Classification

• Primary/Essential hypertension:
  – 90-95% of cases.
  – Unknown etiology
  – Associated with my risk factors

• Secondary hypertension
  – Secondary to underlying organic pathology
  – 5-10%
  – Most renal pathology – e.g. glomerulonephritis
  – Endocrine causes – e.g. hyperthyroidism, pheochromocytoma
  – Neurogenic – e.g. acute stress such surgery, ICP
Essential Hypertension

• Many risk factors interact and play role in its pathogenesis.

• Determinants of essential hypertension:
  – Family history – high risk in those with +ve Hx

• Environmental factors:
  – Dietary sodium intake: high risk with high intake
  – Stress – high stress related to high risk
  – Other factors: obesity, smoking, physical inactivity
Target Organs for Complications

- CVS (Heart and Blood Vessels – big & small vessels)
- The kidneys – essentially it is a vascular pathology
- Nervous system
- The Eyes
Effects On CVS

- Heart: Ventricular hypertrophy, dysfunction and failure.
- Conduction: Arrhythmias
- Small vessels: Coronary artery disease, Acute MI
- Large vessels: Arterial aneurysm, dissection, and rupture.
Vascular Pathology

- Hypertension accelerates atherogenesis
- Induces **hyaline arteriosclerosis**
- Induces **hyperplastic arteriosclerosis**
  - Related to more acute or severe hypertension
  - Characteristic in malignant hypertension (but not limited to it)
Effects on The Kidneys

- **Glomerular sclerosis** leading to impaired kidney function and finally end stage kidney disease.
- **Ischemic kidney disease** especially when renal artery stenosis is the cause of HTN
Nervous System

- Acute event: Stroke, intracerebral and subarachnoid hemorrhage.
- Chronic hypertension: Cerebral atrophy and dementia
Pathological changes in stroke

Massive hypertensive hemorrhage into a lateral ventricle

Hypertensive hemorrhage in the pons
The Eyes

- Retinopathy: retinal hemorrhages and impaired vision.
- Vitreous hemorrhage, retinal detachment
- Neuropathy of the nerves leading to extraocular muscle paralysis and dysfunction
Results of Essential hypertension

- Eye – hypertensive retinopathy
- Cardiac:
  - LVH & cardiac failure
  - IHD
  - Stroke: hemorrhagic stroke
  - Conduction abnormalities
Hypertensive Retinopathy

Hypertensive Retinopathy

Image from: www.pathology-india.com
# Retina Normal and Hypertensive Retinopathy

<table>
<thead>
<tr>
<th>Normal Retina</th>
<th>Hypertensive Retinopathy</th>
</tr>
</thead>
<tbody>
<tr>
<td>![Normal Retina Image]</td>
<td>![Hypertensive Retinopathy Images](A: Hemorrhages, B: Exudates (Fatty Deposits), C: Cotton Wool Spots (Micro Strokes))</td>
</tr>
</tbody>
</table>

### Hypertensive Retinopathy Features:
- **A**: Hemorrhages
- **B**: Exudates (Fatty Deposits)
- **C**: Cotton Wool Spots (Micro Strokes)
### Classification of Hypertensive Retinopathy

<table>
<thead>
<tr>
<th>Grade</th>
<th>Classification</th>
</tr>
</thead>
<tbody>
<tr>
<td>Grade I</td>
<td>Mild generalized retinal arteriolar narrowing or sclerosis</td>
</tr>
</tbody>
</table>
| Grade II | Definite focal narrowing and arteriovenous crossings  
Moderate to marked sclerosis of the retinal arterioles  
Exaggerated arterial light reflex |
| Grade III | Retinal hemorrhages, exudates and cotton wool spots  
Sclerosis and spastic lesions of retinal arterioles |
| Grade IV | Severe grade III and papilledema |
Hypertensive Retinopathy - Grade 4

- Flame haemorrhage
- Hard Exudates
- Cotton Wool Spot
- Papilloedema

Image from: www.theeyepractice.com.au
This left ventricle is very thickened (slightly over 2 cm in thickness), but the rest of the heart is not greatly enlarged. This is typical for hypertensive heart disease. The hypertension creates a greater pressure load on the heart to induce the hypertrophy.

Image from: http://library.med.utah.edu/
The left ventricle is markedly thickened in this patient with severe hypertension that was untreated for many years. The myocardial fibers have undergone hypertrophy.
LVH

- Pressure hypertrophy due to ventricular outflow obstruction.
- Left ventricle wall grossly enlarged.
Normal heart

Pressure hypertrophied heart

Dilated heart

Increased mass with thin walls
Enlargement of cardiac muscle figures

Case B: Recent and healed myocardial infarction with hypertrophy

Ref: interactive pathology manual, UTAS.
Hypertensive vascular changes

• 2 forms of small blood vessel diseases:
  – Hyaline arteriosclerosis
  – Hyperplastic arteriosclerosis

• Large vessel diseases:
  – Aortic dissection & Aneurysm
  – Cerebrovascular haemorrhage
Aortic Dissection - Macro

Right carotid artery compressed by blood dissecting upwards

Image from: http://library.med.utah.edu/WebPath
Aortic Dissection - Micro

Image from: www.library.med.utah.edu/WebPath
Aneurysm

Image from: www.quizelet.com

Aortic aneurysm with organised blood clot
Atherosclerosis

- Most frequent cause of vascular disease worldwide.
- Characterised by: fibrous plaques or atheromas in intima or arteries affecting coronary arteries, carotid arteries, circle of Willies, large vessels of lower limbs, renal and mesenteric arteries.
- Plaques have a central core of cholesterol & cholesterol esters, lipid-laden macrophages or foam cells, calcium and necrotic debris.
- Core covered by a subendothelial fibrous cap made up of smooth muscles, foam cells, fibrin and coagulation proteins, collagen, elastin, glycosaminoglycans, proteoglycans and ECM.
Atherosclerosis

• Plaques maybe complicated by: Ulceration, haemorrhage into plaque or calcification of plaque, thrombus formation at the site causing obstruction to blood flow or embolization of an overlying thrombus/plaque material.

• Consequences of atherosclerosis: IHD, MI, stroke, ischaemic bowel disease, peripheral vascular occlusive disease & hypertension.
Normal Histology Review

ATHEROMA: MORPHOLOGY AND EFFECTS

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Pathology Department
University of Newcastle upon Tyne

Return to Cardiovascular Pathology Index Page
Atheroma with intraluminal thrombus
Case 6: Atherosclerosis

Gross Pathology

The mounted specimen consists of the lower thoracic and abdominal aorta. *It shows multiple variable sized atheromatous plaques which become confluent in the abdominal aorta.* These plaques are well circumscribed, slightly raised and yellow/white in colour. Some of the larger plaques are complicated by superficial ulceration with adherent thrombus and focal dystrophic calcification.
...multiple variably sized atheromatous plaques which become confluent in the abdominal aorta.
Case 6: Atherosclerosis

core of necrotic cell debris, haemorrhage, cholesterol clefts and foam cells

fibrous cap
Case 6: Atherosclerosis

1. history
2. macro
3. slide
4. micro
5. comment

- Collagen
- Macrophages
- Proliferating smooth muscle cells
- Cholesterol clefts
- Foam cells
Case 6: Atherosclerosis

1. history
2. macro
3. slide
4. micro
5. comment

- foam cells
- cholesterol clefts
Case 8: Recent and healed myocardial infarction with hypertrophy.

atherosclerosis involving epicardial coronary vessels
Case 7: Recent myocardial infarction

- **History**
- **Macro**
  - LAD Infarct
  - Hyperaemia
  - Mural thrombus
- **Comment**
the myocardial fibre outlines can be recognised and most fibres lack nuclei
Case 7: Recent myocardial infarction

viable and degenerate neutrophils

the myocardial fibre outlines can be recognised and most fibres lack nuclei
striations are still recognisable but appear fragmented and the cell cytoplasm of the necrotic cardiac fibres is more eosinophilic than the surviving fibres
irregular areas of virtually acellular, dense connective tissue (scar tissue)
Case 8: Recent and healed myocardial infarction with hypertrophy

- Associated neutrophilic infiltrate
- Muscle fibres with increased eosinophilia, loss of nuclei
Case 8: Recent and healed myocardial infarction with hypertrophy

organising granulation tissue

scar tissue
Case 8: Recent and healed myocardial infarction with hypertrophy

accumulation of brown granules at the nuclear poles of the cardiac muscle fibre cytoplasm
Case 8: Recent and healed myocardial infarction with hypertrophy

adipose cells are seen extending as finger-like projections between the muscle bundles
Hypertensive Nephropathy

**Benign Nephrosclerosis.** The smaller arteries in the kidney have become thickened and narrowed. There is patchy ischemic atrophy with **focal loss of renal parenchyma** that gives the surface of the kidney the **characteristic granular appearance** as seen here.
Vascular Changes in Kidneys

- **Hyaline arteriosclerosis:**
  - Arterioles show homogenous, pink hyaline thickening with associated luminal narrowing.
  - Due to increased smooth muscle cell matrix synthesis.
  - Due to protein leakage across injured endothelial cells

- **Hyperplastic arteriosclerosis:**
  - Exhibit “onion skin lesion”.
  - Concentric laminated thickening of walls & luminal narrowing.
  - Laminations consists of smooth muscle cells with thickened BM
Vascular Changes in Kidney

High Blood Pressure: The Microvascular Lesions

Healthy Arteriole

Hyaline Arteriole

diabetes, radiation, FSGS

Hyperplastic Arteriole

HUS, radiation, scleroderma, malignant, ↑BP

Necrotic Arteriole

malignant, ↑BP

Healthy Small Artery

Fibroelastic Hyperplasia

also medial hypertrophy

Ref: www.pathguy.com
Benign Nephrosclerosis (Hyaline arteriosclerosis)

Recall: Hyaline refers to pink, acellular proteinaceous material. Glassy appearance.

Ref: medicalchoices.blogspot.com
Onion skin lesion

Hyperplastic arteriosclerosis

Laminated smooth muscle cells with thickened BM

Ref: lookfordiagnosis.com
Pathological changes in hypertension: Hyperplastic Vs Hyaline Arteriosclerosis in arterioles

Hyperplastic arteriosclerosis

concentric, laminated, “onion-skin” thickening of the arteriolar walls

Hyaline arteriosclerosis


Patient Evaluation Objectives

- To assess lifestyle and identify other cardiovascular risk factors or concomitant disorders that may affect prognosis and guide treatment
- To reveal identifiable causes of high BP
- To assess the presence or absence of target organ damage and CVD
(1) Cardiovascular Risk factors

- Hypertension
- Cigarette smoking
- Obesity (body mass index ≥30 kg/m²)
- Physical inactivity
- Dyslipidemia
- Diabetes mellitus
- Microalbuminuria or estimated GFR <60 mL/min
- Age (older than 55 for men, 65 for women)
- Family history of premature cardiovascular disease (men under age 55 or women under age 65)
(2) Identifiable Causes of HTN

- Sleep apnea
- Drug-induced or related causes
- Chronic kidney disease
- Primary aldosteronism
- Renovascular disease
- Chronic steroid therapy and Cushing’s syndrome, other endocrine disorders
- Pheochromocytoma
- Coarctation of the aorta
- Thyroid or parathyroid disease
Laboratory Work Up

- ECG/Echo.
- Urinalysis.
- Blood glucose and hematocrit; serum potassium, creatinine (or estimated GFR), and calcium.
- HDL cholesterol, LDL cholesterol, and triglycerides.
- Optional tests
  - urinary albumin excretion.
  - albumin/creatinine ratio.
End

References:
Robins Pathological Basis of Diseases
Interactive Pathology CD, UTAS.

Seminar notes available at:
www.pathologyatsmhs.com