

THE EFFECTIVE MANAGEMENT OF HYPERKERATOSIS

There are various skin conditions that fall under the umbrella term 'hyperkeratosis' and this article looks at the aetiology and subsequent modes of treatment in regards to these conditions.

Hyperkeratosis is an umbrella term for a number of skin conditions. It involves a thickening of the stratum corneum (the outer layer of the skin), often associated with a keratin abnormality, and is also usually accompanied by an increase in the granular layer of the skin. As the corneum layer normally varies greatly in thickness across different sites, some experience is needed to assess minor degrees of hyperkeratosis (Kumar et al, 2004).

This thickening is often the skin's normal protection against rubbing, pressure and other forms of irritation, causing calluses and corns on the hands and feet or whitish areas inside the mouth. Other forms of hyperkeratosis occur as part of the skin's defence against chronic inflammation, infection and the radiation of sunlight or irritating chemicals. Less often, hyperkeratosis develops on skin that has not been irritated. These types may be part of an inherited condition, may begin soon after birth and can affect skin on large areas of the body (Freedberg et al, 2003).

Types of hyperkeratosis (hereditary)

Epidermolytic hyperkeratosis

This type of hyperkeratosis is also known as 'bullous congenital ichthyosiform erythroderma', 'bullous ichthyosiform erythroderma', or 'bullous congenital ichthyosiform erythroderma Brocq' and is a rare

skin disease of the ichthyosis family, affecting around 1 in 250,000 people.

It involves the clumping of keratin filaments (Freedberg et al, 2003). This is a hereditary disease, the symptoms of which are hyperkeratosis, blisters and erythema. At birth, the skin of the individual is entirely covered with thick, horny, armourlike plates that are soon shed, leaving a raw surface on which scales then reform.

Multiple minute digitate hyperkeratoses (MMDH)

MMDH is a rare familial or acquired cutaneous eruption of filiform keratosis, typically found across the trunk and extremities. Histopathology, distribution and history can distinguish it from other digitate keratoses. Goldstein described the first case of MMDH in 1967, suggesting the term 'multiple minute digitate hyperkeratosis' because the projections were numerous, and finger-like, comprising a horn of dense orthokeratin (Goldstein, 1976). MMDH presents with numerous digitate keratoses across the trunk and limbs, with sparing of the face, palms, and soles. The keratoses are skin-coloured, rod-shaped, non-follicular, between 1–5mm in length, between 0.3–2mm in diameter, and arise from otherwise normal skin (Caccetta et al, 2010).

Focal acral hyperkeratosis

Also known as 'acrokeratoelastoidosis

- ▶▶ Fungal infection
- ▶▶ Hyperkeratosis
- ▶▶ Stratum corneum
- ▶▶ Keratin

ANN JAKEMAN is a Tissue Viability Nurse at the Royal Brompton and Harefield Foundation Trust, London

lichenoides' this condition is a late-onset keratoderma, inherited as an autosomal dominant condition (single parental gene), characterised by oval or polygonal crateriform papules developing along the border of the hands, feet and wrist (Freedberg et al, 2003).

Lamellar ichthyosis

This is a rare skin condition that appears at birth and continues throughout a person's life. It is passed down through families and both parents must have at least one abnormal gene to pass it on to their children. People with this condition are born with a collodian membrane — a shiny, waxy layer of skin that sheds within the first two weeks of life. Red, scaly skin remains underneath — this resembles the surface of a fish (Morelli, 2007).

X-linked ichthyosis (XLI)

XLI is also known as 'steroid sulfatase deficiency', as well as 'X-linked recessive ichthyosis' (from the Ancient Greek 'ichthys', meaning 'fish') and is a skin condition caused by the hereditary deficiency of the steroid sulfatase (STS) enzyme, affecting between one in 2,000 and one in 6,000 males. XLI manifests with dry, scaly skin and is due to deletions or mutations in the STS gene. XLI can also occur in the context of larger deletions, causing contiguous gene syndromes. Treatment is largely aimed at alleviating the skin symptoms (Gelmetti and Caputo, 2002).

Keratosis pilaris

Also known as 'follicular keratosis', keratosis pilaris is a common, autosomal dominant, genetic follicular condition that is manifested by the appearance of rough bumps on the skin. It most often appears on the back and outer sides of the upper arms (although the lower arms can also be affected), and can occur on any body part except glabrous skin (naturally hairless, such as the palms or soles of the feet). Less commonly, lesions appear on the face, which may be mistaken for acne. This

condition has been shown in several small-scale studies to respond well to supplementation with vitamins and fats rich in essential fatty acids. Some research suggests this is due mainly to vitamins E and B. Vitamin A is also thought to be connected to the pathology (Nadiger, 1980).

Site specific

Plantar hyperkeratosis

This condition is a circumscribed keratotic area which may or may not be associated with a hammered great toe. Keratosis often resembles verruca plantaris, for which it is mistakenly treated, until the keratosis breaks down and ulcerates or leaves a permanent scar. Although keratosis under the first metatarsal head is common, it is more usual to have disabling symptoms underneath the middle three metatarsals (Skinner and Fitzpatrick, 2008).

Hyperkeratosis of the nipple and areola

This is an uncommon benign, asymptomatic, acquired condition of unknown pathogenesis.

Lichen planus

Lichen planus may appear as a lacy, white patch on the inside of the mouth or as an itchy, violet, scaly patch elsewhere on the skin. In some cases, it affects the mouth, genitals, hair, nails, and rarely, other parts of the body. About one in 50 people develop lichen planus and it occurs equally in men and women. More than two-thirds of cases occur in people aged 30–60 years, however, it can occur at any age. It is not an inherited disease, nor is it an infection, and the rash cannot be caught or passed on to others. Although the cause of this condition is unknown, researchers suspect that it may be related to an abnormal reaction of the immune system. One theory is that the immune system may be triggered by a virus or other factor in the environment to attack cells in the skin, which leads to the inflammation (Lehman et al, 2009).

Seborrheic keratosis

These are small, non-cancerous skin growths. They can be tan, brown or black and are found on the face, trunk, arms or legs. These are very common and most people develop between one and 20 during their lifetime. Their cause is unknown (Freedberg et al, 2003).

Corns

A corn is a small area of skin which has become thickened due to the pressure exerted on it. They are roughly round in shape and press into the deeper layers of skin, becoming painful. Hard corns commonly occur on the top of the smaller toes or on the outer side of the little toe. These are the areas where poorly fitted shoes tend to rub the most. Soft corns sometimes form in between the toes, most commonly between the fourth and fifth toes. These are softer because the sweat between the toes keeps them moist. Soft corns can sometimes become infected (Freeman, 2002).

Calluses

A callus is larger, broader and has a less well-defined edge than a corn. These tend to form on the underside of the foot (the sole). They commonly form over the bony area just underneath the toes. This area takes much of your weight when you walk. They are usually painless but can become painful (Freeman, 2002).

Other

Hyperkeratosis lenticularis perstans (HLP)

Also known as Flegel disease, HLP is a cutaneous condition characterised by rough, yellow-brown keratotic, flat-topped papules of irregular outline measuring 1–5mm in diameter and up to 1mm in depth (Rapini et al, 2007). Lesions are located primarily on the dorsal feet and lower legs, with a decreasing likelihood of manifestation proximally. Most cases have been reported in Europe. No instigating factor has been identified clearly, however;

Continued on p70

Table 1 – Treatment of Hyperkeratosis

Type	Treatment	Side effects
<i>Epidermolytic hyperkeratosis</i>	<ul style="list-style-type: none"> ▶▶ Gene therapy is really the only true therapy for people with EHK. Until gene therapy solutions finally become a reality, EHK sufferers must treat their fragile skin carefully. Most have learned that taking regular, extended baths allows patients to care for their fragile skin and keep it manageable. Baths that include sea salt seem to improve the process of softening and removing the thickened skin (Brecher and Orlow, 2003) ▶▶ Oral retinoids – etretinate, acitretin, isotretinoin (Brecher and Orlow, 2003). Contraindicated in hyperlipidaemia, renal and hepatic impairment ▶▶ Acitretin 	<ul style="list-style-type: none"> ▶▶ Oral retinoid therapy frequently causes reversible changes in liver function and serum lipid ▶▶ Up to 75% of retinoid-treated patients experience dose-related hair loss, which is reversible upon discontinuation of therapy ▶▶ Acitretin can cause major foetal abnormalities and is therefore contraindicated in pregnant women ▶▶ Topical retinoids are also contraindicated in pregnancy. Local reactions include: burning, erythema, stinging, pruritus, dry/peeling skin (BMJ/RPS, 2012)
<i>Multiple minute digitate hyperkeratosis</i>	<ul style="list-style-type: none"> ▶▶ Topical keratolytics combined with topical emollients ▶▶ Tretinoin cream and oral vitamin A ▶▶ Topical 5-fluorouracil cream – apply thinly to the affected area once or twice per day (max area 500cm² for three to four weeks (BMJ/RPS, 2012). ▶▶ Surgical care - the lesions can be trimmed or clipped as needed ▶▶ Diet – gluten-free diet plus frequent follow up 	<ul style="list-style-type: none"> ▶▶ Topical keratolytics – can cause pain, itching, burning, irritation, inflammation, dryness, swelling and tenderness at site of application. This will heal once treatment is complete ▶▶ Tretinoin cream can cause stinging, burning, dry and peeling skin, sensitivity to UVB light. Eye irritation and oedema ▶▶ Topical 5-fluorouracil cream can cause local irritation and photosensitivity (BMJ/RPS, 2012)
<i>Focal acral hyperkeratosis</i>	<ul style="list-style-type: none"> ▶▶ Treatment is not indicated in most patients ▶▶ Mild keratolytics occasionally help, but recurrences are common ▶▶ Topical retinoids are not effective 	n/a
<i>Lamellar ichthyosis</i>	<ul style="list-style-type: none"> ▶▶ Moisturisers containing urea, ammonium lactate, or other alpha-hydroxy acids may help ▶▶ Retinoid medications, such as tazarotene, may be used on the skin (topically). Apply once daily to the affected areas in the evening, usually for 12 weeks. Not recommended for child under 18 years of age ▶▶ Gene therapy to correct the genetic defect may be possible in the future (Morelli, 2007) 	<ul style="list-style-type: none"> ▶▶ Close monitoring of fluids, electrolytes, signs of sepsis and placement in a high-humidity incubator for all newborns. ▶▶ Mild skin irritations, redness and flaking ▶▶ Topical tazarotene can cause local irritation, pruritus, burning, erythema, non-specific rash, dry or painful skin. (BMJ/RPS, 2012)
<i>Keratosis pilaris (follicular hyperkeratosis)</i>	<ul style="list-style-type: none"> ▶▶ Daily exfoliating and moisturising. These actions may make the appearance less visible ▶▶ Suggest spending some time with the affected area in sunlight for a few minutes each day to minimise the appearance 	

Table 1 (continued) – Treatment of hyperkeratosis

Type	Treatment	Side effect
<i>X-linked ichthyosis (XLI)</i>	<ul style="list-style-type: none"> ▶▶ There is no cure for ichthyosis. The main goal of treatment is to moisturise and exfoliate. This helps prevent dryness, scaling, cracking and build-up of skin ▶▶ Oral retinoids, such as acitretin or isotretinoin, can help to reduce scaling ▶▶ Topical isotretinoin – apply thinly once or twice daily ▶▶ Lanolin creams and products containing urea, lactic acid and other alpha hydroxyl acids may help to exfoliate and moisturise skin (BMJ/RPS, 2012) 	<ul style="list-style-type: none"> ▶▶ Oral isotretinoin — dry skin, epidermal fragility, dry lips/eyes/mouth, inflammatory bowel disease, depression, hypersensitivity, hypertension, blurred vision. Contraindicated in hypervitaminosis A, hyperlipidaemia, renal and hepatic impairment and pregnancy ▶▶ Topical isotretinoin — contraindicated in pregnancy. Women of childbearing years must use effective contraception. Local reactions include burning, erythema, stinging, pruritus. Eye irritation and oedema, blistering or crusting of skin have been reported (BMJ/RPS, 2012)
<i>Plantar hyperkeratosis</i>	<ul style="list-style-type: none"> ▶▶ Topical cream formulation containing 10% urea and 8% glycerine ▶▶ Palliative measures such as reduction of the horny accumulation and then padding of the area to distribute pressure give relief in most cases ▶▶ Intractable conditions over a non-weight bearing area respond to excision of the condylar prominence (Skinner and Fitzpatrick, 2008) 	<ul style="list-style-type: none"> ▶▶ The water-binding, keratolytic, exfoliative, and epidermal-thinning activities of urea and the skin softening and skin barrier repair properties of glycerine, combine to deliver significant relief (Loden, 2003)
<i>Hyperkeratosis of the nipple and areola</i>	<ul style="list-style-type: none"> ▶▶ The disease has a benign course and may only be a cosmetic problem. Treatment with topical retinoic acid can induce an acceptable response (Perez-Izquierdo et al, 1990) 	n/a
<i>Lichen planus</i>	<ul style="list-style-type: none"> ▶▶ No treatment is an option if symptoms are mild ▶▶ A steroid cream or ointment can reduce inflammation. Steroid pastes or mouthwashes may help to ease painful mouth ulcers ▶▶ A course of steroid tablets may be advised ▶▶ Ciclosporin and azathioprine are recommended if lichen planus is severe. These reduce inflammation. Potential serious side effects mean that they are not used routinely ▶▶ PUVA (psoralen and UVA) is a special light therapy that may be advised by a skin specialist if you have extensive and severe lichen planus ▶▶ Psoralen increases the skin's sensitivity to UV light and can be administered topically or orally 1.5–2 hours before exposure to ultraviolet light (BMJ/RPS, 2012) ▶▶ Antihistamine medicines may help to ease the itch ▶▶ Emollients are often also given to provide moisture to your skin. These can also help to reduce the itching 	<ul style="list-style-type: none"> ▶▶ Steroid tablets taken for longer than a few weeks are not usually advised due to possible side effects. Therefore, the rash may reappear after the tablets are stopped ▶▶ Oral ciclosporin — abnormal renal function, uncontrolled hypertension, infections not under control, malignancy, UVB sensitivity. Contra-indicated in hepatic and renal impairment/pregnancy ▶▶ Oral azathioprine — malaise, dizziness, vomiting, diarrhoea, fever, rigors, myalgia, hypotension and interstitial nephritis. Also bone marrow suppression, liver impairment, jaundice and hair loss

Table 1 (continued) – Treatment of hyperkeratosis

Type	Treatment	Side effect
Seborrheic keratosis	<ul style="list-style-type: none"> ▶▶ A variety of techniques may be used to treat seborrheic keratosis. They include cryotherapy with carbon dioxide (dry ice) or liquid nitrogen, electro-desiccation and curettage, curettage alone, shave biopsy or excision, or laser or dermabrasion surgery. However, some of these techniques destroy the lesion without providing a specimen for histopathologic diagnosis ▶▶ Ammonium lactate and alpha hydroxy acids have been reported to reduce the height of seborrheic keratosis. Superficial lesions can be treated under expert supervision by applying these treatments and repeating if the full thickness is not removed on the first treatment. There is some evidence that superficial peeling will hasten the transition of closed lesions to the surface of the epidermis resulting in a quicker clearance (Kempiak and Uebelhoer, 2008) ▶▶ Topical treatment with tazarotene applied once daily to the affected areas in the evening usually for 12 weeks ▶▶ Not recommended for children under 18 years of age 	<ul style="list-style-type: none"> ▶▶ Ammonium lactate — do not apply to the face unless advised by a doctor. Do not apply on sunburned, wind-burned, dry, cracked, irritated, or broken skin ▶▶ Avoid exposure to sunlight or artificial UV rays ▶▶ Contraindicated in pregnancy ▶▶ Alpha hydroxy acids — (as above)
Corns and calluses	<ul style="list-style-type: none"> ▶▶ Paring and trimming. The thickened skin of a corn or callus can be pared down (trimmed) by a podiatrist by using a scalpel blade ▶▶ Correcting poor footwear will reduce any rubbing or friction on your skin. In many cases, a corn or callus will go away if rubbing or pressure is prevented through improved footwear ▶▶ Depending on the site of a corn or callus, a cushioning pad or shoe insole may be of benefit ▶▶ Surgery is recommended for foot or toe abnormality causing recurring problems (Hogan et al, 2008) 	n/a
Hyperkeratosis lenticularis perstans	<ul style="list-style-type: none"> ▶▶ Topical 5% fluorouracil and a synthetic vitamin D-3 derivative over several months have been used together with effective results ▶▶ Local excision may be successful, especially if the number of lesions is small ▶▶ Dermabrasion is a possible surgical modality. However, a large number of lesions, as well as lesion location, make this an impractical approach ▶▶ Cryotherapy is an additional possibility however this causes pain, swelling and blistering ▶▶ Oral retinoids have been successful only with continuous therapy. Patients tend to relapse when therapy has ended (Metze et al, 2000) 	n/a

Table 1 (continued) – Treatment of hyperkeratosis

Type	Treatment	Side effect
Actinic keratosis	<ul style="list-style-type: none"> ▶ An emollient may be sufficient for mild lesions ▶ Diclofenac sodium 3% gel is suitable for the treatment of superficial lesions in mild disease. Apply thinly to affected areas for 60 to 90 days (BMJ/RPS, 2012) ▶ 5-fluorouracil cream is effective against most types of non-hypertrophic actinic keratosis. Apply thinly to the affected area once or twice daily for 3–4 weeks (BMJ/RPS, 2012) ▶ Imiquimod (immune response modifier) is used topically for lesions of the face and scalp when cryotherapy or other topical treatments cannot be used. Apply to lesions three times a week for four weeks, assess and repeat four-week course if lesions persist — maximum two-week course (BMJ/RPS, 2012) ▶ Cryosurgery with liquid nitrogen, by freezing off the actinic keratosis ▶ Photodynamic therapy is a new therapy involves injecting a chemical into the bloodstream, which makes actinic keratosis more sensitive to any form of light ▶ Laser therapy, notably CO² and Er:YAG lasers. A Laser resurfacing technique is often used with diffuse actinic keratosis ▶ Electrocautery — this involves burning off actinic keratosis with electricity ▶ Different forms of surgery (Quaedvlieg et al, 2006) 	<ul style="list-style-type: none"> ▶ Diclofenac sodium 3% gel — may cause gastrointestinal disturbances including nausea, diarrhoea and occasional bleeding and ulceration. Contraindicated in hepatic, liver, respiratory, renal and cardiac impairment ▶ 5-fluorouracil — local irritation (use a topical corticosteroid for severe discomfort associated with inflammatory reactions). Photosensitivity. Max area of skin to be treated at one time is 500cm² ▶ Imiquimod — avoid normal or broken skin and open wounds. Itching, burning sensation, erythema, erosion, oedema, excoriation and scabbing, headache and influenza type symptoms. Less commonly local ulceration and alopecia (BMJ/RPS, 2012)
Warts	<ul style="list-style-type: none"> ▶ No treatment may be required. Warts may regress on their own and treatment may only be required if they are painful, unsightly, persistent or cause distress. Treatment usually relies on tissue destruction ▶ Salicylic acid is suitable for treating warts of the hands and feet but not suitable for anogenital warts. Apply carefully to wart ensuring to protect the surrounding skin with soft paraffin or appropriate dressing. Rub wart gently with file or pumice stone once weekly. Treatment may need to continue for up to three months ▶ Cryotherapy causes pain, swelling and blistering and may be no more effective than topical salicylic acid 	<ul style="list-style-type: none"> ▶ Salicylic acid is not suitable for diabetic patients at risk of neuropathic ulcers. Avoid broken skin. Not suitable for application to face, anogenital region, or large areas and may cause skin irritation

some investigators have implicated ultraviolet light (Miljkovic, 2004).

HLP, described by Flegel in 1958, is considered to be an autosomal dominant inherited keratinisation disorder, although most cases are sporadic, affecting patients aged 40–50 years with no noted predominance in either sex. The condition has

been described in association with endocrine abnormalities, including diabetes and hyperthyroidism, while a possible relationship with digestive and cutaneous tumours is more open to debate. Many treatment options have been discussed, including topical and systemic retinoids, 5-fluorouracil, vitamin D derivatives, psoralen, UV-A therapy, excision and dermabrasion of

the lesions. At present, all of these are considered unsatisfactory due to high rates of recurrence (Metze et al, 2000).

Actinic keratoses

This is a premalignant condition of thick, scaly, or crusty patches of skin. It is more common in fair-skinned people. It is associated with those who are frequently exposed to the sun, as it is

usually accompanied by solar damage. Since some of these pre-cancers progress to squamous cell carcinoma, they should always be treated. When skin is exposed to the sun constantly, thick, scaly, or crusty bumps appear. The scaly or crusty part of the bump is dry and rough. The growths start out as flat scaly areas, and later grow into a tough, wart-like area.

An actinic keratosis site commonly ranges between 2–6mm in size, and can be dark or light, tan, pink, red, a combination of all of these, or have the same pigment as the surrounding skin. It may appear on any sun-exposed area, such as the face, ears, neck, scalp, chest, backs of hands, forearms or lips (Quaedvlieg et al, 2006).

Warts

These are small rough lumps on the skin. They are caused by a virus (human papillomavirus HPV) that causes a reaction in the skin. Warts can occur anywhere on the body, but are found most commonly on hands and feet.

Verrucas

Verrucas are warts that occur on the soles of the feet. They are the same as warts on any other part of the body. However, they may look flatter, as they tend to get trodden in (note: anal and genital warts are different).

General Complications

The most common complication encountered in all types of hyperkeratosis is a foul smelling odour, produced by bacteria, when the macerated scales become infected. Without intervention, heavy bacterial colonisation may result in sepsis at any age.

Epidermolytic hyperkeratosis is generalised redness, with thick, generally dark, scales that tend to form parallel rows of spines or ridges, especially near large joints. The skin is fragile and blisters easily following trauma, however, the extent of blistering and amount of scale is variable. Neonatal patients may need to be transferred to the neonatal ICU for monitoring. Widespread areas

of denuded skin can lead to infection, sepsis and electrolyte imbalance and intravenous fluids or antibiotics will be necessary. Other complications include a variety of benign and malignant skin lesions, including melanocytic lesions (Hutcheson et al, 2004).

Although some authors have proposed a relationship between MMDH and malignancy and/or inflammatory disorders, this contention was based on a broad definition of MMDH that included disorders that are now recognised as distinct from MMDH. However, adopting the more stringent criteria proposed by Caccetta et al (2010), their findings did not find evidence for associating MMDH with malignancy or systemic disease. That stated, malignancy screening could still be considered (Caccetta et al, 2010).

Babies with lamellar ichthyosis are at risk of infection when the collodian membrane is shed. Later in life, eye problems may occur because the eyes cannot close completely. Pre-natal morbidity due to sepsis is a concern, therefore, an intensive care environment should be considered for affected children. Widespread areas of denuded skin may also lead to fluid and electrolyte imbalances, especially in infants (Di Giovanna and Robinson-Bostom, 2003).

In the case of XLI, aside from the skin scaling, it is not typically associated with other major medical problems. Corneal opacities may be present but do not affect vision. Cryptorchidism is reported in some individuals (DiGiovanna and Robinson-Bostom, 2003). Mental retardation can also be seen in some affected individuals, and is thought to be due to deletions encompassing neighboring genes in addition to STS (Van Esch et al, 2005).

Larger deletions that include the SHOX gene can result in short stature. Female carriers generally do not experience any of these problems, but can sometimes have difficulty during

childbirth, as the STS expressed in the placenta plays a role in normal labour. For this reason, carriers should ensure their obstetrician is aware of the condition (Morelli, 2007).

Untreated lesions in actinic keratosis have up to 20% risk of progression to squamous cell carcinoma and should be treated immediately. Preventive measures recommended are similar to those for skin cancer (Quaedvlieg et al, 2006).

With plantar hyperkeratosis, calluses are basically caused by shoes with pointed toes, short toes, or both. If untreated these can force the toes to buckle and thus produce a 'hammer toe' deformity at the joints (Skinner and Fitzpatrick, 2008).

After the rash has cleared up, a change in skin colour may occur (a brown or grey mark), which can sometimes last for months. This is known as post-inflammatory hyperpigmentation, and tends to be more noticeable in people with darker skin. Additionally, there is some evidence that lichen planus may increase the risk of certain cancers, such as squamous cell carcinoma, and oral, penile and vulvovaginal cancer.

Lesions affecting the vulva and vagina often do not respond well to treatment and are difficult to manage. Therefore, the condition can result in significant sores or permanent changes to vulvovaginal tissues that at times may scar. Because severe itching, pain and burning sensations are common, the condition can result in subsequent sexual dysfunction (Lehman et al, 2009).

Complications of seborrheic keratosis include, skin abscesses, cellulitis, impetigo and scarring. Malignant melanoma associated with seborrheic keratosis has rarely been reported in the literature, with disagreement regarding whether it is coincidental or whether malignant transformation occurs. Because seborrheic keratoses are common and association with malignant melanoma

is very rare, many experts conclude that the association is coincidental. However, because of the association of other malignancies, a biopsy of any suspect or changing seborrheic keratosis is essential (Zabel et al, 2000).

Corns and callus that are not treated will become painful. They will not heal independently unless the associated pressure is removed. If it is not, then the skin will continue to thicken and become more painful. After a while, the body will start treating it as a foreign body and an ulcer (abscess) can develop. This can get infected and the infection can spread. Instances of infections of corns on the toe are more common than calluses. This can be a serious complication for those with poor circulation, peripheral neuropathy and those requiring diabetic foot care (Freeman, 2002).

Conclusion

Although the symptoms of all types of hyperkeratosis can be difficult and uncomfortable, the disease can be successfully managed. Hyperkeratotic skin disorders are characterised by red, dry cracked and scaling skin affecting 10–20% of the Western population. While the rate of incidence of hyperkeratotic condition increases with age, different types affect persons of all ages, gender and racial/ethnic groups.

A variety of treatment options exists for hyperkeratosis, including keratolytics, moisturisers and corticosteroids that promote skin hydration and promote an increased lipid content in the stratum corneum. Emollients and moisturisers are frequently used to disrupt the cycle of skin dryness and other skin barrier disorders.

Due to the visual similarities in some these conditions, it is vital to ensure that the correct diagnosis is confirmed prior to initiating treatment. A multidisciplinary approach is key to early detection, correct diagnosis

and management of these patients. A clinician usually reaches the diagnosis of hyperkeratosis using a detailed history and embarking on a thorough skin inspection.

Laboratory investigations are often needed in order to arrive at a correct diagnosis, as many of these conditions share clinical similarities. Skin biopsy specimens from involved areas may be required for histopathological purposes. The exact mode of treatment depends on the type of hyperkeratosis.

The prognosis is good for most types of hyperkeratosis, however, actinic keratosis and, very rarely, seborrheic keratosis, can develop into skin cancers. As researchers learn more about the condition and what causes it, they continue to move closer to effective treatments, and perhaps, ultimately, a cure. **WE**

References

Brecher AR, Orlow SJ (2003) Oral retinoid therapy for dermatologic conditions in children and adolescents. *J Am Acad Dermatol* 49(2): 171–82

BMJ/RPS (2012) *British National Formulary* 63. March, BMJ Group Macmillan, London

Caccetta T, Dessauvagie B, McCallum D, Kumarasinghe S (2010) Multiple minute digitate hyperkeratosis: A proposed algorithm for the digitate keratoses. *J Am Acad Dermatol* Nov: 1 [Epub ahead of print]

Gelmetti C, Caputo R (2002) *Pediatric Dermatology and Dermatopathology: A Concise Atlas*. Informa Healthcare, London

Connetics Corporation (2004). Soriatane (acitretin) capsules. Complete product information. Available at: <http://www.connetics.com>, <http://www.prnewswire.com> (accessed 20 June, 2012)

Di Giovanna JJ, Robinson-Bostom L (2003) Ichthyosis: etiology, diagnosis, and management. *Am J Clin Dermatol* 4(2): 81–95

Flegel H (1958) Hyperkeratosis lenticularis perstans. *Hautarzt* 9: 362–64

Freedberg IM, Eisen AZ, Wolff K, Austen KF et al (2003) *Fitzpatrick's Dermatology in General Medicine*. (6th ed). McGraw-Hill, Columbus, USA

Freeman DB (2002) Corns and calluses resulting from mechanical hyperkeratosis. *Am Fam Physician* 1: 65(11): 2277–80

Goldstein N (1976). Multiple Minute Digitate Hyperkeratoses. *Arch Dermatol* 96(6): 692–3

Hogan DJ, Basile AL. *Corns*. eMedicine. January 2008

Hutcheson AC, Nietert PJ, Maize JC (2004) Incidental epidermolytic hyperkeratosis and focal acantholytic dyskeratosis in common acquired melanocytic nevi and atypical melanocytic lesions. *J Am Acad Dermatol* 50(3): 388–90

Kempiak S, Uebelhoer N (2008) Superficial chemical peels and microdermabrasion for acne vulgaris. *Seminars in Cutaneous Medicine and Surgery*. 27(3): 212–20

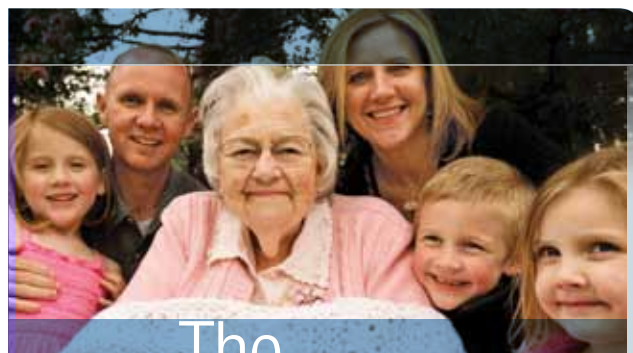
Kumar V, Fausto N, Abbas A (2004) *Robbins & Cotran Pathologic Basis of Disease* (7th ed). Saunders, US: 1230

Lehman JS, Tollefson MM, Gibson LE (2009) Lichen planus. *Int J Dermatol* 48(7): 682–94

Loden M (2003) Role of topical emollients and moisturizers in the treatment of dry skin barrier disorders. *Am J Clin Dermatol* 4(11): 771–88

McClure SL, Valentine J, Gordon KB (2002) Comparative tolerability of systemic treatments for plaque-type psoriasis. *Drug Saf* 25(13): 913–27

3M™ Cavilon™ Skin Care Products



The Protective Instinct In wound care

When skin is vulnerable, it needs protection.



3M™ Cavilon™ No Sting Barrier Film provides a barrier to protect peri-wound areas from maceration and further breakdown from exposure to wound exudate. It will also protect skin from adhesive damage when removing wound dressings and tapes.



3M™ Cavilon™ Durable Barrier Cream provides an effective moisturising barrier for intact skin at risk from breakdown. It is associated with a significant decrease in grade 1 pressure ulcers when used as part of a sacral care protocol.¹



3M™ Cavilon™ Durable Barrier Cream + Honey provides a barrier and moisturiser suitable for lymphoedema, dermatology, leg ulcer or other patient groups with potential sensitivities. Honey acts as a natural preservative replacing parabens and perfumes.

If you would like more information about Cavilon skin care products, to receive product samples or to arrange a representative visit, call **0800 616066** (answer phone) or visit **www.cavilon.co.uk**.

1. Bale, S. The benefits of implementing a new skin care protocol in nursing homes. *Journal of Tissue Viability*, Vol 14, No. 2. April 2004.

Metze D, Lübke D, Luger T (2000). [Hyperkeratosis lenticularis perstans (Flegel's disease) — a complex disorder of epidermal differentiation with good response to a synthetic vitamin D3 derivate]. *Hautarzt* 51(1): 31–5

Miljkovic J (2004) An unusual generalized form of hyperkeratosis lenticularis perstans (Flegel's disease). *Wien Klin Wochenschr* 116 Suppl 2: 78–80

Morelli JG (2007). Disorders of keratinization. In: Kliegman RM, Behrman RE, Jenson HB, Stanton BF, eds. *Nelson Textbook of Pediatrics* (18th ed). Philadelphia, Pa: Saunders Elsevier; chapter 657

Perez-Izquierdo JM, Vilata JJ, Sanchez JL, et al (1990) Retinoic acid treatment of nipple hyperkeratosis. *Arch Dermatol* 126: 687–88

Nadiger, HA (1980) Role of vitamin E in the aetiology of phrynoderma (follicular hyperkeratosis) and its interrelationship with B-complex vitamins. *Br J Nut* 44(3): 211–14

Quaedvlieg PJ, Tirsi E, Thissen MR, Krekels GA (2006) Actinic keratosis: how to differentiate the good from the bad ones? *Eur J Dermatol* 16(4): 335–39

Rapini RP, Bologna JL, Jorizzo JL (2007) *Dermatology: 2-Volume Set*. Mosby, St Louis

Skinner H, Fitzpatrick M (2008) *Current Essentials: Orthopedics*. McGraw-Hill Medical, USA

Van Esch H, Hollanders K, Badisco L, Melotte C, et al (2005) Deletion of VCX-A due to NAHR plays a major role in the occurrence of mental retardation in patients with X-linked ichthyosis. *Hum Mol Genet* 14 (13)

Zabel RJ, Vinson RP, McCollough ML (2000) Malignant melanoma arising in a seborrheic keratosis. *J Am Acad Dermatol* 42(5): 831–33