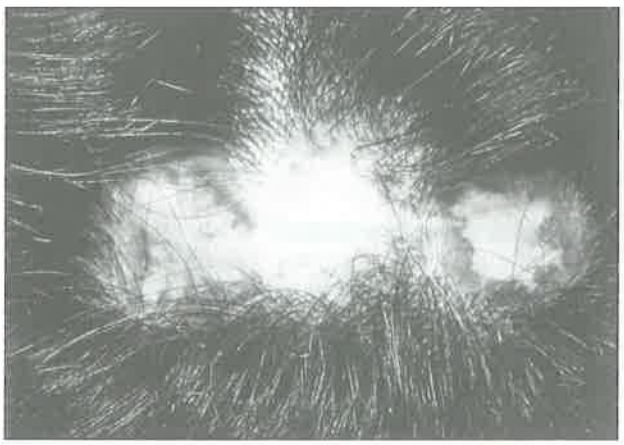
Androgenetic alopecia – male



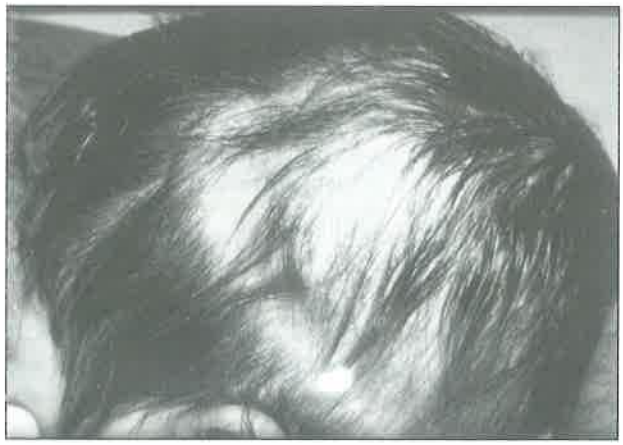
Tinea capitis



Scarring alopecia – folliculitis decalvans



Scarring alopecia – discoid lupus erythematosus



Alopecia areata

ATOPIC DERMATITIS — AN UPDATE

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INTRODUCTION

Atopic dermatitis is a common dermatological diagnosis encountered in family practice. Terms such as atopic eczema, infantile eczema, flexural eczema, disseminated neurodermatitis and *prurigo diathésique* (Besnier) have been used to describe the same condition[1]. The term atopy was coined in 1925 to mean "out of place" or "strange" in the hereditary tendency of patients to develop allergies to food and inhalant substances resulting in eczema, asthma and hay fever. In 1933 Wise and Sulzberger detailed the diagnosis and named the condition atopic dermatitis.

DISTRIBUTION AND INCIDENCE

In 1989-1990, 67% of new cases seen in a tertiary dermatological referral center in Singapore were endogenous eczema which encompassed atopic dermatitis[2]. The prevalence of endogenous eczema has been noted to increase sharply from 31% in 1973 to 67% in 1989/90.. The frequency of atopic dermatitis is also rising in developed countries. In another study[3], 8.9 - 20.4% of patients born after 1970 compared to 1.4 - 3.1% of those born before 1960 had atopic dermatitis at some stages in their lives. No racial group is spared. There is a high prevalence in Asian emigrants to temperate North America. About 90% of cases appear to occur by the age of 2 years. Adult onset occurs rarely and most cases seen in adults are recurrences of childhood disease.

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SYMPTOMS AND SIGNS

1) Itch is the most important symptom in atopic dermatitis. Itch causes scratching and rubbing. The resulting excoriation can lead to secondary infection. Chronic trauma results in lichenification, xerosis and dryness of the skin. This may in turn induce itch and a vicious cycle is established.

2) Rash is another important symptom. The erythema which initially may be localised can become generalised resulting in erythroderma and desquamation. Vesicles may form and rupture, predisposing again to infection. The rash tends to originate at characteristic sites.

3) A linear transverse fold just below the edge of the lower eyelids, known as the Dennie-Morgan fold, has been widely held as an early cutaneous sign of atopic dermatitis signifying the predisposition of the patient to develop the condition.

4) Thinning of the eyebrow on its lateral aspect known as *Hertoghe's sign* is sometimes present in patients with this condition.

5) Infraorbital darkening and facial pallor may be seen in some atopic children and adults.

6) Some patients have whitish scaly patches on the face and arms known as pityriasis alba which is likely to be atopic in origin.

7) Atopic dermatitis often coexists with keratosis pilaris. Large number of keratotic papules can be found on the upper arms, anterior thighs and occasionally on the face.

8) Juvenile plantar dermatitis is a dry scaly form of foot eczema. This almost is always atopic in origin and may be the only manifestation of the disease. It is especially prevalent in older children and adults, particularly males.

9) The rash in dark-skinned atopic patients may be more papular and leaves areas of hyperpigmentation and hypopigmentation.

10) A history of atopic dermatitis is frequently obtained in young adult women with chronic hand eczema. The hand eczema is often caused by primary household irritants.

11) Irritant industrial contact dermatitis is also common in atopic individuals.

12) About 8% of patients develop lens opacities. This is most frequent in women in their thirties. The opacities are unilateral in about half the cases, and posterior cortex involvement is twice as often as the anterior cortex.

13) Keratoconus occurs uncommonly in about 1% of atopic patients.

14) Other illnesses such as asthma and allergic rhinitis may co-exist with atopic dermatitis. Studies have shown that there is one in three chance of an atopic child developing asthma or hay fever. However, atopic dermatitis does not occur exclusively in families with history of asthma or hay fever.

COMPLICATIONS

As a result of excoriation and rupture of vesicles, secondary infection can occur and lead to formation of crusts, pustules and indurations. Infection by coagulase-positive staphylococci can cause boils, carbuncles, folliculitis and impetigo. In addition to secondary bacterial infection, patients are susceptible to herpes simplex infection such as Kaposi's varicelliform eruption, varicella-zoster virus infection, molluscum contagiosum and fungus *Pityrosporum ovale* infection which can lead to severe exacerbation. Scabies may also complicate the picture.

CLINICAL PRESENTATION

The natural history of the illness may be described under the different age groups:

Infancy

In infancy, at between two months to two years of age, a child may develop an itchy erythemathous rash on the cheeks. The rash may develop into minute epidermal vesicles which can rupture and produce moist crusted areas. It may then rapidly extend to other parts of the body like the scalp (cradle cap), neck, forehead, wrists, buttocks, diaper areas and extremities. Sometimes it becomes generalized causing erythroderma and desquamation. In the acute stage when the lesion is moist, exudation may be marked with associated secondary effects of scratching and rubbing. Infection results in crusts, pustules and indurations. In the chronic stage, the indurations take on a characteristic lichenified appearance with dry lesions and xerosis. In older children, this usually appears at the cubital and popliteal fossae. In most instances, the skin symptoms disappear towards the end of the second year. However, in some cases, it progresses into the childhood phase.

Childhood

In the childhood phase, the rashes are usually less acute, less exudative, drier and more papular. The lesions occur at classical locations like the antecubital and popliteal fossae, wrists, eyelids, face and collar regions. Lichenified, slightly scaly or infiltrated patches may intermingle with isolated, excoriated papules over the exposed parts. Pruritis is a constant feature and many cutaneous changes are secondary to it. About 60-70% resolve before adulthood.

Adolescence and adulthood

In the adolescent and adult stage, the lesions may appear as localised erythematous, scaly, papular or vesicular patches. Or they may appear in the form of pruritic, lichenified patches. They usually involve the antecubital and popliteal fossae, the front and sides of the neck, the forehead, and around the eyes. The hands and wrists are frequently involved. Hyperlinearity of the palm is a manifestation of ichthyosis vulgaris which

accompanies 30-40% of cases. At times the eruption is generalized, being severe in the flexures, and consists mostly of lichenification. These lesions are papular, dry, slightly elevated and flat-topped that tend to coalesce to form lichenified, slightly scaly plaques with excoriation. The plaques are erythematous and hyperpigmented. With trauma from scratching, the plaques may become exudative and crusted when infected. The skin is usually dry and has a tendency to become thickened. Widespread involvement results in disseminated neurodermatitis with a leather quality skin with exaggerated markings. Pruritis is again the symptom that causes the skin changes.

DIAGNOSTIC CRITERIA

A summary of diagnostic criteria proposed by Hanifin and Lobitz may be helpful in difficult cases. To make a diagnosis, there must be:

1. Pruritis
2. Typical morphology and distribution with
   a. facial and extensor involvement in infancy
   b. flexural lichenification in adults
3. Tendency towards chronic or chronically relapsing dermatitis

Plus either two or more of the following features:

1. Personal or family history of atopic disease e.g. asthma, allergic rhinitis, atopic dermatitis
2. Immediate skin test reactivity
3. White dermographism or delayed blanch to cholinergic agents
4. Anterior capsular cataracts

Or else, four or more of the following features:

1. Xerosis / icthyosis / hyperlinear palms
2. Pityriasis alba
3. Keratosis pilaris
4. Facial / infraorbital darkening
5. Elevated serum IgE
6. Keratoconus
7. Tendency towards nonspecific dermatitis
8. Tendency towards repeated cutaneous infection

DIFFERENTIAL DIAGNOSES

1. Seborrheic dermatitis
2. Contact dermatitis
3. Nummular dermatitis
4. Psoriasis
5. Keratosis follicularis (Darier's disease)
6. Wiscott-Aldrich syndrome
7. Hyper-IgE syndrome
8. Ataxia-telangiectasia
9. Swiss-type agammaglobulinemia
10. Histiocytosis X (Letter-Siwe or Hand-Schüller-Christian disease)
11. Phenylketonuria

Particular attention should be paid to the following two conditions especially in infancy:

1. Wiscott-Aldrich syndrome – This is a X-linked recessive condition causing thrombocytopenic purpura, severe eczema and predilection to infection in early infancy. The serum IgM is low, IgG is normal, IgA and IgE are elevated. The inability to form antibody to bacterial capsular polysaccharide antigens is the most commonly reported immunological defect. T cell numbers decrease with age. Some patients also manifest a partial T-lymphocyte response. They usually succumb to infection, haemorrhage, leukaemia or lymphoma.

2. Hyper-IgE syndrome – This is an autosomal recessive condition. These young children present with rash resembling atopic dermatitis and recurrent pyogenic infection in the lungs and skin. The serum IgE is markedly elevated (often more than 10000 IU/ml). IgD is also elevated with eosinophilia. There is diminished level of resistance to staphylococcal infection because of impaired chemotaxis of neutrophils and monocytes. There is also abnormal cell-mediated immunity. Infection caused by relatively low virulent organisms such as *Candida albicans* may occur. These children may have coarse facial features.

AETIOLOGY

The exact aetiology of atopic dermatitis has yet to be elucidated. Studies have shown a strong genetic link in its transmission. Autonomic nervous system disturbances and changes in fatty acid metabolism and phosphodiesterase activity have been implicated. But the interactions of immune-response cells appear to be the most likely underlying mechanism in atopic dermatitis[4].

Genetic factor

The tendency to develop atopic dermatitis is probably inherited in a polygenic fashion and strongly modulated by the environment. About 70% of atopic patients have family history of atopic dermatitis. In the first three months of life, more than 25% of children with atopic mothers developed atopic dermatitis. By two years of age, more than 50% of the children will develop allergic symptoms if one parent has allergies. If both parents have allergies, the incidence increases to 79%. Environmental factors such as exposure to allergens are thought to be involved in the phenotypic expression of atopic dermatitis.

Biochemical factors

The degrees of dryness of the skin may be explained by lichenification, increased transepidermal water loss and changes in the lipid composition of the epidermal barrier. This change in lipid composition may lead to increased sensitivity to irritants[5]. A deficiency in 8-6 desaturase activity has been postulated as the origin of the decreased linoleic and linolenic acid metabolites. This leads to dry skin and disturbed cellular immune reactions through the secondary deficiency of PGE_1. The abnormal, although not pathognomonic, skin responses to a variety of pharmacological stimuli, such as acetylcholine-induced delayed blanching and other vascular abnormalities, remain unexplained. These are linked to increased secretion of vasoactive mediators including histamine and leukotrienes. Prutitis and inflammation may result in subsequent immunological reactions.

Immunological factors

Atopic dermatitis is part of the atopic syndrome that includes genetically determined phenotypes such as bronchial asthma, allergic rhinitis, allergic conjuntivitis and gastrointestinal allergy. Atopic syndrome is always associated with abnormal IgE production. Human IgE production is promoted by interleukin-e(IL-4) and suppressed by interferon-gamma(IFN-γ). In atopy, IgE-type antibodies are produced against a variety of environmental antigens, especially air and food allergens. Certain T-cells clones in atopic individuals are considered to have an **aberrant lymphokine-production profile**. These cells are potent inducers of in-vitro IgE production due to their **high IL-4/IFN-γproduction ratio**. They are also potent inducers of eosinophil proliferation because of their **high IL-5 secretion**[6]. When peripheral blood T lymphocytes are cloned at random in an antigen-independent fashion, the resulting clones with high IL-4/IFN-γ ratio are defined as Th2 lymphocytes and those with high IFN-γ/IL-4 ratio as Th1 lymphocytes. In atopy, a preferential expansion of Th2 cells occurs, possibly directed by antigen-presenting cells. However, not all allergens lead to specific IgE production in atopic individuals. It is possible that the forces that drive differentiation into Th2 type cells in these patients could be the result of an **abnormality in antigen-presenting** cells in certain tissues or the intrinsic properties of the allergic proteins itself. The observation that stimulated leukocytes of atopic patients have a **high phosphodiestrerase activity** and **low cyclic adenosine monophosphate concentrations** have been suggested to be related to this supposed antigen-presenting cell role in directing Th0 to Th2 differentiation. The increased skin sensitivity to irritants of atopic patients results in increased production of cytokines by keratinocytes which leads to the increased production of adhesion molecules by endothelial cells and subsequent influx of immunocompetent cells.

Presence of IgE on Langerhans cells was identified in atopic dermatitis in 1986 by Bruijnzeel-Koomen et al[7]. The binding of IgE is the result of the high affinity receptors for IgE on Langerhans cells. Langerhans cells and other antigen-presenting cells in the skin also express low-affinity Fc receptors that efficiently bind allergen-precomplexed IgE. The expression of these Fc receptors for IgE on antigen-presenting cells in the skin amplify the response of in-situ Th1 and

Th2 cells to minute quantities of allergens. As a result of this facilitated antigen-processing, only minute quantities of allergens are needed to be presented to T-cells. The IgE-receptor-allergen complex helps processing and subsequent presentation to T-cells up to a thousand fold!

Consistent abnormalities in systemic cell-mediated immunity have not been found in patients with atopic dermatitis. Patients with atopic dermatitis do not develop diseases that are characteristically associated with systemic immunosuppression. Systemic viral or microbial opportunistic diseases do not occur with higher frequency in these patients. But within the skin of these individuals there is evidence of decreased cell-mediated immunity with increased susceptibility to skin infections with viruses and chronic dermatophyte infections. There is reduced responsiveness to contact allergens such as poison ivy, cutaneous anergy to intracutaneous skin tests with several recall antigens and a decreased sensitisation to dinitrochlorobenzene. These observations may be best explained by a partial defect in the effector responses of the skin immune system and a decrease in epidermal barrier function due to excoriation. Therefore, atopic dermatitis cannot be interpreted as the clinical manifestation of decreased cell-mediated immunity. On the contrary, in atopic eczematous skin, there are vigorous allergen-directed immune responses, partially mediated by Th2 cells as mentioned above. The exact relation between the different cell populations such as keratinocytes, endothelial cells, Th1 and Th2 cells is still unclear.

Therefore, the aetiology of atopic dermatitis may be closely related to the interplay between the number of skin-infiltrating allergen-specific T cells, serum concentration of allergen-specific IgE and the degrees of expression of Fc receptors on antigen-presenting cells in the skin as a consequence of allergen exposure.

PRECIPITATING FACTORS

Seasonal changes have been known to worsen the condition. Exacerbations have also been related to stress and exposure to environmental antigens such as food or animals. The role of diet is controversial but in infancy some foods do play a role in the flare-ups of the disease. Precipitating factors may vary with age of the patients.

In infancy, food allergy plays a significant role in a selected population of young atopics. 56% of severe atopic dermatitis patients in a study[8] exhibited at least one reaction to food challenge. Skin symptoms appeared in 84%, gastrointestinal reactions in 52% and respiratory symptom in 32% of the patients. Most of the patients developed reaction to one (57%) or two (32%) food items only. Foods most commonly tested positive are egg, peanuts, milk, wheat, fish, soy and chicken. Food additives such as preservatives, food colours and biogenic amines have also been considered as provocation factors[9]. The positive challenge is associated with increased plasma histamine. Abstinence from the affected food results in decreased serum IgE and improved skin condition. About 50% will lose their reactivity to food over 1-2 years. Exacerbations are often seen after vaccinations. Colds are often followed by flare-ups. Partial or complete remission in summer and relapse in winter may occur in those aggravated by wool irritation and low humidity.

In childhood, there is a decrease in frequency of sensitisation to egg, wheat and milk. Increased sensitisation to noningested substances such as wool, cat hair, dog hair and pollen are more common. Wool irritation appears as pruritic eczema on the neck, face, hands, wrists, and legs. Feather sensitivity has its onset in the second year but is commoner in adults. Hairs of cats and dogs and lacquer paint on toys may exacerbate the eczema in children. Sensitivity to nickel, neomycin and ragweed oleoresin are more common. However, patch test may be negative because of a diminished ability to acquire delayed hypersensitivity. Excessive humidity and high temperatures, prolonged exposure to cold and windy weather, and emotional factors in older children and adults may aggravate the condition.

In adolescence and adulthood, the itch may occur in crises or paroxysms. It is often nocturnal or triggered by acute emotional upsets and may be absent at intervals. It usually becomes less severe as the patient grows older and is uncommon after middle life. Triggering factors may be rough clothing, wool irritation, foods or tension.

SPECIFIC LABORATORY INVESTIGATIONS

Cord blood IgE is not sensitive enough to be a good screening parameter for atopic dermatitis (A positive family history is regarded as the best screening parameter)[10 11 12].

Blood and tissue eosinophilia may be found.

Antigen-specific IgE bound to mast cells in the skin can be detected in cutaneous challenge tests or in peripheral blood in radioallergosorbence test (RAST). A negative skin prick test is a reliable indicator of nonsensitivity to the allergen. A positive test is a less reliable indicator of sensitivity. The RAST (radioallergosorbent test) has no advantage over the prick test.

MANAGEMENT

GENERAL PRINCIPLES

1. Hospitalisation

Patients with mild and moderate disease can be treated as outpatients. Those with severe or intractable condition should be admitted to hospital. This will facilitate therapy and reduce exposure to precipitating factors such as mites and stress. Intensive nursing, ultraviolet light, baths and supervised administration of ointments and oral sedation will lead to rapid resolution in many cases. It is important to realise that more aggressive treatment during the initial stages of each recurrences which results in rapid improvement in the patient's condition can give the patient confidence, minimise suffering and improve compliance which may modify the prognosis favourably.

2. Corticosteroids

Topical corticosteroids are the single most important agent. Potency, concentration, and the vehicle of topical corticosteroids have to be selected with regard to the localisation and morphology of the lesions. The least potent steroid that is effective should be used. 1% hydrocortisone cream may be quite effective in young children. Sometimes a diluted form of a more potent steroid may be useful. High potency topical steroids may have a place in localised intractable atopic dermatitis. Intralesional injection of triamcinolone acetonide suspension may be helpful for persistant pruritic nodules. Potent or medium strength topical corticosteroid therapy can be used twice daily to induce rapid and extensive remission of acute generalised exudative lesions. This is to reduce patient's symptoms, restore skin barrier function and reduce effect of irritants and allergens. With proper treatment, improvement is usually achieved within 3-7 days. Intensity of treatment can be then tapered off using less potent preparations, increased interval between administrations or overlapping with emollients. The efficacy has to be outweighed against potential side-effects which mostly manifest on the skin. Widespread use for prolonged period of time should be avoided because of the risk of systemic absorption leading to hypercortisolism and adrenal suppression causing delay or reduced growth spurt in adolescents. This should never occur when topical corticosteroids are used correctly. Nothing stronger than 1% hydrocortisone should be used for intertriginous sites or face because of risk of telangiectasia, connective tissue atrophy and striae formation. Systemic corticosteroids may be indicated for severe flare-ups that cannot be controlled by topical therapy alone. These have to be tailed off and withdrawn as soon as possible. Corticosteroid should not be used on infected skin.

3. Anti-histamines

Antihistamines may be useful for their sedative and antipruritic properties but are not the most important pruritogen in atopic dermatitis[13]. Its theraputic value remains to be defined. Older sedating antihistamines may be prefered to newer non-sedating agents in the initial treatment phase because many anti-histamines exert pharmacological activity beyond blockade of the H1-receptor such as interference with mediator release. Hydroxyzine and antidepressants have also been used.

4. Adjuvant Therapy

In severe inflammatory lesions, hydrophilic creams, milks, or wet dressings[14] should be used. Crusts should be removed gently by tepid water bath, supplemented with anti-inflammatory agents e.g. wheat brans, bath oil or wet dressings. Wet dressings should be applied only for 20-30 minutes and not longer than a total of 2 hours a day. They should not be used without underlying cream or ointment to prevent extreme drying out. With the remission of acute inflammation, more lipophilic preparations should be introduced for the dry skin. Emollients may be applied after bath for ichthyosis. Unscented bath oil may be used to moisturise the skin. This is to be carried out regularly to obtain maximum benefit. For hydration of xerotic skin, 10% hydrophilic cream or 1% hydrocortisone in 10% urea cream may be effective. Hydration and alteration of skin lipids can prevent irritant contact dermatitis. The efficacy and acceptibility of the different types of preparations differ from patient to patient. They should be chosen and tried individually. Pastes and occlusive dressings are suitable for chronic lichenified eczema. Too greasy application may cause irritation on the lesions and normal skin. Tar preparations may also be helpful in florid disease. Preparations containing lactate like urea are also beneficial. Natural sunlight and shortwave ultraviolet light may be helpful in some patients. However, home sun lamps and tanning facilities are best avoided.

5. Antimicrobials

Antibiotics may be used for frank infections. Treatment of infection involves removal of crusts, wet dressing on weeping lesions and oral antibiotics e.g. erythromcycin and dicloxacillin. Topical application of fusidic acid cream or ointment can be used for small lesions. In some recalcitrant atopic dermatitis, empirical use of antibiotics for 7 to 10 days may result in clearing of the disease. Allergy to penicillin in atopic dermatitis is also more frequent. Eczema herpeticum can be missed because it may run a mild course but it can become life-threatening. Intravenous acyclovir should be given and wet compresses or lotions applied on the lesions.

6. Patient Education

Patients should be educated as "co-therapists". Provision of information of the clinical picture, natural history and pathophysiology will enable the patient to understand and hopefully cope with the disease more effectively. Therapeutic measures such as application of dressings and ointments should be demonstrated. Information on avoidance of skin irritation is also important. Individual tuition is preferable to sheets of information. Patient's knowledge should be checked and widened at each consultation. Sometimes apparent reluctance to co-operate with treatment may require repeat counselling and psychotherapeutic or psychiatric intervention. Systematic training has been found to improve the effect of therapy[15]. The attitude of the therapist is also important who must be open to discussion and willing to provide adequate support at any time.

7. Prevention

Primary prevention

In infants with high genetic risk of developing atopic dermatitis, preventive measures taken during early infancy may help to delay the occurrence of symptoms[16]. These include:

1. Avoidance of tobacco smoke before and after birth.

2. Avoidance of potent aeroallergens such as pets, house dust mites and moulds.

3. Dietary restrictions such as breast feeding for the first 3 months because breast milk contains hypoallergenic sources of protein as well as protecting immunogenic factors which have been found to have protective effects in children with atopic dermatitis[17]. Infants who cannot be supplied with sufficient breast milk for one reason or another may benefit from a hypoallergenic infant formula. It has also been suggested

that the child should not be given solid food in the first six months and only allowed egg and fish after 12 months.

4. Reduce risk of infection by staying away from creches.

Secondary prevention

Avoidance of irritants

This involves private and occupational aspects of the patient's life. The commonest irritants are water and detergents when patients use them for cleansing purposes. Cleansing should not be done too frequently, for too long, at too high temperatures and with too concentrated solution. Double rinsing may sometimes be necessary to remove excess detergents. Regular use of alkaline soap is contraindicated. They should use bland non-irritating soap or just bath oil. Application of emollients after bath helps to reduce water loss from the skin. Frequent application of bland lubricants both soothes and physically protects the skin. Patients should avoid excessive bathing. Potent contact allergens or aeroallergens should be avoided because of the risk of sensitization. Mineral oils, solvents, dust and sand are other skin irritants which must be cautiously avoided. Environment with constant temperature is favorable to these patients. They should avoid extreme cold and hot, excess humidity and extreme dryness. Profuse perspiration, exposure to heat or ultraviolet irradiation should also be avoided. Humidifiers in rooms are helpful for patients with xerotic skin. Wool and synthetic fabrics whether on clothing or carpets and rough, occlusive and poor fitting clothing should be avoided. Clothing should be absorbent, non-irritating and laundered with bland soap and thoroughly rinsed. Cotton garments and socks are preferable. Limiting the use of coffee, tea, cola beverages, chocolate and other stimulants is desirable. Tomato and citrus fruits may produce a circumoral erythema and these should be avoided in susceptible individuals. Birds, cats, dogs, horses and guinea pigs should be avoided in those who are affected by them. Finally, patients should be aware that emotional stress is an important factor in causing exacerbation. Adequate rest and relaxation like taking a vacation may be helpful for some patients.

PROGNOSIS

Fortunately, most patients can easily by controlled satisfactorily. However, as individual tolerance differs and cannot be predicted, careful assessment of response is necessary. Complete or nearly complete clearing of eczema is usually achieved within 10 days to 2 weeks. Sometimes there is no or inadequate response. The causes of this failure may be:

1. Inadequate therapeutic measures due to inadequate strength of medications e.g. weak corticosteroids, insufficient quantity, incomplete duration and the use of wrong vehicle e.g. greasy emollients for acute eczema.

2. Noncompliance from refusal to co-operate with treatment due to willful patient negligence, following the wrong instructions due to insufficient information, misunderstanding or poor self-esteem.

3. Inability to avoid precipitating or provocative factors like exposure to air-borne allergens or haematogenous systemic allergens from food.

4. Infections e.g. unrecognized or intercurrent infections locally or systematically.

5. Contact dermatitis e.g. persistent contact with exogenous irritants or allergens; allergy to components of the topical preparations e.g. antibiotics such as neomycin, preservatives such as paraben, fragrances and also sometimes allergy to corticosteroid itself.

6. Natural history of illness e.g. persistent lichenified areas of eczema takes longer period to heal and in some children, their disease may take a few months to clear[18].

REASSESSMENT OF PATIENTS

Reassessment and alteration of therapy may be needed if patients still fail to respond. In these patients, individual provocation factors need to be reevaluated. These include:

1. *Food allergy*

The identification of non-tolerated food or food ingredients is made by history, skin tests, determination of specific IgE antibodies and provocation tests[19]. History alone is not sufficient because many patients are not aware of their food hypersensitivity or accuse the wrong type of food. Challenges should be done after admission to hospital because correct judgment is impeded in outpatients and severe reactions need immediate medical attention. If an elimination diet is planned, its effect on the nutritional status of the patient must be considered. Improvement of the condition seen during undirected exclusion diets e.g. avoidance of eggs or cow's milk cannot be attributed to the effectiveness of these diets. This is because a radical change in diet, restriction of nutrition or other ingredients of substituted dietary regimen rather than the avoidance of these suspected food ingredients are the ones that exert the beneficial effects. As most patients react only to one or a few food items, in daily practice routine undirected atopic eczema diets with a greatly restricted spectrum of allowed foods make no sense. Such diets may be associated with life-threatening risks such as nutritional deficiencies, anaphylaxis after unsupervised reintroduction of non-tolerated food in addition to social isolation, particularly in children, and therefore should be avoided.

2. *Aeroallergens*

House dust mite, particularly *Dermatophagoides pteronyssinus*, is a potent aeroallergen in atopic dermatitis. Skin test with mite-allergens can produce eczematous reactions[20] and allergen avoidance may be associated with an impressive and long-lasting remission of the condition[21]. A suggestive history such as deterioration associated with pets, with the pollen season, or exposure to environment that may be rich with house dust mites and test reactions like positive skin tests, specific IgE antibodies in the serum may be helpful. An eczematous skin test reaction to an aeroallergen alone is not diagnostically sufficient because this test is neither standardized nor evaluated with regard to clinical significance. If an aeroallergen alone is suspected, avoidance is the measure of choice. Measures include encasing of mattresses, washing of all bedding at least every 10 days at above 57°C and removal of bedroom carpets. Allergen-specific hyposensitisation (immunotherapy) might be beneficial[22] but is still experimental and not a routine treatment for atopic dermatitis patients. Recently, modified hyposensitisation with *D pteronyssinus*-allergen complexed with autologous specific antibodies improves atopic eczema[23] although this awaits further confirmation.

3. *Microbes*

Staphylococcus aureus is considered to be a provocation factor of atopic eczema in addition to its role in impetiginisation. Skin of atopic patients has high frequency of colonization and *S aureus*-specific IgE antibodies can be found. Antibacterial treatment may be beneficial[24]. Pityrosporum yeasts are also thought to be important in the lesions affecting the head and neck[25]. Although the definite role of these as elicitors of atopic dermatitis remains to be established, appropriate antimicrobial therapy seems reasonable when the clinical findings are suggestive. Occurrence or exacerbation of atopic dermatitis related to HIV infection has also been reported[26].

4. *Psychological factors*

In clinical practice the interaction of soma and psyche in patients with atopic eczema is noted. However, the scientific basis of assuming that psychological factors directly provoke atopic eczema remains weak even though patients whose condition deteriorates in association with psychological and / or social stress are encountered. These patients may benefit from specialized psychological care and / or changes in their social situation.

5. *Other factors*

Hormonal influence has not been established as a cause of atopic dermatitis. Some patients

have skin lesions which are provoked by sun exposure. These should not be confused with photo-dermatitis. Other rare provocation factors such as haematological malignancies should be considered when the disease course is atypical.

NEW TREATMENTS

Other modalities of treatment may be considered when individual provocation factors cannot be identified, eliminated or their elimination does not improve the disease course. Those treatments that have been investigated include:

1. Chinese medicinal herb, e.g. Zemaphyte® which contains components of plants with widespread use in china, has been investigated[27]. Liver toxicity is an important complication which warrants further studies.

2. Ketotifen 1mg two times daily for 3 months given to 56 adult patients in a double-blind study produced statisticaly significant improvement in their condition with no notable drug-related side-effects[28].

3. Cyclosporine. A low dose oral treatment may be useful in more severe form of atopic dermatitis. Clinical manifestations may reappear after discontinuation of treament[29].

4. Topical sodium cromoglycate solution with oral oxatomide produced improvement after 4 weeks of treatment in moderate to severe cases of atopic dermatitis[30].

5. Oral psoralen photochemotherapy (oral PUVA) produced clearance or near clearance of severe atopic dermatitis in 74% of the children after a mean period of 9 weeks. There is a possibility of sustained remission and improved growth[31].

6. Oral treatment with evening primrose rich in linoleic and gamma-linolenic acid produced a small but significant clinical improvement in patients with atopic dermatitis[32]. Higher dosages were shown to be more efficacious than lower dosages and no side-effects were noted[33].

However, alpha interferon 2b was found to be ineffective in short-term treatment of atopic dermatitis[34].

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UPDATE IN MANAGEMENT OF TINEA PEDIS

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Fungal infections are common in warm humid tropical climates. Wearing closed shoes or boots for long hours and during sports and improperly dried feet after showers and swimming provide optimal conditions for fungal growth on the feet.

Over the last five years we have newer and more potent oral and topical agents for antifungal therapy but the main challenge in the management of tinea pedis is in accurate diagnosis.

CLINICAL FEATURES INADEQUATE FOR ACCURATE DIAGNOSIS

Clinical features alone are inadequate as some of the common dermatologic conditions affecting the feet like eczema and psoriasis have similar dry, scaly or vesicular-scaly inflammatory features of tinea pedis. Sometimes the eczema may be complicated by fungal infection especially if potent topical steroids are used. Even the experienced dermatologists have to seek the help of the microscope as it is often difficult to exclude fungal infection on clinical grounds alone.

The duration of the disease and the presence of itch are not helpful in differentiating eczema from tinea. The well-defined edge and failure or worsening of lesions with topical steroids are useful markers.

Tinea pedis is uncommon in children under age 12.

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LABORATROY METHODS ESSENTIAL FOR ACCURATE DIAGNOSIS

For accurate diagnosis clinical features alone are inadequate. Microscopy, supplemented by fungal culture, is necessary to confirm the diagnosis. For the general practitioner this means aquisition of skills in KOH microscopy and proper collection of specimen for examination. Appropriate skin scales can be sent to the laboratory if it is too inconvenient for the patient to present himself at the laboratory.

TYPES OF TINEA PEDIS

There are 3 main types of tinea pedis as based on the clinical features:

(a) The **intertriginous type** is characterised by whitish, macerated, soggy lesions between the toes; most frequently between the 3rd and 4th toe, though all interdigital spaces may be affected.

The differential diagnosis includes a simple intertrigo due to heat, sweat and maceration and bacterial toe-cleft infection. Bacterial infection may be associated with plantar hyperhidrosis and foul odour.

Both candida and dermatophytes may be involved in this form of tinea pedis.

(b) The **squamous-hyperkeratotic** type may present as focal well-defined scaly patches or affecting the entire sole and the sides (Mocassin type). Associated nail infection by the fungus is common.

The differential diagnosis includes eczema (endogenous or contact from shoes), plantar psoriasis, lichen planus and plantar keratodermas.

The dry scaly fore-foot dermatitis affecting the toes of children or so-called Juvenile Plantar Dermatosis is often mistaken by non-dermatologists to be tinea pedis. Juvenile plantar dermatosis can occur by itself or as part of atopic dermatitis, atopic diathesis or part of ichthyosis vulgaris.

(c) The **dyshidrotic type** is characterised by localised vesicles containing a clear or yellowish fluid which may appear on the sole or the sides of the feet. These may be associated with or preceeded by interdigital tinea pedis. The vesicles occur in hot humid environment and can clear spontaneously leaving behind scaly patches.

The differential diagnosis includes Dyshidrotic eczema (often associated with plantar hyperhidrosis) or plantar pustular psoriasis.

MYCOLOGICAL DIAGNOSIS

As discussed earlier, the diagnosis of tinea pedis by purely clinical criteria leads to inaccurate diagnosis. As tinea pedis may warrent expensive and species-specific oral antifungals, laboratory diagnosis is essential. The easy availability of laboratory facilities to all practitioners in Singapore should prompt us for a laboratory confirmation of diagnosis.

Confirmation can be made by:
(1) direct microscopy (KOH microscopy)
(2) fungal culture

Histological diagnosis is not usually necessary in tinea pedis.

THERAPY OF TINEA PEDIS

The principle of treatment of dermatomycoses is to use topical therapy when the infection is localised, focal and not a relapsed infection.

Topical therapy would be considered for the focal interdigital tinea pedis. Terbinafine (Lamisil) cream is most active against dermatophytes. However, if a mixed infection or candidal infection is suspected one of the imidazoles—ketoconazole (Nizoral) or amorolfine (Loceryl) cream would be useful. Ciclopiroxolamine (Batrafen) is effective against dermatophytes and yeast. The older imidazoles (miconazole), though less efficacious, may be used if cost is a major consideration. The duration of treatment is for two to four weeks depending on clinical response. The patient should be reviewed in two weeks' time to assess the clinical response. In addition the patient should be advised to dry the interdigital spaces after shower and before applying the cream.

Systemic oral therapy would be indicated for both the squamous (mocassin) and dishidrotic types of tinea pedis and if concomitant onychomycosis is present. For dermatophytosis the most efficacious is Terbinafine (Lamisil) tablets as the drug is fungicidal. Itraconazole (Sporanox) is efficacious against both dermatophytes and candida. However, except for interdigital infection and immunocompromised patients (like AIDS), candida is not a common cause of tinea pedis. This may be different in the case of onychomycoses.

Ketoconazale (Nizoral) tablets have generally given way to terbanifine and itraconazole due to the small but known risk of hepatotoxicity especially if the drug is given to the elderly and for four weeks or more. Griseofulvin may be used, but with the squamous-hyperkeratotic type commonly caused by Trichophyton rubrum failure with griseofulvin is well known.

Treatment schedule for tinea pedis

(1) Terbinafine 250mg daily 2 to 4 weeks
(up to 3 months if onychomycosis is present)

(2) Intraconazole 100mg daily 2 to 6 weeks.

(3) Itraconazole 200mg daily 2 to 4 weeks.

(4) Griseofulvin (ultramiconised) 500mg to 1gm daily 4 to 12 weeks.

Of the three antifungals, terbinafine gives the best cure rates (of more than 80%) clinically and mycologically after two weeks of treatment. The duration of treatment with itraconazole can be reduced to two weeks, if a higher dose (200mg or 400mg daily) is used. As both drugs (terbinafine and itraconazole) are concentrated and have great affinity and storage in the epidermis and keratin layers, both clinical and mycological improvement continues even after the drug has been stopped. Further clinical trials based on pulse therapy (once weekly) are being carried out.

However, in the therapy of tinea pedis (like other dermatomycoses) local and systemic host factors and the type of causative agent would determine the duration of therapy and the cure rates. Therefore, it is important for the practitioner to follow up the patient, to assess cure (clinical and mycological) and follow up for a period of at least three months if not six months to ensure that there is no relapse.

Fortunately the oral antifungals have minimal adverse effects and clinical monitoring alone would be adequate in the generally healthy young adults. None of the oral antifungals are safe for use in pregnancy.

PREVENTIVE DERMATOLOGY

In addition to therapy and follow-up it is imperative on the physician to advise the patient on prevention. This includes:

(1) Keeping interdigited spaces dry after showers and swimming. In some cases separation of the toes using cotton gauze for a few hours would be necessary.

(2) Use cotton socks. Regular or daily change of socks.

(3) Regular change of shoes. Allow shoes to dry out, especially for sports shoes and when shoes have been wet by rain.

(4) Leather shoes are preferred.

(5) Use of antifungal or antiperspirant powder to feet, between toes and in socks is useful for prevention.

CONCLUSION

With the newer oral and topical antifungals shorter duration of treatment and more effective cure rates are possible for tinea pedis. However, as these agents are expensive, accuracy of diagnosis of tinea pedis is imperative to ensure cost / benefit before starting on therapy.

AN APPROACH TO NAIL PROBLEMS

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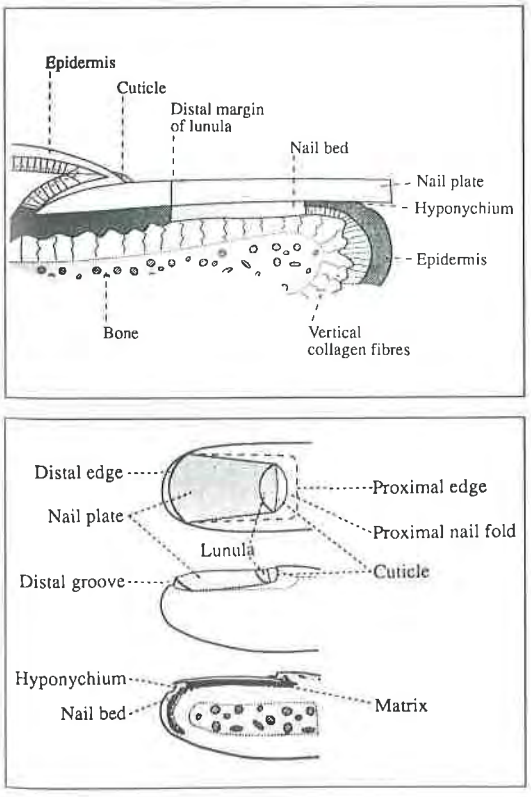
To understand nails from a practical point, the signs of the diseased nail can be easily understood when they are related to the anatomy of the nail. For this reason, learning the anatomy is mandatory, especially if surgery is undertaken.

The nail is to protect, allow dexterity of the hand and is aesthetic. Imagine a famous model with diseased nails or severe onycholysis; it would be truly traumatic for the patient. Can you manage the problem, and in the first place, are you sure you have the right diagnosis?

Fig. 1 Parts of the nail

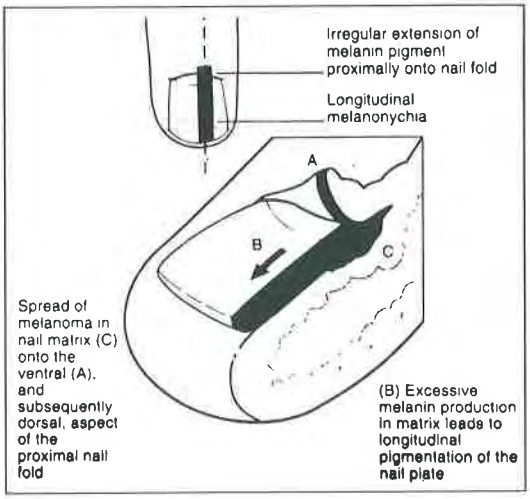


THE PARTS OF THE NAIL (Fig. 1)

1. The **matrix**, which is the growing part of the nail, which produces the nail plate. It is covered by the proximal nail fold and the cuticle. The matrix has two parts; the proximal forms the superficial nail plate, the distal forms the deeper nail plate.

   Thus, a melanocytic naevus will form a dark line along the entire nail, while a melanoma will form an irregular line, and grow onto the proximal nail fold, a sign called the Hutchinson sign (Fig 2).

Fig. 2 Melanoma



2. The **nail plate** consists of superficial and deep layers. In psoriasis, the superficial part of the matrix shows parakeratosis: this immature layer is seen as pits as the scales fall away on growing out as a nail. A superficial white onychomycosis affecting the nail plate does not cause dystrophy of the nail bed, and so appears as a white nail.

3. The **nail bed** is a non-growing part that the nail plate rests on, but is connected by epidermis. Onycholysis due to disease separates them, chemicals only separate the nail transversely and distally.

4. The **hyponychium** attaches the distal nail bed to the skin at the distal end.

5. **Blood supply:** The arterial supply branches into two and supplies the pulp. A separate branch in the centre supplies the nail base as well. Thus, scleroderma of the distal pulp often show normal nails as the blood supply is not compromised.

SOME PHYSICAL SIGNS (Table 1):

1. Changes superficially on the nail surface: due to the proximal matrix or external agents:

   Pitting: These are due to parakeratosis (immature stratum corneum cells) in the proximal matrix. These fall off as the nail grows out, and leave depression or pits. Superficial pits are due to psoriasis, eczema, alopecia areata (hair loss related to autoimmune cause), lichen planus, trauma etc.

Trachyonychia (rough, sandpaper nails): The nail is rough, grey, splits at the edges; trachyonychia is acquired, congenital or idiopathic. Common conditions in which it is seen are 20-nail-dystrophy of childhood, alopecia areata, lichen planus or psoriasis without skin lesions, eczema or trauma. It can be idiopathic; it can also occur with age.

Lines: Longitudinal grooves are normal with age and can be seen in rheumatoid arthritis, circulatory disorders, lichen planus. Some tumours press on the matrix, e.g. wart and myxoid cyst, and cause the groove. Transvere lines like Beau lines mark a sudden inhibition of the matrix, marking the event e.g. febrile illness, Steven Johnson syndrome, or trauma like pushing back the cuticle.

Onychomadesis: Occurs if the inhibition of nail matrix is for one to two weeks; the nail growth is arrested, the nail splits transversely and stops growing.

Onychoschzia: Nail plate splits; occurs with housewives' hands, trauma and chemical injury if seen distally.

TABLE 1

Nail Pathology	Causes
Pitting	Eczema, psoriasis, trauma, alopecia areata, lichen planus
Trachyonychia	20 nail dystrophy, alopecia areata, psoriasis, lichen planus, eczema, trauma, idiopathic
Beau's lines	Sudden illness, psoriasis, Steven Johnson syndrome
Onychomadesis	Trauma, febrile illness, pemphigus, Steven Johnson syndrome
Onychoschzia	Trauma, injury
Onycholysis	Psoriasis, hyperthyroidism, candida, trauma, eczema, chemical
Pachyonychia	Psoriasis, Reiter's, fungus, trauma, eczema, lichen planus
Subungual hyperkeratosis	Psoriasis, lichen planus, fungus, eczema
Acute and chronic paronychia	Eczema, psoriasis, lichen planus, fungus, bacterial infection

2. Nail dystrophy affecting the whole nail:

Onycholysis: This is a separation of the nail plate from the nail bed at the distal or lateral margin. The nail changes from the normal pink colour to yellow green, red or black colour.

Causes: Most common are psoriasis, hyperthyroidism, candida, trauma (shoes, manicure), eczema, chemicals etc.

Onychomadesis: It is nail loss partially or wholly as the nail stops growing and is shed off. Common causes are acute paronychia, trauma, febrile illness, pemphigus, Steven Johnson syndrome, drugs.

Thick nails (Pachyonychia): A normal finger nail is 0.5 mm thick, toenail 1.0 mm thick. The pathology is a thickening of the matrix, or the nail bed (apparent thickening or subungual hyperkeratosis).

Common causes: Psoriasis, Reiter's disease, fungal, trauma, contact eczema, lichen planus.

Subungual hyperkeratosis: apparent thickening under the distal nail plate seen in psoriasis, Reiter's, lichen planus, fungal infection, chronic eczema.

Onychogryphosis: thickening and curling of the nails due to neglect of nails and distortion, due to ill-fitting footwear. It occasionally may be related to vascular abnormalities.

3. Periungual tissue problems

Acute paronychia: shows as localised erythema and periungual swelling due to trauma and aggressive clipping of the nails. There may be pustulation.

Treatment: Cloxacillin and antibiotic cream

Chronic paronychia: affects usually the index and middle fingers of the dominant hand and there is periungual erythema and swelling and loss of cuticle with opening of the proximal nail fold. Fungus and bacteria become lodged here. A secondary nail dystrophy may develop.

Causes: Eczema, psoriasis, lichen planus, fungus.

Treatment: Protection with gloves, emollients, and treat the infection with antimicrobial preparation e.g. miconazole.

NAIL CHANGES IN SOME COMMON DERMATOLOGICAL CONDITIONS:

Psoriasis

Nail changes are common in psoriasis and are seen as pitting, subungual thickening, onycholysis, discoloration, oily spots, splinter haemorrhages and paronychia.

Treatment: generally not helpful till the generalised condition improves as with etretinate, PUVA, methotrexate, topical steroid and Calcipotriol (Vit D) cream.

Eczema

This includes atopic, contact and endogenous eczema. Nail feature include pitting, periungual eczema, transverse ridging, subungual hyperkeratosis and discoloration. The first three fingers of the dominant hand are affected.

In some professions like in hairdressers and mechanics, they often have endogenous and exogenous eczema and a patch test to exclude allergy is necessary. Splits and cracks in the distal edge are due to detergent and water. If the feet are affected, contact allergy is less likely.

Treatment: Protection with gloves is essential, and moderately potent steroid creams.

Fungal infection / Onychomycosis

The fungus can involve the nail bed matrix or nail plate, singly or together. These are manifested as proximal white subungual onychomycosis, superficial white (nail plate) onychomycosis and distal and lateral subungual onychomycosis.

The most common is Trichophyton species (dermatophyte) or candidiasis. Confirmation is by nail clipping culture. The fungal infection may be a secondary event to a primary nail dystrophy e.g. epidermolysis bullosa nail dystrophy. The latter

accounts for the lack of complete cure after antifungal treatment. In 30%, cultures can be negative despite the presence of a fungus.

Treatment: Medical nail avulsion with 40% urea paste dressing detaches the diseased nail in 2 weeks and allows topical antifungal to be used. Systemic treatment is recommended, e.g.:

Terbinafine :	6 weeks for finger nails, 12 weeks for toe nails.
Itraconazole :	3 months for finger nails, 6 months for toe nails.
Griseofulvin:	6 months for finger nails, 1 to 1.5 years for toe nails.

Topical amorolfine (Loceryl) is better with avulsion and is applied weekly for 6 to 9 months.

Tumours

Benign:
Warts: Periungual and subungual warts are best treated by CO_2 laser but there is still a 50% chance of recurrence. Mild residual warts are treated with keratolytics (17-20% salicylic acid).

Malignant:
These include
i) Squamous cell carcinoma (SCC): presenting as "pyogenic granuloma".
ii) Melanomas: presenting as pyogenic granuloma, or black line on the nail.

A biopsy is mandatory. Early SCC can present as eczema, warts, acute or chronic paronychia.

USEFUL TIPS

1. If you see a dystrophic nail:

   i) exclude fungal infection — take nail clippings, put a drop of 30% KOH and look for fungal hyphae.
   If negative, put some nail clippings on a black paper and send for culture.

   ii) It can be psoriasis — check the body and scalp for papulosquamous lesions or arthropathy.

   iii) do not forget dystrophic nails in genetic diseases like epidermolysis bullosa.

2. If you see onycholysis and scaling of finger tips and periungal swelling:

   i) exclude psoriasis

   ii) then candidal infection: do a nail clipping or culture

   iii) or else it is chronic eczema (exogenous cumulative insult due to water, detergent, chemicals, trauma) like housewives' hands.

3. If you see lumps around nails:

   i) exclude warts, mucoid cyst, glomus tumour (blood vessel), exostosis (bone), granulation tissue (in-growing toe nail).

   ii) do not miss squamous cell carcinoma or melanoma. Do a biopsy.

4. If you see pits:

   i) exclude psoriasis

   ii) eczema or alopecia areata.

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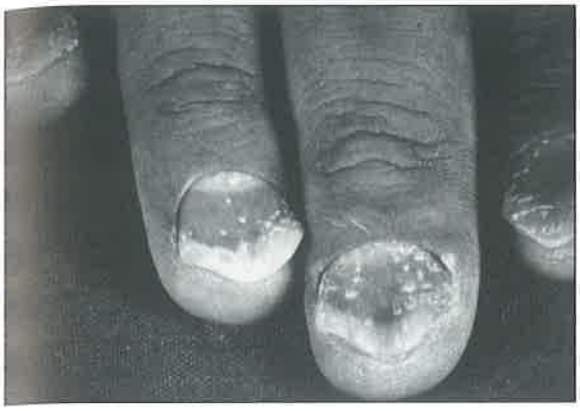
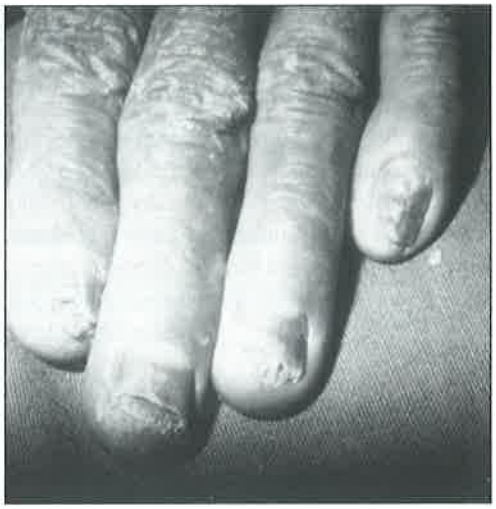
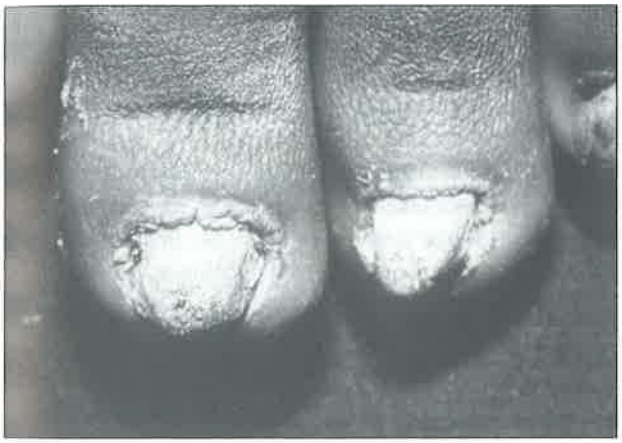


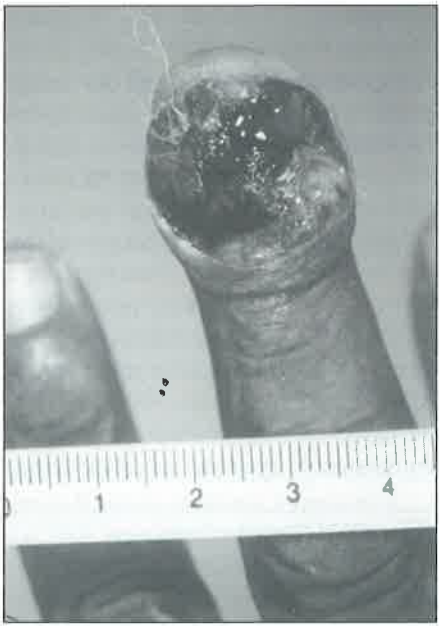
Nail Psoriasis



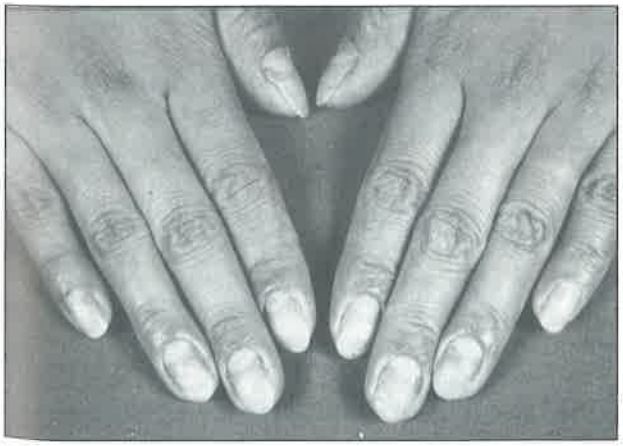
Nail Fungal Infection (dermatophyte)



Nail Candidiasis



Malignant Melanoma (Subungual)



Chronic Paronychia / Eczema

MINIMIZING ADVERSE EFFECTS OF CORTICOSTEROIDS IN SKIN DISEASE

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SUMMARY

Corticosteroid therapy for steroid responsive dermatoses over the past 35 years has considerably reduced patient suffering and chronicity arising from many skin diseases. Despite widespread use of this modality of treatment, significant and severe adverse effects are infrequently seen to-day because of the judicious use of this very useful medicine. This paper describes ways of prescribing the optimum topical, intra-lesional injection, oral and intra-muscular injection corticosteroid to achieve the maximum therapeutic response with the minimum significant adverse effects.

INTRODUCTION

Better understanding of the aetiology and pathogenesis of various skin diseases and the widespread use of corticosteroid therapy for steroid responsive dermatoses over the past 35 years has considerably reduced patient suffering and chronicity arising from many skin diseases. Corticosteroid therapy itself has brought on the problem of adverse effects because these cannot be totally divorced from the desired therapeutic effects. Despite widespread use of this modality of treatment, significant and severe adverse effect are infrequently seen today because of the judicious use of corticosteroids. Nevertheless, there are many physicians and many skin patients who are afraid to use corticosteroids. This fear is often based on only knowledge of the possible side effects, but not on how much corticosteroid is necessary to cause these side effects. As a consequence, patients are often deprived of a helpful treatment for their skin diseases, or are treated with an under-effective corticosteroid regime. There is enough cumulative experience and documented information for physicians to prescribe and patients to be treated with the optimum corticosteroid to achieve the maximum therapeutic response with the minimum adverse effects. This paper is a collection of the recommended methods of prescribing corticosteroids to achieve this aim. For a better understanding of the subject the reader is referred to the original articles referenced at the end of this paper and textbooks of dermatology on the different types of topical, oral and injectable corticosteroids, their potency group, their duration of action, and their side effects.

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PRESCRIBING TOPICAL CORTICOSTEROIDS

Topical corticosteroids come in the following *classes of potency:*

(1) mild (hydrocortisone 1% and equivalent)

(2) medium (betamethasone 17-valerate 0.025% and equivalent)

(3) strong (betamethasone 17-valerate 0.1% and equivalent) and

(4) very strong (clobetasol propionate 0.05% and equivalent).

Others fall in between the 4 classes of potency. Each class of corticosteroid can have *side effects* that vary with

(1) patient age

(2) site of application and

(3) type of skin lesion

Within the same class and concentration ointments are stronger in effect than creams. Prolonged application of strong and very strong corticosteroids, even in small quantity over a small surface area, masks skin infections and leads to striae and atrophy of the skin amongst other effects. Prolonged application of large quantities of strong and very strong corticosteroids over large body surface area causes severe systemic side effects of growth retardation, Cushing's syndrome, immuno-suppression and adrenal cortical insufficiency.

PRESCRIBING MILD CORTICOSTEROIDS (Hydrocortisone 1% and equivalent

Mild corticosteroid is ideally suited for treating eczema and dermatitis in infants, young children, the aged, adult muco-cutaneous junction, genitals, face, armpit, and groin flexures. The steroid is applied once to twice daily. Small quantities are safe if application is not occluded.

PRESCRIBING MODERATE STRENGTH CORTICOSTEROIDS (Betamethasone 17-valerate 0.025% or equivalent)

Indications for this class of corticosteroids include eczema and dermatitis lesions that do not respond to mild corticosteroids, adolescent and adult body and limbs. Application is once to twice daily. When lesions become better go down to corticosteroids one grade lower. Quantities exceeding 240 g per day in adults are known to cause adrenal cortical suppression.

PRESCRIBING STRONG CORTICOSTEROIDS (Betamethasone 17-valerate 0.1% or equivalent)

Indications for use of this category of corticosteroids include lesions that do not respond to moderate corticosteroid, adult hands and feet. Application is once to twice daily. When lesions are better and milder, continue on corticosteroids one grade lower. Quantities exceeding 60 g/day of strong corticosteroid in adults cause adrenal cortical suppression.

PRESCRIBING VERY STRONG CORTICOSTEROIDS (Clobetasol propionate 0.05% or equivalent)

Indications for use of very strong corticosteroids include thick lesions that do not respond to strong corticosteroids, lichen simplex, hypertrophic lichen planus, lichen amyloidosis, plaque type psoriasis as additional treatment to tar, anthranol or calcipotriol. Method of application is:

(1) once daily for 2-3 days a week, and moderate corticosteroids on other days of the week, or

(2) once daily for 1 week, and moderate corticosteroids for 2 weeks. When lesions are better and milder, stop the very strong corticosteroid and continue on the moderate corticosteroid. Quantities in excess of 10g/day of very strong corticosteroids in adults cause measurable adrenal cortical suppression.

COMBINING TOPICAL CORTICOSTEROIDS WITH ANTIBIOTIC AND ANTIFUNGAL AGENTS

Combination therapy is indicated for lesions in anatomical sites where candida, p acnes and pityrosporum flourish, or where infections occur easily, e.g. feet dermatitis, chronic paronychia, flexural dermatitis, seborrheic dermatitis, chronic eczema affecting scalp, face and upper body, excoriated dermatitis. Medication of this type include vioform-hydrocortisone, combination of corticosteroid and imidazole (Daktacort, Pevisone, Triderm, Quadriderm, Celestoderm with

Garamycin and others) and combinations of topical corticosteroid with coal tar solution. Method of use is by application for 1-2 weeks till better or cleared of lesion, then continuing with the corticosteroid component. Allergic contact dermatitis to the antibiotic and antifungal component has to be borne in mind.

PRESCRIBING INTRA-LESIONAL INJECTIONS OF CORTICOSTEROID

Triamcinolone acetonide injection acts and suppresses endogenous steroid production for 3 weeks. Too much and too frequent intra-lesional corticosteroid injections cause

(1) atrophy of skin and striae and

(2) Cushing's syndrome, immuno-suppression and adrenal cortical insufficiency.

Indications for intra-lesional injections include alopecia areata, lichen simplex, prurigo nodularis, and keloids. Recommended method is using 5 mg/ml solution for flat lesions like alopecia areata, and 10 mg/ml solution for thick lesions like lichen simplex, hypertrophic lichen planus and keloids. 0.1 ml of solution is injected into the lesion at 1 cm apart. Quantities exceeding 20 mg triamcinolone acetonide causes HPA axis suppression. Injections more frequent than at three weeks' intervals cause continuous HPA axis suppression and are undesirable. Four to six weeks interval between injections allows time for HPA axis suppression to recover.

PRESCRIBING ORAL CORTICOSTEROIDS

Prolonged high dose oral corticosteroid therapy causes systemic as well as cutaneous side effects. Prolonged high dose oral corticosteroid therapy leads to severe systemic effects of growth retardation, Cushing's disease, immuno-suppression and adrenal cortical insufficiency. One high dose of oral prednisolone (short acting) suppresses HPA axis for 12-36 hours. One high dose of dexamthasone (long acting) suppresses HPA axis for 60 hours. Multiple doses of prednisolone a day cause more prolonged and sustained HPA axis suppression. Alternate day oral prednisolone causes less significant HPA axis suppression and allows time for HPA axis suppression to recover before the next dose. Recommended methods of prescription include the following:

1) One single course of 1-2 weeks of oral corticosteroid

Indications: extensive dermatitis of >20% body surface area, acute severe dermatitis of face, genitals, hands and feet, acute exacerbation of chronic dermatitis. Method: 1/4 to 1/3 mg/kg body weight / day given in one single morning dose. It is not necessary to tail down the dose.

2) One to two weeks per month oral corticosteroid

Indications: chronic extensive dermatitis of more than 20% body surface area. Dose of 1/4 to 1/3 mg/kg body weight / day is given in one single morning dose. It is not necessary to tail down the dose. Stop oral corticosteroid when body surface area of involvement is less than 20%.

3) Long term oral corticosteroid

Indications: bullous pemphigoid, pemphigus vulgaris, collagen vascular disease and chronic cutaneous vasculitis. Method: starting dose of 1/2 to 1 mg/kg body weight / on alternate days, reducing slowly with improvement to lower doses in one single dose on alternate mornings.

4) Combination of Oral Corticosteroid with Another Agent

Oral corticosteroids are combined with other therapeutic agents to prevent and counter side effects arising from long term use of the higher doses of the medicine. These include

(1) *antacids* in those with gastric symptoms

(2) *oral potassium supplement* in those taking high doses

(3) *anti-hypertensives* in those with elevated BP

(4) *anti-diabetic agent* in those with steroid diabetes

(5) *dapsone* in dermatoses that respond

(6) *colchicine* in dermatoses that respond

(7) *immuno-suppressives* (azathiaprine, cyclophosphamide) in dermatoses that benefit from them. Combining immuno-suppressives with corticosteroids makes it possible to use lower doses of corticosteroid.

PRESCRIBING INTRA-MUSCULAR INJECTION (IMI) OF CORTICOSTEROID

One IMI of hydrocortisone lasts only a few hours. Several injections of hydrocortisone a day for many days can cause significant HPA axis suppression. One IMI of methylprednisolone 40-80 mg suppresses HPA axis for 4-8 days. One IMI of betamethasone sodium phosphate 9 mg suppresses HPA axis for 1 week. One IMI of triamcinolone acetonide 40 mg suppresses HPA axis for 3 weeks or longer. Because of their long action and suppressive effect many physicians are not happy with using the longer-acting corticosteroids because once injected into the body the medication cannot be stopped until its effect wears off. There are no chronic skin conditions that need to be treated with IMI corticosteroid. It is much safer to use short-acting oral corticosteroid such as prednisolone, the dose and frequency of which can be easily adjusted according to patients' needs and response.

PRESCRIBING INTRA-VENOUS INJECTION (IVI) OF CORTICOSTEROID

The indications for IVI injections are life threatening conditions like acute anaphylaxis, acute angio-oedema of the mouth and throat, and adrenal cortical insufficiency. Hydrocortisone is injected intra-venously slowly or by IV drip until the patient is better, then by IMI injection subsequently. In anaphylaxis the corticosteroid injections are often given after the more important adrenaline and antihistamine injections.

MONITORING OF PATIENTS ON CORTICOSTEROID THERAPY

Even though the above methods of administering corticosteroids are rarely associated with severe side effects it is always prudent to constantly check all patients who are under corticosteroid treatment. At each visit the patient's skin is checked for scabs, boils, fungal infections, acne, folliculitis, peri-oral dermatitis, rosacea-like dermatitis, signs of atrophy, striae, echymoses and hypertrichosis. Questions are asked and observations made of any systemic side effects of increasing appetite, weight gain, obesity, moon face, increasing blood pressure, gastric discomfort and pain, and intercurrent infections. Once in six months for patients on long term corticosteroid the following tests are carried out: urine analysis, blood counts, X-ray chest. At any time when there are unexplained symptoms and sickness, checks are made of urine, blood count, x-ray chest, serum electrolytes and morning serum cortisol, and attempts made to find infection and other sickness. It is only by awareness of the possible side-effects and regular checking for them that serious side-effects can be detected early and the medication stopped.

CONCLUSION

Much experience on the use of corticosteroids for skin diseases has been accumulated over the past 35 years. This has enabled physicians to prescribe corticosteroids judiciously to achieve the maximum therapeutic effect with the minimum adverse side effects. Corticosteroids have drastically changed the way skin diseases are being treated. With corticosteroid therapy many skin patients have been cured, and many others have had their suffering reduced.

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CARE OF DIABETIC PATIENTS IN TOA PAYOH POLYCLINIC

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SUMMARY

This paper describes the existing state of care and profile of diabetic patients in a Government Polyclinic in Singapore. It is a descriptive, cross-sectional study of 2264 patients, being all diabetic patients on follow-up at the Toa Payoh Polyclinic till 30 September 1993. Information on the provision and utilisation of services were obtained from interviews with clinic staff and computer entries. The profile of patients was drawn from the case notes.

Continuing care for diabetic patients was provided by a team approach, consisting of the doctor, nurse practitioner and dietitian. Services available to the patient in the clinic included laboratory and pharmacy. Clinics ran included follow-up clinics and annual checkup clinics. All follow-up care and available services were subsidised. Diabetic consultations formed 11% of all consultations from January to September 1993. Attendance rate for follow-up consultations was 89%. Ninety-eight percent of patients were non-insulin dependent, and about half had diabetes for less than 10 years. Forty percent had associated hypertension, 10% associated ischaemic heart disease. One third of patients had past record of hospital admissions, mainly for diabetic control and treatment of complications. The majority of patients were on oral hypoglycaemics, with 11% on dietary control alone. Glycaemic control was satisfactory in 66% of patients. Complications noted by clinical criteria included 7% with peripheral vascular disease, 17% with peripheral neuropathy, 11% with retinopathy, and 15% with renal impairment.

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INTRODUCTION

Diabetes mellitus is an important health problem in Singapore. It was the sixth principal cause of death, accounting for 1.6% of total deaths in 1992[1]. In the National Health Survey conducted in 1992, 8.6% of Singaporeans aged between 18 and 69 years were noted to have diabetes, as diagnosed by oral glucose tolerance test[2].

Compared with a prevalence of 4.7% in adults aged 18-69 years in 1984, diagnosed also by oral glucose tolerance test[3], and that of 1.99% in adults aged 15 years and above in 1975 (oral glucose tolerance tests for those screened positive for glycosuria)[4], there may be a real increase in prevalence over the past 20 years, giving allowance for differences in survey methodology.

Diabetes mellitus, especially the non-insulin-dependent type (NIDDM), is a major disease condition seen in Primary Care in Singapore, being the third most common problem at Government Outpatient Service Clinics, after upper respiratory tract infections and hypertension, accounting for 10.5% of total attendances in 1992[5]. It ranks eighth in the list of common conditions seen in General Practice clinics, accounting for 2.1% of all consultations[6]. More patients with chronic problems are seen at Government clinics because of the lower costs.

To date, not much is known about the pattern of care of diabetic patients in Primary Care clinics in Singapore. Fong et al[7] described the management of diabetic patients in General Practice in a series of 499 patients, and Lee el al[8] reported on the pattern of care in a single-doctor Government outpatient clinic.

The present study aims to describe the pattern of care as well as the profile of diabetic patients in one of the new-generation Government Polyclinics offering comprehensive primary health care.

MATERIALS AND METHODS

The study was conducted in Toa Payoh Polyclinic, one of 13 Government polyclinics. It is located in the Toa Payoh Housing Estate, in the central region of Singapore. The patient population is not, however, restricted to those residing in the Housing Estate itself, as there is no fixed list for each clinic. Some of these patients may attend the Polyclinic as well as the neighbourhood general practice clinic, for the same or different health problems.

The authors obtained information on services provided for the care of diabetic patients from interviews conducted with medical and paramedical staff of the clinic.

In addition information was also extracted from computer entries on patient registration, follow-up appointments, and appointments for annual checkup.

For the description on the profile of diabetic patients, all diabetic patients currently on follow-up at the Polyclinic since its opening in February 1988, up till 30th September 1993 were included in the study. There were 2264 patients on the diabetic follow-up register. The case notes of patients on this register were examined by two physician investigators, and information extracted from them included date and mode of diagnosis, records of associated conditions, hospital admissions, medications used and annual checkup findings. Information on annual checkups and results of these checkups were also collected from the case notes.

All diabetic patients on the register were required to undergo annual checkups. Patients who have not had annual checkups were identified by doctors during follow-up clinic sessions and booked for appointments by the nurse practitioner. At the time of booking, the patient would be given appointments to go for fundal photography and chest X-ray, if none was done in the past two years. Patients were also instructed to go for electrocardiogram, urine microscopy for protein and casts, and blood investigations which included glycaemic control, lipid profile and renal function. In addition to the investigations mentioned above, the content of the annual checkup protocol also included records of height, weight, blood pressure, smoking status, clinical examination with particular reference to clinical indicators of complications such as absent peripheral pulses and tendon jerks.

Of the 2264 patients on the register, 833 patients had had at least one annual checkup done at the time of the study. Data from the first annual checkup were used for further analysis of patient profile.

Data collected were entered using DBase IV programme and analysis done using the SAS package for statistical analysis[9].

RESULTS

I. PROVISION OF SERVICES

In the Toa Payoh Polyclinic, diabetic care was provided by a team approach, consisting of the primary care doctors, the nurse practitioners, the dietitian and the medical social worker. Patients were either walk-in patients who were diagnosed to be diabetic from symptoms or routine health checkups, or known diabetics referred from general practitioners, other Government clinics or hospitals for continuing care.

Personnel

Doctor
There were four full-time and one part-time resident doctors providing outpatient care in the clinic. These were experienced doctors, of whom two had the postgraduate degree of Master of Medicine in Family Medicine.

In addition, 3-5 junior doctors were posted to the Polyclinic on six-monthly rotation. There were also two visiting doctors providing part time service at the clinic.

Nurse Practitioner
Several qualified staff nurses at the clinic received additional training in chronic care and functioned as nurse practitioners. They gave health education talks, counselling and dietary advice,. taught patients glucose home-monitoring and self-administration of insulin injections, and monitored patient progress.

Dietitian
The dietitian provided dietary counselling services once a week on Mondays. She saw patients by appointment, and patients who required special dietary needs or monitoring were referred to her.

Medical Social Worker
The medical social worker also saw patients by appointment, once a week on Saturdays. Patients with financial or home problems were referred to her.

Services Provided

Follow-up clinic
All diabetic patients were seen in the General Outpatient Clinics. They were, however, given appointments and had shorter waiting times compared to patients presenting with acute problems. Diabetic patients on follow-up appointments were usually seen at two-monthly intervals by the same resident doctor, though this may not be possible all the time due to the constraints of the service. During these visits, patients' compliance and control were checked, as well as the presence of any additional problems which may or may not be related to patients' diabetes.

Annual checkup clinics
Annual checkup clinics were conducted one afternoon a week. A maximum of 8 patients were booked per session, about 20 minutes per patient. These sessions were conducted by both the doctor and the nurse practitioner. A structured protocol was used for the annual checkup.

Laboratory services
These were provided at a subsidised rate to all patients, with a maximum or $7 at any one time regardless of the number of investigations done. Blood donors were not charged for laboratory services.

Pharmacy
Medications were also subsidised. The cost to the patient was $1 per item per week to a maximum of $4 per week. Charges were halved for senior citizens and waived for blood donors. There is a standard drug list from which doctors could prescribe. Patients using medications outside the standard drug list could apply to the medical director for approval of purchase at subsidised rates on grounds of poverty, or obtain the medication from private pharmacies.

II. UTILISATION OF SERVICES

From the opening of the polyclinic in February 1988 till end September 1993, diabetic patients formed 2% of all patients registered with Toa Payoh Polyclinic (2,264 / 119,191). Diabetic consultations, however, formed an estimated 11% of all consultations in the first 9 months of 1993.

Eighty-nine per cent (2,105) of the patients came for regular follow-up, at four to eight week intervals. Of the 11% (248) who were irregular with follow-up appointments, the majority (88.3%, 219) were lost to follow-up. The rest were followed-up by their own general practitioners or by doctors in the hospital.

The proportion of diabetic patients who had annual checkup done till 30.9.1993 is indicated in Table 1. About 31% of all diabetic patients had undergone at least one annual checkup in the four years since its implementation. A small proportion had more than one annual checkup done.

Table 1. Diabetic patients who had undergone annual checkup from Feb'88 to Sep'93

Number of annual checkups	Number of patients	Percent
0	1431	63.2
1	697	30.8
2	125	5.5
3	10	0.4
4	1	0.0
Total	2264	100.0

Table 2 shows the attendance rate for the annual checkup appointments in the last three years. It can be seen that the defaulter rate was less than 15%, and of those who defaulted annual check-up appointments, the majority had come for the blood tests, but did not turn up for appoinments with the doctor.

Table 2. Defaulters of annual checkup appointments

Year	Number booked	Number attended (%)	Defaulters (%)	
			blood test done, no check-up	blood test not done, no check-up
1993	292	248 (84.9)	30 (10.3)	14 (4.8)
1992	292	258 (88.4)	29 (9.9)	5 (1.7)
1991	230	202 (87.8)	26 (11.3)	2 (0.9)

Eight per cent (186) of all diabetic patients had been referred to the dietition for dietary counselling. Of these, 31% (58) did not turn up for the appointments. In 20% (439) of diabetic patients, records of dietary counselling by the nurse practitioner were found in the case notes.

III. PATIENT CHARACTERISTICS

Demographic Profile

Gender and Ethnic Groups

More than half (55.2%, 1250) of the patients on follow-up at Toa Payoh Polyclinic were females. The majority were Chinese (79.7%, 1805). Malays formed 5.8% (177) and Indian 10.2% (230) of the patient population respectively.

Age Distribution

The bulk of the patients were between 50 and 79 years of age, with about one-third in the 60-69 age group, as shown in Table 3.

Table 3. Age distribution of diabetic patients in Toa Payoh Polyclinic

Age group	Number	Percent
<40 years	108	4.8
40 - 49	289	12.8
50 - 59	539	23.8
60 - 69	753	33.3
70 - 79	460	20.3
80+	115	5.1
Total	2264	100.0

Clinical Profile

Diagnostic category

Ninety eight per cent (2227) of patients were non-insulin-dependent (NIDDM), and only 1.4% (31) were insulin-dependent (IDDM). Six patients (0.6%) had impaired glucose tolerance.

Duration Since Diagnosis

In 80% (1807) of patients the dates of diagnosis of diabetes were recorded in the case notes. Of these, more than half (55%, 1246) were diagnosed less than 10 years ago. Twenty per cent (457) had known diabetes of 10 to <20 years duration, and about 5% (104) had had diabetes for 20 years or more.

Presence of Associated Conditions

Three out of ten patients (30.2%, 683) had associated hypertension alone. Four percent (98) had associated ischaemic heart disease and 0.8% (19) had a past history of cerebrovascular problems. About 10% of diabetic patients had more than one associated condition, with various combinations of hypertension, ischaemic heart disease or cerebrovascular problems.

Tuberculosis was seen in 3.5% (79) of patients and 2% of patients had associated thyroid diseases (49) and gout (45) respectively.

Of the 833 patients who had annual checkups done, one third (33.5%, 357) were found to have

a serum total cholesterol level of >6.5 mmol/L, and almost half (42.7%, 357) had a borderline raised cholesterol level of 5.2 -<=6.5 mmol/L. Triglyceride levels were found to be elevated (>1.7 mmol/L) in 47.5% (396) of the patients. (Table 4)

Table 4. Diabetes with associated conditions

Disease condition	Number (%)
	(N = 2264)
Hypertension	683 (30.2)
IHD	98 (4.3)
CVA	19 (0.8)
HT and IHD	140 (6.2)
HT and CVA	59 (2.6)
IHD and CVA	4 (0.2)
	(N = 833)
Hyperlipidaemia	
TC 5.2 -<= 6.5 mmol/L	357 (42.7)
> 6.5 mmol/L	279 (33.5)
TG >= 1.7 mmol /L	396 (47.5)

Record of Hospital Admissions for Diabetes Related Conditions

More than two-thirds of the patients (71.1%, 1624) had no records of hospital admissions in the case notes. About one-fifth (22.3%, 505) had been admitted once, and the rest twice or more.

Of those admitted, 30% (224) were for diabetic control, 2.9% (22) for hypertension control, and the rest were for complications or problems in conditions associated with diabetes (Tables 5 and 6).

Table. 5 Record of hospital admissions

Number of admissions	Number (%)
0	1624 (71.7)
1	505 (22.3)
2	104 (4.6)
>2	31 (1.4)
Total	2264 (100.0)

Table 6. Admission diagnosis

Admission diagnosis	Number (%)
Poor diabetes control	224 (29.9)
IHD and CCF	171 (22.8)
Skin infections	118 (15.8)
Cerebrovascular problems	72 (9.6)
Lung infections (including PTB)	45 (6.0)
Amputations	30 (4.0)
Poor hypertension control	22 (2.9)
Others	67 (8.9)
Total	749 (100.0)

Diabetic Medications

Table 7 shows the types of diabetic medications the patients were on. The majority of the patients obtained their supply of medication from the Polyclinic Pharmacy. A small proportion of patients (2.2%, 49) were given private prescriptions to buy medications not on the standard drug list from other pharmacies. Eleven percent (259) of all diabetic patients were not on any diabetic medication. Of those who obtained medication from the Polyclinic Pharmacy, 40.9% (925) were on only one drug. The majority were using sulphonylureas. A comparable proportion of patients (41.4%, 937) were on a combination of sulphonylurea and metformin. Patients on insulin alone comprised only 2.6% (59), and a small proportion (1.5%, 35) were on a combination of insulin and oral hypoglycaemics.

Table 7. Diabetic medications

Medication	Number (%)
	(N = 2264)
None	
Dietary control	259 (11.4)
Single medicaiton	
Glibenclamide	451 (19.9)
Tolbutamide	397 (17.5)
Chlorpropamide	17 (0.8)
Metformin	60 (2.7)
Insulin (medium acting)	58 (2.6)
Insulin (short acting)	1 (0.0)
Combination	
Any sulphonylurea and metformin	937 (41.4)
Oral hypoglycaemics and insulin	35 (1.5)
Private Prescription	49 (2.2)

Annual Checkup Findings

These were based on information of the first annual checkup done in 833 patients.

Smoking and Alcohol Intake
Only 9.6% of patients (80) were smokers. Few took alcohol (5.9%, 49).

Body Mass Index
In the 781 patients whose height and weight readings were available, about half (51.5%, 402) had normal BMI of less than 25. Thirty-seven percent (286) were overweight (BMI 25 to -<30), and about 12% (92) were obese.

Blood Pressure
Table 8 shows the blood pressure readings of patients at the time of their first annual checkup. It can be seen that in those with a history of hypertension, the diastolic pressure was well controlled in nearly 88%, and the systolic pressure was below 160 mmHg in 72%. A small proportion of patients had elevated blood pressure readings in the absence of history or record of hypertension.

Table 8. Blood pressure readings of patients on first annual checkup

	(N = 833)			
	Hypertensive		Non-hypertensive	
	No.	%	No.	%
SBP >= 160 mmHg	102	27.7	57	12.3
< 160 mmHg	266	72.3	408	87.7
DBP>= 95 mmHg	45	12.2	14	3.0
< 95 mmHg	323	87.8	451	97.0

Glycaemic Control
Fasting blood glucose was found to be in the acceptable range in only 42.4% (353) of patients. The longer term HbA1C, however, was acceptable in almost 66% (547) of patients (Table 9).

Table 9. Glycaemic control

Parameter	Number (%) (N = 833)
Fasting blood glucose	
< 8 mmol/L	353 (42.4)
>= 8 mmol/L	480 (57.6)
HbA1C	
< 9	547 (65.7)
>= 9	286 (34.3)

Complications
Table 10 shows the prevalence of complications detected both clinically and by investigations done.

Table 10. Diabetic complications

Complication	Number (%)
Macrovascular	
Ischaemic heart disease	
(see 'Associated condition')	
ECG abnormalities	
suggestive of IHD	93 (11.2)
Peripheral artery disease	
Diminished / absent pulses	
Dorsalis pedis R	53 (6.4)
Dorsalis pedis L	61 (7.3)
Post. tibial R	53 (6.4)
Post. tibial L	63 (7.6)
Microvascular	
Peripheral neuropathy	
Diminished / absent reflexes	
Knee R	85 (10.2)
Knee L	85 (10.2)
Ankle R	143 (17.2)
Ankle L	139 (16.7)
Diminished / absent sensations	
Foot R	31 (3.7)
Foot L	31 (3.7)
Retinopathy	(N = 663)
Nil	574 (86.6)
Background retinopathy	44 (6.6)
Maculopathy	12 (1.8)
Proliferative retinopathy	6 (0.9)
Retinal detachment	1 (0.2)
Retinopathy (not specified)	10 (1.5)
Others	16 (2.4)
Renal complications	
Urine examination	(N = 806)
Proteinuria	140 (17.4)
WBC / RBC / Pus cells	62 (7.7)
Serum creatinine	
> 110 umol/L	123 (14.8)

DISCUSSION

Provision and Utilisation of Services

Diabetic consultations formed >10% of all outpatient consultations in Toa Payoh Polyclinic. This reflected the importance of diabetes in the spectrum of diseases seen at the polyclinic, and the workload of the staff with regard to this disease.

In the Polyclinic, the primary health care physician, nurse practitioner and other paramedical staff all worked together as a team in the care of the patients. The time allocated for annual checkup was, however, deemed to be inadequate, as one afternoon per week (with appointments for a maximum of eight patients per afternoon) for a whole year would only cover 400 odd patients, less than a quarter of the present patient load, not to mention any further additions to the register. To improve on this, the number of sessions per week for annual checkup should be increased, provided service constraints can be overcome. Alternatively, it is suggested that one resident doctor be assigned every clinic day to run a separate 'chronic disease clinic' where patients are seen both for follow-up and more detailed examination. The detailed examination according to the annual checkup protocol could be staggered over two or three visits, so that consultation time for each visit would not be too long.

The regular follow-up rate of 89% was good. Given the fact that patients had the freedom to choose where and with whom they wanted to be followed up, and also that some in the population believed in the use of traditional or alternative medicine, it was inevitable that a small proportion of patients would be lost to follow-up, as they did not see the need to inform staff at the polyclinic of their alternative plans. The solution to this problem is not easy, and lies a lot in the education of the public.

Of the patients who defaulted annual checkup appointments, most had actually had blood tests done. The patients could have been mistaken that the series of tests represented the checkup itself, or that the review by the doctor was actually part of their regular follow-up. This could be remedied by making sure the patient understood the necessity to undergo all the various components that made up the 'annual checkup'. Also, if both the follow-up clinic and the annual checkup clinic were incorporated into the 'chronic disease clinic' as outlined above, this problem would also be minimised.

The service of dietitians was in great demand throughout Singapore, particularly in the continuing care of diabetics, where dietary counselling is an integral part of management. In Toa Payoh Polyclinic, the need for dietary counselling advice was in part fulfilled by trained nurse practitioners. The number of patients receiving dietary advice was under-reported, as these were based on the presence of the dietary counselling sheets filled up by the nurse practitioners, and did not take into account the advice given orally after follow-up visits.

Patient Characteristics

When compared with the national racial distribution[10], there were relatively more Indian diabetic patients. This observation has also been noted in previous population surveys[2,3]. This has important cultural bearing, especially in dietary advice and meal planning for individual patients.

About 40 % of patients in this study had associated hypertension. This figure is comparable with that reported by Lee et al on diabetic patients in a single-doctor primary health clinic in Singapore[8], as well as in the population study conducted by Thai et al[11]. The increased prevalence of hypertension in diabetic subjects compared to nondiabetic subjects had been reported in several studies[12,13]. In the continuing care of diabetic patients, this has bearing on the choice of appropriate antihypertensives, as well as ensuring a tight control of blood pressure, to minimise the already increased risk of adverse cardiovascular outcome in these patients. Any proposed 'chronic disease clinic' should encompass both diabetes and hypertension as these two conditions are so closely associated.

Besides hypertension, other preventable cardiovascular risk factors of importance identified in this group of patients included the fifty odd percent of patients who were overweight or obese,

a similar proportion who had raised triglyceride levels, and three-quarters with serum cholesterol levels above the desired level. Even though the presence of these risk factors in diabetics carry the same risk for the development of macrovascular disease as in nondiabetic individuals[14], and that to date there had been no randomised trial that shows any beneficial effect secondary to an improvement of these risk factors in diabetic individuals[14], it would still be prudent to aim for a reduction of the risks, as the prevalence of cardiovascular diseases is reported to be two to four-fold in diabetics compared with non-diabetics.

The detection of complications is an important part of continuing care of diabetic patients. Often the primary care physician has only clinical assessment and basic laboratory investigations to aid him in this task. The use of sophisticated methods such as electrophysiology studies or ultrasound doppler for measurement of ankle blood pressure is mainly for research purposes and not practical for day-to-day practice in a busy primary care clinic. In this study, about 17% of patients were noted to have absent ankle jerks, and 4% had diminished sensations in the lower limbs, as clinical indicators of peripheral neuropathy. This was comparable with that reported by Thai el at[11], but lower than the prevalence of 25.8% reported by Franklin et al[15] in a population based study, or similar results reported by Boulton et al[16] in a study on patients attending hospital clinics. The difference could be because of selection bias, as patients attending hospital clinics tend to have more severe diabetes than those attending outpatient clinics. It could also be due to the difference in clinical criteria used, as for example, Boulton et al included touch and vibration sense in addition to tendon jerks and response to pain. Observer variation should also be taken into account, as the physical examinations were conducted by different doctors. Similarly, about 7% of diabetic patients attending Toa Payoh Polyclinic were noted to have decreased or absent pulses on the feet, as a clinical indicator of peripheral vascular disease. This was again comparable with that reported by Thai et al[11], who used the same clinical criteria.

As a follow-up to detection of abnormalities, the task of the primary care physician is to refer the patients to the appropriate specialised centres for further evaluation and management. This was done for patients with cardiovascular and renal problems as well as those with retinopathy. Records of outcome of such referrals were incomplete, as often no replies were received, for various reasons. This could be improved upon, as it is important for the primary care physician to know about his patients' progress.

CONCLUSION

A study on the care of 2264 diabetic patients in the ambulatory care setting in Singapore showed that diabetic consultations formed about 10% of all consultations; most patients were of the NIDDM type; the regular follow-up rate was 89%; blood HbAlc levels were acceptable in 66% of patients, and 6-17% were noted to have complications.

The system of care is comprehensive and affordable. Increase in manpower number will no doubt further improve the service.

Acknowledgements

The authors would like to thank the Ministry of Health, Government of Singapore, for permission to conduct this study; Professor Lee Hin Peng, Head, Department of Community, Occupational and Family Medicine, and Associate Professor Goh Lee Gan, Head of Family Medicine Division, Department of Community, Occupational and Family Medicine, for support and encouragement. They would also like to acknowledge contributions made by Nursing Officer Tan Miah Huan, Staff Nurse Wong Wai Meng and other staff members of Toa Payoh Polyclinic, as well as Ms Angela Chan of the Department of Community, Occupational and Family Medicine.

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HOW I CHART MY DIABETICS

S U Wong MBBS, MCGP(S), MCGP(M), FRACGP

"... informed decisions rely on instant information."
Teleview advertisement.

In the group setting where I work, diabetics are registered by the thousands and seen by the score. Each resident doctor has hundreds of diabects on his list. Most diabetics are seen two monthly, and for some very stable cases, 4 monthly. It is a practice policy to follow-up every diabetic in the same consultation room, which improves continuity of care.

This paper is about one aspect in the routine care of diabetics: charting.

WHY CHART?

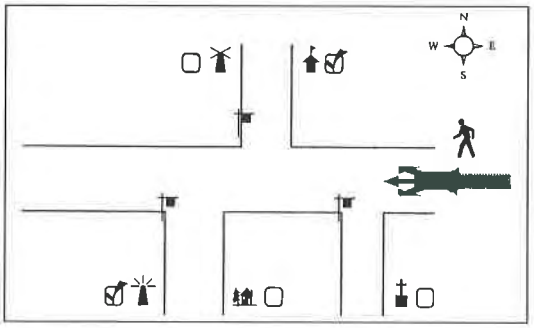
Paperwork can be burdensome during consultantion. Question is: does a diabetic chart bring more benefits than burden?

The number of tasks and variety of activities involved in the care of a diabetic are unequalled by any other common chronic condition. They span over the years, indeed, decades, of the patient's lifetime, and cannot be comfortably accommodated even by the most ingeniously designed routine records.

The purpose of a diabetic chart is to show information in a visual manner, like a map, as illustrated in Figure 1.

Registrar
Family Health Services
Queentown Polyclinic
Singapore 0314

Figure.1 Going Places?



Diabetic care is like a complex journey with many stations to call on. It has to be planned. Progress must be carefully recorded, which provides information to guide decisions and prompt actions. Both oversights and duplications are costly.

THE IDEAL CHART

The ideal chart:

- looks *simple*, and 'intuitive'.
- is *focused* in purpose.
- consists of *only one page*.
- can be *used by all* members of the team.
- is *comprehensive*, but not too clustered.
- allows *quick* entry and retrieval of information.
- is *integrated* with the rest of medical records.
- is *congruous with thinking / action* during each and over several consultations.

That is a tall order, but certainly should be strived for.

WHAT TO CHART?

The chart must cover the tasks and activities in diabetic care. These are:

1. Finding out the other risk factors and associated conditions, e.g. smoking, hyperlipidaemia and hypertension. These are synergistic cardiovascular risks.

2. Detecting complications which threaten sight, limb or life. The eyes and feet are easily accessible to examination and early treatment prevents disabilities.

3. Monitoring blood sugar and weight. They provide feedback essential for achieving control, mainly through diet modification. Other biochemical monitoring includes lipid levels, urine albumin, and serum creatinine.

4. Educating the patient continuously, since diabetic care is essentially self care. The educational needs are many. They vary among individuals and, within the individual over time.

To recapitulate, the chart as shown in the next page, helps to:

1. Assess Other Risks and Conditions.
2. Check fo Complications regularly
3. Monitor Blood Glucose / Weight and Selected Tests
4. Identify and fulfil Educational Needs.

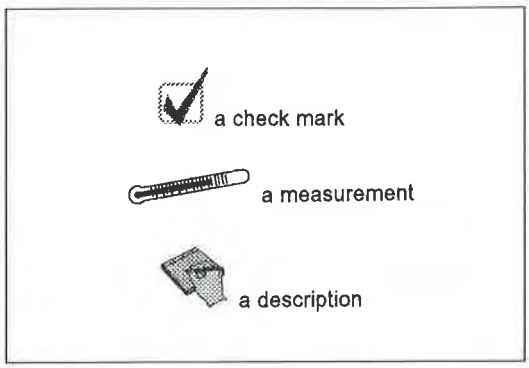
FILLING UP THE CHART

First, it is useful to appreciate that information entered may be one of a few *types*. It could be:

1. a *check*, in the form of a date when an action has been taken,.
2. a *measurement*, often of a test, or
3. a *description* of findings / abnormalities / actions taken.

In other word, it could be logical, numeric, or a memo.

Figure 2. Forms of Entry



Secondly, a few important principles are worth reiterating:

- Information must be recorded at the level of certainty,
- entered in the right place, so that
- it can be retrieved quickly.

Thirdly, a few practical points:

- Check a selection by circling it.
- The ' / ' sign in shaded areas indicate where dates should be entered for an action taken. Enter only the month and year. The precise date offers no advantage.
- There should be no need to use special coloured ink.

Now we are ready to chew on the chart, section by section.

1. General

The top section consists of relatively changeless information.

- *Period* refers to the years during which the chart is in use.
- *History* of *Ketosis* indicates dependence on insulin, in contrast to others who are just insulin-requiring.
- *Associated conditions* include only cardiovascular diagnoses, e.g. hypertension, stroke, ischaemic heart disease. Other conditions should be recorded in the problem list.
- *Desired weight range* is preferred to the more fashionable but abstract BMI. It offers the patient a more concrete goal to work on.
- Circle the self monitoring method. This reminds the doctor to discuss it with the patient.

FHS DM CHART

Name: ______________________

RN : ______________________

Period : __________ - __________

Onset • Year : ________ H/O Ketosis : Y / N

• BSL / OGTT : ______________________

Assoc. Conditions : ______________________

Ht : ______________ cm

Desired wt : ______________ kg

SelfGM : US BS Meter Nil

OTHER RISKS Assesed on / since : (/)

Smoking + -	Overweight ++ + -	↑ Cholesterol ++ + ± -
Alcohol + -	Others:	FH

REGULAR CHECKS

EYE Date	/	/	/	/
VA				
Cotaract				
Fundi/DRP				
FOOT Date	/	/	/	/
Skin				
Pulses				
sensations				

SELECTED TESTS

Date	TC	HDL	LDL	TG	U.Alb	Cr.	ECG	CXR	
/									
/									
/									
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/									
/									
/									

EDUCATION Refer Dietician : (/) NP (/) R

1 Diet/Meal Planning	/		/		/	
2 Glu Monitoring: U/B	/		/		/	
3 Lifestyle/Exercise	/		/		/	
4 Skin/Foot care	/		/		/	

5 What is DM/Guidebk	/		9 Sickdays/Travel	/	
6 Oral Medication	/		10 Complications/Eye	/	
7 Insulin Injection	/		11	/	
8 Hypo/Hyperglycaemia	/		12	/	

B. GLUCOSE

Date	F/P/H	Wt
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2. **Other Risks**
   - Together with the *associated conditions*, this provides a more complete assessment of cardiovascular risk for the patient.
   - It needs not be completed in one consultation.

3. **Regular Checks**
   - Eyes and feet are essential. The convention is to record the right side first, then the left, i.e. 'R' 'L' columns.
   - Describe findings or abnormalities and actions taken.
   - If the patient is already on follow-up with the ophthalmologist, record when this information is elicited, and check it periodically.
   - Diabetic retinal photography makes screening easier. Since the photograph is returned to the patient, early diabetic changes must be recorded as diagrams in the progress notes, so that subsequent photographs can be compared with.

4. **Selected tests**
   - May be done as a panel.
   - Enter measurements in SI units.
   - For ECG / CXR, enter diagnosis. Record details in progress notes.

5. **Education**
   - Referral to Dietician or Nurse Practitioner for more structured sessions is usually done upon diagnosis.
   - Circle 'R' when registered as a diabetic in the practice.
   - The topics may be covered with the patient during structured sessions or at opportune occasions.
   - Enter 'D' / 'N' / 'G', to indicate that the topics has been covered by the doctor, nurse or as a group, respectively. Enter '+L' if leaflet is given.
   - If the patient buys the guidebook, circle it.
   - Additional topics include smoking / weight reduction / low cholesterol diet.
   - Extra coloumns are provided for repeating 4 common topics.
   - The practice may wish to work out what constitutes adequate coverage for each topic; one-page information sheets would be useful.

6. **Blood Glucose and weight**
   - Record Fasting / PostPrandial blood sugar levels and HBAlc, e.g. F 7.8; P 10.2; H 8.5.
   - Use 'R' for the occasional random blood glucose.
   - The normal range for HBA1c is numerically close to that of BSL; this makes it easy to scan the figures.
   - Weight monitoring can be easily correlated with the blood sugar levels.
   - Self-monitored blood sugar levels are too numerous to be accommodated. Record a sample or a range instead.

7. **Others**
   - BP and current medications are recorded in the progress notes.
   - The dietician records her/his notes separately.

8. **Diet history and advice** (see next page)
   - An important supplement, printed on the back of the same sheet as the chart.
   - The main strategy is diet modification rather than prescription. First, know the patient's dietary pattern and lifestyle, then negotiate qualitative and quantitative changes in manageable degrees. A prescription taken wholesale from a textbook will never work.

This chart, hopefully, can adequately replace all forms of charting used so far, including blood sugar levels, other biochemistries, education, diet advice, and annual check-up. Each sheet is estimated to last for about four years.

A QUESTION OF APPROACH

My practice employs an *opportunistic* approach in attending to the various tasks in diabetic care, including detection of complications. The alternative is to set up an afternoon clinic dedicated to 'annual diabetic check-up'. However, it is estimated that it would take a few years to screen every diabetic one round! This 'cohort' approach does not allow flexibility in urgency of action: eye screening is imperative in every patient upon diagnosis (may be already overdue), while foot care in the younger patients can be safely left to the 'next convenient lesson'.

DIET REVIEW

Diet History	(Date: ____ / ____ / ____)	Meal Plan / Changes Advised
Breakfast		
Tea		
Lunch		
Tea		
Dinner		
Bedtime		

Remarks : _____

_____

Diet History	(Date: ____ / ____ / ____)	Meal Plan / Changes Advised
Breakfast		
Tea		
Lunch		
Tea		
Dinner		
Bedtime		

Remarks : _____

_____

Whatever the approach, no diabetic can tolerate a heavy meal of interview, examination, investigations,and education in a single session, especially if 'driven in herds'.

The chart helps the doctor to focus on the outstanding problems and tasks in every visit. A pertinent example is the foot at risk, which needs continuous vigilance and repeated education, if it were to outlast the patient. If detecting complications is reckoned as a sort of annual affair, problems tend to be neglected during 'the rest of the year'.

PUTTING THE CHART INTO ACTION

The chart is considerable paper burden — multiplied by tens of thousands of times — because of the number of diabetics there are, care providers involved, and consultations that occur each year. It must, therefore, be tested and agreed to by all involved before implementation. The practice team must feel its necessity and benefits. It is not there to enforce standard of service.

The chart is not a rigid protocol for mindless compliance of the team. A map in hand does not guarantee that the destination will be reached. The whole team must understand the rationale of all aspects of care. and, each member — the doctors, nurses, and dieticians (but where is the chiropodist?) — must communicate effectively about any aspect of care in a patient. This includes the nurse in the dressing room whose humble contribution is in laboriously dressing the foot ulcers, for weeks and months on ends.

THE REST OF THE RECORDS

Besides the progress notes, there must be a place and a way to record and update information such as the problem list, the social and family information and the 'last normals'. Placed side by side, the chart and the rest of the records complement each other, without too much overlap in content.

BEYOND THE CHART

The doctor (and patient) who values continuing relationship, and the team which is committed to planned care, are more likely to work effectively. Even with the best effort, however, success in biochemical terms cannot always be guaranteed. The reason: there are many personal, social and economic determinants of health that doctors are often unable, sometimes unwilling, to influence.

CONCLUSION

Diabetic care is planned care, with the patient doing most of the work. There is no cure: the doctor and patient must define the end-points of their efforts. A diabetic chart does not in itself ensure good care. It helps, more or less.

THE PRACTICAL MANAGEMENT OF PSORIASIS

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INTRODUCTION

Psoriasis is a common worldwide skin disorder. It is estimated to affect in Northern Europe 3% and in the Far East 0.5% of the population. 80-100 million people worldwide are affected. It affects all races and both sexes and does not discriminate between age or socio-economic status.

AETIOLOGY

In terms of understanding the aetiology of psoriasis, one could use the model of the 3Ps of chronic illness:

(1) Predisposing factors
(2) Provocative factors
(3) Perpetuating factors

Basically as far as pathogenesis is concerned, there is an epidermal / upper dermal immunological-inflammatory component and a component of proliferation and terminal differentiation in the epidermis

Table 1: Aetiological factors in psoriasis

Inherent — genetic familial
Injury: Scratching
Irritation: Overtreatment
Infections
Stress
Drugs : Medical . Nonmedical
Alcohol
Hormones
Climate: Dryness, Sunlight
Obesity

Consultant Dermatologist
Specialists' Centre
277 Orchard Road #08-16
Singapore 0923

In managing patients with psoriasis it is of outmost importance to try and establish the provoking and perpetuating factors.

One of the most common provoking factors is **stress** in areas of work, relationships and the family. It is worthwhile talking to patients and finding out the areas of stress and their lifestyles.

Another common problem which is not only a provocative factor but also a perpetuating factor is **scratching**. There is a certain amount of itch. Patient feel they need to remove the scales. Scratching delays healing and it is one of the reasons why sometimes the treatment does not work. The trauma of scratching can also result in a Koebner's phenomenon.

Trauma can also be seen in terms of work and sports (eg amongst footballers). Trauma is definitely something that can be discussed with the patient, the removal of which can be of help.

Infections like streptococcal pharyngitis (in the young), cystitis (in the elderly) and sinusitis can be provocative factors especially of guttate psoriasis. Appropriate antimicrobial treatment is indicated.

Chemical injury is quite common and sometimes associated with overtreatment. Patients tend to overtreat with preparations or medications given by somebody else and non-qualified traditional medicine practicers.

Drugs: This is a difficult area. Most studies do not give a clear-cut answer. The temporal relationship between the use of drugs and worsening of psoriasis may not be very clear. Betablockers, antimalarials, lithium and NSAIDS have been implicated. It is

worthwhile taking a drug history at time of onset and establishing the relationship leading to worsening of psoriasis. Traditional medicines - eg herbs, ginseng – can sometimes have a provocative effective.

Climate: Dryness is a major problem. Airconditioning, hot showers, strong soaps and winters can provoke dehydration and make psoriasis worse. Excess heat and humidity in the tropics can provoke itch and exacerbation of psoriasis.

Hormonal factors: In the post-partum state, there is often an exacerbation of psoriasis.

Alcohol: Some studies done in Singapore and elsewhere show alcohol to be a perpetuator. If the patient gives a history of such an aggravation it is important to get the patient to abstain. Alcohol overuse could be a reaction to the depression following the psoriasis. Except for special situations, all psoriasis patients should be discouraged from taking alcohol.

If you do find an aetiological factor it is important to remove or diminish it so as to help in clearing and preventing relapses.

TREATMENT VS CURE

Very often the doctor tells the patient that psoriasis cannot be cured. In literal translation to the patient, it means you cannot do anything about the disease. You cannot cure hypertension. There are very few diseases we can cure (eg tuberculosis or gonnorhoea). In other diseases which require long-term management the patient gets very positive messages. In diabetes, you are told to control your blood sugar. But psoriasis patients are told "you cannot be cured; you have to live with it". This is another major psychological impediment in the establishment of a good doctor-patient relationship.

Empirical therapy is very effective and we should not get obsessed by cure because that does not help us to achieve a therapeutic relationship. At no time should one claim a cure.

Table 2: Management of psoriasis - Aims of therapy

• Slowdown overgrowth of epidermal cells
• Control symptoms and signs
• Reduce disease extent
• Prevent recurrence
• Prevent complications

The management plan (Table 3) is developed as:

(1) Clearance Phase
(2) Maintenance Phase

Table 3: Treatment objectives in psoriasis

Clearance phase - to achieve a 100% clearance if possible
Maintenance phase - to prevent and control recurrence

At the initial visit, you should aim to achieve a 100% clearance. You may not always be able to achieve that. Achieving one good clearance for the patient is a major boost for the patient. Be more aggressive in terms of trying to achieve a clearance phase.

If you do achieve a good clearance phase, using UVB, retinoids, methotrexate (MTX) they tend to clear for a longer period of time rather than if you treat just enough and leave a few patches and ask patients to continue treating with an ineffective modality. The maintenance phase basically involves both prevention and coping with aggravting factors and treating early when the first lesions arise.

TREATMENT MODALITIES

There is a range of treatment modalities available for the management of patients with psoriasis (Table 4). The treatment of psoriasis whether in the clearance phase or maintenance phase has to be tailored for each patient's needs and unique features. In most situations, a combination of therapies is used in one individual patient at any one time.

In other words, there is no standard treatment that works for everyone. Treatments that may have not worked before can be effectively utilised at a different time or in combination.

Table 4: Psoriasis therapy

Topical therapy: tar, anthralin, emollients, topical steroids, calcipotriol ointment (Vitamin D)
Phototherapy: ultraviolet B (UVB)
Photochemotherapy: PUVA
Cytotoxics: methotrexate, hydroxyurea
Retinoids: etretinate, etretin
Cyclosporin

The **tar** shampoo is actually meant to be treatment for the scalp and patients should use it at least 15 minutes before a shower. They then take a shower, massage the scalp and wash off the shampoo. As a modality by itself, it may not be very useful for thick plaques but you find as the plaques become less thick and less scaly, it works better.

Anthralin is effective for chronic plaque psoriasis. A major disadvantage is irritancy and staining of skin. You need to educate and motivate patients to use it carefully. With short contact therapy and a few small plaques you can achieve a good effect.

Topical steroids have their limitations. Do not use steroids as a monotherapy. It is useful to reduce the inflammatory component and itch. Combination does not mean mixing together. You can use anthralin and after washing off use topical steroid. Prolonged use of potent topical steroids can destabilise psoriasis and on stopping the topical steroids a rebound exacerbation is often seen.

A recent major advance in topical therapy is **calcipotriol ointment** (Daivonex). The great advantage of this is its relatively low irritant effect. It is also odourless, colourless, does not stain clothes and is well-tolerated. In terms of efficacy, it is at least as efficacious as anthralin. Calcipotriol ointment can be combined with topical steroids (one in the night and one in the day), UVB, methotrexate and PUVA.

Its major disadvantage is that it is not recommended for use in the scalp and head and neck area. Less irritant formulations are expected to be available. It works very well for chronic plaque psoriasis, (palms, soles included). The irritant reaction is minimal which can be overcome by using an appropirate emollient and cleansing preparation. In view of its good efficacy, minimal side effects and convenience of use, calcipotriol ointment (Daivonex) should be considered therapy of first choice in limited chronic plaque psoriasis.

Phototherapy with UVB is extremely useful for guttate and plaque psoriasis. Topicals in combination with UVB is the preferred treatment for chronic plaque moderate disease. It works very well for psoriasis on the trunk and proximal limb. It can also be used in the maintenance phase on a weekly basis.

With **PUVA** we are a little guarded because of the long-term side-effects. It is an extremely effective modality which can be used alone or in combination with retinoids for clearance. The problem is that the patient must have strict compliance with the treatment regimen. Patients need to take medication 2 hours before and they must come on time. The initial outlay in a PUVA machine is expensive.

Methotrexate (MTX) has been used in psoriasis therapy for more than 20 years and is extremely effective. If the necessary basic precautions are taken it is useful when used for short periods (4-6 months) for clearance. You do not always have to commit patients to long-term treatment; monitoring of liver, blood and kidney function is important. In over 90% of patients, one can achieve a remission provided they can tolerate the acute side effects of nausea and gastrointestinal discomfort.

In reference to concerns on reproductive needs, generally we advise either parent to stop MTX at least 3 months before starting a family.

Etretinate is an alternative but is more expensive than MTX. It works very well, especially the first time. It is the treatment of choice for pustular psoriasis and generalised erythrodemic psoriasis but after about 6 months the effect tends to plateau off. You can get a more complete remission by combining with UVB or PUVA.

Cyclosporin: A useful agent for erythroderma psoriasis in achieving a remission. Renal function and blood pressure must be monitored.

CHOICE OF THERAPY

Basically there are several modalities, each with its own advantage and disadvantage. Each will suit a patient better at a particular time. Table 5 shows the factors to be considered in choosing therapy.

Table 5: Factors in the choice of therapy in psoriasis

- Severity of psoriasis: extent, disability, distress
- Clinical type of psoriasis
- Age, sex, reproductive needs
- Site of psoriasis
- Systemic illness
- Tolerance - previous response
- Ability to comply - personality and needs, cost, home support

Severity of psoriasis can be measured by the extent of skin involvement, the disability and the distress caused by psoriasis. The severity will influence the choice of therapy.

Age and sex are related to reproductive needs, liver and renal function are important considerations when using systemic therapy.

Table 6: Psoriasis therapy guidelines

Mild disease - extent less than 10%	
Topicals ± UVB	Outpatient therapy
Moderate disease - extent 10 to 30%	
Calcipotriol ± UVB SCAT ± UVB	Day Care Centre
Anthralin ± UVB Systemic therapy	Inpatient therapy
PUVA, REPUVA Retinoids + UVB	Outpatient
Severe disease - extent more than 30%	
Anthralin + PUVA	Day care / inpatient
PUVA, REPUVA	Outpatient / inpatient
MTX, Retinoids	Outpatient / inpatient

In flexures, face and genitals it is necessary to use milder preparations eg topical steroids or some tar preparations.

Compliance is determined by personality and needs. Some people do not have the time. Then there is the importance of cost of medication. Home support in terms of helping the patient to apply medication, bath or shower can be very important.

You have to monitor the patient to follow his progress. If you have been using topical treatment and the patient has not been making progress, then you have to move to other treatment modalities.

PSORIATIC ARTHROPATHY

Psoriatic arthropathy is common, manifesting as an oligo - asymmetrical pattern and there is a combination of central (spinal) and peripheral arthritis.

Quite a number of patients do have aches and pains apart from arthropathy eg soft tissue inflammation (entesopathy) and tendinitis. It is important to treat and explain to the patient that this is part of psoriasis. MTX is extremely useful for patients with arthritis that is disabling.

PATIENTS' VS DOCTORS' PERSPECTIVE

There is more to psoriasis that what you see on the skin.

Table 7: Psoriasis - Burden of illness

- Medical morbidity
- Physical disfigurement
- Psychosocial stress
- Economic stress

The psychological stress and distress caused to the patient are often not adequately addressed by the medical practitioner.

There is a significant psychological stress induced by a chronic or recurrent condition like psoriasis (Table 8). Psoriasis can lead to depression, low

self esteem, frustration, anger and social withdrawal. All these must be explored and dealt with appropirately. Anxiolytics, antidepressants and hypnotics can be useful adjuncts to psychotherapy and positive psychological support.

There are beliefs, values and misconceptions of psoriasis and its therapy that you need to spend time discussing with your patient.

Table 8: Psoriasis - Psychosocial stress

Depression - low self esteem
Avoid games
Avoid social functions
Worry about offsprings
Avoid public places

Itch in Psoriasis

Itch can be a major problem. Sometimes antihistamines help. Treatment of itch is important as scratching is a major perpetuating factor. Xerosis, strong soaps and overtreatment can contribute to itch. Itch in psoriasis is sometimes a manifestation of anxiety or depression. For patients with psoriasis and atopy with a considerable amount of itch, topical steroids can be useful.

Treatment routines

The burden of applying the treatment, in terms of time and energy needed to comply, can lead to non-compliance. It is important to tell the patient to build the treatment program into their everyday routine (eg brushing teeth every day) and ask them to do it just immediately after their bath (eg 20 minutes or so after bath). If they build it into their routine then it will not appear to be such a burden.

Acceptance

Acceptance of their psoriasis is of utmost importance. Often patients do not accept the disease. You have to tell patient not "Just live with it", you have to tell the patient "You have this problem in your skin. I will help you to give the dermatological expertise but you have to comply with the treatment and together we can actually sort this problem out".

With acceptance of the disease and building the treatment into their routine, the burden of illness becomes much lighter.

It is importance to educate people and not only parents but also relatives, neighbours, employers, teachers, schoolmates and working colleagues. If there is social acceptance, you find that half the burden is actually reduced.

Another important aspect is to recommend a healthy lifestyle to improve general well being.

Medical Quackery

In a chronic disorder like psoriasis, there are all sorts of people making all sorts of claims. Patients are quite desperate looking for a cure and they go all over the place. We have to protect the patient from medical quackery as this is one of the major causes of complications of severe arthritis, disability and also erythrodermic psoriasis.

Partners in Therapy

The other important thing is not to make the patient feel that he is a "guinea pig" to whom you just give some creams and lotions. Explaining why the treatment is being used and how it works brings the patient in as an active partner in therapy.

Educating the patient on the disease with the use of written patient information is extremely important. This will not only allay fears but remove myths thus enabling the patient to be an active informed partner in therapy. Issues in relation to infectivity, heredity and offsprings, risk of cancer and treatment side-effects are common concerns of patients.

Doctors need to educate and emphasize to the patient to accept responsibility for their treatment. Patients must be encouraged to be committed and compliant with their treatment, report any side-effects and openly discuss issues with their doctors. Patients should have the right to choose their doctors, but doctor hopping is detrimental and leads to further frustration.

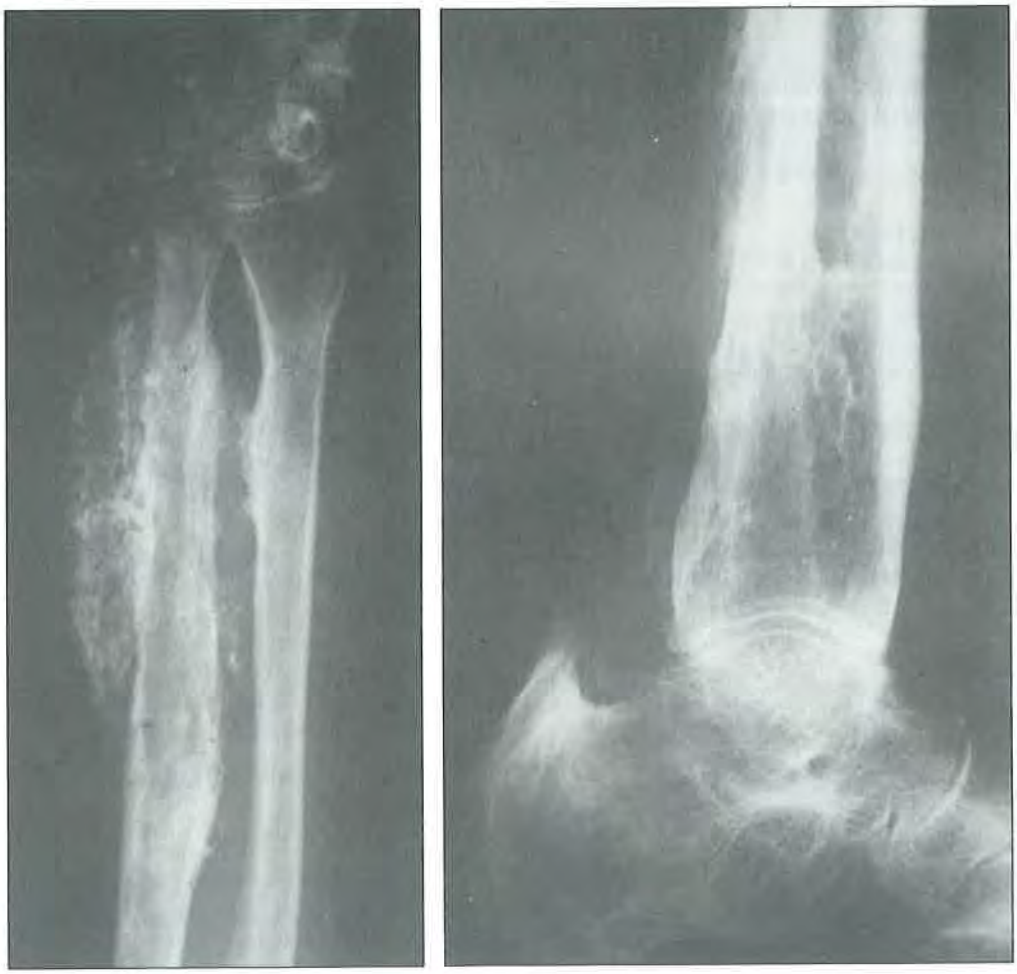
In the management of psoriasis the role of the physician is that of healer, psychotherapist, educator and friend. The enthusiasm of the doctor must never be wanting.

Although there are no "magic bullet cures" in psoriasis, there is a wide range of effective treatment modalities. The success of therapy is crucially linked to a good doctor patient relationship which is a partnership of working, sharing and creating an effective management plan. The partnership should be honest, open and based on mutual trust. A good doctor patient relationship is the centre of the healing process in psoriasis.

X-RAY QUIZ

Submitted by Dr Ng Hweena
MBBS (S'pore), FRCR (UK)

Both radiographs are from different individuals. One had swelling of forearm and the other swelling of leg.



Questions:
1. What are your differentials?
2. Do you think they are of similar disease process?

Radiologic Clinic
#05-09 The Promenade
300 Orchard Road
Singapore 0923

Answers on next page

X-RAY QUIZ ANSWERS

Soft tissue calcifications are noted on both radiographs. Their configurations differ slightly.

Those of the forearm are more conglomerate, curvilinear to rounded with reactive periosteal bone changes. Some have appearances of phleboliths.

The ankle film shows more linear sheet-like ossification with more profuse periosteal reaction.

CAUSES OF SOFT TISSUE CALCIFICATION

Metabolic

Hyperparathyroidism — more commonly seen in secondary hyperparathyroidism. Vascular calcification is common.

Gout — calcified tophi, usually associated with joint changes.

Traumatic

Haematoma

Burns

Myositis Ossificans — usually outer part is more densely calcified than the centre.

Paraplegics at pressure points

Dystrophic — calcification in damaged tissue without generalised metabolic disturbances.

Collagenoses

Skin, subcutaneous and connective bone.

Scleroderma, CREST Syndrome, tumoral calcinoses

Dermatomyositis, Ehlers-Danlos Syndrome.

Neoplastic

Benign — Parosteal lipoma – usually a lucent mass ± pressure erosion of adjacent bone.

— Haemangioma– Phleboliths present in an unusual site ± soft tissue mass with or without adjacent bone destruction.

Malignant — Parosteal osteosarcoma – Lobulated calcification around metaphysis. Inner part is more densely calcified than periphery. It can have features of osteosarcoma, i.e. Codman's triangle, cortical destruction, raised periosteum.

— Juxta-cortical chondrosarcoma - usually affects the pelvis.

— Liposarcoma

DIAGNOSIS

Forearm lesion turned out to be a haemangioma confirmed by venogram. The curvilinear calcifications are typically calcifications along the intima with calcified phleboliths.

However, the changes at the ankle are more due to dystrophic calcification from stasis and chronic inflammation related to varicose veins.

WORLD HEALTH ORGANIZATION PUBLICATIONS

NEW BOOK ANNOUNCEMENT

EVALUATION OF RECENT CHANGES IN THE FINANCING OF HEALTH SERVICES
Report of a WHO Study Group
Technical Report Series, No. 829
1993, v + 74 pages
ISBN 92 4 120829 5

This book provides a critical evaluation of changes, over the past decade, in the methods used in different countries to finance health services. Recent developments in the financing of health services are considered within the context of a global climate characterized by economic recession, heightened consumer expectations, and increasing demands, as chronic and degenerative diseases gain importance worldwide.

Apart from identifying overall trends, the report makes a special effort to determine, on the basis of available empirical evidence, the ways in which specific types of changes will influence the provision and utilization of health services, alter health status, and thus affect a country's health policy objectives. Both the negative and positive consequences of recently introduced methods are critically explored.

Throughout the report, examples of experiences in both developing and developed countries are used to illustrate the complex and far-reaching consequences of changes in the method of financing health services. Information ranges from an alert to administrative problems in the implementation of user-fee schemes, through a discussion of strategies for making health systems more sensitive to consumer preferences, to the simple warning that attempts to limit private practice incomes by law have proved unworkable.

The opening section provides an overview of the immense variety and complexity of changes made in health care financing throughout the world. Trends identified include liberalization, increased use of non-government sources of finance, and greater emphasis upon market mechanisms and incentives as a means of structuring health sector operations. The opening section also develops a framework for evaluating the impact of specific changes on the efficiency, equity, and viability of health services and on the health status of various population groups.

Using this evaluation framework, the second section analyses the impact of changes in each of the four principal sources of finance for the health sector: government, private sources, health insurance, and external funding. The increasing use of user fees for government services is identified as one of the most significant changes during the past decade.

The third section, devoted to payment mechanisms, describes the menu of options that can be used to pay hospitals, health centres, and providers. In considering the impact of different payment mechanisms, the report alerts readers to the complex set of behavioural incentives for providers, which influences their relationship with payers and affects both the type and price of services. A section devoted to organizational changes considers the changing roles of consumers, purchasers, providers, and governments. Particular attention is given to the consequences of the tendency, seen in many countries, to reduce the role of government as a funder of services.

The final section establishes research priorities and draws a number of conclusions concerning the contribution of different methods of financing to the functioning of health systems based on primary health care. While noting the range of policy instruments available to improve the financing and organization of health systems, the report also warns that each is likely to have some bad as well as some good consequences.

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