

# CEREBRAL VASCULAR ACCIDENT - TRANSIENT ISCHEMIC ATTACK

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## CVA

- Thromboembolic infarctions
- Cerebral and cerebellar hemorrhage
- Dissection of carotid and vertebral vessels
- Subclinical hemorrhage
- Subdural and extradural hemorrhage and hematoma
- Cortical venous and dural venous sinus thrombosis

Pathophysiologic etiologies: common

- Arterial embolism from distant site and subsequent brain infarction
- Atheromatous carotid or vertebral artery occlusion -> brain infarction
- Atheromatous arterial thrombosis within cerebral vessel -> brain infarction
- Hemorrhage into the brain

Pathophysiologic etiologies: less common

- Venous infarction
- Dissection of carotid or vertebral arteries and subsequent cerebral infarction
- Air embolism
- Multiple sclerosis: plaque of demyelination
- Mass effects of expanding lesions: brain tumor, abscess, subdural hematoma

## TIA

- Usually secondary to microemboli into brain
- Fall in cerebral perfusion
  - Cardiac dysrhythmia
  - Postural hypotension
  - Decreased flow through atheromatous carotid/vertebral arteries
- Brain tumors or subdural hematoma: both rare causes
- Stroke is averted via autoregulation: lysis of thrombus

## CLINICAL PRESENTATION

COMMON SYNDROMES ASSOCIATED WITH TIA

Amaurosis fugax: sudden transient loss of vision in one eye

- Due to passage of emboli through retinal arteries
- Obstruction may be visible via ophthalmoscope
- Often first evidence of internal carotid artery stenosis -> CVA
- Benign event in migraine

Transient global amnesia

- Transient amnesia and confusion -> complete recovery
- Lasts for several hours
- Common in age over 65
- Exact cause unknown: possibly ischemia in posterior cerebral circulation

## PRESENTATIONS

- Presentation (symptoms resolve within 24 hours)
  - Hemiparesis and aphasia most common
  - Consciousness usually preserved
  - Compromised anterior circulation/ carotid system
    - amaurosis fugax
    - aphasia
    - hemiparesis
    - hemisensory loss
    - hemianoptic visual loss
  - Compromised posterior circulation/vertebrobasilar system
    - diplopia, vertigo, vomiting
    - choking, dysarthria
    - ataxia
    - hemisensory loss
    - hemianoptic visual loss
    - transient global amnesia
    - tetraparesis
    - loss of consciousness (rare)
- Amaurosis fugax: sudden transient loss of vision in one eye
  - Due to passage of emboli through retinal arteries
  - Obstruction may be visible via ophthalmoscope
  - Often first evidence of internal carotid artery stenosis -> CVA
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## ETIOLOGY

- Clinical evidence of emboli source(s)
  - Carotid artery bruit (stenosis)
  - Atrial fibrillation or dysrhythmia
  - Valvular heart disease or endocarditis
  - Recent myocardial infarction
  - Difference in BP re: right and left brachial artery (subclavian stenosis)
- Clinical evidence of underlying disease process
  - Atheroma
  - Hypertension
  - Postural hypotension
  - Bradycardia or low C.O.
  - DM
  - Rarely: arteritis, polycythemia
  - Antiphospholipid syndrome:
    - Recurrent miscarriage
    - Thrombosis, thrombocytopenia

## DIFFERENTIAL DIAGNOSIS

- Brain tumor/space occupying lesion
- Focal epilepsy
  - Usually presents w accompanying irritative phenomenon (limb jerking)
  - Characteristic progress of events over minutes
- Migraine with facial prodrome
  - Headache common in migraine (not invariable) is rare w TIA
  - Typical visual disturbances seen w migraine are not seen in TIA

Versus TIA where maximum deficit is immediate apparent

## PROGNOSIS

- 40% of patients will progress to stroke w 5 years
- 25% of patients will succumb to CVA or CDH
- TIA in anterior cerebral circulation carries more serious prognosis than posterior circulation

## WORKUP

- *Complete* history and physical w emphasis on *neuro* exam
- **Cat scan or MRI**
- Bloodwork
  - Comprehensive metabolic: glucose, bun/creatinine, electrolytes
  - Liver function testing
  - Ca/PO4
  - Vitamin B12
  - TSH, T4, T3
  - Syphilis serology
  - HIV antibodies

## CVA

- Major cerebral infarction from thromboemboli (TE) -> CVA - small infarcts may cause TIA
- Clinical picture very variable: (f) extent and site of infarct
- Clinical deficits
  - Often inaccurate in mapping precise vascular territory
  - General site of major infarct may be determined from presentation

## CLINICAL FEATURES

- Hemiplegia is most common
  - Infarction of internal capsule -> TE of branch of middle cerebral artery
  - Total internal artery occlusion: contralateral paresis/paralysis
- Paralysis/paresis of hands and face (contralateral side to lesion)
  - Signs similar to acute complete contralateral upper motor neuron lesion
- Aphasia common when dominant hemisphere involved
- Affected limbs flaccid and are hyperreflexic
- Headache unusual
- Loss of consciousness uncommon

- Reflexes recover over days and become exaggerated
- Extensor plantar response appears
- Deficits are maximal at first; recovery is gradual over days, weeks, months

#### **SPECIFIC PATTERNS OF INFARCTION**

##### **- Brainstem infarction**

Causes complex patterns of dysfunction (f) site of lesion and relationship to cranial nerve nuclei, long tracts and brainstem connections

- **lateral medullary syndrome** (Wallenberg's syndrome)
  - aka *posterior inferior cerebellar artery (PICA) thrombosis*
  - caused by PICA or vertebral artery TE
  - vertigo, vomiting, dysphagia, unsteadiness, contralateral loss of pain and temperature sensation on face
- **coma**: bilateral brainstem infarction damages reticular formation
- **locked-in syndrome**: upper brain stem infarction

State of unresponsiveness due to massive brainstem infarction. The patient has functioning cerebral cortex, and thus is aware, but cannot move or communicate except by vertical eye movement

##### **- pseudobulbar palsy: (PBP)**

- Bilateral supranuclear (UMN) lesions of the lower cranial nuclei producing weakness and poverty of movement of the tongue and pharyngeal muscles.
- Findings: stiff, slow, spastic tongue (which is not wasted), dysarthria with a stiff, slow, spastic voice which sounds dry and gravelly, and dysphagia. The gag reflex and palatal reflex are preserved. The jaw jerk is exaggerated. Emotional lability (inappropriate laughing or crying) often accompanies
- Causes
  - Motor neuron disease where there are often both UMN and LMN lesions (elements of both PBP and bulbar palsy)
  - M.S. where lesions occur mainly as late event
  - Cerebrovascular disease where it may occur with MID
  - following severe head injury
- Different from similar findings with Parkinson's disease  
great difficulty w swallowing, dysarthria and slow-moving tongue also develop in late stages of Parkinson's disease which is etiologically different from PNP and bulbar palsy

**- Lacunar infarction (LI)**

- Small areas of infarction seen on MRI or autopsy
- HTN commonly present
- Minor strokes typically caused by LI
  - Pure motor stroke, pure sensory stroke, sudden unilateral ataxia and sudden dysarthria with a clumsy hand
- May be symptomless

**- Hypertensive encephalopathy**

- Neurological sequelae of severe accelerated HTN with occlusion of small arteries
- Severe headaches, TIA, CVA, subarachnoid hemorrhage (rare)
- Papilledema may develop
  - Due to ischemic optic neuropathy or following brain swelling from multiple acute infarcts

**- Multi-infarct dementia (MID)**

- Multiple lacunas or large infarcts result in generalized intellectual loss
- Seen in pts with advanced cerebrovascular disease
- Occurs stepwise progression months/years w each subsequent infarct
- Dementia, pseudobulbar palsy
- Shuffling gait w small steps (*marche a petits pas*)
- May confuse w Parkinson's disease
  - Called *atherosclerotic Parkinsonism* in the past

**- Binswanger's disease**

Low attenuation areas in cerebral white mater on CT, with dementia, TIAs and stroke episodes in patients who are hypertensive

**- Infarction of the visual cortex**

- Combos of hemianopic visual loss, cortical blindness and Anton's syndrome follow infarction of the posterior cerebral arteries and middle cerebral branch to the macular cortex

**- Weber's syndrome**

- Ipsilateral third-nerve paralysis
- Contralateral hemiplegia due to a lesion in 1/2 of the midbrain
- Paralysis of upward gaze - due to lesion in region of red nucleus

**- Watershed infarctions**

- Multiple cortical infarcts
- Results from periods of prolonged lower cerebral perfusion
- Common precipitant: Hypotension from massive MI or CABG
- Border zones between areas supplied by ACC, MCC and PCA affected
- Cortical visual loss, memory loss and intellectual impairment

## EXAMINATION

- Neuro exam
- Exam for source of emboli
  - Carotid bruit
  - Atrial fibrillation
  - Valve lesions/ endocarditis
- Identify hypertension or postural hypotension a factor
- History of other emboli or TIA
- Brachial BP in each arm: difference of > 20 suggests *subclavian artery stenosis*

## CLINICAL MANAGEMENT

### Immediate management

- Thrombolytic therapy
- Admission to specialized stroke unit has better prognosis than general unit

### Long term management

- Antihypertensive therapy
- Antiplatelet therapy
- Anticoagulants: indications
  - Atrial fibrillation or paroxysmal dysrhythmias
  - Uninfected cardiac valve lesions
  - Cardiomyopathies

Note: wide controversy in use in clinical practice; potentially dangerous in 2 weeks following CVA due to risk of provoking cerebral hemorrhage

- Treatment of polycythemia if present
- Antispasmodics (Baclofen - a GABA agonist) may be helpful w spasticity

### Surgical management

- Internal carotid endarterectomy
  - ICA stenosis of > 70%
  - Risk further TIA/CVA reduced by 75% if surgery is successful
  - Mortality rate is 3%
  - Not recommended for stenosis less than 70% or if an incidental finding
- Extracranial-intracranial bypass is non-therapeutic and obsolete

Anastomosis of superficial temporal artery (external carotid) through skull-burr-hole to a cortical branch of middle cerebral artery (internal carotid)

### Rehabilitation

- PT particularly valuable in first few months
- Speech therapy following aphasia
- Occupational therapist

- Depression and loss of self esteem very common - devastating event
- Various ADL aids available
  - Stair rails, toilet adaptations, sliding boards, wheelchairs, tripods, modification of doorways, sleep arrangements, stair lifts, kitchen modifications

### PROGNOSIS

- 1/3-1/2 who die do so in first month
- Early mortality lower from thromboembolic infarction (<1/4) than for intracerebral hemorrhagic events (>3/4)
- Poor outcome likely w coma, defect in conjugate gaze, sever hemiplegia
- Recurrence is common (10% in first year)
- Many die of subsequent MIs
- Initial CVA survivors: 30%-40% alive after three years
- Gradual symptom improvement follows; severe residue deficit may persist
- 1/3 return to independent mobility; 1/3 require permanent institutional care
- Language recovery
  - Good prognosis for recovery of fluent speech where sufficient language to be intelligible after 3 weeks
  - Residue word-finding difficulties are common.

### ADDITIONAL COMMENTS (applies to CVA/TIA)

Source of Thrombus in CVA/TIA (main source outside brain -70% TIA; 80% CVA)

- Thrombus and atheromatous plaques
  - Great vessels
  - Carotid and vertebral systems,
  - Heart
- Mural/valvular thrombi following atrial fibrillation
- Carotid artery

### Risk Factors

- HTN
- Smoking
- Sedentary lifestyle
- Excess consumption ETOH
- hyperlipidemia - LDL
- Diabetes
- Obesity
- Ethnicity: African Americans at higher risk
- High dose OCs (no longer in use; low dose OCs no increased risk)

### Prevention

- Statin therapy for hyperlipidemia
- ASA q d (now additional agents are available e.g. Plavix)
- Wt loss, control HTN, smoking cessation
- HRT thus far no definitive benefit but studies in progress