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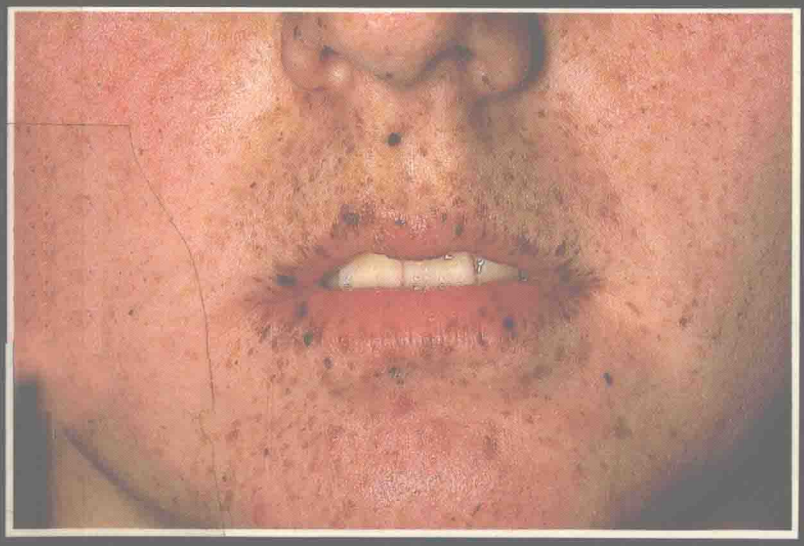
Dermatology Revision

MCQs, Case Histories and Picture Interpretation

S. K. Goolamali



Dermatology Revision:

MCQ's, Case histories and Picture interpretation

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Foreword

Unlike most other 'developed' countries, Great Britain still clings to the time-honoured 'apprenticeship' system for training its specialist dermatologists. Exams in dermatology play no part in acquiring recognised accreditation. It is at first sight surprising that a London dermatologist has seen fit to produce a substantial book, much of which comprises examination questions and answers in dermatology. The readership of the book is obviously not restricted to this country. But more importantly, the need for dramatic action to improve dermatological training in Britain, coupled with the desirability of reciprocity with EEC and other countries, will surely lead to the introduction of a qualifying dermatology exam sooner or later. Dr Goolamali has had the foresight to anticipate this event; his collection of dermatological problems, teasers and conundrums should quickly establish itself as valuable study material for the prospective dermatological examination candidate. In the meantime, there is much for the contemporary unexamined trainee or consultant to gain from Dr Goolamali's efforts. The best way to learn more about spots and rashes is to be in the clinic or at the bedside. Regular reading of current dermatological and relevant general medical and basic science literature is also important, in order to keep up with the growing edge of clinical dermatology. But reading dermatological text books is at best supplementary and at worst useless, unless it is firmly patient-based. Dr Goolamali has espoused a different innovative approach which brings together valuable components of these different modes of learning. Beware of cheap foreign imitations. Other publications containing MCQ compendiums are available, but 'Dermatology Revision' offers well-balanced explanatory comments for each set of answers, and comprehensive reference lists. As icing on the gingerbread the author has included 20 dermatological case problems for the reader to flex his mental muscles. Each is well illustrated and I found it an enjoyable, though somewhat chastening, experience to go through them. I am sure that Dr Goolamali, who is nothing if not enterprising, has succeeded in finding and filling an important gap in existing world dermatological literature; and his approach should appeal to dermatologists of all levels of ignorance. 'Dermatology Revision' deserves and will no doubt enjoy initial success. Its long term future will depend upon the willingness of the

author to carry out sufficiently frequent revision to maintain the freshness and relevance of its contents.

London, 1984 M.W.G.

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Preface

This is not a textbook of dermatology. There are several textbooks available which cater admirably for the student who wishes either for a basic understanding of skin diseases or requires a detailed knowledge of the subject.

The purpose of this book is threefold. The first is to provide a means of self-examination which hopefully will help the reader consolidate information gleaned from so called 'straight' reading and identify areas of weakness. The second is to introduce students and non-dermatologists to the idea that recognition of certain skin diseases may help in the diagnosis of internal disease. Finally, it is hoped that the MCQs and cases for 'spot' diagnosis will allow practice for those examinations where such methods are now used routinely.

There are 10 papers, each of which consists of 20 questions. Each question is composed of a 'stem' followed by a variable number of statements. Any number of statements may be correct. The reader is invited to identify both 'true' and 'false' statements. For those who wish to assess their skills, a correct answer, which may be either 'true' or 'false', gains one mark whilst an incorrect answer loses a mark. No marks are awarded for 'don't know'. The answers given with each question relate to those statements which are 'true' only. As with all such tests it is important to read the question carefully and not to guess at the answer.

The references after each paper have been chosen as a guide to further reading and refer to the topics covered in that paper. The information in this book has been confirmed whenever possible by referral to at least three different sources. Those texts which have been particularly useful are listed under 'Selected Bibliography'.

Acknowledgements

It gives me much pleasure to thank Professor Malcolm Greaves for writing the Foreword. I am grateful to Dr Michael Liberman and Dr Elliott Larson for helpful comments.

I owe much to my family for their patience with me during the preparation of this book and finally my special thanks go to the Publishers, for their encouragement and support.

Contents

Paper one

1
The following are inherited as autosomal dominant traits

A tuberous sclerosis
B Peutz–Jeghers syndrome
C hereditary haemorrhagic telangiectasia
D diabetes mellitus
E psoriasis
F acne vulgaris

2
An increased incidence of vitiligo is found in

A diabetes mellitus
B Addison's disease
C thyrotoxicosis
D pernicious anaemia
E alopecia areata
F tinea versicolor

3
Neurofibromatosis

A *café au lait* spots are the most common disturbances of pigmentation
B pathognomonic sign is presence of freckle-like pigmentation in axillae
C commonest solitary intracranial tumour is an optic nerve glioma
D inherited as a sex-linked recessive trait
E neurofibroma may undergo sarcomatous transformation
F endocrine abnormalities may be found

(Answers overleaf)

1 A, B and C

Comment

Diabetes mellitus, psoriasis and acne vulgaris are considered to have a polygenic mode of inheritance. Twin studies provide evidence that psoriasis is hereditary but the mode of inheritance remains speculative since it is difficult to distinguish between multifactorial inheritance and two alleles at a single locus, with empiric data alone. Acne shows a distinct familial bias and is commoner in certain races. In one survey almost five out of 10 boys with acne had one or both parents who had also suffered from acne whereas less than one in 10 boys without acne had a parent with a history of acne.

2 A, B, C, D and E

Comment

Tinea versicolor is a mild chronic fungus infection of the skin. It is caused by the conversion of a normal saprophyte of skin *Pityrosporon orbiculare* to the pathogenic form *Malassezia furfur*. The term versicolor implies that it changes from one colour to another and in dark skin it may appear as whitish patches and be mistaken for vitiligo whilst in fair skin it can present as brown macules. When viewed under a Wood's lamp (long-wave ultra-violet light) it fluoresces a golden yellow.

3 A, B, C, E and F

Comment

Pigmentation is present in most patients and commonly seen as well defined brown patches often 2–5 cm in length. Axillary freckling is considered to be pathognomonic and found in one of five patients. Neurological manifestations are found in some 40% of patients and the commonest solitary intracranial tumour is an optic nerve glioma. Astrocytomas and schwannomas also occur. Sarcomatous change within a neurofibroma arises in 5–15% of cases. Neurofibromatosis is inherited as an autosomal dominant trait with an incidence estimated at one in 2500–3000 births.

Endocrine abnormalities which may be associated include precocious puberty, hyperparathyroidism, Addison's disease and acromegaly.

4
Phenylketonuria

A may manifest itself as infantile eczema
B phenylalanine hydroxylase usually absent
C mental deficiency is a common sequel
D therapy involves a diet low in phenylalanine
E electroencephalogram abnormal in the majority

5
Underlying malignancy may be heralded by

A diffuse hyperpigmentation of the skin
B Paget's disease of the nipple
C acanthosis nigricans
D blue-green pigmentation of the finger tips
E dermatomyositis
F herpes zoster

6
Gynaecomastia may occur with

A bronchial carcinoma
B cholera
C spironolactone administration
D chronic liver disease
E malnutrition
F testicular tumour
G digitalis therapy

(Answers overleaf)

4 A, B, C, D and E

Comment

Cutaneous manifestations include fair hair and skin (due to impaired melanin synthesis), eczema which may be of the atopic variety and dermographism. The incidence of pyogenic infection is also increased. The enzyme phenylalanine hydroxylase which converts phenylalanine to tyrosine is usually absent.

A low phenyalanine diet is essential in therapy though in some instances may safely be stopped after the first eight years of life. PKU account for 0.04–1% of mental defectives in institutions. The EEG is abnormal in 80% of patients with PKU.

5 A, B, C, E and F

Comment

Diffuse hyperpigmentation is recognised as one of the many signs of neoplasia. In the ectopic ACTH syndrome associated with an oat-cell carcinoma of the bronchus pigmentation is usual. Paget's disease of the nipple is associated with an intraductal carcinoma of the breast. Acanthosis nigricans can be a marker of underlying malignancy particularly when it develops in adult life. Childhood dermatomyositis is not normally associated with neoplasia but the adult onset variety (40–60 years) is frequently found with malignancy, (15–20% of patients). Herpes zoster occurs more commonly and is more likely to be disseminated in patients with an underlying reticulosis.

6 A, B, C, D, E, F and G

Comment

The multiplicity of causes of gynaecomastia reflects the complex hormonal mechanisms responsible for breast enlargement. Some breast enlargement occurs at puberty in about 60% of boys. Patients with testicular teratoma often have a high serum concentration of Human Chorionic Gonadotrophin (HCG) which stimulates oestrogen production. Other non-endocrine tumours which produce HCG are hepatoma and large cell bronchial carcinoma (10% of cases). When drug-related, withdrawal of the offending drug reduces the discomfort associated with gynaecomastia. In the majority of symptomatic patients an anti-oestrogen such as tamoxifen or a gonadotrophin inhibitor, danazol, is useful.

7
Generalised pruritus can be a feature of

A trichinosis
B lymphatic leukaemia
C drug reactions
D chronic renal failure
E Hodgkin's disease
F biliary cirrhosis
G iron deficiency

8
Clubbing may be found with

A metastatic cancer to the thorax
B cirrhosis
C ulcerative colitis
D coarctation of the aorta
E thyrotoxicosis
F bronchogenic carcinoma

9
The following may result in a false positive test for syphilis

A systemic lupus erythematosus
B lepromatous leprosy
C Hashimoto's thyroiditis
D malaria
E infectious mononucleosis
F Sjögren's syndrome

(Answers overleaf)

7 A, B, C, D, E, F and G

Comment

Trichinosis is caused by a round worm, *Trichinella spiralis*. Generalised pruritus can occur at the stage of muscle invasion by the parasite. Pruritus is seen more commonly with the lymphatic rather than the granulocytic variety of leukaemia. The pruritus of chronic renal failure has been related to the level of the blood urea, the skin surface urea and the dryness of the skin. In many, parathyroidectomy relieves the pruritus suggesting that secondary hyperparathyroidism may also be relevant. Pruritus occurs in some 30% of patients with Hodgkin's disease. In biliary cirrhosis generalised pruritus can precede all other signs and may occur 1–2 years before the onset of jaundice. A low serum iron level has been correlated with pruritus.

8 A, B, C, D, E and F

Comment

In clubbing increased fibrous tissue separates the nail from the phalanx. In cyanotic congenital heart disease clubbing is seldom noticeable before the second year of life whereas in malignant pulmonary disease the onset may be rapid and painful. The pathogenesis of clubbing of the fingers remains uncertain but a low arterial oxygen tension and increased blood flow cannot account for all the cases encountered. In ulcerative colitis clubbing is rare in the absence of coexisting small bowel involvement.

9 A, B, C, D, E and F

Comment

Two types of antibodies form in response to infection by *Treponema pallidum*, treponemal and non-treponemal antibodies (reagins). The latter form in response to tissue damaged by the action of spirochaetes and are detected by tests which are not specific but indicate the presence of a chronic disease. Patients with a chronic biologically false positive (BFP) reaction develop a high incidence of auto-immune diseases especially SLE, Sjogren's syndrome and Hashimoto's thyroiditis.

10
The oral mucosa can be involved in

A lichen planus
B Peutz–Jeghers syndrome
C Addison's disease
D discoid lupus erythematosus
E pemphigoid
F infection with viral warts

11
Trophic ulceration may occur in

A tabes dorsalis
B sciatic nerve injury
C diabetes mellitus
D spinal dysraphism
E syringomyelia
F leprosy
G clostridium botulinum infection

12
Candida albicans

A can exist as a saprophyte in healthy adults
B untreated diabetes mellitus predisposes to candida infection
C fluoresces green under Wood's light
D increased candidiasis in hypoparathyroidism
E may cause endocarditis after vascular surgery
F frequent secondary pathogen in napkin rash

(Answers overleaf)

10 A, B, C, D, E and F

Comment

In lichen planus mucous membrane lesions are very common and occur in 30–70% of cases. The buccal mucosa and tongue are most often involved. In the Peutz–Jeghers syndrome pigmented macules develop on the oral mucosa in association with perioral pigmentation. In chronic discoid lupus erythematosus lesions within the mouth are commonly seen on the palate and buccal mucosa. In pemphigoid bullae occur in the mouth in one of five patients and usually late in the course of the disease.

11 A, B, C, D, E and F

Comment

Essential factors in the production of a trophic ulcer are loss of pain sensation and trauma. In leprosy, tabes dorsalis, spinal vascular disease and syringomyelia there is sensory loss. In diabetes mellitus peripheral neuropathy and vascular insufficiency both contribute to the development of a trophic ulcer. In one series diabetes mellitus was the most frequent cause of neurotrophic ulcers of the foot.

The presence of pain does not rule out the diagnosis of a neurotrophic ulcer as referred pain can be present in an anaesthetic ulcer.

12 A, B, D, E and F

Comment

Candida albicans is a frequent inhabitant of the gastrointestinal tract in otherwise healthy individuals. In hypoparathyroidism, hypothyroidism and Cushing's syndrome increased susceptibility to candidiasis is thought to be related to depressed immune especially T cell function. *Candida albicans* is commonly isolated from the skin in a napkin eruption. The organism may be the cause of the rash though it is frequently a secondary invader.

In fungal endocarditis, most frequently caused by *Candida* or *Histoplasma*, a characteristic is embolic occlusion of large arteries. It is suggested that this occurs because of the bulky vegetations induced by these organisms.