# **CEREBRALVASCULAR DISEASE**

Dr Rodney Itaki Lecturer Anatomical Pathology Discipline Division of Pathology



University of Papua New Guinea School of Medicine & Health Sciences Division of Pathology

# **Cerebral vascular Disease**

## ← Definition of term:

- The term cerebrovascular disease designates any abnormality of the brain resulting from a pathologic process of the blood vessels.
- Sudden loss of neurological function is the hallmark of cerebrovascular disease.
- $\leftarrow$  3<sup>rd</sup> most common cause of death in affluent societies
- Its incidence increases with age and is somewhat higher in men than in women.

# Fundamentals:

- Stroke is the third leading
   cause of death in the USA (200
   000)
- Incidence 160/100 000
- HTN most common cause
- 30% associated with
   extracranial carotid stenosis
- Significant disability
   <u>\$7.5 billion (1976)</u>



Ref: Murray Shames, M.D. Teaching Slides

# **Relative Risk for Ischemia:**

- Age:
  - 10/100,000 < 45 yr
  - 1000/100,000 > 75 yr
- Hypertension: 6x
- Atrial fibrillation: 6x
- Prior stroke/TIA: 5 x
- Asymptomatic carotid bruit: 3x
- Smoking: 2x

# **Risk factors for stroke**

Systolic or diastolic hypertension
Diabetics
Hypercholesterolemia
Heart disease (afib)
Cigarette smoking
Heavy alcohol consumption
High homocystine
Oral contraceptive use

# The major types of cerebrovascular disease

#### ← Cerebral ischaemia and infarction

- Transient Ischemic Attacks
- Atherosclerotic thrombosis
- ← Lacunes
- ← Embolism
- ← Hemorrhage
- ← Hypertensive hemorrhage
- Ruptured aneurysms and vascular malformationsOther

# I、 Cerebral ischaemia and infarction ←Anatomy and pathology

- The principal pathological process is the occlusion of arteries supplying the brain.
- The two internal carotid arteries and the basilar artery form the Circle of Willis at the base of the brain.
- Efficient anatomotic device in the event of occlusion of arteries proximal to it.













# Pathology:

#### **Carotid Artery Lesion**



#### 90 % atherosclerosis



10% fibromuscular dysplasia, intimal dissection, inflammatory lesions, radiation



# Anatomy and pathology

Cocclusion leads to sudden severe ischaemia in the area of brain tissue supplied by the occluded artery, and recovery depends upon rapid lysis or fragmentation of the occluding material:

Reversal of neurological function within minutes or hours gives rise to the clinical picture of a transient ischaemic attack.

# Anatomy and pathology

When the neurological deficit lasts longer than 24 hours, it may be called a reversible ischaemic neurological deficit (RIND) if it recovers completely in a few days, or a completed stroke if there is a persistent deficit. Sometimes recovery is very slow and incomplete.

# Neurological symptoms and signs

The loss of function that the patient notices, and which may be apparent on examination, entirely depends on the area of brain tissue involved in the ischaemic process.



1. Transient Ischemic Attacks(TIA)

*Content of term* 

TIAs are brief, reversible episodes of focal, nonconvulsive ischaemic neurologic disturbance, Duration should be less than 24 h.

## **Transient Ischemic Attacks:**

reversible, painless neurologic deficit, lasting 1-5 minutes
Complete recovery < 24 h</li>



# Normal Histology Review



## Atherosclerosis:

- Similar L-R distribution
- 40% of lesions at proximal ICA
- 20% of lesions at proximal vertebral
- Aortic arch disease in 10% of patients



#### Fibromuscular Dysplasia:

- Carotid second most common site of disease
- 92% Women
- 30% of patients intracranial aneurysms
- Degenerative process involving long, unbranched medium sized vessels
- Hormonal, mechanical, unusual distribution of vasa vasorum
- Histology
  - Intimal fibroplasia
  - Medial Hyperplasia
  - Medial Fibroplasia (most common)- replacement of media
  - with dense fibrous connective tissue
  - Perimedial dysplasia (renals)

## 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 debries.
- 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 (from renal ischaemia).

## Pathogenesis of Atherosclerosis:

- Intimal injury (hemodynamics)
- Nodular deposition of fat in arterial intima
- Associated inflammatory response fibroblast, smooth muscle cell proliferation
- Slow accumulation of lipoproteins
- Calcium precipitation in the primary fatty plaque.

#### ATHEROMA: MORPHOLOGY AND EFFECTS



## ATHEROMA: MORPHOLOGY and EFFECTS

**Brian Angus** 

Pathology Department

University of Newcastle upon Tyne



Return to Cardiovascular Pathology Index Page



# Atheroma with intraluminal thrombus



# Carotid Dissection:

Traumatic disruption of intima
Compression of true lumen



# Aneurysm

Localised abnormal dilations of either arteries or veins. They can erode adjacent structure or rupture.

- Types: Atherosclerotic aneurysm, Berry aneurysms, aneurysms due to cystic medial necrosis, syphilitic (luetic) aneurysm, dissecting aneurysm or arterio-venus fistula.
- Study guide: describe the characteristics of each of the types of aneurysms. Describe the gross and micro anatomy of the different types of aneuryms.

## Atherosclerosis:

Flow reducing
Embolic
Clot
Platelets
Cholesterol debris
Thrombosis





Left: Vessels of the circle of Willis showing multiple aneurysms. Right: Inferior view of brain showing subarachnoid hemorrhage.



Subacute lateral medullary plate infarct and associated basilar artery showing atherosclerotic plaque with hemorrhage and occlusion of vessel.

## **Complex Carotid Plaques:**

- Calcification
- Loss of intimal continuity
- Ulcer formation
- Subintimal necrosis
- Plaque hemorrhage



#### Relationship between TIA & Stroke

- a harbinger of stroke (30-40% of patients with surgically accessible carotid stenosis)
- No loss of consciousness syncope
- Amaurosis fugax: embolus to ipsilateral retinal artery
- Aphasia
- Contra-lateral paralysis, paresis, paresthesias
- Stroke rate at 1,3, 5 years 23%, 27%, 45%
- Crescendo TIA's/ Stroke in evolution

# Stroke

- Brain infarction
- Hypertension common cause (common cause at PMGH)
- 50% preceded by TIA
- Embolic or thrombosis with inadequate collaterals
- Symptoms greater than 24 hours
- 1/3 resolve, 1/3 deteriorate, 1/3 remain the same



Lateral View of Cerebral Cortex
Normal (left)
Acute hemorrhagic infarct (lower left)
Remote infarct (lower right)







Brain showing superior sagittal sinus thrombosis and bilateral hemorrhagic infarcts.



Coronal section of cerebrum showing lacunar infarct in right internal capsule.



Hypereosinophilic (red) neurons indicative of recent infarction and coagulative necrosis (HE stain).

#### Sequalae of Internal Carotid Artery Occlusion:



propagation of thrombus to intracranial arteries
embolization of thrombus
both can result in cerebral infarction
outcome depends on adequacy of collateral flow:

#### Circle of Willis

# **Presentation:**

- Asymptomatic
- Transient Ischemic Attacks
- Cerebral Infarction (Stroke)

# **Clinical picture**

In general, evolution of the clinical phenomena in relation to cerebral thrombosis is more variable than that of embolism and hemorrhage.

The loss of function that the patient notices, and which may be apparent on examination, entirely depends on the area of brain tissue involved in the ischaemic process.

# **Clinical Evaluation:**

- History and Physical Exam
  - Degree, duration of symptoms
  - Extent of recovery
  - Presence of infarction on CT/MRI
- Cerebrovascular Imaging
  - Duplex
  - Angiography
  - MRA

#### Magnetic Resonance Angiography:







#### Contrast Angiography:





#### High grade ICA stenosis







# Asymptomatic:

Natural historyprogression of disease
>80% stenosis associated with 35% risk of symptoms or occlusion in 6 months



#### Duplex scan q 6-12 months

## Surveillance:



The ultimate goal of vascular testing is to identify <u>clinically</u> <u>significant</u> carotid disease, so that treatment can be applied and risk of stroke reduced.

Diagnose hypertension early and treat.

**PREVENTION IS ALWAYS BETTER THAN CURE!!** 

# Summary:

- Risk of stroke from extra-cranial carotid atherosclerosis related to stenosis severity.
- Patients with carotid territory TIAs or minor stroke; & >60% ICA stenosis benefit from surgical intervention.
- High-grade ICA stenosis (>70%) increases the risk of stroke in asymptomatic patients.

#### END

Reference: Robins Pathological Basis of Diseases.

Murry Shames, MD, Teaching slides at

http://usfvascularsurgery.com/