VASCULAR DISEASE IN OPTOMETRIC PRACTICE

Outline notes to accompany City University 3rd year undergraduate Clinical Practice course lecture

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Introduction

Optometrists in the UK are responsible not only for detecting ocular disease but also systemic disease with ocular manifestation. In optometric practice, the practitioner will frequently observe the effects of vascular disease. This may be local to the eye or may affect the broader visual system. Thus a full range of signs may be diagnosed from visual field defects, paretic squints, gaze palsies and nystagmus due to stroke or aneurysms within the brain, to specific retinal haemorrhages secondary to a wide range of disease processes.

Vascular hypertension

1. Prevalence. Approx 10% to 15% of UK population
2. One of the commonest contributing causes of death or morbid events
3. Fundus signs may be graded e.g. Keith, Wagener & Barker (1939)
   - Grade 1 Narrowing of the arterioles (general or focal)
   - Grade 2 More marked than grade 1
   - Grade 3 Grade 2 + cotton wool spots + haemorrhages (often superficial flame shaped
   - Grade 4 Grade 3 + papilloedema
4. Aetiology. Cause of primary or essential hypertension unknown. Hereditary component. Causes of acute secondary grade 4 hypertension include phaeochromocytoma
5. Case finding - fundus examination + sphygmomanometry + family history
6. Sphygmomanometric criteria. Refer one reading of diastolic > 110 mm Hg otherwise 2 or more readings of :

Guideline upper levels of blood pressure

Age (years)	BP (mm Hg)
18 - 44	140/90
45 - 64	150/90
> 64	160/95

7. Chronic hypertension $\Rightarrow$ arteriosclerosis

Arteriosclerosis

A.. ***Atheroma or atherosclerosis***

1. Common degeneration of intima of arteries.
2. Hyaline $\Rightarrow$ cholesterol + foam cells $\Rightarrow$ plaques $\Rightarrow$ obstruction

3. Can occur in retinal arterioles
4. Plaques may be observed in larger vessels

B. ***Involutionary sclerosis***

1. Replacement fibrosis involving the media of the arteries. Associated with advancing age.
2. Almost universal after age 60 years

C. ***Arteriosclerosis associated with vascular hypertension***

1. Hypertension ⇒ hypertonus of media ⇒ hyperplasia (increase in cell numbers) and hypertrophy(increase in cell size) of media (especially in younger patients) + new elastic fibres
2. If acute and overwhelming ⇒ cellular necrosis in artery wall ⇒ circulatory breakdown
3. If chronic then hyperplasia is followed by reduction in number of cells with corresponding laying down of collagen ⇒ acellular fibrous vessel wall
4. "Corkscrew" appearance of arterioles in macular region
5. "Copper wire" = thickening and hyaline degeneration
   "Silver wire" = entire vessel is reflecting light
   Irregular reflexes
   Vessel sheathing. First appears at vessel crossings. May lead to "pipe stem"
6. A/V crossing changes:
   (i) concealment of vein
   (ii) deflection (Salus' sign) with humping, depression, or deflection
   (iii) apparent nipping & tapering (Gunn's sign)
   (iv) banking
7. Fundus arteriosclerotic signs may be graded e.g. Keith, Wagener & Barker (1939)
   Grade 1 Mild changes in vascular reflexes including widening of reflex and concealment of vein
   Grade 2 Changes in vascular reflex + sclerosis at a/v crossings
   Grade 3 Grade 2 + copper wiring + banking
   Grade 4 Grade 3 + silver wiring. Predisposition to vein occlusion
8. Refer for cardiovascular examination including serum cholesterol levels (HDL/LDL)

Arteritis (e.g. temporal or cranial)

1. Inflammation of larger arteries usually carotid and often temporal branch but can be systemic
2. 24 cases/100,000 > 55 years; female > male; familial predisposition
3. Unknown aetiology. ? cell-mediated immune response
4. Fever, anaemia, weight loss, sweating, arthralgia
5. Symptoms continuous or intermittent and last weeks/months
6. Temporal arteritis ⇒ headaches, tender scalp, nodular and sensitive temporal artery, claudication on chewing

7. Ischaemic optic neuropathy, amaurosis fugax ⇒ may lead to permanent visual loss
8. Other signs: peripheral claudication, CVA
9. Diagnosis from signs, symptoms, temporal artery biopsy. High ESR.
10. Excellent response to systemic steroids; Prednisone 80-100 mg/day ⇒ 10 mg/day maintenance

Retinal vaso-occlusive disease

A. ***Branch vein occlusion (BRVO)***

1. strong association with systemic disease
2. occurs most frequently at a/v crossings
3. Non-ischaemic BRVO ⇒ dot-blot haemorrhages and lipid
   Ischaemic BRVO ⇒ as above + cotton-wool spots and flame-shaped haemorrhages
4. Collaterals may form to drain affected area (NB this is not neovascularisation)
5. and 25% ⇒ neovascularisation of disc or retina
6. Visual field defect crosses horizontal raphe as oedema spreads
7. Check BP and carotid auscultation. Refer for evaluation of lipids, glucose, blood counts and others.
8. Fluorescein angiography indicated if macular threatened
9. Follow-up for neovascularisation

B ***Central retinal vein occlusion (CRVO)***

1. Strong association with systemic disease
2. May be associated with localised optic nerve compression
3. Occlusion occurs at lamina cribosa
4. More likely to occur if artery and vein are juxtapositioned
5. Non-ischaemic CRVO ⇒ dot/blot haemorrhages to periphery , macular oedema, dilated tortuous veins, choked disc; minimal threat for neovascular glaucoma
6. Ischaemic CRVO ⇒ signs as above + cotton-wool spots and flame-shaped haemorrhages; 60% chance of neovascular glaucoma
7. Rule out raised IOP as cause
8. Refer for medical and ophthalmological evaluation
9. Possible anticoagulation drugs
10. Possible pan-retinal photocoagulation

C. ***Branch retinal artery occlusion (BRAO)***

1. Strong association with systemic disease especially internal carotid and cardiovascular disease

2. Secondary to embolus trapped in retinal artery
3. Embolus usually lodges at a bifurcation
4. ⇒ total anoxia in the region of arteriole ⇒ white retina
5. May have associated cotton-wool spots
6. Field defect respects the horizontal raphe
7. Attempt to dislodge embolus by massage and refer urgently
8. Systemic evaluation

D. Central retinal artery occlusion (CRAO)

1. Strong association with systemic disease especially internal carotid and cardiovascular disease
2. Sudden (seconds) painless loss of vision
3. Often embolus at lamina cribosa
4. Narrowing of arteries , haziness of retinal tissues due to anoxia
5. Fragmentation appearance to venular blood
6. "Cherry red spot" (choroidal supply) at macular
7. ? Cilioretinal artery ⇒ small area of residual centrocaecal field
8. Urgently attempt dislodging embolus by massage and refer urgently to a Hospital Eye Casualty Department
9. ? Paracentesis
10. Systemic evaluation

Diabetes mellitus

1. May be considered as a capillary vascular disease
2. Background retinopathy (deep dot haemorrhages, lipid) ⇒ pre-proliferative (venous beading, retinal ischaemia, cotton wool spots, IRMA) ⇒ proliferative retinopathy (neovascularisation of venules into sub-hylaoid space) ⇒ haemorrhaging into vitreous, fibrosis and retinal detachment. Rubeosis iridis.
3. Refer any changes within one disc diameter of fovea, pre-proliferative or proliferative retinopathies for ophthalmological evaluation. Photocoagulation.
4. Formal report to GP (UK law) following examination of diabetic
5. Report to GP/Diabetologist any changes in background retinopathy - treatment regime may need fine tuning

Radiation retinopathy

1. Usually occurs 3 months to 3 years after x-ray irradiation near orbital region
2. Microaneurysms, ptelangiactasia, haemorrhages, cotton-wool spots, exudates, optic disc oedema, RPE changes, retinal and iris neovascularisation
3. Photocoagulation management similar to diabetes

Retinal periphlebitis (Eales' disease)

1. Healthy men 20 - 40 years

2. May be bilateral
3. Perivasculitis, occasional anterior uveitis, vitritis, peripheral neovascularisation
4. Rule out systemic disease e.g. tuberculosis
5. Photocoagulation

Sickle-cell retinopathy

1. Normal population has haemoglobin A
2. Sickle-cell patient have abnormal haemoglobin
3. Considerably more common in blacks
4. Retinopathy results from alteration of retinal circulation
5. Intra-retinal haemorrhages, RPE disturbances, venous tortuosity, neovascularisation, vitreous haemorrhage
6. Neovascularisation may benefit from photocoagulation

Retinopathy of prematurity (ROP)

1. Risk factors include : prematurity < 36 weeks, under 4lb 6 oz, supplemental oxygen
2. The last area of retina to be vascularised is the temporal periphery. ROP is an alteration to this development
3. Distinct demarcation line between vascular and avascular retina develops into a ridge.
4. May lead to neovascularisation and retinal detachment
5. Precautions to prevent ROP - improved neonatal care
6. Prophylactic cryotherapy

Behçet's disease

1. Systemic occlusive vasculitis
2. Immune complex disorder
3. Young adults 18 - 40 years; Japan & Mediterranean
4. Genital and oral ulcers, dermatological problems, GI & CNS involvement
5. Recurrent uveitis
6. Peripheral retinal vasculitis ➔ rubeosis iridis, glaucoma, cataract, retinal neovascularisation, retinal detachment
7. Steroids, photocoagulation

Angiomatosis retinae

1. Benign capillary hemangioma
2. May be associated with the phakomatosis, von Hippel-Lindau disease
3. 50% bilateral
4. Appearance of very tortuous vessels feeding orange tumour
5. Genetic predisposition
6. Genetic counselling
7. Possible photocoagulation

Retinal ptelangiectasia (Coat's disease)

1. Ptelangiectasia ➔ exudative retinopathy
2. Usually unilateral and in males < 20 years
3. No genetic association
4. No systemic association
5. Irregular dilatation of retinal vessels may ➔ progress to intra and sub retinal exudation
6. Possible photocoagulation

MCQs

In order to obtain the greatest benefit from these notes

1) read through them carefully
2) attempt the MCQs
3) do not look at the answers until after you have completed the test

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1. Hypertrophy may be defined as
   (a) an increase in the size of cells
   (b) a decrease in the size of cells
   (c) an increase in the number of cells
   (d) a decrease in the number of cells

2) Routine measurement of a 40 year-old patient's blood pressure gave one casual reading of 150/95. The correct course of action is to
   (a) refer the patient to the GP
   (b) ask the patient to return for a routine eye examination in two years
   (c) re-schedule the patient for a further BP reading in the near future
   d) ask the patient to lose weight

3) A possible cause of "choked disc" is
   (a) a space occupying lesion in the brain
   (b) papillitis
   (c) phaechromocytoma
   (d) all of the above

4) Cotton wool spots may best be described as
   (a) lipid
   (b) soft exudates
   (c) retinal ischaemia and axonal debris
   (d) areas of plasma leakage

5) Banking of a retinal venule may best be described as
   (a) grade 3 arteriosclerosis
   (b) a larger venular lumen on the distal side due to embarrassment to venous flow secondary to arteriosclerotic impingement by an arteriole
   (c) a sign of vascular hypertension
   (d) a spasm of a venule

6) A 60 year-old female presents complaining of feeling unwell for the last few weeks and of an episode of transient visual loss and headache. Her VAs are 6/4 with each eye and visual fields are full. Intraocular pressures are 21 mmHg in each eye and anterior chamber angles are Grade 3 open. There is a family history of Type 1 diabetes. Her mother had glaucoma.

   Your correct course of action is to:
   (a) request she return immediately if visual symptoms reoccur
   (b) refer her to her GP requesting an examination to rule out diabetes
   (c) refer her to her GP suggesting an ophthalmological opinion

concerning suspected glaucoma

(d) refer her to her GP suggesting a medical examination including erythrocyte sedimentation rate

Answers

1. a
2. c
3. d
4. c
5 d
6 d