DIAGNOSIS AND MANAGEMENT OF HEMIPLEGIA

INTRODUCTION

Hemiplegia is paralysis of one half of the body—which includes arm, leg and often face on the affected side.

Terms used to describe weakness
Hemiplegia: Total Paralysis on one side of the body
Hemiparesis: Weakness on one side of the body.

MAIN POINTS: (Hemiplegia)

- Usually acute in onset
- Results from upper motor lesion/ most commonly pyramidal tract lesion
- Associated symptoms and signs aid the diagnosis of level of the lesion
- A detailed history taking is of great value
- Initially weakness is flaccid; later-spastic
- Diagnostic Features:
  - Involvement of one half of the body, hypertonia, hyper-reflexia, extensor plantar- response and characteristic hemiplegic gait.
- Speedy reach to hospital at the earliest warning signs is a must.
- Treatment of risk factors- ideal for prevention

Chart 1. HIGHLIGHTS
1A. Points in favor of UMN lesion:

1. No muscle wasting
2. Groups of muscles are involved.
3. Hypertonia
4. Exaggerated deep tendon reflexes
5. Extensor plantar
6. Abdominal and cremastric reflex absent.

1B. Site of the lesion in hemiplegia:

A unilateral lesion of the cortico spinal tract, results in hemiplegia on contralateral side.

So to diagnose and treat patients with hemiplegia knowledge about the pathways that control - the movements of the body is required.

1. Bi. Anatomy of the pyramidal tract (fig.1)

Origin and course of pyramidal tract:
- Originates from Betz cells (in 5th layer) of motor cortex – area 4
- Pyramidal tract also receives fibers from supplemental motor area 6 and from parietal lobe areas 1, 2, 3, 5 and 7.
- The fibers then pass through - Corona radiata,
- Next through, internal capsule i.e. through genu and anterior 2/3 of posterior limb of internal capsule, thence through
  - Cerebral peduncle of midbrain–( through the middle 3/5th ) and enters
  - Pons – in pons the compact bundle is broken into number of small bundles by pontine nuclei and transverse ponto cerebellar fibers and then the tract passes on to
  - upper medulla – forming again a compact tract
  - In the lower medulla most of fibers of pyramidal tract decussate to form the crossed pyramidal tract which descends in –
  - The lateral column of spinal cord ; 90% fibers pass through lateral column after crossing in the lower medulla. 10% fibers do not cross – descend ipsilaterally as uncrossed fibers in anterior funicle
  (Rarely all fibers cross).
- **Termination**: axons terminate or synapse in internuncial neurons which in turn synapse with motor cells in anterior horn cells. Throughout the brain stem, pyramidal tract gives out *cortico bulbar fibers* to opposite side which end in nuclei of cranial nerves of opposite side.
1. B ii. Anatomical diagnosis in hemiplegia
Diagnosis of anatomical site of the lesion in hemiplegia depends upon associated
-clinical features of involvement of other ascending /descending tracts and nuclei

**Features of hemiplegic lesions at various levels:**

1. **cortical lesions:**
   - Loss of consciousness
   - Monoplegia or hemiplegia with predominant
     - involvement of one limb
   - Cortical sensory loss
   - Fits can occur
   - Flaccidity
   - Aphasia when dominant hemisphere involved
   - Visual field defects

2. **Corona radiata:** same features as above but denser hemiplegia

3. **Internal capsule:**
   1. dense motor hemiplegia with upper motor neuron type of
      facial palsy
   2. **But emotional facial movements are retained despite facial palsy**
   3. no fits, no visual field defects and no sensory symptoms
      usually.

4. **Brain stem lesions:**
   1. Typically cause ipsilateral cranial nerve palsy and contra lateral
      hemiplegia. This condition being known as crossed hemiplegia
      - **Mid brain:** example of crossed hemiplegia –e.g. Weber’s syndrome
      - **Pons:** crossed hemiplegia –e.g. Millard –Gubler’s syndrome
      - **Medulla:** lower cranial nerve involvement

*Learning point:*

*Internal capsular lesion- causes contra lateral hemiplegia*
Brain stem lesion causes Crossed hemiplegia

Cortical lesion causes contra lateral flaccid Monoplegia

Lower medulla lesion below the decussation: causes ipsilateral spastic hemiplegia without facial involvement

1. C. Etiological diagnosis in hemiplegia (FIG.2)

Causes of hemiplegia:

- Congenital
- Acquired

**Congenital:**
- atrophy of the lobe, cerebral agenesis, porencephaly

**Acquired:**

**a. Vascular:** stroke- most common cause (Fig3.)
  1. Arterial: -
     a. Cerebral thrombosis
     b. Cerebral embolism
     c. Cerebral hemorrhage/Sub arachnoid hemorrhage
     d. Hypertensive encephalopathy
  2. Venous: cortical vein thrombosis /thrombophlebitis

**b. Traumatic:** subdural /epidural hemorrhage ,birth injury ,cerebral contusions

**c. Infective:** meningo encephalitis of various origins
  Common being tuberculous, pyogenic

**d. Neoplastic:** primary /secondary (Seizures common)

**e. Other space occupying lesions:**
  Cerebral abscess, cysticercosis, hydatid cyst

**f. degenerative:** disseminated sclerosis, tuberous sclerosis, epiloia

**g. Autoimmune -Collagen Vascular diseases**

(In Subarachnoid hemorrhage hemiplegia may result from,
1. Tearing in of blood into brain substance,
2. from softening of brain distal to point of rupture or
3. due to spasm of the vessel
(Diagnosis: sudden headache, unconsciousness, blood in C.S.F.)

1. D. Details in history which help in etiological diagnosis:

Sudden Onset:
Vascular - commonest cause
Trauma and demyelination- less common

Insidious onset:
Space occupying lesions
Degenerative conditions

Rapidity of onset
Cerebral embolism ----- within seconds
Cerebral thrombosis --- within hours
Cerebral hemorrhage ---- very sudden and progresses Relentlessly

Time of occurrence of hemiplegia:
During sleep or soon after rising from bed: cerebral thrombosis
During day time: cerebral embolism
During day time after exertion /emotion : cerebral - hemorrhage

Progress: Quick recovery in embolism
FIG. 2. SITES OF LESIONS AND ETIOLOGIES FOR HEMIPLEGIA

Table 1. Differential diagnosis of cerebral Embolism, Thrombosis, Hemorrhage:

<table>
<thead>
<tr>
<th>Features</th>
<th>Embolism</th>
<th>Thrombosis</th>
<th>Hemorrhage</th>
</tr>
</thead>
<tbody>
<tr>
<td>Age</td>
<td>Young</td>
<td>Old</td>
<td>Old</td>
</tr>
<tr>
<td>Onset</td>
<td>Abrupt</td>
<td>Over few hours</td>
<td>Catastrophic</td>
</tr>
<tr>
<td>Progress</td>
<td>Abrupt recovery</td>
<td>Amelioration</td>
<td>Progressive</td>
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<tr>
<td>Past history</td>
<td>CVS disease</td>
<td>TIA/stroke</td>
<td>HT/DM</td>
</tr>
<tr>
<td>Pulse</td>
<td>May be Irregular</td>
<td>---</td>
<td>---</td>
</tr>
<tr>
<td>BP</td>
<td>Normal</td>
<td>High</td>
<td>Very high</td>
</tr>
<tr>
<td>CVS</td>
<td>MS/MVPS/arrhythmia</td>
<td>Hypertension</td>
<td>Hypertension</td>
</tr>
<tr>
<td></td>
<td>Nil</td>
<td>Nil</td>
<td>Present</td>
</tr>
<tr>
<td>----------------------</td>
<td>-----------</td>
<td>-----------</td>
<td>-----------</td>
</tr>
<tr>
<td>Neck stiffness</td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>Conjugate deviation</td>
<td>Not present</td>
<td>Not present</td>
<td>Present</td>
</tr>
<tr>
<td>of eyes</td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>Pin point pupils</td>
<td>-----</td>
<td>-----</td>
<td>Present in pontine -hemorrhage</td>
</tr>
<tr>
<td>CSF</td>
<td>-----</td>
<td>-----</td>
<td>Uniformly blood stained</td>
</tr>
<tr>
<td>Prognosis</td>
<td>Good</td>
<td>Fair</td>
<td>Bad</td>
</tr>
</tbody>
</table>

Fig.3. Vascular causes of hemiplegia
Transient ischemic attack (TIA) Fig. 4

It is episodic neurological deficit which lasts less than 24 hrs; patient usually recovers within 1 hour leaving no residual deficit — generally lasts for 15 min to 1 hr. Has to be differentiated

- From migraine — by history
- From partial seizures — by EEG
- From hypoglycemia — by blood sugar estimation, altered sensorium

In migraine and partial seizures symptoms build up more gradually. All symptoms start together and reach maximal intensity within seconds in TIA.

Symptoms of TIA/ Cerebral ischemia

- Carotid territory TIA; sudden monocular blindness, hemianesthesia, hemiparesis, transient aphasia (dominant lobe affected)
- Vertebrobasilar territory:
  - unconsciousness (reticular formation) Drop — attacks
  - (Non specific dizziness on its own is not diagnostic of TIA of posterior circulation.)
  - Bilateral limb motor /sensory dysfunction
  - Diplopia, Dysarthria, vertigo, tinnitus,
Dysarthria and ataxia of cerebellar lesion
Cortical blindness / homonymous hemianopia

Natural history of TIA: 10% develop infarction in the following year
Another 10% in next year. Greatest incidence in 3-6mths after initial TIA
• **OFTEN THERE IS NO WAY TO DIFFERENTIATE TIA FROM ACUTE STROKE.**
• **DO NOT WAIT TO SEE IF SYMPTOMS GO AWAY**
• **HOWEVER RISK OF TIA DEVELOPING FUTURE STROKE CAN BE EVALUATED USING ABCD SCORE**

**Learning point:**
It is important for lay person to know and recognize stroke symptoms so that speedy reach to the hospital is made.

1. G. Stroke in evolution:

   Neurological deficit that progresses under observation (sometimes fluctuates)
   
   *Possible mechanism*: (Physical and chemical changes)
   
   A. propagating thrombus obliterating collateral branches thus enlarging territory of ischemia.
   B. progressive narrowing of the vessel by thrombus
   C. cerebral edema.
   D. enlarging intra cerebral hematoma from continued hemorrhage.
   E. emboli – propagating, migrating, lysing and dispersing.
   F. recurrent artery to artery embolisation.

1. H. Completed stroke:
   Where neurological deficit does not progress further.

1. I. Lacunar infarct:
   - Very small infarct measuring \(\frac{1}{2} - 1\)cm in diameter.
   - *Cause* – local small vessel disease – Block of local small penetrating branches of major intra cranial arteries.
   - *Resolution*: the infarct which consists of neurons and glial tissue degenerate and eventually are absorbed by activated microglial cells. Finally a cystic cavity or glial scar remains.
   - it is found in poorly controlled hypertensive.
   - Hypertension occludes these vessels by – lypohyalinisation, fibrin deposition, and micro atheroma.
   - Final outcome: Lacunar infarcts are multiple and eventually lead to, dementia and pseudo bulbar palsy.

**Recognized Lacunar syndromes:**
   1. Pure motor Hemiplegia 60% ; - Lesion in internal capsule
2. Pure sensory stroke 10%: lesion in thalamus – posterio-lateral aspect

3. Dysarthric clumsy hand syndrome: lesion in base of the pons or genu of Internal capsule. Features: Dysarthria, clumsiness of hand, facial weakness (contralateral)

4. Ataxic hemiparesis: Hemiparesis (or leg weakness) with ipsilateral ataxia. Severe dysarthria with facial weakness

**2. Blood supply of internal capsule (fig. 5 and fig 5a.)**

Upper half: supplied by Middle cerebral artery (MCA) through its Lenticulo striate branches

Lower half: 1st part by anterior cerebral

Middle part - by posterior communicating branch of Posterior cerebral artery

3rd part – anterior choroidal branch of MCA
(Through their lenticulo striate branches.)

Mnemonic: **APC** (Antr. Cerebral, Posterior communicating, Choroidal-antr)

![Blood supply of internal capsule](image)

**Fig 5. Blood supply of internal capsule**
**Key Neuro anatomical point**

Major source of blood supply to internal capsule is through several small branches given off -from circle of willis (called perforate branches)

They are named **LENTICULO STRIATE ARTERIES**

These small arteries are different from large cerebral vessels functionally.

They are end arteries and do not anastomose

They may occlude and cause cerebral infarct or rupture causing cerebral hemorrhage.

A relatively small lesion may cause major motor/sensory deficit on the opposite side

This is because the fibers of internal capsule are densely packed

They enter the brain by perforating the gray matter: hence the name perforating branches

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![Image of blood supply of internal capsule]

**Fig 5a. Blood supply of internal capsule**

Upper half: Middle cerebral artery

Lower half: Anterior cerebral, Posterior communicating and choroidal
3. Structures passing through internal capsule: (fig6)

Internal capsule contains largely the descending 1. cortico spinal and 2 cortico bulbar fibers 3. Non pyramidal motor tracts and 4. ascending thalamo cortical fibres.

Non Pyramidal motor tract are
• fibers from cortex to cerebellum via pons,
• and from cortex to striatum (thalamus, caudate nucleus, putamen, substantia nigra, red nucleus, brain stem reticular formation).
• These pathways are adjacent to pyramidal tract and so can be involved in lesions of pyramidal tract.

Anterior limb- Fronto pontine and Thalamo cortical fibers mainly.

Genu: CORTICO BULBAR tract – passes through genu


The retro lenticular part contains fibers from auditory radiation

Posterior to this - travel fibers of optic radiation

4. CAUSES OF VASCULAR DISORDERS RESULTING IN HEMIPLEGIA-

4a. Causes of cerebral thrombosis (Fig8)

Commonest- atherosclerosis of intra cerebral vessels
Others causes: blood disorders, Polycythemia, sickle cell anemia
Antiphospholipid syndrome, Hyperhomocysteinemia
Oral contraceptives,
Dehydration in extremes of age, slowing of blood stream

Fig 7. Cerebral thrombosis

Fig 8. Cerebral Hemorrhage
4. b. Causes of cerebral hemorrhage: (Fig8)
Hypertensive hemorrhage /Subarachnoid hemorrhage
Other hemorrhages-
- atherosclerotic aneurysm
- mycotic aneurysm,
- vascular malformation

4. c. Causes of cerebral embolism: (Fig9)
Source of emboli-A.F, Valvular heart disease
Thrombosis of great vessels including common carotid bifurcation /arch of aorta
In the heart: LA thrombus, LA myxoma, infective Endocarditis, and myocardial thrombus

4. d. Cerebral venous thrombosis
   
   - d. Bilateral involvement common
   - e. Convulsions –50%
   - f. Common in post partum females
   - g. CSF is hemorrhagic
   - h. CT scan shows delta sign

4. e. Causes of bilateral stroke;

   - Venous thrombo phlebitis
   - Moya –moya disease
   - Arteritis –TB, syphilitic
Multiple brain abscesses.

**RISK FACTORS IN HEMIPLEGIA**

Modifiable risk factors
- Hypertension
- Diabetes mellitus
- Ischemic heart disease
- Obesity
- Hyperlipidemia
- Smoking
- Use of oral contraceptives
- Prior stroke, TIA

Non modifiable risk factors:
- Heredity, male gender, increasing age, Asian/African American descent

**COMPPLICATIONS**

**Acute:**
- Coning due to cerebral edema
- Thrombus propagation to occlude collateral vessels
- In embolic stroke – recurrent embolism
- In ischemic stroke hemorrhage into infarcted area
- Cerebral edema especially in cerebellar infarct
- Seizures, when hemorrhage extends to cortical white matter junction

Increased ICT is *most dangerous complication*—manifests with altered sensorium, unequal pupils, XI nerve palsy, papilloedema and periodic breathing

**Late complications:**

Urinary tract infection, pneumonia
Bed sores
Deep vein thrombosis
Septicemia
Contractures

**INVESTIGATIONS**

In cases of stroke ‘**time is brain**,’ and thus loss of time due to an extensive diagnostic work-up is contraindicated

**IMAGING STUDIES:**

Urgent imaging studies are done to differentiate cerebral ischemia from cerebral hemorrhage.

**CT brain**

Immediately excludes hemorrhage  
Can detect cerebral edema  
Smallest infarct that can be made out –0.5 –1 cm  
Demerits: cannot detect most infarcts for at least 24 hrs  
  *Does not detect lesions in cortical surface or brain stem*

**MRI:**

More sensitive than CT in detecting ischemic stroke; better resolution;  
Within hours, infarct can be detected including infarcts in posterior fossa, cerebral surface and lacuna infarcts of less than ½ cm  
Hemorrhagic infarcts can be made out.  
Blood flow in many intracranial arteries may be imaged.  
Sections in all planes possible; can diagnose demyelinating diseases  
Demerits: Expensive, Not available all 24hrs; claustrophobia

3. **Positron emission Tomography**  
Advantage: assesses the function of the neurons

4. **SPECT**: *Single Proton Emission Computerised Tomography*

5. **DSA; Digital Subtraction Angiography of cerebrum**
Usually performed by retrograde femoral artery catheterization

- Four vessel cerebral angiography if indicated in TIA
- MR angiography in Vertibro basilar involvement

Other studies

6. Spinal tap: To diagnose encephalitis or multiple sclerosis

7. ECG, ECHO, Holter monitor: for evaluation of suspected embolic stroke TIA.

8. Coagulation studies: including PT and PTT – especially in patients receiving anticoagulant or thrombolytic therapy and in patients with cerebral hemorrhage.

9. Look for hypercoagulable states in young.

10. Cardiolipin antibodies, lupus inhibitors if antiphospholipid syndrome is suspected.

11. Routine blood tests; blood count, Hb, sugar, urea, creatinine
12. Serum electrolytes
13. X-ray chest-to detect cardiac disease, lung abscess, empyma
14. Non invasive carotid tests:, Carotid ultrasound techniques, Ophthalmo dynamometry, Oculo plesthismography ‘Directional supra orbital Doppler-’Trans cranial Doppler’
15. Blood culture

TREATMENT

I. Treatment of STROKE: (CHART 2)

4main features in treatment

- Quick transport to hospital, assessment, and CT scan in one hour
- Dissolving the blood clot with thrombolytic t-PA in 3hrs.- (if t-PA criteria are met.)
- Also treat the risk factors- hypertension, diabetes and others
• Post stroke rehabilitation started at the earliest.

*Pearls for practice:*

In patients presenting soon enough, for t-pa therapy, time allotted for different steps are:

for physician’s examination-10 min:

(Most important thing in evaluation is time of onset of symptoms)

For CT testing-within 25 min of arrival

CT interpretation within 20 min of test completion

IV t-PA(Tissue plasminogen Activator) administration –within 3hrs of stroke onset

patient if not candidate for iv t-PA, after excluding hemorrhage on CT, should be put on aspirin at earliest indicated.

BP if persistently high above 220 syst and 120 diastole only must be treated and hyperglycemia in the range of 140-180 mg/dl.
Treatment of Ischemic Stroke:

1. Urgent restoration of blood flow; (if diagnosed within 3 hours)-With thrombolytic t-PA (tissue plasminogen activator).
   *But before that hemorrhagic stroke is to be ruled out definitely.*
   - t-PA is to be administered within 3hrs of stroke onset and
   - in less than 60 min.-of arrival to causality.

2. Aspirin or aspirin with Clopidogrel;
   *But aspirin is contraindicated* within 24 hrs of treatment with t-PA.
   Still must be given within 48 hrs of symptoms.
   After ruling out hemorrhage, and if ischemic patient is found unfit
   -for t-PA, is to be promptly put on aspirin
3. Control of blood sugar, fever, seizures
4. Hypertension is not treated immediately unless level is 220/120

Treatment of raised intracranial tension:
(a) elevate the head end, (b) Restrict fluids, (c) administer osmotic diuretics, (d) hyperventilate

Treatment of Hemorrhagic stroke

1. No specific medicine available for hemorrhage as such.
2. Drugs for control of blood pressure, cerebral edema, fever, seizures
3. Monitor for signs of raised intracranial tension; if raised treat with intravenous mannitol with or without frusemide
4. Avoid straining while coughing, passing stools, vomiting, changing posture

Surgery for hemorrhagic stroke:

a. In ‘huge’ cerebral hematoma, blood removed- to reduce ICT
b. In cases of berry aneurysm, repair by clamping off with metal clip/or by endovascular coil embolisation which blocks it.
c. Indication for surgery depends on site of aneurysm
Lacunar infarcts: Aggressive treatment of hypertension.

TIA:

- Often there is no way to differentiate TIA from acute stroke.
- Do not wait to see if symptoms go away.
- Using ABCD score, assess risk of TIA developing stroke in future.

Athero thrombotic large/small vessels

- Aspirin
- Clopidogrel
- ASA/dipyridamole

- Anticoagulation;
  - Heparin: For thrombotic stroke in evolution or in posterior circulation involvement.
  - Warfarin in Atrial fibrillation
- Antiplatelet agent: Aspirin-especially in TIA and as an alternative to anticoagulation
  - Recurrent TIA after aspirin- aspirin plus dipyridamole
- Also treat the underlying cause e.g. -anticoagulants in atrial fibrillation
- Surgical treatment: For carotid thrombosis: carotid end-arterectomy.

Education:
Patient and family is to be educated regarding-
Early stroke symptoms, risk factors, diagnostic procedures treatment options

REHABILITATION

1. Therapy by health care professionals
   - Physiotherapy to be started at the earliest.
   - Occupational therapy
• Speech therapy
• Helping to cope with psychological changes with clinical psychologist’ help

2. Gadgets, equipments and home adaptation
   Mobility aids like walking stick and wheel chair
   Adaptations at home such as ramps and hand rails
   Special equipment and gadgets to make it easier to manage tasks at home.

3. Support from family and friends is important

4. Rehabilitation in the society
   Stroke club or rehabilitation and support services in the society

STROKE PREVENTION

Primary Prevention

Treat hypertension, obesity, and hypercholesterolemia, council to stop smoking and to adapt healthy life style
Also detect and treat atrial fibrillation, administer Aspirin for stroke prevention and myocardial infarction

Secondary prevention entails

All features of primary prevention and also the following
   Carotid endarterectomy/stent (In carotid atheroma)
   Anticoagulation for cardiac emboli
   Antiplatelet therapy.