Overview of pigmentary disorders

Disorders of pigmentation are a common presentation in dermatology and general practice. The aesthetic appearance of either hyperpigmentation or hypopigmentation, especially in the visible parts of the skin, can have major psychosocial implications. Although it is more of a cosmetic problem, a disorder of pigmentation can occasionally be associated with an underlying systemic disorder. It is therefore important to recognise the causes of pigmentary disorders to achieve the best treatment outcome.

This article discusses pigmentary disorders in general including the underlying pathological mechanisms and management. It then looks at individual hyperpigmentary and hypopigmentary conditions in more detail.

The single most important substance determining human skin colour is the pigment melanin. Other compounds that may contribute to the skin colour include carotenoids and haemoglobin.

Melanin is produced by melanocytes during melanogenesis. This occurs within melanosomes, which are small membrane-bound packages. When melanosomes become full of melanin, they move along the arms of melanocytes and are transferred to the keratinocytes. One melanocyte supplies melanin to about 36 keratinocytes. People have the same number of melanocytes but their skin colour is determined by the amount and types of melanin the melanocytes produce. Melanocytes are found in the skin or epidermis, hair follicles, eye, nervous system, leptomeninges and the inner ear. Melanocytes in the skin and other body sites have a common embryological origin from the neural crest. They migrate from here into these different body sites.

The production of melanin is mainly regulated by the enzyme tyrosinase. Melanocytes produce two types of melanin that give the skin its colour. The most common form of melanin is eumelanin, which gives a brown-black colour and is more abundant in people with dark skin. Pheomelanin gives a red-yellow colour to the skin. Both the amount and type of melanin produced is controlled by several genes, such as the genes for the MSH cell surface receptor and the melanosomal P-protein. These genes can regulate the variability of the skin colour in humans by mainly regulating the level and activities of enzymes involved in the melanin biosynthesis, mostly the enzyme tyrosinase.

Disorders of pigmentation — Part 1: Hyperpigmentation

This is the first of a two-part series on disorders of pigmentation. In Part 1, we discuss the nature of pigmentary disorders and then focus on hyperpigmentary disorders. In Part 2, we cover hypopigmentary disorders.
How To Treat – Disorders of pigmentation — Part 1: Hyperpigmentation

**Pathogenesis**

Disorders of pigmentation can be due to a variety of genetic and acquired causes. This is because there are many genes involved in the different stages of melanin production and transport, abnormalities may arise from any of these stages. In piebaldism, for example, melanocyte migration from the neural crest is defective. Other abnormalities may occur in the formation of melanocytes and melanosomes, or during the secretion of melanosomes to keratinocytes.

Certain pigmentary disorders such as Waardenburg syndrome may also be associated with visual and neurological defects, owing to the defects’ common embryological origin and migration from the neural crest. Acquired conditions of pigment loss can arise from melanocyte destruction. This may be due to an underlying autoimmune disorder such as vitiligo, or inflammato-

dory disorders, or be secondary to chemicals or drugs. Increased pigmentation similarly can be induced by many factors such as UV radiation, drugs, inflammation and hormonal factors. Abnormalities in the other pigment compounds such as carotenoids and haemoglobin may also result in certain pigmentary disorders.

**Diagnosis**

Many pigmentary disorders can be diagnosed clinically. A detailed history and examination is important and often gives clues to the underlying cause. A lesion presenting from birth may be a naevus or a genetic disorder. A family history of a similar disease also favours a genetic disorder. Diffuse types of pigmentary changes may be due to metabolic, hormonal or nutritional causes. A preceding rash or injury to the skin suggests post-inflammatory hyper- or hypopigmentation.

A detailed drug history may help to determine if the hyperpigmentation is caused by a photosensitive or allergic reaction, or from fixed drug eruptions.

**Dermatoscope and Wood’s lamp**

The dermatoscope and Wood’s lamp can assist the clinician in diagnosing certain diseases. Table 1 shows clinical features that may be detected under a Wood’s lamp in a variety of pigmentary disorders. Dermatoscopy is a very useful tool in differentiating a benign melanocytic naevus, pigmented seborrhoeic wart from a malignant melanoma. Histological diagnosis will be required in some cases.

**General principles of management**

While mostly benign, both hyperpigmented and hypopigmented lesions, such as vitiligo or facial melasma, can profoundly affect a patient’s emotional and psychological wellbeing and self-esteem.

**Patterns of hyperpigmentary disorders**

**DISORDERS of hyperpigmentation**

may be localised (circumscribed) or diffuse (table 2). Certain conditions such as polikoidermatosus disorders cause reticulate and mottled type of hyperpigmentation (figure 1). Because of their cosmetic significance, facial and periorbital pigmentation need special attention. There is no set algorithm or specific way to approach a patient with a hyperpigmented lesion. However, figure 2 will help the clinician in approaching a patient.

**Table 1: Wood’s lamp features in pigmentary disorders**

<table>
<thead>
<tr>
<th>Condition</th>
<th>Features</th>
</tr>
</thead>
<tbody>
<tr>
<td>Phrygian versicolor</td>
<td>Yellowish-white or copper-orange fluorescence</td>
</tr>
<tr>
<td>Vitiligo</td>
<td>Bright areas with sharp borders, varying in location and extent</td>
</tr>
<tr>
<td>Tuberculosis</td>
<td>Patches; characteristic ash-leaf shape</td>
</tr>
<tr>
<td>Hypermelanosis of its</td>
<td>Whorled or streaked patterns.</td>
</tr>
<tr>
<td>Enhydraha</td>
<td>Shows corral red fluorescence</td>
</tr>
<tr>
<td>Melasma</td>
<td>Pigments in the outer epidermal layer of the skin are accentuated while the colour of the deeper dermal pigments is decreased</td>
</tr>
</tbody>
</table>

**Table 2: Patterns of hyperpigmentary disorders**

<table>
<thead>
<tr>
<th>Circumscribed</th>
<th>Freckles, lentigines, melanocytic naevi, melasma, achromatosis nigricans, post-inflammatory, fixed drug eruption, café-au-lait macules, mastocytosis, phytocirosis versicolor</th>
</tr>
</thead>
<tbody>
<tr>
<td>Diffuse</td>
<td>Drug-related, endocrinopathies, haemochromatosis, HIV/</td>
</tr>
<tr>
<td>Reticulate and mottled</td>
<td>Polikoidermas of Civatte, erythema ab igne, genetic reticulate pigmentary disorders, polikoidermatosus disorders (eg, mycosis fungoides and dermatomyositis), confluent and reticulate papilomatosis, associated with host disease, post-kala azar dermal leishmaniasis, systemic sclerosis</td>
</tr>
<tr>
<td>Blaschiod</td>
<td>Pigmentary mosaicism, incontinencia pigmentosa</td>
</tr>
</tbody>
</table>

**Freckles**

FRECKLES are small flat brown circular spots arising on the face and other sun-exposed areas. They usually fade in winter with less sun exposure but are often seen in fair-skinned, red-haired people. Genetic tendency and sun exposure can predispose to the development of freckles. Freckles represent an increase in the amount of melanin, not an increase in the total number of melanocytes. Increased tendency to develop freckles is seen in genetic disorders such as xeroderma pigmentosum. Axillary freckles are a feature of neurofibromatosis (table 3).

While freckles are a harmless condition, some patients may be distressed by their appearance and seek treatment. Sun protection is essential in managing this condition if treatment is requested. There are several effective and safe methods that are available to help lighten or reduce the appearance of freckles. Treatment options include bleaching or fading creams (table 4), light application of liquid nitrogen, laser treatment and chemical peels. Frequently, multiple or a combination of treatment methods may be required for the best results. Preventive photoprotection starting in early childhood is important because freckles can easily recur with repeated UV exposure.

<table>
<thead>
<tr>
<th>Lentigines</th>
<th>Lentigines are small, brown to black-coloured macular melanoses. They have several points of difference to freckles: there is an increased number of melanocytes in lentigines, lentigines are darker, persist throughout the year, and do not darken on exposure to sunlight. There are several types of lentigines, as listed in the box below. Multiple lentigines can occur in association with an underlying genodermatosis (table 3).</th>
</tr>
</thead>
<tbody>
<tr>
<td>Café au lait macules</td>
<td>Café au lait spots are macules varying from light brown to dark brown with smooth or irregular borders. While having a few small</td>
</tr>
</tbody>
</table>
Melasma presents as bilateral symmetric hyperpigmented macules commonly in the cheeks, the upper lip, the chin and the forehead (figure 4). The most significant cause is the combination of topical steroids and sunscreens. Patients with darker skin types may worsen the hyperpigmentation, therefore sun protection is necessary to minimise complications. Oral contraceptives are associated with melasma and should always be combined with adequate sun protection. As shown in table 5, epidermal melasma has well-defined borders and is dark brown in colour. Dermal melasma has ill-defined borders and is light-brown to black in colour. In table 6, the types of lentigines are listed. The complications include transient hyperpigmentation and hypopigmentation, slight scarring, permanent hyperpigmentation and recurrence.

**Post-inflammatory hyperpigmentation**

Post-inflammatory hyperpigmentation is a frequent complication following various cutaneous disorders. These cutaneous disorders may be infections (e.g., tinea), dermatitis, drug reactions and inflammatory skin disorders (e.g., lichen planus). Epidermal post-inflammatory hyperpigmentation appears light-brown to black whereas deeper dermal pigmentation has a grey to bluish appearance. A variety of topical agents have been used to treat epidermal post-inflammatory hyperpigmentation, with varying degrees of success. These agents include hydroquinone, tretinoin cream, corticosteroids, glycolic acid (GA) and azelaic acid (table 4). A combination of topical creams and gels, chemical peels and sunscreens may be necessary and is only effective for epidermal hyperpigmentation. Laser treatment may be able to address dermal pigment deposition. Broad-spectrum sunscreens should be combined with any treatment regimen.

Melasma can be epidermal, dermal or mixed. Epidermal melasma has well-defined borders and is dark brown in colour. Dermal melasma is the common type with ill-defined borders and is bluish in colour. The mixed type has a combination of clinical features (see table 5). Melasma can be difficult to treat and can be recurrent. There are several treatment modalities; these should always be combined with adequate sun protection. As shown in table 5, epidermal melasma has a good response to treatment while dermal melasma has a poor response.

Topical hydroquinone 2.4% cream twice a day with a sunscreen remains the first-line treatment. There are several other topical medications used in melasma that are listed in table 4. A combination of topical treatments (‘Triple Combination Cream’) with hydroquinone 4%, tretinoin 0.05% and fluorocine acetone 0.01% has been shown to work faster. Superficial chemical peels with glycolic acid, trichloroacetic acid (TCA), tretinoin or salicylic acid are being used to treat melasma in resistant cases. Three steps are involved in treating with chemical peels: careful patient selection, proper priming and post-procedural care (see box, next page). Lasers that selectively target the hyperpigmentation in the correct depth can be used in resistant cases but with caution. Highly pigment-selective short pulsed Q-switched lasers that target the melanosomes are used in the treatment of both dermal and epidermal pigmentation. As they are selective of the target, there is minimal damage to the surrounding tissue. Repeated treatments are necessary to achieve a desirable outcome.

Fractional laser is a non-invasive treatment that uses a device to deliver a laser beam divided into thousands of microscopic treatment zones that target a fraction of the skin at a time. This laser treatment works at both the epidermal and dermal layers of the skin. Fractional lasers are FDA approved and have made lasers safer, especially for darker-skinned patients. Hyperpigmentation, hypopigmentation and scarring are potential complications. Patients with darker skin should be treated with caution as even slight inflammation may worsen the hyperpigmentation. Prior treatment with topical bleaching creams and post-procedure sun protection is necessary to minimise complications. Oral tranexamic acid may be effective for some patients, but further studies are needed.
How To Treat – Disorders of pigmentation — Part 1: Hyperpigmentation

from page 31

Ashy dermatosis (erythema dyschromicum perstans)

Erythema dyschromicum perstans, also called lichen planus pigmentosus, causes ash discoloration of skin and is therefore also known as ‘ashy dermatosis’. Ashy dermatosis is an asymptomatic eruption of oval, polycyclic, or irregularly shaped, grey-blue hyperpigmented macules on the trunk, the arms, the face, and the neck (figure 5). There are no systemic symptoms or associations. There are close similarities with lichen planus although the exact relationship is not clear. The disease is more common in Asian and Latin American people.

Different treatments have been tried but ashy dermatosis is usually resistant to treatment. Clofazamine has shown satisfactory results in some patients. Other treatments such as dapsone, potent steroids and bleaching creams have not shown satisfactory results.

Acanthosis nigricans

Acanthosis nigricans causes increased pigmentation with a soft velvety appearance on the sides of the neck, in the axillae and groin. There is also often an increased pigmentation of mucosal surfaces.

The mechanism responsible for the pigmentation is unknown, but increased melanocytes-stimulating hormone activity is suspected. There are several types of acanthosis nigricans (see box, right).

The aim of treatment is to correct the underlying disease. This may involve multidisciplinary evaluation.

Treatment of the lesions of acanthosis nigricans is for cosmetic reasons only. Topical medications that have been effective include keratolytics (eg, topical tretinoin 0.05%, ammonium lactate 12% cream, or a combination of the two) and triple-combination depigmenting cream (triazine 0.05%, hydroquinone 4%, fluorocaine acetate 0.01%) nightly with daily sunscreen.

Erythrasma

Erythrasma is a chronic superficial infection of the intertriginous areas of the skin caused by Corynebacterium minutissimum. The typical appearance of erythrasma is well-demarcated, uniform, brown-red macular patches located over the inner thighs, crural region, scrotum and toe webs (figure 6). The axillae, submammary area, periumbilical region and intergluteal fold are less commonly involved in erythrasma.

Excessive sweating/hyperhidrosis, obesity, diabetes mellitus, warm climate and poor hygiene can predispose to the disease. Erythrasma can be confused with acanthosis nigricans (which has a velvety texture and ill defined borders), candidiasis (which shows more erythema and fissuring at skin creases and shows satellite papules around the lesion), psoriasis (which has more pinkish well-defined lesions), and tinea cruris (which shows an active scaly edge). Wood’s lamp examination of erythrasma lesions reveals coloured fluorescence of lesions and is a helpful bedside test.

Oral erythromycin 250mg four times daily for two weeks is the treatment of choice. Topical fusidic acid and miconazole twice daily for 2-4 weeks are also helpful.

Table 5: Melasma categorised by depth of pigmentation and ease of treatment

<table>
<thead>
<tr>
<th>Melasma type by depth of pigmentation</th>
<th>Description</th>
<th>Relative response to treatment</th>
</tr>
</thead>
<tbody>
<tr>
<td>Epidermal</td>
<td>Well-defined borders</td>
<td>Good</td>
</tr>
<tr>
<td></td>
<td>Dark brown in colour</td>
<td></td>
</tr>
<tr>
<td></td>
<td>Becomes prominent with Wood’s lamp examination</td>
<td></td>
</tr>
<tr>
<td>Dermal</td>
<td>Bi-defined borders</td>
<td>Poor</td>
</tr>
<tr>
<td></td>
<td>Blush in colour</td>
<td></td>
</tr>
<tr>
<td></td>
<td>Become less obvious with the Wood’s lamp examination</td>
<td></td>
</tr>
<tr>
<td>Mixed</td>
<td>Combined features</td>
<td>Partial</td>
</tr>
</tbody>
</table>

Figure 5: Ashy dermatosis causes hyperpigmentation on the face of a middle-aged woman.

Figure 6: Hyperpigmentation due to erythrasma in the right armpit.

Facial hyperpigmentation

FACIAL hyperpigmentation is one of the most common cosmetic complaints and requires special consideration. The presentation may be either diffuse or patchy. The causes may be multifactorial (see box, right), including genetic factors, sun-light, cosmetics and hormonal factors.

Periorbital hyperpigmentation

Dark circles around the eyes or periorbital hyperpigmentation can cause much psychological distress to the patient. Even though the condition is usually benign, it can be very resistant to treatment. It is a common condition and often familial. There are several multiple cause factors involved, including epidermal hypermelanosis, dermal melanosis, increased vascularity, and normal anatomic variants.

The vascular type can present with erythema predominantly involving the inner aspect of the lower eyelids, with prominent capillaries or telangiectasia. Blush discolouration of the lower eyelid can be seen as a result of visible blue veins. Constitu- tional type can present with a curved band of brownish to black hyperpigmentation with a velvety texture and often involving the upper eyelids (figure 8). Post-inflammatory hyperpigmentation may be secondary to contact or atopic dermatitis. Shadow effects due to an overhang of the tear trough can cause dark eye circles more commonly seen with ageing. Dry skin, hormonal disturbances, nutritional deficiencies and other chronic illnesses can also contribute.

Treatments that have been tried include skin-lightening creams, chemical peels, intense pulsed light, Q-switched ruby laser, antologous fat transplantation, combinations of fat grafting and blepharoplasty, and fillers. However, none have provided a satisfactory treatment.

Naevus of Ota

Naevus of Ota is a hamartoma of dermal melanocytes presenting as a blue or grey patch on the face, within the distribution of the ophthal-mic and maxillary branches of the trigeminal nerve. The naevus can be unilateral or bilateral, and, in addition to skin, it may involve ocular and oral mucosal surfaces (figure 9).

Cosmetic camouflage makeup can minimise the disfiguring facial pigmentation. Other topical therapy is of no value. Pulsed Q-switched laser, Q-switched ruby, Q-switched alexandrite, and Q-switched NdYAG
lasers have shown good results. Naevus of Ota can be associated with increased intracocular pressure and ophthalmic referral is necessary.

Hor’s naevus
Hor’s naevus is an acquired bilateral naevus of Ota-like multiple brown-grey to blue-brown macules, in the malar region of the face. It is differentiated from the naevus of Ota by the later age of onset and lack of conjunctival, mucosal and sympathetic membrane involvement. Lasers are the most commonly used treatment for this condition and achieve good results.

Riehl’s melanosis (melanoderma toxicum)
Riehl’s melanosis is caused by frequent and repeated contact with small amounts of sensitising allergens in cosmetics at a concentration that is too low to produce typical eczematous dermatitis. However, the repeated contact over time and repeated sun exposure causes cumulative allergic contact dermatitis that results in basal cell damage and pigment incontinence. Many cases are preceded by mild erythema, oedema and pruritus, followed by a diffuse to reticulated pattern of hyperpigmentation.

Photo-patch testing
Patch testing and photo-patch testing will help in making a diagnosis. A photo-patch test is similar to a skin patch test except that the allergens are applied on the skin in duplicate. One set is exposed to a small dose of ultraviolet radiation (UVA) to detect a photosensitivity reaction. Standard patch test series, cosmetic series, fragrance series, and patient’s personal products can be used depending on the suspected allergens.

Other management measures
Complete avoidance of the suspected allergen is necessary, leading to a gradual improvement. Topical creams containing 2-4% hydroquinone twice daily for 4-8 weeks combined with tretinoin may hasten the resolution of the hyperpigmentation.

Poikiloderma of Civatte
Poikiloderma of Civatte is a reddish-brown, reticulate pigmentation with atrophy of the skin and telangiectasia that occurs on the lateral cheeks and sides of the neck. It is seen more commonly in middle-aged women. Photosensitising chemicals in perfumes or cosmetics together with chronic sun exposure have been implicated in the pathogenesis of poikiloderma of Civatte. Hormonal changes related to menopause or low oestrogen levels may also be a causative factor.

There is no specific medical treatment for poikiloderma of Civatte. Educating the patient about avoiding sun exposure and the proper use of sunscreens is most important.

Berloque dermatitis
Berloque dermatitis has drop-like or streak-like hyperpigmentation on the face or nose that arises from the application of perfumes and subsequently being exposed to the sun. The principal management is discontinuation of the offending substance and/or limiting the use the perfumes to covered areas. A short course of topical corticosteroids may help if there is inflammation and discomfort.

Diffuse or systemic causes of hyperpigmentation

**DIFFUSE** hyperpigmentation may be due to an underlying systemic disease (eg, Addison’s disease, hyperthyroidism, haemochromatosis) or it may be a side effect of medication use.

**Addison’s disease**
In Addison’s disease, hyperpigmentation occurs over the entire body with accentuation in old scars and in skin creases. The nail beds and the oral mucosa may also become hyperpigmented. The hyperpigmentation is caused by an increased activity of the melanocyte-stimulating hormone and adrenocorticotrophic hormone, both of which are capable of stimulating pigment production. Vitiligo, which is also an autoimmune disorder, can be associated in patients with Addison’s disease as part of a multisystemal deficiency syndrome.

**Other systemic conditions**
Hyperthyroidism causes a pattern of hyperpigmentation similar to that in Addison’s disease, especially in patients with darker complexion. Haemochromatosis, a disorder of iron storage and deposition, can cause a slate-grey hyperpigmentation owing to the deposition of haemosiderin. In scleroderma, hyperpigmentation is generalised, but there is accentuation of the brown colour on the dorsal surface of the arms and hands. Occasionally, vitiligo-like hypopigmentation will be interspersed within areas of darkened skin. Hyperpigmentation associated with malignancies is most commonly found with carcinoma of the lung. The pigmentation occurs because of the melanocyte-stimulating hormone-like activity of polyphenol substances produced by such tumours. Generalised melanosis may also be seen with advanced, widespread melanoma, in which case the colour is due to the pigmented compounds produced directly by the malignant cells.

Reticulate and mottled hyperpigimentary disorders

**RETICULATE** pigmentary disorders cause net-like patterns of cutaneous hyperpigmentation that are seen in both congenital and acquired conditions (see tables 2 and 3). A formal diagnosis of any underlying disorder should be made because it may be treatable and/or associated with malignant and systemic diseases.

**Erythema ab igne**
Erythema ab igne is characterised by localised areas of reticulate erythema and hyperpigmentation that result from chronic heat exposure (eg, heating pads, heating blanket, laptop computer). It is typically seen in middle-aged or elderly patients, and is more common in women.

There may be an initial erythema which then, with repeated exposure to heat, evolves into a non-blanching dusky hyperpigmentation with epidermal atrophy. Hyperkeratosis and bullae may be seen in the late stage. Squamous cell carcinoma may develop as a possible long-term consequence. **Confluent and reticulate papillomatosis** The lesions of confluent and reticulate papillomatosis are red, verrucous, minimally scaly papules, which coalesce to form brown plaques (figure 10). It usually develops between the breasts and in the midline of the back, and gradually spreads over the breasts and abdomen. With progression, the lesions acquire the characteristic reticulate appearance. This condition is commonly seen in young females.

The treatment of choice for confluent and reticulate papillomatosis is minocycline 100-200mg per day for weeks to months. Other antibiotics that have been tried include clarithromycin, erythromycin and azithromycin. Isotretinoin, tacalcitol and calcipotriol are useful topical treatments.

**Prurigo pigmentosa**
Prurigo pigmentosa is a rare inflammatory skin disease characterised by pruritic, erythematous urticarial papules that are symmetrically localised on the trunk. The lesions resolve, leaving reticulate pigmentation. Lesions in different stages are usually present together at any one time; the condition has a waxing and waning course. Several causative factors have been implicated such as ethnic predisposition, environmental causes, seasonal variation, mechanical stimuli and contact allergens. Different medications have been tried with variable results. Antibiotics and steroids have been found to be ineffective while minocycline, tetracycline and doxycycline have shown promising results.

Medication-induced hyperpigmentation

**MANY drugs are known to cause diffuse or localised hyperpigmentation.** These are summarised in table 6, see next page.

**Drug-induced pigmentation**
The following features point to a drug-induced pigmentation:

- The increased pigmentation starts gradually after initiation and fades when drug is discontinued.
- The hyperpigmentation shows photo-aggravation.
- Involvement of cartilage, mucosa, nail and scar.

**Fixed drug eruptions**
These are eruptions at sites following re-exposure to the same drug. Local symptoms may include pruritus, burning, and pain. The initial eruption is frequently located on the lip or genitalia.

The common medications that may cause fixed drug eruptions are analgesics, muscle relaxants, sedatives, anticonvulsants and antibiotics. Patients should be counselled on medication avoidance and possible cross-reactions to similar medications.
Conclusion

PIGMENTATION disorders are usually considered to be cosmetic in nature. However, it is important to recognise that they can cause psychological distress to the patient. It is also important to remember that some hyperpigmentation disorders may be associated with an underlying systemic disease or be caused by a medication. A thorough history and clinical examination is required, and investigations or specialist referrals should be made where appropriate.

How to Treat Quiz

Disorders of pigmentation — Part 1: Hyperpigmentation

1. Which TWO statements are correct regarding the pathophysiology of pigmentedary disorders?
   
   a) Melanin, carotenoids and haemoglobin all contribute to human skin colour
   b) Disorders affecting the enzyme tyrosinase may disrupt the production of melanin
   c) Melanocyte numbers are never affected in acquired conditions of pigment loss
   d) Darker skin colour is due to increasing numbers of melanocytes

2. Which THREE statements are correct regarding clinical features associated with pigmentedary disorder under Wood’s lamp examination?
   
   a) Phakomatosis pigmentosa has a yellowish-white or copper-orange fluorescence
   b) Vitiligo has increased sharpness of borders
   c) Melasma has accentuated outer epidermal layer pigmentation
   d) Tuberculosis has ovoid yellow patches

3. Which THREE statements are correct regarding the general management principles of pigmentedary disorders?
   
   a) Formal counselling from a mental health professional is important
   b) Management of patient expectations is important
   c) Tanning is a good strategy to improve depigmented skin
d) Sunscreen should be advised for both hyperpigmentary and hypopigmentary disorders

4. Which TWO statements are correct regarding the clinical features of circumscribed hyperpigmentary disorders?
   
   a) Melanocytic naevi, arthropathy naeviridescent and mastocytosis all have circumscribed hyperpigmented lesions
   b) Melasma presents as bilateral symmetric hyperpigmented macules
   c) Lentigines, unlike freckles, darken on exposure to sunlight
   d) Freckles in the axilla are a clinical feature of fibromyalgia

5. Which TWO statements are correct regarding the management of circumscribed hyperpigmentary disorders?
   
   a) Both epidermal and dermal melanosis have a good, curative response to treatment
   b) 60% of café au lait macules may clear with pigment laser
c) Maintenance therapy is not required after a successful chemical peel for melasma
d) The primary aim of treatment of acanthosis nigricans is to correct the underlying disease

6. Which TWO statements are correct regarding the clinical features and management of reticulate and mottled hyperpigmentary disorders?
   
   a) Confluent and reticulate papillomatosis usually forms between the breasts and on the back of young women
   b) Erythema ab igne is usually caused by prolonged cold exposure
   c) Squamous cell carcinoma may develop as a long-term consequence of erythema ab igne
d) Prurigo pigmentosa may be effectively treated with an antihistamine and steroids

7. Which TWO statements are correct regarding the clinical features of medication-induced hyperpigmentation?
   
   a) Photo-patch testing helps the management of Riehl’s melanosis by identifying the causative agent to avoid
   b) The principal treatment for Berloque dermalitis is discontinuation of the offending substance
c) Periodic hyperpigmentation is readily treated with a chemical peel
d) Ophthalmology referral is essential for patients with Hor’s naevus

8. Which TWO statements are correct regarding the management of facial pigmentation?
   
   a) Drug-induced pigmentation usually fades when the drug is discontinued
   b) Drug-induced pigmentation usually occurs in areas not exposed to sun
   c) Fixed drug eruptions are usually gradual in onset
   d) Common medications that may cause diffuse or systemic hyperpigmentation include: antihistamines and antibiotics

9. Which TWO statements are correct regarding the clinical features of medication-induced hyperpigmentation?
   
   a) Drug-induced pigmentation usually fades when the drug is discontinued
   b) Drug-induced pigmentation usually occurs in areas not exposed to sun
   c) Fixed drug eruptions are usually gradual in onset
   d) Common medications that may cause diffuse or systemic hyperpigmentation include antihistamines

10. Which THREE medical conditions may cause diffuse or systemic hyperpigmentation?
    
    a) Lupus
    b) Addison’s disease
    c) Raynaud’s disease
    d) Hypothyroidism

Instructions

Complete this quiz online and fill in the GP evaluation form to earn 2 CPD or PDP points.

We no longer accept quizzes by post or fax.

The mark required to obtain points is 80%. Please note that some questions have more than one correct answer.

GO ONLINE TO COMPLETE THE QUIZ


CPD Quiz Update

The RACGP requires that a brief GP evaluation form be completed with every quiz to obtain category 2 CPD or PDP points for the 2014-16 triennium. You can complete this online along with the quiz at www.australiandoctor.com.au. Because this is a requirement, we are no longer able to accept the quiz by post or fax. However, we have included the quiz questions here for those who like to prepare the answers before completing the quiz online.

Next Week

We conclude this series on disorders of pigmentation with a close examination of hypopigmentary disorders.