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Dermatology

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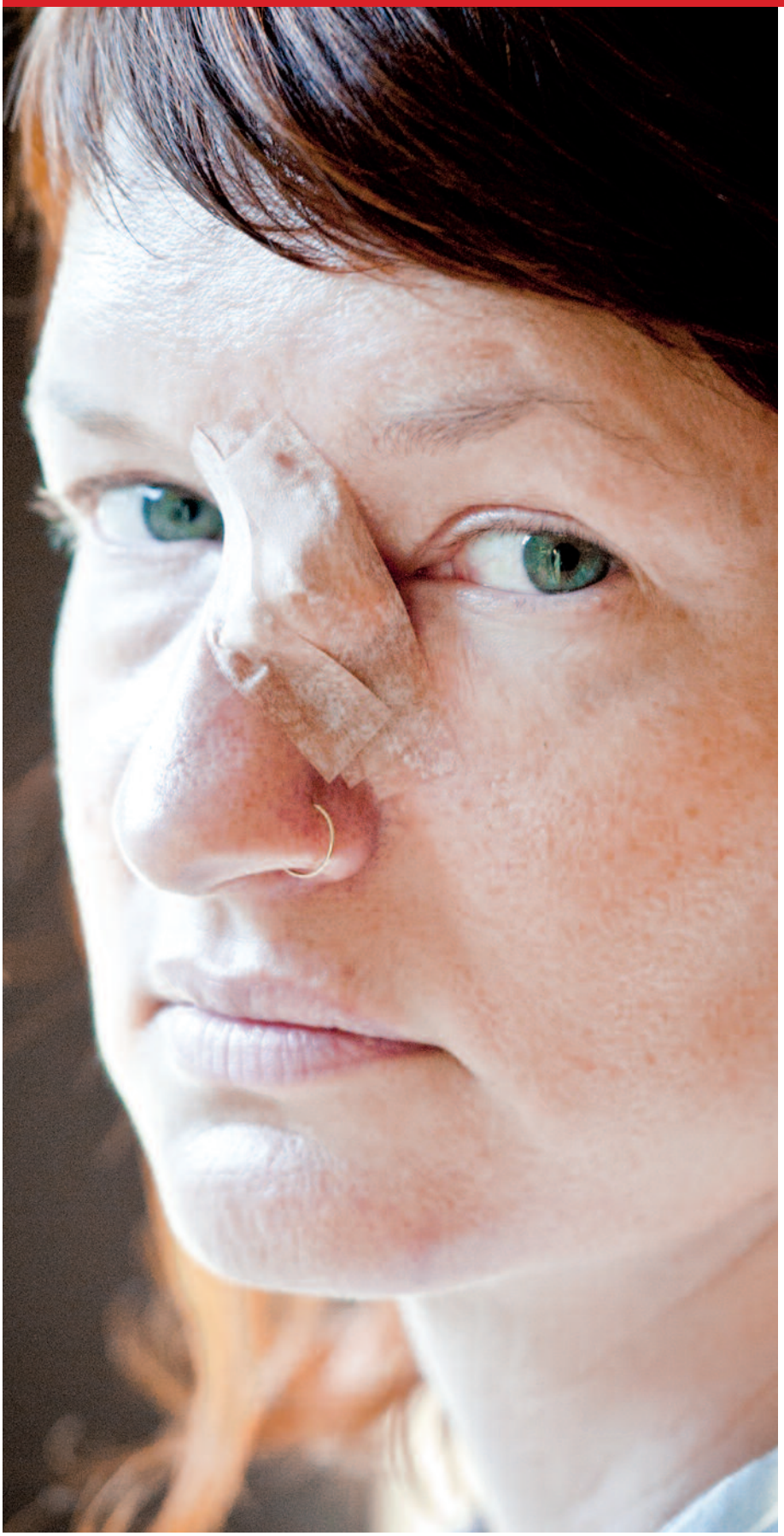

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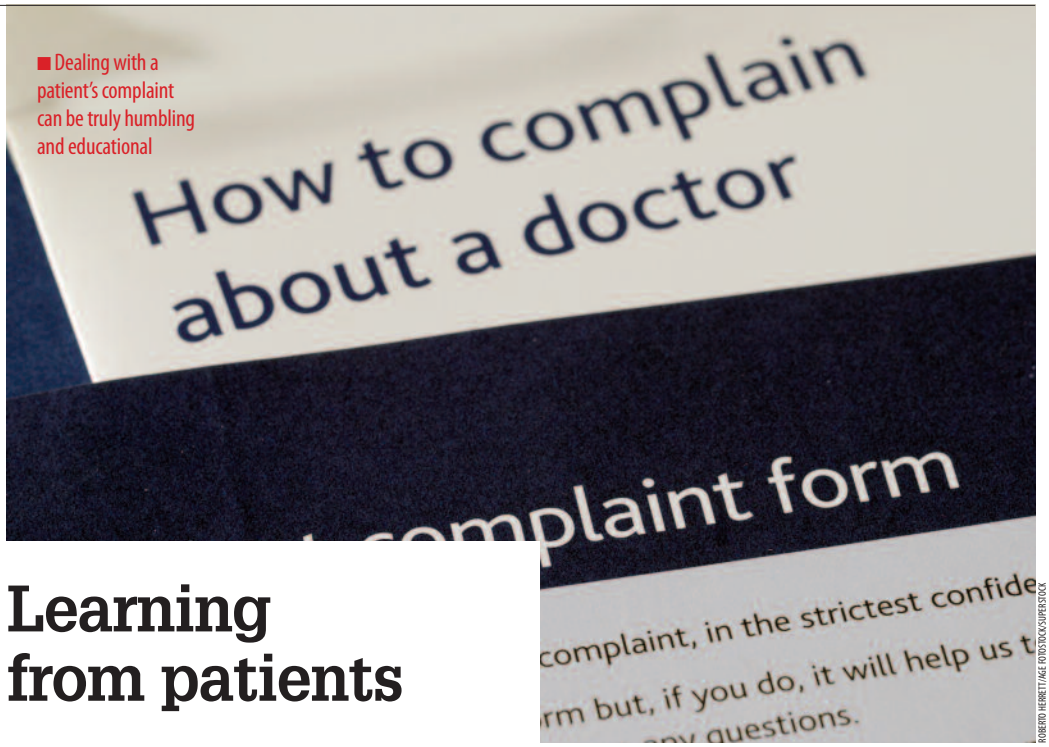

ROBERTO HERRETT/AGE FOTOSTOCK/SUPERSTOCK

■ Dealing with a patient's complaint can be truly humbling and educational

Learning from patients

Complaints from patients have been dominating my life for the past few weeks. Following the Keogh Review we have, as a trust, been reviewing and changing our complaints processes. As part of that exercise, I have 'signed off' many of the complaints about medical care. I have also spoken to, and met, many of the complainants. What a thoroughly humbling and educational time it has been! Reading Nicholas Collier, Faisal Ali and John Lear's article on HIV, I recalled the last complaint made about me personally. I had failed to test a man with severe idiopathic pruritus for HIV. While I could be 'defensive' and point out that he had been very selective in the history he had given, it is also true that I should have at least thought of testing him. My index of suspicion had been too low. Patients who itch without an obvious cause are always something of a challenge to me – so that patient's complaint and the book reviewed on page eight of this issue have both helped me.

The rapidly changing world of therapeutics for non-melanoma skin cancer is an exciting one. Stephen Keohane and Sweta Rai introduce some of these agents in the first of a two-part article. Medical dermatology's fight against surgical dermatology continues! It is quite fascinating to see these evolving therapies coming into daily practice in such a short time.

Andrew Affleck's and Zoe Chouliara's article about psychological adjustment to disfiguring skin disease is quite different. I find it difficult to judge how much patients are affected by their problem. From time to time I just get it wrong! The routine use of questionnaires may stimulate further questioning, possibly leading to further insights, more informed management strategies and improved patient outcomes. In my current practice I only use them routinely in the biologics clinic, but I should adopt them more generally. It might even reduce complaints!

I sometimes spend several days in London teaching management skills to dermatology specialist registrars (SpRs). It's refreshing to find that – although I spend a significant portion of my time wrestling with the competing pressures of advancing medical technology, increasing numbers of older people, rising patient expectations and restrictions on funding – our trainees appear blissfully unaware of these issues. While their time to confront these issues will come, it's good to see that they have been protected during most of their training. After all, this is their time to learn about the diseases, their management and most importantly how to help patients in the years to come. Barry Monk expresses his frustration with the world of medical management, and I am sure many readers share his view, but the SpRs I spoke to this week have yet to encounter the realities of that world. They will soon become familiar with the tension involved in balancing the needs of the individual patient with the ability of the health community to fund and deliver care, but at least for the present it appears to be a relatively unknown world to them.

Neill Hepburn, Editor

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Therapy update on HIV and skin disease

Skin disease is highly prevalent in patients with HIV and causes significant morbidity through severe symptoms, as well as stigmatising, disfiguring lesions. It is associated with mortality, either as a manifestation of generalised conditions or, less commonly, due to a primary cutaneous disorder. Early treatment of HIV infection provides near normal life expectancy; however, currently an estimated 24% of HIV-infected people are undiagnosed in the UK, with 47% presenting as a late diagnosis (CD4+ cell count <350 mm^3). The integumentary system provides a window to immune function, enabling detection of derangement, therefore facilitating earlier diagnosis. Although some HIV-associated skin diseases may present pathognomonically, many do not. Thus, clinicians must adopt a low threshold of suspicion of HIV, particularly in conditions that are atypical, severe, occur in combination or are recalcitrant to treatment. Antiretroviral treatment has improved life expectancy and reduced opportunistic infections while newer medications have improved side effect profiles; however, adverse drug reactions are common and awareness of drug interactions is vital. This review aims to provide a brief overview of HIV-associated skin disease with a focus on cutaneous infections, inflammatory diseases and adverse drug reactions.

Epidemiology

HIV is estimated to affect 34 million people worldwide, with 95% of infections occurring in low- and middle-income countries. In 2011 in the UK, an estimated 96,000 people were living with HIV, a prevalence of 0.15%; higher-risk groups included men who have sex with men (MSM) (4.7%) and the black African community (3.7%). Overall incidence in the UK is declining, but continues to rise in the MSM community.

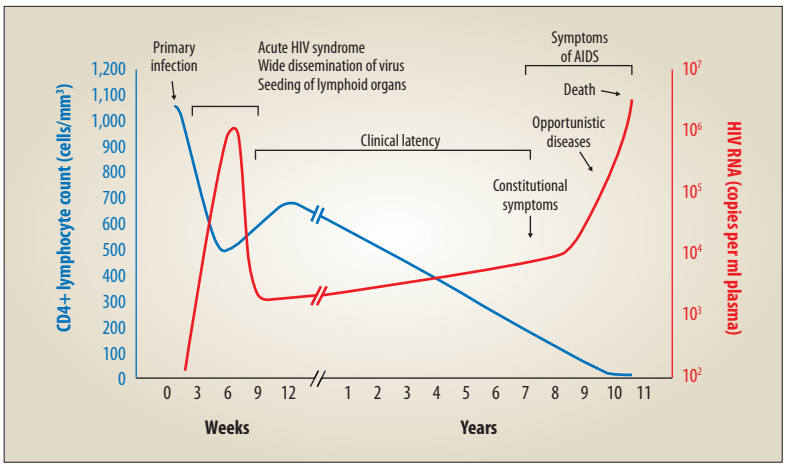

Figure 1. Change in HIV viraemia and CD4+ lymphocyte count in an untreated patient

Pathogenesis

Following HIV infection, there is marked viraemia and rapid decline in CD4+ cell counts (see Figure 1), particularly in the gut, as well as a robust immune response comprising innate, cell-mediated and humoral responses. Seroconversion illness occurs in about half of patients and is associated with more rapid progression. Symptoms occur one to six weeks after exposure and patients may present with a mononucleosis-like illness, exhibiting an exanthematous rash. HIV is a lentivirus; patients often exhibit a clinical latency period of six to eight years before developing AIDS. However, the spectrum ranges from 'elite controllers,' who do not progress in 12 years, to 'rapid progressors', who exhibit a clinical latency of less than four years.

HIV and skin disease

Skin disease is highly prevalent in HIV-positive patients; 90% develop a dermatological disease, which is often inversely proportional to CD4+ cell count in frequency and severity.[1] HIV-associated skin diseases can be categorised into primary non-infectious diseases and secondary infections.[2] Treatment may need to be longer or more aggressive than in the immunocompetent. Often, conditions improve with antiretroviral treatment, but may worsen on initiation. Current medications should be scrutinised when initiating systemic treatments to avoid adverse drug reactions.

Primary skin diseases

Much is unknown about the pathogenesis of HIV and of many skin disorders, but their association provides further insight into both. Chronic HIV infection is associated with sustained immune activation and CD4+ T-cell depletion, creating a vicious circle and leading to increased immunosuppression. Mechanisms for immune activation include: increased response to gut pathogens; viral reactivation; direct response to HIV; and disturbance of T-helper cell subsets.

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1. Gallagher J. *et al.* Poster presented at EADV Congress 2009.
2. Dermol Range – Total Unit Sales since launch. Dermal Laboratories Ltd. Data on file.

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Seborrhoeic dermatitis

The prevalence of seborrhoeic dermatitis is much higher in seropositive patients, particularly in those with CD4+ cell counts less than 200/mm^3, who have a 20-fold increased risk. Manifestations can be more florid, with widespread involvement of the trunk. In these cases, if HIV status is not known, testing is highly recommended.

Xerosis

Xerosis occurs in approximately 20% of HIV patients, frequently causing pruritus and worsening with disease progression.[2,3] It has been associated with changes in microcirculation and mast cell populations.[2,4,5] Pruritus in HIV is a widespread problem that has multiple causes. Idiopathic pruritus is common and frequently leads to complications, such as prurigo nodularis.

Atopic dermatitis

Atopic dermatitis is associated with impaired epidermal barrier function and filaggrin mutation. It is more prevalent in HIV patients and is seen in about 30–50%. It is frequent in atopic individuals and has been linked with eosinophilia secondary to a T-helper cell imbalance. HIV patients develop a Th2 immune-reactivity pattern, which may promote dermatitis.[6] Isolates of *Staphylococcus aureus* have often been found on HIV-positive patients and may stimulate Langerhans cells and cytokine release.

HIV-associated psoriasis is often more severe and recalcitrant

Psoriasis

The prevalence of psoriasis in HIV patients is roughly equal to that in the immunocompetent, but the incidence of psoriatic arthritis is increased. Plaque psoriasis remains the most common form; however, in HIV patients, there is an increased prevalence of guttate, flexural and erythrodermic forms. HIV-associated psoriasis is often more severe and recalcitrant to treatments. Pathogenesis is both unclear and paradoxical as HIV infection and psoriasis cause differing T-helper cell imbalances. Psoriasis often improves with antiretroviral therapy. Improvement with systemic immunosuppressive therapies has been documented but their use needs careful balancing against potential risks.[7]

Eosinophilic folliculitis

Eosinophilic folliculitis is almost pathognomonic of HIV infections and is characterised by scattered pruritic papular lesions located above the nipple line. The disease typically presents with CD4+ cell counts below 300/mm^3.[8] It is possible that the *Demodex* mite is implicated. Topical steroids are the mainstay of treatment. Topical tacrolimus and phototherapy have also shown efficacy.[9]

Secondary skin diseases

Bacterial infections

Staphylococcus aureus

S aureus is the most frequent bacterial skin infection in HIV patients, in whom colonisation and methicillin-resistant *S aureus* strains are prevalent.[10,11] It may occur as a primary or secondary infection causing folliculitis, impetigo, cellulitis, abscess formation and, rarely, botryomycosis.

Bacillary angiomatosis

Bacillary angiomatosis presents as smooth, violaceous, vascular cutaneous papules which may mimic Kaposi's sarcoma; however, lesions are often painful. It is caused by *Bartonella* species, notably *B henselae* (from cats), and *B quintana* (via human lice), and it is strongly associated with immunosuppression, in particular HIV positivity where the CD4+ cell count is less than 100/mm^3.

Viral infections

Viral infections are ubiquitous. In the immunocompetent, they are often asymptomatic or transitory. In HIV immunosuppressed patients, disease is often atypical, severe, prolonged and can disseminate, requiring aggressive treatment.[12]

Herpes simplex virus

Herpes simplex virus (HSV) and HIV are strongly linked; chronic herpetic ulceration for more than one month is an AIDS-defining illness. Genital ulcers can increase the risk of HIV transmission and trials have demonstrated reduced transmission with topical antiretroviral microbiocides.[13–15] HSV-2 has been shown to increase HIV susceptibility by affecting the function of Langerhans cells.[16] Ulcers can measure up to 20 cm, or present atypically, resembling condyloma acuminatum.[17] Trials of the effect of daily aciclovir in patients co-infected with HIV and HSV-2 have shown modest reductions in HIV progression.[18] Aciclovir and related antivirals are the mainstays of therapy. HIV patients have an increased prevalence of viral mutations which disable thymidine kinase and so may require alternative treatments, such as foscarnet.

Varicella zoster

Varicella zoster reactivation is higher in those with HIV co-infection, with an estimated 15-fold increase in risk. Recurrent and multi-dermal presentations are indications for HIV testing. Reactivation may occur at the relatively high CD4+ cell count of 400/mm^3 and is common with immuno-suppression. Patients should be exam-

ined for Hutchinson's sign, which precedes ocular involvement. Reactivation is not uncommon as a reconstitution syndrome following anti-retroviral treatment.[19,20]

Epstein–Barr virus

Epstein–Barr virus (EBV) can cause oral hairy leucoplakia in the presence of HIV immunosuppression, particularly at lower CD4+ cell counts (<200/mm^3).[21] It presents as corrugated painless plaques on the lateral tongue which cannot be scraped away. EBV also has an aetiological role in the development of Hodgkin's lymphoma, which is markedly more prevalent in HIV co-infection.

Kaposi's sarcoma

Kaposi's sarcoma is an AIDS-defining malignancy caused by the human herpesvirus-8. It has an estimated relative risk of 23 in those with HIV. Its incidence has fallen since the introduction of highly active antiretroviral therapy (HAART) but it still represents a significant burden of HIV-associated malignancy. Staging is based on the extent of tumour, immune status and systemic involvement. Lesions are often clinically apparent as violacious, mucocutaneous lesions. Diagnosis should be confirmed histologically. Treatments of mucocutaneous lesions include HAART, topical retinoids and intralesional vinblastine.

Fungal infections

Fungal infections, particularly candidiasis and dermatophytosis, occur frequently in HIV patients and there is increased severity, additional complications and increased resistance to treatment. Furthermore, fungal skin infections may provide a sentinel clue of systemic disease, which may prove fatal. Cryptococcosis is the most frequently occurring systemic fungal infection in these patients; 10% demonstrate cutaneous features.

Scabies

Scabies occurs more frequently in HIV patients and may present as highly infested Norwegian scabies, requiring systemic treatment.

Non-melanoma skin cancer

Non-melanoma skin cancer is common in immunosuppressed HIV patients, with high recurrence rates.[22] Prevalence may increase due to the extended life expectancy given by anti-retroviral treatments.

Cutaneous adverse drug reactions

Cutaneous adverse drug reactions (CADRs) have an increased incidence in seropositive patients who have aberrant immune function and take multiple medications. CADRs are more common at decreased CD4+ cell counts until immunosuppression is profound, when an immune reaction is not able to be mounted.[23] Implicated drugs include antiretroviral and antituberculous medications as well as other antibiotics (including trimethoprim, sulphonamides and aminopenicillins). Reactions may be transitory and mild; however, severe hypersensitivity reactions including DRESS (drug reaction with eosinophilia and systemic symptoms), Stevens–Johnson syndrome and toxic epidermal necrolysis can also occur.

Kaposi's sarcoma is an AIDS-defining malignancy

Antiretroviral medications, particularly protease inhibitors, may act as inducers or suppressors of cytochrome P450 enzymes. Elucidation of the causative agent requires careful evaluation of the temporal relationship of medications. Care must be taken with cessation of antiviral treatments as this may promote resistance. Liaison with infectious disease pharmacists and specialists is advisable. The website www.hiv-druginteractions.com provides information on adverse reactions.

Abacavir, a nucleoside reverse transcriptase inhibitor (NRTI), is associated with a severe hypersensitivity reaction; however, as patients are routinely tested for HLA-B*5701, reactions are rare. The earlier NRTIs stavudine and zidovudine, as well as protease inhibitors, are associated with lipodystrophy involving characteristic peripheral fat lipoatrophy with abdominal accumulation. This may threaten continuation of treatment.

Enfuvirtide, a subcutaneously dosed fusion inhibitor, can cause injection site reactions including induration, erythema and nodules.

Immune recovery syndrome

Immune recovery syndrome (IRS) occurs following the commencement of HAART. As immune recovery occurs, with decreasing HIV viral load and recovery of CD4+ cell counts, increased antigenic response may occur. Such reactions occur in approximately 15% of HIV patients and commonly

Key points

- Skin disease is highly prevalent in patients with HIV infection and may represent the first sign of HIV.
- There should be a low threshold for HIV testing in severe, atypical and recalcitrant cases or high-risk groups.
- Adverse drug reactions are common and particular care should be taken with systemic medications.
- Immune recovery syndrome may occur following the commencement of highly active antiretroviral therapy.

arise four to 12 weeks after treatment. They can be categorised as 'paradoxical', where current conditions worsen, or 'unmasking', where latent infections become apparent. The time at which HAART is commenced may be influenced by current infections to reduce the risk of IRS. However, this decision must be balanced against the risk of worsening immunosuppression.[24]

Conclusion

There is still much to be discovered regarding the pathogenesis of HIV but it is illuminated by the skin disorders discussed. Treatment of skin disease is likely to be required for longer and to be more aggressive in those who are HIV-positive, but conditions improve with anti-retroviral treatment. Adverse drug reactions should be minimised by using caution when starting sytemic treatments ■

Declaration of interest
John T Lear has accepted honoraria for speaking at meetings by Leo, Galderma, Almirall, Astellas and GSK. Nicholas J Collier and Faisal R Ali have no conflicts of interest.

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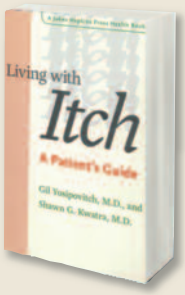
Living with Itch: A Patient's Guide

Yosipovitch G, Kwatra SG. Maryland: Johns Hopkins University Press, 2013: £11



Content: ★☆☆
Teaching: ★★☆
Reference: ★☆☆
Illustrations: ★★☆
Readability: ★★★

This little paperback is one in a series of guides for patient's published by the Johns Hopkins University Press. It is a short overview of living with itch and is easily read from cover to cover in a couple of hours. The misery that itch can cause is brought to life by some harrowing tales from patients. A simple overview of the mechanisms of itching is followed by a consideration of itch associated with common disorders like atopic dermatitis, psoriasis, urticaria, infections and also systemic disorders like diabetes and renal failure. The book also covers psychogenic itching and neuropathic itch. The final part is devoted to non-medical treatments, such as topical therapies and systemic treatments.

The book is American and so cites some problems not often encountered in the UK; for example, poison ivy. It is in the treatment section that this becomes a real problem, as some of the treatments are not readily available in the UK (for example, pramoxine cream and strontium cream) and, where similar products are available, the brand name often differs. On page 109, the table showing the potency of different topical steroids rates mometasone incorrectly as having mild/moderate potency.

I was not really sure this book would be suitable for many of my patients. For a GP, a trainee in dermatology or a dermatology nurse, it is easy to read and gives a useful introduction to the subject in a relaxed and accessible fashion. Indeed, I read it at the gym after my Sunday morning workout. However, I think many patients would find it difficult to assimilate, as they would not be able to put the treatment options into perspective. I suspect there will be the occasional patient who might read it, but beware of the long consultation ahead! ■

Neill Hepburn MD FRCP Consultant Dermatologist, Lincoln County Hospital

PRIMARY CARE DERMATOLOGY SOCIETY

In this column last summer (see *DIP* Vol 19 No 2), I described the unacceptably low profile of dermatology education in the UK. I highlighted how worrying that is, especially when skin disease accounts for such a large a proportion of the workload in primary care, and how extravagantly secondary care dermatology resources are used by GPs. The present arrangement, where most GPs qualify having had virtually no teaching in dermatology, is outrageous. The All Party Parliamentary Group for Skin (APPGS) is extremely concerned about this situation. Last month Sir Edward Leigh, Member of Parliament for Gainsborough, spearheaded a debate in the House of Commons on this predicament.[1] Inevitably, primary care did not look good from this exposure. We can no longer accept this, as we are letting our patients and society down, while the reputation of primary care is being threatened.

The *raison d'être* of the PCDS is to deliver quality dermatology education for GPs. As well as the quarterly meetings where top national and international speakers contribute, the PCDS delivers numerous educational events. These include Essential Dermatology, More Dermatology, Basic Dermoscopy for Beginners, Advanced Dermoscopy, as well as hands-on minor surgery teaching days. We also works closely with the Royal College of General Practitioners (RCGP) to deliver joint whole-day educational events.

Health Education England (HEE) is eager to see the proportion of graduates choosing primary care rise to over 50%. Its desire is to increase the number of GP registrars to over 3,000 per year.

There are only about 650 whole-time equivalent dermatologists in the UK. They are primarily responsible for delivering dermatology services to patients, as well as training future dermatologists and occasionally medical students! There is no way we can expect them to take on the additional burden of educating future GPs. Besides, what we need is for GPs to become better at managing primary care dermatology and that should ideally be delivered from those working in primary care.

I am actively involved in trying to evaluate the size of this problem with the British Association of Dermatologists, the PCDS, the APPGS and the RCGP; however, we urgently need solutions. I would be grateful for any ideas or details of local initiatives that you, the readers, are already involved in. GPs with competence and enthusiasm for dermatology are needed now more than ever, to help design a more comprehensive programme of dermatology education for our profession.

Finally, if you have not recently visited our website, tally no longer. I believe it is now the most useful and comprehensive Internet resource for information on skin conditions and you will be able to discover the full extent of teaching that the society delivers. Furthermore, if you are not already a member of the PCDS, I urge you to join. The more GPs we represent, the more our voice will be heard ■

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PCDS educational events

Surgical meetings
- Litchdon Medical Centre, Barnstaple, 9–10 May 2014

Essential Dermatology
- Crewe Hall, Crewe, 3 April 2014
- Newcastle Marriott Metro Centre, Newcastle, 8 May 2014
- Holiday Inn Luton South, Markyate, Hemel Hempstead, 14 May 2014
- Hilton, Northampton, 21 May 2014

More Essential Dermatology
- Cavendish Conference Centre, London, 11 June 2014
- Hilton Hotel Sheffield Park, Sheffield, 26 June 2014

Dermoscopy for Beginners
- Cambridge Belfrey, Cambridge, 15 May
- Cavendish Conference Centre, London, 12 June 2014

Other Society Meetings
- Summer Meeting, Leeds Marriott Hotel, Leeds, 5 June 2014

DERMATOLOGY IN PRACTICE 2014; Vol 20 No 1

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A non-surgical approach to non-melanoma skin cancer – part 1

Non-melanoma skin cancer (NMSC) is the most common form of skin cancer in the UK, with 99,549 cases registered in 2010 alone. Of these cases, 56% were male and 44% were female, giving a 13:10 male-to-female ratio.[1] The true incidence of NMSC in the UK remains unknown because robust registry data are not available and many NMSCs treated in both the public and private sectors are not recorded. It is estimated that 30–50% of basal cell carcinomas (BCCs) and around 30% of squamous cell carcinomas (SCCs) are unrecorded.[1]

Although NMSC causes little mortality, it can cause significant morbidity if either detection or treatment are delayed. Therefore, both timely recognition and appropriate management of NMSC are important to prevent disfigurement and preserve function. This should also decrease the associated financial burden on the NHS.

In this first part of a two-part article, we give an introduction to NMSC, outlining primary and secondary prevention and describing some emerging therapies for the most common forms of NMSC. In the second part, we will examine in more detail the emerging non-surgical therapies for actinic keratosis (AK), BCC and SCC.

AKs are focal areas of abnormal keratinocyte proliferation associated with chronic ultraviolet radiation (UVR) exposure, which over time may progress to NMSC. There is a 10% transformation rate in patients with an average of 7.7 AKs, which substantiates the need for treatment to prevent NMSC. BCC and SCC represent the majority of NMSCs (74% and 23%, respectively, in the UK); other tumour types include Merkel cell carcinoma, cutaneous lymphoma, microcystic adnexal carcinoma and cutaneous Kaposi's sarcoma – to name a few.

The mainstay of NMSC management is surgical treatments such as cryotherapy, curettage and electrodessication, standard excisional surgery and Mohs micrographic surgery. However, major advances in the medical treatment of NMSC in the last ten years necessitate a comprehensive review of the currently available non-surgical treatment options to enable dermatologists to optimise NMSC management. In this article we discuss the holistic treatment of NMSC, including preventative and curative treatments, focusing on the more common forms of NMSC seen in clinical practice (BCC, SCC and SCC *in situ*).

Box 1. Factors contributing to the development of non-melanoma skin cancer

- Fitzpatrick I–II skin phototype (fair skin that burns easily)
- Male gender and an older age (40–79 years old)
- History of chronic UVR exposure
- Living in, or having previously lived in, tropical latitudes
- Genetic disorders, such as xeroderma pigmentosum, basal cell naevus syndrome, epidermodysplasia verruciformis and albinism
- Immunosuppression after organ transplantation where SCCs are highly prevalent (particularly after heart and lung transplants due to multiple immunosuppressives)
- Exposure to ionising radiation, coal tars, soot, petroleum oils, polycyclic aromatic hydrocarbons and arsenic or burn scars
- Infection with human papillomavirus types 16, 18, 30 and 33 (in SCC)[2]

SCC = squamous cell carcinoma; UVR = ultraviolet radiation

Primary prevention

Identifying high-risk patients

Many factors contribute to the predisposition to, and development of, NMSC (see Box 1). The timely identification of these factors, as well as tailoring treatment and advice to these, will help appropriately identify and treat patients who have an increased risk of developing NMSC.

Sun protection

Excessive cumulative UVR exposure, UVR-induced mutations in the p53 tumour suppressor gene[2] and the hedgehog signalling pathway are causative factors for the development of BCCs.[3] Therefore, it is essential to discuss appropriate ways to protect the skin from the damaging effects of the sun. Exposure to both UVA (315–400 nm) and UVB (280–315 nm) light is implicated in NMSC development, although chronic UVB exposure is more significant in BCC development.

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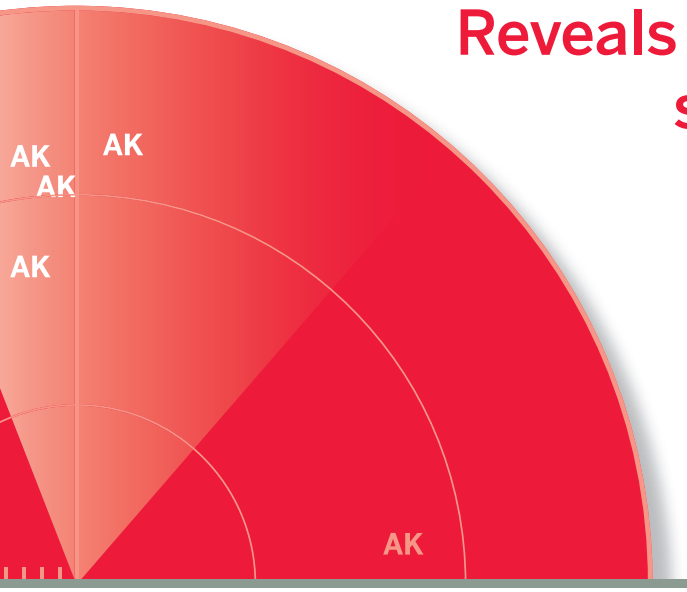

Zyclara 3.75% cream (imiquimod). Indications: Zyclara is indicated for the topical treatment of clinically typical, nonhyperkeratotic, nonhypertrophic, visible or palpable actinic keratosis (AK) of the full face or balding scalp in immunocompetent adults when other topical treatment options are contraindicated or less appropriate. **Dosage:** Treatment should be initiated and monitored by a physician. Apply up to 2 sachets, once daily, before bedtime to the skin of the affected treatment area for two treatment cycles of 2 weeks each separated by a 2-week no-treatment cycle or as directed by the physician. The treatment area is the full face or balding scalp. The safety and efficacy of imiquimod in AK in children and adolescents below the age of 18 years have not been established. For external use only. Contact with eyes, lips, and nostrils should be avoided. The treatment area should not be bandaged or otherwise occluded. Apply as a thin film to the entire treatment area and rub in until the cream vanishes. Partially-used sachets should be discarded and not reused. Leave on the skin for approximately 8 hours; after this time it is essential that the cream is removed by washing the area and the hands with mild soap and water. **Contraindications:** Hypersensitivity to the active substance or to any of the excipients. **Warnings and precautions:** Lesions clinically atypical for AK or suspicious for malignancy should be biopsied to determine appropriate treatment. Not recommended until the skin has healed after any previous medicinal products or surgical treatment. Use of sunscreen is encouraged, and patients should minimise or avoid exposure to natural or artificial sunlight. Not recommended for the treatment of AK lesions with marked hyperkeratosis or hypertrophy as seen in cutaneous horns. During therapy and until healed, affected skin is likely to appear noticeably different from normal skin. Local skin reactions are common but generally decrease in intensity during therapy or resolve after cessation of therapy. Rarely, intense local inflammatory reactions including skin weeping or erosion can occur after only a few applications. There is an association between the complete clearance rate and the intensity of local skin reactions. These local skin reactions may be related to the stimulation of local immune response. Imiquimod has the potential to exacerbate inflammatory conditions of the skin. If required by the patient's discomfort or the intensity of the local skin reaction, a rest period of several days may be taken. Treatment can be resumed after the skin reaction has moderated. The intensity of the local skin reactions tend to be lower in the second cycle than in the first treatment cycle. Flu-like systemic signs and symptoms may accompany, or even precede, intense local skin reactions and may include fatigue, nausea, fever, myalgias, arthralgias, and chills. An interruption of dosing or dose adjustment should be considered. Use with caution in patients with reduced haematologic reserve. Patients with cardiac, hepatic or renal impairment were not included in clinical trials. Caution should be exercised in these patients. Use with caution in immunocompromised patients and/or patients with autoimmune conditions and consider balancing the benefit of treatment for these patients with the risk associated either with the possibility of organ rejection or graft-versus-host disease or a possible worsening of their autoimmune condition. No data are available on re-treating AK that have cleared after two cycles of treatment and subsequently recur. Stearyl alcohol and cetyl alcohol may cause local skin reactions. Methyl parahydroxybenzoate (E 218), and propyl parahydroxybenzoate (E 216) may cause allergic reactions (possibly delayed). No interaction studies have been performed but use with caution in patients who are receiving immunosuppressive drugs. Avoid using with any other imiquimod creams in the same treatment area. No data are available on the use of Zyclara during pregnancy or breast-feeding and there are no data on the risk to human fertility. There is no or negligible influence on the ability to drive or use machinery. **Side effects:** Herpes simplex, skin infection, lymphadenopathy, haemoglobin, white blood cell and platelet counts decreased, anorexia, blood glucose increased, insomnia, depression, headache, dizziness, nausea, diarrhoea, vomiting, erythema, scab, skin exfoliation, skin oedema, skin ulcer, skin hypopigmentation, dermatitis, erythema multiforme, Stevens Johnson syndrome, cutaneous lupus erythematosus, skin hyperpigmentation, myalgia, arthralgia, application site effects, including erythema, scabbing, exfoliation, dryness, oedema, ulcer, discharge, reaction, pruritus, swelling, burning, irritation and rash, fatigue, pyrexia, influenza like illness, pain, chest pain. Consult the Summary of Product Characteristics before prescribing, particularly in relation to side effects, precautions and contraindications. **Legal Category:** POM. **Package quantity and basic NHS price:** Pack of 28 sachets £113.00. **Product licence number:** EU/1/12/783/002. **Marketing authorisation holder:** Meda AB, Pipers väg 2A, 170 73 Solna, Sweden. **Date of preparation of prescribing information:** January 2013. UK/ZYC/13/0003

Adverse events should be reported.
Reporting forms and information can be found at www.mhra.gov.uk/yellowcard. Adverse events should also be reported to Meda Pharmaceuticals Ltd.

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UK/ZYC/13/0005 Date of preparation: February 2013.

MEDA

For this reason, sunscreens offering dual protection should be recommended to patients who are at increased risk as a primary preventative measure.[3] Other measures, including avoiding sun exposure between 11 am and 2 pm and wearing UV protective clothing, are also advisable.

Alfamelanotide (synthetic alpha melanocyte-stimulating hormone) and its role in the prevention of NMSC is currently under evaluation and highly contentious.[4] It may be that it stimulates melanin production *in vivo* with higher melanin levels, thereby offering much greater protection against the sun.

Secondary prevention

Regular skin checks are a simple, yet effective, way of identifying problem skin lesions, thus facilitating early specialist attention and treatment. In the UK, regular, specialist-led, total body examinations are reserved for high-risk patients who have had melanoma or high-risk SCC.

Chemoprevention of NMSC is a preventive strategy where chemical compounds are used, either topically or systemically, to reverse or suppress the conversion of precursor lesions to NMSC. Common agents used to treat precursor lesions in the form of field therapy (that is, over wide surface areas of skin with undersurface subclinical disease) are topical 5-fluorouracil and imiquimod, which act via the non-competitive inhibition of thymidylate synthetase and toll-like receptor 7, respectively. Photodynamic therapy (PDT) and the newer lower-dose imiquimod (Zyclara® 3.75%, Meda) are also being used successfully in the treatment of actinic field damage. The use of 5-fluorouracil and imiquimod in the direct chemoprevention of NMSC is also currently being studied. Field therapy may be the more effective treatment for widespread superficial actinic damage where surgical treatment is impractical.

Specialist-led, total body examinations are reserved for high-risk patients

Retinoids

Commonly used chemopreventive agents include vitamin A derivatives such as topical tretinoin, oral acitretin and isotretinoin. Retinoids induce apoptosis; arrest growth; stimulate differentiation of tumour cells during carcinogenesis;[2] and down-regulate the overexpression of UVR-induced cyclooxygenase-2 (COX-2), causing a decrease in prostaglandins, which are increased in NMSC. Retinoid use in the prevention of NMSC in both solid organ transplant recipients (SOTRs) and non-SOTR populations has been found to be effective.[5]

Systemic retinoid therapy is currently not licensed for the prevention of NMSC. Despite this, it is widely used: in high-risk patients such as those who are chronically immunosuppressed (for example, those with non-Hodgkin's lymphoma, chronic lymphocytic leukaemia or HIV and SOTRs); in psoriasis treated with long-term psoralen combined with UVA (PUVA) treatment; in radiation-induced NMSC; and in patients with xeroderma pigmentosum, naevoid BCC syndrome, Bazex's syndrome, Rombo syndrome or epidermodysplasia verruciformis.[2]

Further candidates for retinoid chemoprevention are patients who develop more than five NMSCs per year; multiple NMSCs in the head and neck area; numerous AKs; metastatic or aggressive NMSC; and patients with eruptive keratoacanthomas.[2] Acitretin (dosed at 0.2–0.4 mg/kg/day) has been used with good effect in the prevention of AK, SCC and BCC. A sustained response was noted in patients while on treatment but there was disease relapse on cessation of the treatment.[6]

Photodynamic therapy

PDT is effective for treating actinic damage in both immunosuppressed and immunocompetent patients. The photosensitisers 5-aminolaevulinic acid (ALA) and methyl laevulinic acid (MAL) are commonly used. The application of ALA and MAL increases the concentration of protoporphyrin IX in sun-damaged skin with subsequent irradiation of the area. This increases the production of reactive oxygen species, which promote selective destruction of diseased tissue. MAL PDT is less hydrophilic compared with ALA PDT, so it is better absorbed, which subsequently enhances its efficacy in thicker lesions.[7] Laser-mediated ALA PDT is a viable alternative offering shorter treatment duration, rapid recovery time and less discomfort compared with blue light PDT devices.[2]

Emerging therapies for non-melanoma skin cancer

Ingenol mebutate

Ingenol mebutate (IM) is the active agent of the plant *Euphorbia peplus* or milkweed, and has recently become available in the UK. It acts on dysplastic keratinocytes by chemo-ablation, which disrupts the plasma membrane because of the loss of the mitochondrial membrane potential and mitochondrial swelling, resulting in rapid cell death by primary necrosis. IM promotes rapid healing by restoring both clinical and histological morphologies. IM also promotes neutrophil infiltration, resulting in tumour-specific antibody production and antibody-dependent cellular cytotoxicity, which helps to clear residual cells. A concentration of 0.015% is used for three consecutive days on the face, while the 0.015%

preparation is used for two consecutive days at other sites. Both of these concentrations have shown promising initial and sustained results in AK treatment.[8]

Perillyl alcohol

Perillyl alcohol (POH) is a natural hydroxylated monocyclic monoterpene found in the essential oils of cherries, spearmint and lavendin. The exact mechanism of POH is unknown, although it is widely known to induce apoptosis of tumour cells, therefore further inhibiting AP-1 activation and cholesterol synthesis. The use of topical POH twice a day for 30 days has proven safety in humans.[2]

T4 endonuclease V

T4 endonuclease V (T4E5) is a polypeptide derived from *Escherichia coli* infected with a T4 bacteriophage, which is able to reverse UV-induced cyclobutane pyrimidine dimer damage in DNA and suppress UV-induced upregulation of tumour necrosis factor-alpha and interleukin-10. Topical application of T4E5 has demonstrated a reduction of NMSC in UVB-irradiated mice and human patients with xeroderma pigmentosum. Topical T4E5 application once daily for one year demonstrated a reduction in the development of AK and BCC with no side effects.[2]

Polyphenols

Polyphenols (PPs) are a large group of natural plant products such as coffee, berries, pomegranate and tannins with established anti-inflammatory, immunomodulatory and antioxidant properties. Epigallocatechenin (EGCC) is the most widely studied PP and is present in green tea. The exact mechanism of action of EGCC is unknown; however, it is thought to inhibit UV-induced tumourigenesis by the inhibition of AP-1 31 and inhibits the autophosphorylation of the epidermal growth factor receptor (EGFR), therefore suppressing cell proliferation.[2]

Alpha-difluoromethyl ornithine

Alpha-difluoromethyl ornithine (DFMO) irreversibly inhibits ornithine decarboxylase, the rate-limiting step in polyamine synthesis, which is increased in UVB-induced NMSC. DMFO therapy once daily (both topical and systemic) has been studied for the treatment, as well as the prevention, of NMSC with demonstrable effect.[2]

Peroxisome proliferator-activated receptors

Peroxisome proliferator-activated receptors (PPARs) are nuclear hormone receptors that modulate the expression of a variety of target genes involved in lipid, glucose and amino acid metabolism. PPAR alpha ligands induce keratinocyte differentiation and inhibit skin tumour production.[2] Studies performed on mice have shown promising results with PPAR ligands in melanoma and further study is ongoing in NMSC to establish their true effect.

Diet

Dietary modification to low-fat food has been studied in animals. There are promising data supporting its role in the prevention of actinic damage and NMSC.[2] A low-fat diet to prevent NMSC and actinic damage in humans, however, is yet to be verified. The dietary effects of beta-carotene, selenium, vitamin C and vitamin E have been studied, but have no proven benefit ■

Part 2 of this article will appear in the Summer 2014 issue (Vol 20 No 2)

Declaration of interest
The authors declare that there is no conflict of interest.

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Key points

- Non-melanoma skin cancer (NMSC) is the most common form of skin cancer in the UK. It causes little morbidity but can cause great morbidity if detection and treatment are delayed.
- Primary prevention involves reducing exposure to ultraviolet radiation.
- Secondary prevention is by treating NMSC precursor lesions with field therapy. Common agents include 5-fluorouracil and imiquimod, while photodynamic therapy is effective for treating actinic damage in both immunosuppressed and immunocompetent patients.
- Systemic retinoid therapy is not currently licensed for the prevention of NMSC, but it is widely used in high-risk patients.

How to use marking sutures on soft tissue lesions

■ **Figure 1.** The first throw is single and placed loose

Sutures are commonly used to mark excised soft tissue lesions in order to orientate the specimens for histological examination.[1] Inappropriate application, leading to tissue trauma, has been reported by our pathology department to compromise histological examination of the specimen.

Here we describe a marker suture technique that will avoid crushing the underlying tissue and therefore minimise the adverse effect on histological analysis. This technique may be applied to the excision of both superficial and deep soft tissue lesions.

Technique

- Place the marking suture in the desired position within the specimen.
- Tie a single, loose first throw (see Figure 1).
- Tie the second throw above the first (see Figure 2).
- Tighten the second throw to lock the first, maintaining a gap between the tissue and the knot (see Figure 3).

■ **Figure 2.** The second throw, also single, is placed above it to create a reef knot, before being tightened and locked

Discussion

The use of marking sutures is common practice,[1] though tight application may crush the edge of the lesion. Several other marking techniques have been described, including incising the edge of the specimen and the use of marker clips and silver nitrate.[2–4]

To our knowledge, the technique discussed here has not been described before. This simple modification of the use of a marking suture results in a cheap, robust method, leading to a more elegant operative technique and better histological examination of the specimen at the lateral margin ■

■ **Figure 3.** The knot is secure and locked with a gap between it and the tissue, to avoid crushing the skin specimen

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Who will guard the guards themselves?

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I am a generous chap by nature and happy to spend my hard-earned money. I am, within limits, even happy for my wife and children to spend it for me. But when complete strangers want to spend my money, well that is quite another matter.

I read in our local newspaper recently that the Bedfordshire Clinical Commissioning Group (CCG) has proposed spending £3.2 million on yet another review of local health provision.[1] It reassured readers that Monitor, the health services regulator designed to ensure that NHS money is spent prudently, will provide the money. I was forced to write back to explain that, in fact, it was not Monitor that was paying, but the taxpayer, you and me, and that £3.2 million would by my calculations be enough to pay for an extra 20 hospital cleaners for the next ten years. Furthermore, I wrote that, since it was my money that the Bedfordshire CCG was planning to spend, I knew which of the two options I would prefer.

It all made me wonder what else Monitor was up to with my chequebook, and its website was an endless source of fascination. With an annual budget of £53.7 million,[2] and advertising for a range of new employees with incomprehensible job titles and mouth-watering salaries, it appears to be spending hundreds of thousands each month on management consultants, no doubt to tell its staff how to do their own jobs. In one instance that I could identify, it even paid for a Monitor employee to attend a conference overseas organised by one of the same management consultants that it was paying in the first place. This may be nitpicking, but my own Trust has just told me that it can't afford the £175 course fee for me to attend a skin cancer update being organised by the British Association of Dermatologists.

This all made me start thinking about that other source of wonder, the Care Quality Commission (CQC). In the last year for which it published figures, it managed to spend £166 million, despite only having an income of £93 million.[3] No doubt next year it will report spending even more money to find out how to explain the imbalance.

These amounts of money may be mere trifles in the overall NHS spend, but it is worth reflecting on the fact that the NHS appeared to run perfectly well for 60 years before anyone dreamed up Monitor and the CQC, and that there is no clear evidence that they have done anything to improve standards.

It is nearly 2,000 years since the Roman poet and satirist Juvenal coined the phrase *'Quis custodiet ipsos custodes?'* Clearly, not much has changed and we must all be on our guard ■

Declaration of interest
The author declares that there is no conflict of interest.

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■ Bedfordshire Clinical Commissioning Group has proposed spending £3.2 million on another review of local health provision

HIROSHI WATANABE/GETTY IMAGES

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Psychological adjustment to disfiguring skin diseases

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After diagnosis of a skin disorder, there follows a period of psychological adjustment during which most people are able to cope. Coping is a process that can be described as 'expending conscious effort to solve personal and interpersonal problems, and seeking to master, minimise or tolerate a stressor'.[1] Many types of coping strategies are possible, including planning; positive re-interpretation; using practical and emotional social support; venting of emotions; suppression of competing activities; emotional, behavioural and cognitive disengagement; and use of humour or religion. Several methods may be used and the methods used may change; skills are used or developed to regain a sense of equilibrium and well-being.

Exploring ways of coping can be useful and an assessment tool such as The Brief COPE Questionnaire (see Box 1) can help in this regard.[2] Some people appear to cope well and adjust quickly despite an objectively severe skin disease, while others may become very stressed and struggle to cope in the face of an objectively minor skin disorder.

Psychological adjustment is a multifactorial and fluctuant biopsychosocial process which centres around three themes: a search for meaning in the experience; an attempt to regain control over the situation and in life generally; and an effort to restore self-esteem.[3]

There are a number of factors that contribute towards adjustment[4] and the potential impact of the skin disorder. Some of these factors are irreversible and objective while others are modifiable and more subjective. A skin disorder can result in physical, psychological, emotional, social and functional types of stress. These can be summarised with the help of a comprehensive quality of life (QoL) measuring tool, like Skindex 29.[5] Recognising that the skin disorder is impacting on the person's QoL is important, but one needs to find out why. An altered appearance may cause stress from body image dissatisfaction, which can be especially acute when the skin condition affects exposed sites.[6] Associated physical symptoms such as pruritus, discomfort and irritation can be addressed with conventional physical treatment. The treatment itself can be a source of stress, especially if topical preparations are messy, sticky or smelly. Time-consuming treatment, like attending phototherapy sessions, can be impractical.

When there is a sudden change in appearance; for example, after skin surgery or acute hair loss, the patient may struggle to come to terms with this and grieve for their previous appearance.[7,8]

Coping mechanisms

Personality factors often contribute towards the coping mechanisms. Resilience, which has been defined as 'the capacity of an individual to successfully maintain or regain their mental health in the face of adversity', is a key factor in coping and has been the focus of many chronic physical disease studies in the last ten years.[9] The objective severity of the disorder, as well as the gender and age of the patient, are poor predictors of adjustment ability. Potential resilience and risk factors have been suggested (see Tables 1 and 2).[4, 7–12] It is the patient's subjective experience and perceived severity that predicts the level of distress, rather than objective clinical severity. Many factors interact, including social support, self-efficacy and perceptions of self-control. Certain comments from others, including healthcare professionals, can be unhelpful: for example, if the skin disorder is trivialised as being 'only cosmetic' and therefore 'not serious'.[13]

Most people cope and adjust by working through psychosocial difficulties themselves. GPs and dermatologists meet patients who have had psoriasis most of their lives and relate their experiences of 'having to live with it'. Social comparisons are a common form of self-enhancement; for example, patients make comments such as, 'I am not that bad compared to other people' or 'It would have been harder if I was young and single'. Learning specific social skills can be helpful when dealing with comments or stares from strangers. Evidence shows that how the patient responds to social skills training, which includes coping with unwanted attention and intrusive questioning, is a better predictor of adjustment than the objective severity of the condition.

Ability to adjust

Research has suggested five main factors which contribute to the stress associated with a skin disorder and predict the patient's ability to adjust:[10]

- Disease and treatment factors
- Cognitive factors – personality characteristics and core beliefs about illness
- Early experiences and cultural beliefs

Box 1. Brief COPE Questionnaire adapted for dermatology patients[2]

Having a skin complaint or condition may be stressful and cause problems for a person. These items ask what you've been doing to cope with this problem. Different people deal with things in different ways; I'm interested in how you've tried to deal with it. Each item says something about a particular way of coping. I would like to know to what extent you've been doing what the item says – 'how much or how often'. Don't answer on the basis of whether it seems to be working or not – just whether you're doing it. Try to rate each item separately from the others in your mind. Make your answers as true for you as you can. Use these response choices please:

1 = I haven't been doing this at all
2 = I've been doing this a little bit
3 = I've been doing this a medium amount
4 = I've been doing this a lot

1. I've been turning to work or other activities to take my mind off things
2. I've been concentrating my efforts on doing something about the situation I'm in
3. I've been saying to myself 'this isn't real'
4. I've been using alcohol or other drugs to make myself feel better
5. I've been getting emotional support from others
6. I've given up trying to deal with it
7. I've been taking action to try to make the situation better
8. I've been refusing to believe that it has happened
9. I've been saying things to let my unpleasant feelings escape
10. I've been getting help and advice from other people
11. I've been using alcohol or other drugs to help me get through it
12. I've been trying to see it in a different light, to make it seem more positive
13. I've been criticising myself
14. I've been trying to come up with a strategy about what to do
15. I've been getting comfort and understanding from someone
16. I've given up attempting to cope
17. I've been looking for something good in what is happening
18. I've been making jokes about it
19. I've been doing something to think about it less, such as going to the cinema, watching TV, reading, day-dreaming, sleeping or shopping
20. I've been accepting the reality of the fact that it has happened
21. I've been expressing my negative feelings
22. I've been trying to find comfort in my religion or spiritual beliefs
23. I've been trying to get advice or help from other people about what to do
24. I've been learning to live with it
25. I've been thinking hard about what steps to take
26. I've been blaming myself for things that happened
27. I've been praying or meditating
28. I've been making fun of the situation

- Coping strategies, including emotion-focused, problem-focused and cognitive-focused coping
- Ongoing social and cultural factors; for example, social support versus social rejection.

Enquiring about the person's understanding and beliefs about the skin disorder is important and the use of the Brief Illness Perception Questionnaire, which measures important factors including the disease identity, cause, timeline, consequences and cure, can be helpful.[14]

Healthcare professionals can help by discussing the impact of the disorder on the person's life, enabling them to form a problem list and identify areas that can be addressed, including coping strategies. The links between feelings, thoughts and behaviour are a useful discussion point. Psychoeducation is helpful to normalise common feelings and behaviours in reaction to a new skin disorder. The level of distress should be documented as high, medium or low.

Some patients demonstrate unhelpful beliefs such as being convinced that they are unattractive, inferior, unlovable or unworthy. Such beliefs should be taken seriously – in severe cases, low self-worth occurs and suicidal ideation can develop. A self-fulfilling prophecy may also develop when the person expects and then experiences unfavourable reactions. This type of cognitive appraisal blocks affective adjustment.

The skin change can be either fixed (a scar) or fluctuating (psoriasis or eczema) and thus pose different challenges. The unpredictable flares of an inflammatory skin disease can cause feelings of a lack of control. As a result, the patient may develop unhelpful strategies such as radical lifestyle or diet changes; repeated internet searches for a 'miracle cure'; repeated purchase of 'miracle' products or behavioural rituals to try to cope (for example, avoiding stressful situations); covering up the affected areas with hair, hands or clothing in public; and use of excessive make-up or repeated mirror-checking or mirror avoidance. Such behaviours can draw more attention than the actual condition itself and are considered maladaptive.

Acceptance is a large part of adjustment and this is underemphasised in clinical practice. It is a positive process: the patient is not in denial and does not 'give up', but accepts the reality of the stressful experience, that some aspects can be actively dealt with and others cannot.[11] Patients can develop unrealistic expectations regarding cure or improvement of their skin condition and may make statements like, 'I won't be able to get married unless my acne is better'. Such convictions can be gently challenged by emphasising that the skin

Table 1. Resilience factors helping patients to cope with a new skin disorder[4,7-12]

- Positive personality factors: good at emotional disclosure, 'thick skinned' to adverse comments, self-determination and independence, self-efficacy, self-acceptance, high self-esteem, optimism
- Optimum psychological well-being
- Popularity among peers
- Leadership qualities
- Strong family bonding
- Trusting relationships with others
- Use of humour

Table 2. Risk factors for maladaptive coping with a new skin disorder[4,7-12]

- Negative personality factors: neuroticism, social sensitivity, 'thin skinned' nature, low self-esteem, shame tendency, appearance consciousness, fear of negative evaluation, Type C personality (emotionally inexpressive coping style), antisocial or dependent personalities
- Co-existing mental health disorder (eg anxiety or depressive disorder)
- Cognitive factors (eg tendency to ruminate and catastrophise)
- Suboptimal or impaired psychological well-being (lack of self-acceptance, dissatisfaction with self, disappointed with what has occurred in past life, troubled by certain personal qualities, wishes to be different)
- Social perfectionism (overconcerned with the expectations and evaluations of others, conforms excessively to social pressures)
- High importance of body image
- Few close relationships with others, difficulty being open, feeling of isolation, lack of support from family and friends
- Dysfunctional family dynamics
- Early maladaptive schemas and ongoing negative self-schemas

disorder is only a small part of them and by emphasising the positive aspects of their life.

Maladaptive schemas

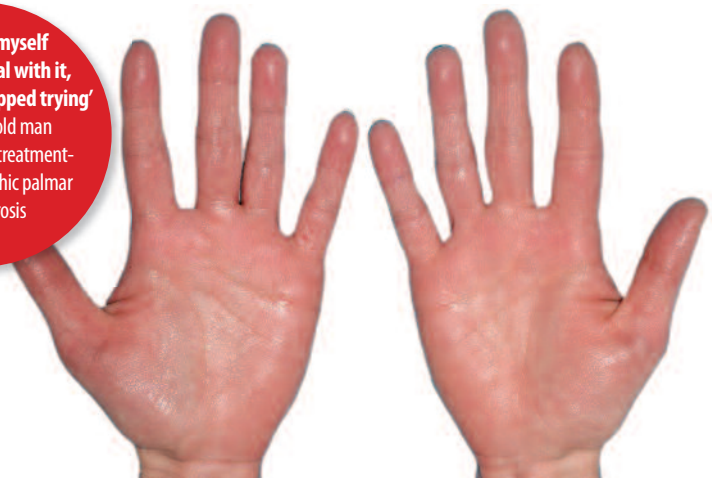
In some patients, a negative event or experience in childhood may cause an 'emotional scarring'. In adults and adolescents this may contribute to ingrained cognitive and emotional patterns (schemas) associated with low self-esteem, shame, appearance consciousness, fear of negative evaluation and social anxiety. There is recent evidence that such negative schemas may have a role in the distress associated with chronic skin disease;[12] for example, psoriasis and atopic eczema in some patients who struggle to adapt. Two early maladaptive schemas, 'vulnerability to harm' and 'defectiveness', were found to be predictive of anxiety, while social isolation and 'vulnerability to harm' were found to be predictive of depression. Patients with these schemas might benefit from high-level schema-focused therapy.

Psychological therapy

Understanding the key factors involved in adjustment can help when discussing coping strategies and finding the appropriate psychological therapy. Ideally, a health psychologist should assess all such patients, but there are minimal resources in the UK for this. It does not seem right that a person with moderate-to-severe psoriasis, which has a high impact on their QoL, may be prescribed a biologic therapy costing approximately £8,000 per year without a discussion about coping. Currently, it is left to GPs, dermatologists and nurses to provide a supportive approach to facilitate self-help.

The assessment of a patient struggling to adjust to a new skin disorder should involve information gathering (to build a detailed picture of emotional morbidity, thoughts and beliefs), behavioural modifications, and social or work-related compromise. This allows triage and a management plan can be suggested. The level of distress and complexity of the issues will determine the nature of the healthcare professional best suited to help. Level 1 intervention consisting of empathy and support may be appropriate, but other patients will need more help from problem-solving and higher-level specific

'I have grown as a person as a result of my experience living with vitiligo' – a 24-year-old woman who initially struggled but over time developed a positive reinterpretation ➜



'I admit to myself that I can't deal with it, and so I have stopped trying' – a 27-year-old man struggling with treatment-resistant idiopathic palmar hyperhidrosis ➜

psychological interventions such as cognitive behavioural therapy and person-centred therapy.

Adjustment disorder

It is common for dermatologists to see patients who are distressed and struggling to adjust. Such patients often have associated minor affective symptoms, including low mood and anxiety, and would fit the diagnostic criteria of adjustment disorder (AD). This is defined as the development of emotional or behavioural symptoms in response to an identifiable stressor, which occur within three months of the onset of the stressor but do not last longer than six months after the termination of the stressor.[15] When suffering from AD, the patient's emotional distress is in excess of what would be expected or there is an excessive impairment in social or occupational functioning. The symptoms may persist if the cause of the stress persists, such as in the case of a chronic skin disorder.

AD can be subdivided into six subtypes according to the predominant symptom:

- With depressed mood
- With anxiety
- With anxiety and low mood
- With disturbance of conduct
- With mixed mood and conduct disturbance
- Unspecified.

The diagnosis is not made if symptoms meet the criteria of another mental disorder. The boundary between AD and normal adaptive stress can be unclear; as can the boundary between transient body image concern and body dysmorphic disorder; and that between AD with predominant low mood and depressive disorder. Psychiatrists may disagree about a psychiatric diagnosis and it is not uncommon for a psychiatric diagnosis to change over time in the same individual. It may be considered best for dermatologists to list symptoms and speculate as to a possible psychiatric diagnosis. There may be overlap between AD and other subclinical psychosomatic factors related to abnormal illness behaviour, including anxiety, somatisation, demoralisation, irritable mood and disease phobia.

Adolescents are particularly vulnerable to AD and this is a risk factor for suicidal ideation and suicide; one study showed that one third of 1,397 adolescents in Finland between March 1987 and April 1988 had been diagnosed with AD prior to suicide.[15] One should be wary of acne in a distressed adolescent. The trigger for the AD may be the direct psychological effect of acne, or episodes relating to the acne like bullying. Such cases should be treated with a thorough history and mental-state examination assessing risk.[16] AD in adolescence is also a risk factor for developing future mental health disorders.

Conclusion

Healthcare professionals can help patients adjust to a skin disease by understanding the principles and practice of successful engagement. The patient's illness perception and main concerns (emotional, psychological, physical and functional) should be determined. To help their patients, healthcare professionals should display encouragement, help them build on their strengths/previous ability to overcome adversity or psychological trauma, and break down the main issues into smaller problems. The plan should be supported by listening to the patient's wishes and providing consistent and positive messages. Barriers and difficulties should be acknowledged and the expression of feelings encouraged. Involving the patient in their own care, including self-management measures, will lead to a perception of increased control over the skin disorder and will help them to adjust ■

Declaration of interest
The authors declare that there is no conflict of interest.

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Key points

- People cope with skin disease in different ways and many coping strategies are possible.
- Psychological therapy is often lacking; for example, a person with moderate-to-severe psoriasis may be prescribed an expensive biologic therapy without any discussion about coping taking place.
- Patients may suffer from adjustment disorder, with a distress level in excess of what would be expected or an excessive impairment in social or occupational functioning.

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