

Problem-Based Medical Case Management

Second Edition

Edited by Kathryn Tan



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Foreword

It is a pleasure to welcome the second edition of this special book. My comments in the foreword to the first edition apply equally here. While the contents and layout are similar, certain sections have undergone modification, and diagnostic approaches and treatment modalities have been brought up to date, in keeping with advances in medical science. There are several new authors, mostly young—it is pleasing to note.

In the sections with advice to the patient, the word “prevention” often appears. The preventive aspect of patient management cannot be over emphasized. Not only can the recurrence of the illness in question be avoided, many current diseases such as those related to lifestyle, including malignancies, are essentially preventable. The physician is well placed to impress this upon patients and their families.

The chapter on medical professionalism has been well rewritten. But as previously pointed out, the teaching of professionalism is not easy. How does one instruct another to be compassionate? The illustrative examples given underscore the fact that the acquisition of professionalism comes with experience—the experience of life. It would be well for us to remember that learning from role models is time-honoured and effective.

The authors are to be congratulated on their ingenuity and scholarship. This book should be of value not only to those preparing for examinations but as a regular reference for all. It is not a replacement for the standard textbook, but is a most useful and unique problem-based guide to diagnosis and patient management.

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3

Endocrinology and General— Short Cases

*Karen Siu-Ling Lam, James D. Best, Kathryn Choon-Beng Tan,
and Chi-Keung Yeung*

Overview

In the endocrine and general short cases examination, do exactly what you have been asked by the examiner. However, you should always spend 10–15 seconds just looking at the patient as well as the part or system you have been asked to evaluate before even attempting an examination. The general cases include endocrinology (usually spot diagnosis), skin conditions, and fundi.

In a quick and systematic way, start looking at the face, followed in turn by the head and neck, the trunk and limbs. Inspect the body build, the skin, and finally the bones and joints. Many endocrine and general short cases are spot diagnoses, as the patients usually have obvious and typical clinical features; important clues become evident just by quickly looking at the patient. Become familiar with the possible short cases commonly used in such examinations and the physical signs associated with each condition. If you have a clear idea of what to look for, you are less likely to miss essential features under the stressful conditions of an examination. Familiarity with typical cases will also make it easier to present your findings in a fluent and professional manner.

3.1 Acromegaly

Classical signs

- Prominent supraorbital ridges
- Large nose
- Protrusion of the lower jaw (prognathism)
- Deep voice
- Oily skin
- Mouth: thick lips, malocclusion, and increased interdental separation; large tongue (macroglossia)
- Eye/vision: bitemporal hemianopia, optic atrophy
- Neck: goitre
- Hands: large hands with broad palms, spatulate fingers, sweaty palms
- Feet: large feet, thick heel pads
- Others: increased blood pressure, osteoarthritis and glycosuria

Causes

- Acromegaly due to excessive growth hormone from a growth hormone secreting pituitary adenoma

Important investigations

- Basal insulin-like growth factor 1: elevated
- Oral glucose tolerance test (glucose and growth hormone measurements). Growth hormone falls to $< 1 \mu\text{g/L}$ in normal individuals, but is not suppressed in acromegaly.
- Magnetic resonance imaging of the pituitary: may demonstrate suprasellar extension and compression of the optic chiasm
- Other anterior pituitary hormones: look for evidence of hypopituitarism and concomitant secretion of prolactin by adenoma.

Discussion

- Regulation of growth hormone secretion:
The hypothalamus controls growth hormone synthesis and release by means of growth hormone releasing hormone and somatostatin. Growth hormone secretion can be stimulated by stress, a fall in blood sugar, prolonged fasting, some amino acids (e.g., arginine) and exercise.
- Complications of acromegaly:
 - Facial and skeletal disfigurement
 - Jaw malocclusion and overbite
 - Arthropathy
 - Nerve entrapment, carpal tunnel syndrome
 - Hypertension and left ventricular hypertrophy
 - Obstructive sleep apnoea
 - Diabetes mellitus
 - Colonic polyps and cancer
 - Hypopituitarism secondary to mass effect
 - Visual defects due to optic chiasm compression
- Management of acromegaly:
 - Transsphenoidal surgery
 - Medical therapy includes: dopamine agonists such as bromocriptine and cabergoline; somatostatin receptor agonists (e.g., octreotide), and growth hormone receptor antagonist (pegvisomant).
 - Adjuvant radiotherapy
- Relationship between acromegaly and goitre:
Non-toxic goitres are quite commonly present, as part of the visceromegaly seen in acromegaly, and hyperthyroidism may occasionally occur.

Pitfalls and tips

- Acromegaly is differentiated from Paget's disease by the presence of soft tissue involvement such as large tongue and thick skin, apart from the typical skull and facial deformities.

- Compare the facial appearance with an old photograph of the patient.
- Note that in children and adolescents, growth hormone hypersecretion will lead to pituitary gigantism as epiphyseal closure of the long bones has not yet taken place.

3.2 Atopic eczema

Classical signs

- Generalized dry skin, symmetrical eruption with lichenification and excoriation, morphology depends on stages (acute, sub-acute, chronic).
- Head: predilection for eyelids, Dennie-Morgan infraorbital folds, infra-auricular fissure, periorbital pigmentation
- Neck: post-inflammatory hyperpigmentation with “dirty neck” appearance at sides of neck
- Trunk: sometimes erythrodermic, ill-defined erythematous patches, papules or plaques, with or without scale, post-inflammatory hyperpigmentation and lichenification, excoriation and erosions/crusting indicating secondary infection
- Limbs: flexural distribution with ill-defined patches or plaques with excoriation and lichenification, frequent involvement of wrists
- Hands and feet: lichenified papules with pigmentation over finger knuckles, hyperlinearity of palmar creases, frequently involves anterior aspect of ankles, dorsa of feet and hands; painful fissuring and cracking of fingers and palm
- Associated asthma

Causes

- Atopic tendency: genetic predisposition of hypersensitive response to environmental antigens

Important differential diagnoses

- Contact dermatitis (allergic/irritant): distribution corresponding to sites of contact with irritant or allergens, +ve patch test
- Drug eruption: tends to affect trunk and proximal limbs
- Seborrhoeic dermatitis: distributed along hairline, medial eyebrows, and nasolabial folds, axillae, groins and scalp
- Psoriasis: well-defined erythematous plaques with silver scaling on extensor surface of limbs and back, associated arthropathy
- Dermatophytosis: annular scaly rash with advancing edge and central clearing

Important investigations

- IgE level: elevated
- Skin swab for bacterial culture
- Skin patch test/skin prick test for allergens

Pitfalls and tips

- Not recognizing the lichenification and distribution of lesions
- Not treating the secondary infection, e.g., staphylococcus or streptococcus
- Personal/family history of atopy

3.3 Cushing's syndrome***Classical signs***

- "Moon face", acne, hirsutism (due to androgen excess and so not seen in cases due to excess steroid intake), plethora
- Pigmentation (ACTH-dependent Cushing's)
- Truncal obesity, thin arms and legs
- Limbs: wasting of limbs, bruising, weakness of the muscles of the shoulders and hips (ask the patient to stand up from squatting position)

- Trunk: buffalo hump, thinning of skin and purple striae over the upper arms, thighs, and abdomen
- Hypertension and glycosuria
- Look for clues that may suggest use of steroid (e.g., renal transplant, asthma).

Causes

- Cushing's syndrome due to excessive glucocorticoids

Important differential diagnoses

- Iatrogenic: due to exogenous steroids
- ACTH-dependent: pituitary adenoma secreting ACTH, ectopic ACTH syndrome
- Non-ACTH dependent: adrenal tumours

Important investigations

- 24-hour urinary free cortisol
- Loss of diurnal rhythm of cortisol secretion
- Screening test: 1 mg overnight dexamethasone suppression test (a.m. level suppressed: normal)
- Confirmation of Cushing's syndrome: 48-hour low dose dexamethasone test (suppressed: normal)
- ACTH: distinguish between adrenal cause (low level) vs ACTH-dependent Cushing's syndrome (high level)
- High dose dexamethasone suppression test: used in the differential diagnosis of ACTH-dependent Cushing's syndrome; suppression of cortisol and ACTH consistent with pituitary-dependent Cushing's disease
- Corticotrophin releasing factor stimulation test: used in the differential diagnosis of ACTH-dependent Cushing's syndrome; rise of ACTH and cortisol consistent with pituitary-dependent Cushing's disease

- MRI of pituitary gland, CT or MRI of adrenals as appropriate, or search for underlying tumour if Cushing's due to ectopic ACTH is suspected
- Inferior petrosal sinus sampling for ACTH: confirmation and localization of pituitary adenoma producing ACTH

Discussion

- Complications of Cushing's syndrome:
 - Cardiovascular: hypertension, fluid retention
 - Metabolic risks: glucose intolerance, central obesity, dyslipidaemia, hypokalaemia
 - Musculoskeletal and connective tissue: thinning of skin, bruises and striae, proximal muscle weakness, osteoporosis
 - Immune system: immunosuppression with risks of opportunistic infection
 - Psychiatric: irritability, depression, psychosis
 - Androgen excess (not seen in exogenous Cushing's): acne, hirsutism
 - For iatrogenic Cushing's (rarer in endogenous Cushing's): avascular necrosis, glaucoma, posterior subcapsular cataract
- Management of Cushing's syndrome:
 - Management of concomitant problems: hypertension, diabetes, hypokalaemia
 - Surgery (aim for cure): transsphenoidal surgery for pituitary lesion; adrenalectomy for adrenal lesion
 - Medical (control of hypercortisolism while awaiting definitive therapy or if residual disease persists post-surgery): metyrapone or ketoconazole (inhibits adrenal steroidogenesis); cabergoline or pasireotide (inhibits ACTH secretion from pituitary tumours)
- Interpretation of dexamethasone suppression tests:
 - Overnight 1 mg or low-dose 48-hour dexamethasone suppression test: 9 a.m. serum cortisol suppressed to less than 50 nmol/L in normal individuals. High dose 48-hour dexamethasone suppression test: 9 a.m. serum cortisol suppressed

to less than 50% of basal level in pituitary-dependent Cushing's syndrome.

- Interpretation of corticotrophin releasing factor stimulation test: in patients with pituitary-dependent Cushing's disease, there is a rise above baseline of plasma ACTH of over 50% and cortisol of over 20%.

3.4 Diabetic retinopathy

Classical signs

- Non-proliferative: microaneurysm, dot and blot haemorrhages, and hard exudates
- Pre-proliferative: cotton wool spots, venous beading, haemorrhages, and intraretinal microvascular abnormalities (IRMA)
- Proliferative: new vessels at the disc/elsewhere, photocoagulation scars; vitreous haemorrhage

Important differential diagnoses

- Hypertensive retinopathy (see 3.7)
- Central retinal vein thrombosis:
 - Venous tortuosity and dilatation
 - Flame-shaped haemorrhages
 - Cotton-wool spots
 - Papilloedema
 - Secondary neovascularization

Important investigations

- Blood for sugar, haemoglobin A1c
- Urine for proteinuria
- Screening for other diabetic complications such as renal, neurological, and cardiovascular diseases

Pitfalls and tips

- Haemorrhages and exudates also found in hypertensive retinopathy.
- Laser scars may be widespread in the periphery of the fundus (grid pattern), appearing like exudates with associated pigment deposition; restriction of the visual field may result.
- Vitreous haemorrhage “organization” may result in widespread fibrous scarring and retinal detachment.

Discussion

- Management of diabetic retinopathy:
 - Medical: good glycaemic and blood pressure control.
 - Laser photocoagulation is indicated for proliferative retinopathy and some cases of pre-proliferative retinopathy and maculopathy.
 - Vitrectomy; for persistent vitreous haemorrhage.
 - anti-VEGF (vascular endothelial growth factor) agents: for diabetic macular oedema and proliferative retinopathy; (as adjunct to laser therapy/vitrectomy).

3.5 Erythema nodosum

Classical signs

- Bilateral multiple tender, erythematous, round, subcutaneous nodules on anterior aspects of legs and knees, occasionally on forearms
- Resolving into bruise-like or brownish patches
- Associated arthralgia especially at ankle joints

Causes

- Immunologic reaction triggered by a wide range of stimuli
- Streptococcal infections
- Tuberculosis

- Drugs, e.g., sulphonamide, oral contraceptive
- Sarcoidosis, inflammatory bowel disease, Bechet's syndrome

Important differential diagnoses

- Another panniculitis (inflamed subcutaneous fat nodule), such as erythema induratum
- Vasculitis, e.g., polyarteritis nodosa, types of panniculitis versus vasculitis revealed by skin biopsy
- Pre-tibial myxoedema: patient usually has associated features of Graves' disease.
- Superficial thrombophlebitis: patient usually has varicose veins and brownish pigmentation at lower parts of the legs.

Important investigations

- Chest X-ray; for tuberculosis (hilar lymphadenopathy in sarcoidosis)
- Mantoux test
- Blood tests: ESR/C-reactive protein, anti-streptolysin titre, anti-neutrophil cytoplasmic antibodies

3.6 Graves' disease

Classical signs

- Eyes: proptosis, periorbital oedema, lid lag, lid retraction, chemosis, ophthalmoplegia
- Hands: sweaty palms, action tremor, thyroid acropachy, palmar erythema
- Neck: possible thyroidectomy scar, diffuse enlarged mass over the neck (goitre) that moves on swallowing both on inspection and palpation; auscultate for bruit over the thyroid
- Limbs: pre-tibial myxoedema (bilateral pinkish, brown dermal plaques); proximal myopathy (ask the patient to stand up from

squatting position, observe whether assistance from hands is required)

- Others: sinus tachycardia, atrial fibrillation, and signs of high output heart failure

Causes (neck swelling and thyrotoxicosis)

- Graves' disease: Graves' disease is distinguished from other causes of thyrotoxicosis by presence of typical diffuse thyroid enlargement and ophthalmopathy.
- Toxic multinodular goitre
- Toxic thyroid adenoma
- Subacute thyroiditis

Important investigations

- Thyroid function test: the screening test is serum TSH; in primary hyperthyroidism TSH is suppressed and serum T4 and/or T3 elevated.
- Thyroid autoantibodies: positive TSH receptor autoantibodies (TRAb) in Graves' disease. In other autoimmune thyroid diseases, anti-thyroglobulin and anti-thyroperoxidase antibodies are present but not TRAb.
- Radionuclide scan: diffusely increased uptake of radioactive iodine in Graves' disease; patchy inhomogeneous uptake in toxic multinodular goitre; localized area of increased uptake with suppression of uptake in the rest of the thyroid gland with toxic adenoma; decreased/no uptake in subacute thyroiditis
- Ultrasound: diffuse enlargement with homogeneous echogenicity

Discussion

- Aetiology of Graves' disease: autoimmune disorder associated with the production of stimulatory autoantibodies against TSH receptor (TSH receptor antibody, TRAb)

- Management of Graves' disease: manage associated hyperthyroidism by antithyroid drugs, thyroidectomy or radioactive iodine. The thiourea group of agents (carbimazole or propylthiouracil) is especially indicated in children and pregnant women. 50% of patients may relapse after one course of antithyroid drug treatment for 18 months. Thyroidectomy or radioactive iodine may be considered if the patient relapses after medical treatment or as first line treatment. (Note: thyrotoxicosis should be controlled with antithyroid drugs before surgery.)
- Management of ophthalmopathy: 70% of patients with Graves' disease may have eye problems, ranging from soft tissue involvement (such as periorbital oedema) to severe proptosis, diplopia, and visual impairment. The cause of the ophthalmopathy is unclear; autoimmunity has been implicated. There is inflammation and swelling of retrobulbar tissues. If severe, corticosteroids or other immunosuppressants may be useful.

Pitfalls and tips

- Absence of thyroid enlargement makes the diagnosis of Graves' disease less likely but does not exclude it.

3.7 Hypertensive retinopathy

Classical signs

- Grade 1: silver wiring
- Grade 2: above + arteriovenous nipping
- Grade 3: above + cotton wool spots, flame-shaped haemorrhages
- Grade 4: above + papilloedema
- Arteriosclerotic changes: silver-wiring (increased arteriolar light reflex); arteriovenous nipping (deflection of venule at arteriovenous crossing points)
- Measure blood pressure

Important investigations

- Examine urine for proteinuria.
- Assess the heart for left ventricular hypertrophy and heart failure.

Discussion

- Causes of secondary hypertension:
 - Renal (e.g., chronic renal failure, renal artery stenosis, IgA nephropathy)
 - Vascular (e.g., coarctation of aorta)
 - Metabolic (e.g., Conn's syndrome, Cushing's syndrome, pheochromocytoma)
 - Drugs (e.g., mineralocorticoids and glucocorticoids)
- Management of hypertension: treat primary cause; lifestyle changes; management of other coronary risk factors such as cigarette smoking, diabetes, hyperlipidaemia; discuss the types of anti-hypertensive drugs.

Pitfalls and tips

- Haemorrhages and exudates are also found in diabetic retinopathy.

3.8 Optic atrophy***Classical signs***

- Pale disc with clearly delineated margin (except if the optic atrophy is due to long-standing papilloedema, which is then termed secondary optic atrophy).
- Central scotoma may occur.

Causes

- Compression of optic nerve by tumour (e.g., pituitary tumour: look for bitemporal hemianopia) or aneurysm
- Glaucoma
- Ischaemic optic neuropathy
- Friedreich's ataxia
- Long-standing papilloedema (from any cause)
- Multiple sclerosis (causing optic neuritis)
- Vitamin B12 deficiency

3.9 Osler-Weber-Rendu syndrome (hereditary telangiectasia)

Classical signs

- Telangiectasia on face, mucosa (mouth, lips, tongue) and on fingers
- May have pallor (due to anaemia)
- No signs of systemic sclerosis

Discussion

- Osler-Weber-Rendu syndrome is an autosomal dominant condition. Lesions may occur elsewhere, especially in the gastrointestinal tract. Patient may present with epistaxis, gastrointestinal haemorrhage and anaemia.
- Facial and mucosal telangiectasia are also present in patients with systemic sclerosis. However, they have other systemic features (e.g., smooth, shiny, and tight skin over the face and fingers; sclerodactyly, atrophic nails etc.).

3.10 Papilloedema

Classical signs

- Loss of physiologic cup
- Elevation of disc head
- Blurring of disc margins
- Distended non-pulsatile veins
- Sub-hyaloid haemorrhages at disc margin
- Enlargement of blind spot and constriction of peripheral visual field

Causes

- Increased intracranial pressure
- Central retinal vein occlusion
- Grade 4 hypertensive retinopathy
- Carbon dioxide retention

Important differential diagnoses

- Papillitis (a form of retrobulbar neuritis): visual acuity considerably reduced in papillitis, visual field defect (usually central) and eye movement may be painful.

Important investigations

- Visual acuity and visual field
- Blood gases
- Imaging of brain for evidence and cause of raised intracranial pressure

Examination case scenarios

i. Examine the neck of this patient.

Important signs	<ul style="list-style-type: none"> • Diffuse neck mass that moves with swallowing • Examine for thyroid bruit. • Ask for permission to examine for other hyperthyroid signs.
Diagnosis	Graves' disease.
Question	<p>What are the differential diagnoses if patient is euthyroid?</p> <ul style="list-style-type: none"> • Controlled Graves' disease, euthyroid goitre, Hashimoto's thyroiditis.

ii. What do you notice about the physical appearance of this patient?

Important signs	<ul style="list-style-type: none"> • Prominent supra-orbital ridge, nose and lips, prognathism • Spade-like hands
Diagnosis	Acromegaly
Question	<p>What investigations will you order?</p> <ul style="list-style-type: none"> • Raised basal level of insulin-like growth factor 1. • Oral glucose tolerance test for non-suppressed growth hormone level. • Pituitary magnetic resonance imaging (skull X-ray may show double floor in sella turcica).

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