


VISUAL DYSFUNCTION IN OPTIC CHIASM SYNDROME

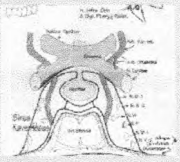
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- Optic chiasm, most important
 - Arrangement of visual fibers
 - Characteristic of visual field
- Bitemporal defects:
 - a) Superior
 - b) Inferior
 - c) Complete
 - d) Peripheral, central

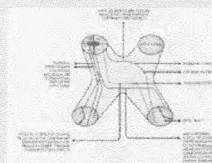
ANATOMY OF CHIASM

- Width : 12 mm
- Length : 8 mm (antero posterior)
- Inclined : 45°
- Location : anterior hypothalamus & anterior third ventricle
- 10 mm above sella
- Vascular supply :
 - Anterior communicating artery
 - Anterior cerebri artery
 - Circle of Willis



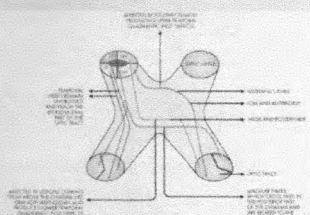
OPTIC CHIASM

- 53% fiber from nasal retina crossed to opposite — contra lateral.
- Inferior nasal fibers cross anterior loop in to contra lateral (Willbrand's knee)
- Macular fiber cross posterosuperior



VISUAL DEFECTS BY LESION DAMAGE

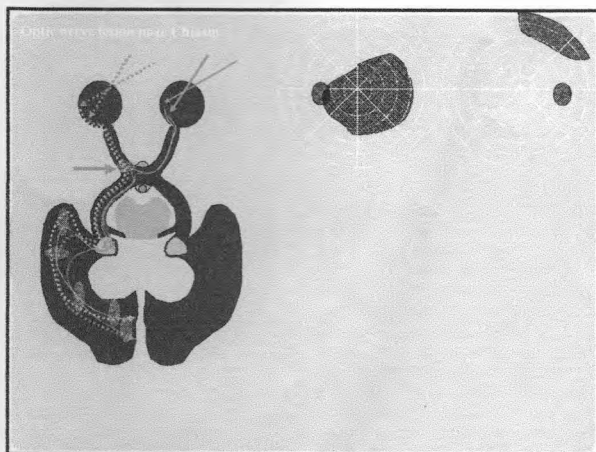
- a) Anterior angle of chiasm
- b) The body of chiasm
- c) Posterior angle of chiasm



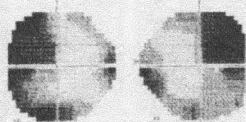
ANTERIOR ANGLE OF CHIASM

Compression to anterior angle of chiasm

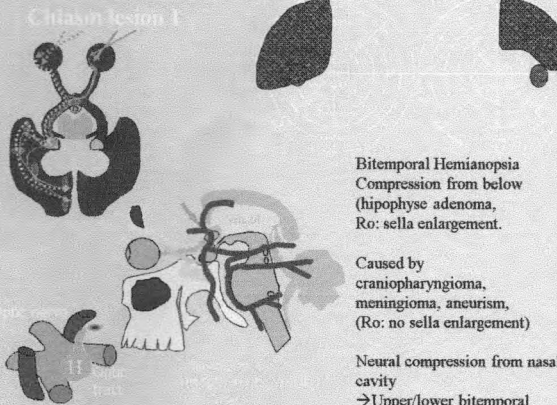
- Small lesion damages the crossing fibers of ipsilateral eye → field defect: monocular and temporal
- Damage of macular crossed fibers : monocular, temporal defects and parasentral scotoma
- Damage fiber from nasal contralateral, anterior extension : central ipsilateral scotoma and contralateral upper temporal quadrant ("Willbrand's Knee")



- Chiasmal compression from below → defects stereotyped pattern : bitemporal defect
Example: pituitary adenoma
- Peripheral fiber damage, defects begin from superior quadrants of both eyes
Can be not similar
- Similar defects causes from tuberculum sellae, meningioma, craniopharyngiomas, aneurysm




Chiasm lesion 1



Bitemporal Hemianopsia
Compression from below (hipophyse adenoma, Ro: sella enlargement).
Caused by craniopharyngioma, meningioma, aneurism, (Ro: no sella enlargement)

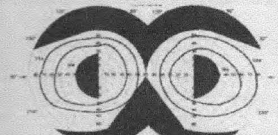
Neural compression from nasal cavity
→ Upper/lower bitemporal hemianopsia

Sella or supra sella lesion : damage superior fiber → defect bitemporal inferior
Example: angioma cavernous
meningioma, craniopharyngioma, aneurism, germinoma, glioma.
If lesion spread to third ventricle → papilledema

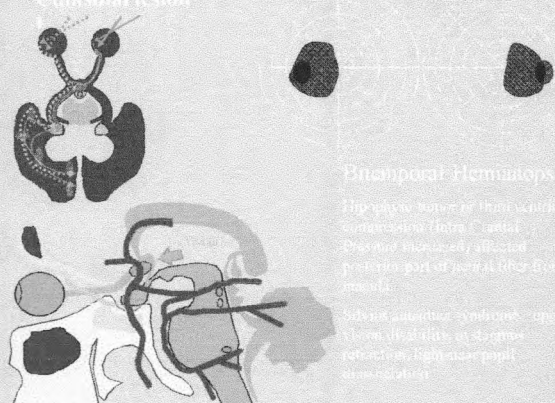


POSTERIOR ANGLE OF CHIASM

- Typical defect: hemianopsia bitemporal, cecocentral bitemporal
Visual acuity, color vision always normal
- Cecocentral defect by other causes → impairment of visual acuity and color vision



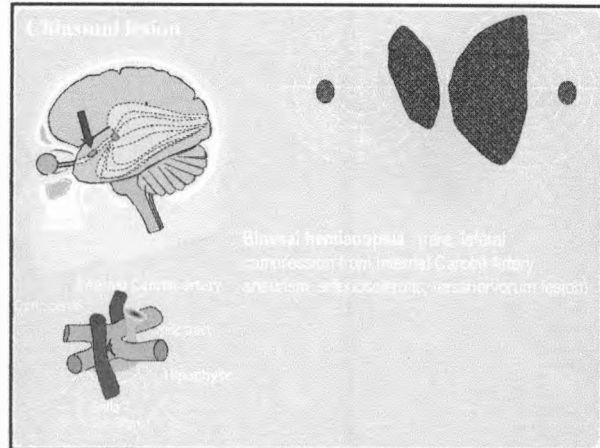
Chiasm lesion



Bitemporal Hemianopsia
The optic fibers of both sides, superior fibers from Central. Superior fibers affected posterior part of neural fiber from macula.
Bilateral parietal syndrome, optic chiasm, bilateral optic atrophy, retrochiasm, high steep papill. atrophy

DAMAGE LATERAL OF THE CHIASM

- Contralateral Homonim Hemianopsia
- Hemianopsia binasal caused by sclerotic of internal carotid arteries
- If lesion spread from optic nerve or optic tract to chiasm → ipsilateral blindness



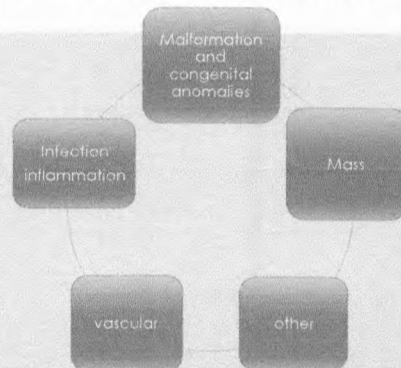
ETIOLOGY OF OPTIC CHIASM SYNDROME

Damage of optic chiasm :

- direct
- indirect
- iatrogenic

- Bitemporal field defects : 80% mass lesion

CAUSES OF VISUAL DYSFUNCTION



• Malformation and congenital anomalies

- Encephalocell
- Craniopharyngioma

• Masses

- Pituitary adenomas
- Sphenoid ridge
- Ectopic sinus cavernosus
- Glíoma
- Supra sella meningioma
- Craniopharyngiomas

• Infection and inflammation

- Chiasmal arachnoiditis
- Sarcoid
- Meningitis

• Vascular

- Compression by aneurysm internal carotid artery
- Radiation necrosis
- Dalichoectatic vessels

- Other

- a) Hydrocephalus
- b) Dilated third ventricle
- c) Empty sella syndrome
- d) Multiple sclerosis
- e) Trauma

COMMON CAUSES

- Adenoma pituitary
- Meningioma supra sella
- Craniangioma
- Glioma
- Internal artery carotid aneurism

Rare

- Multiple sclerosis
- Sarcoidosis
- SLE

UNCOMMON

- During pregnancy:
- Enlargement of pituitary Gland → compression of chiasm → visual impairment → recovery after delivery
- Adenohypophysitis limphositic
- Apoplexy pituitary

Empty sella syndrome :

- Spreading of subarachnoid to sella tursica, spontaneously or arachnoid cyst
- Chiasm disgenesis, achiasm, with congenital nistagmus
- b. Marked by bitemporal visual field loss with or without visual acuity and dyschromatopsia

Complete Hemianopsia bitemporal:
caused by optic chiasm trauma

Hemianopsia defect , causes by posterior fossa lesion.

Increased Intra Cranial Pressure and compression from third ventricle enlargement

Ventricle compression to posterior inferior chiasm :

- a) Bilateral central scotoma
- b) Bilateral nasal scotoma
- c) Arcuata scotoma
- d) Superior hemianopsia scotoma

IATROGENIC OPTIC CHIASM SYNDROME

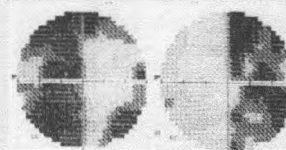
- After lesion removal or chiasm infiltration
- Radiation
- Dopamin Agonist

PATHOPHYSIOLOGY

- Chiasm was compressed : initially, lower nasal and later the upper nasal
- Ischemic → chiasm infarct : visual acuity decrease and visual field defect
Visual improvement: after compression removal
- Visual impairment will happen caused by compression (conduction block, demyelination and axon transport loss)

SIMTOMPS OPTIC CHIASM SYNDROME

- Progressive loss of central acuity
- Thing suddenly disappearing, tilting, diplopia, loss of depth perception
- Bitemporal field defect :
Complete Scotoma



Depth perception impairment

- Complain : difficulty to do activity with precision
Convergence results in crossing of two blind temporal hemifield
This produces a completely blind triangular area of field with its apex at fixation

Testing for stereopsis can also be helpful in patients with suspected chiasm disorders.



Diplopia or reading difficulty

- Caused by a horizontal or vertical deviation of images
" Hemifield slide phenomenon"
none ocular motor nerve paresis
- Diplopia may be caused by ocular motor paresis in subarachnoid or sinus cavernosus space
Pain → trigeminal affected
- Strabismus) → apoplexy of pituitary or process extrinsic of chiasm

- Hemianopsia bitemporal with optic atrophy, appearing as a band across the disc (band atrophy)

- Hemianopsia bitemporal with papilledema caused by pre and post chiasm tumor (supra chiasmal tumors like compression by third ventricle)





Certificate of Attendance

This is to Certify that

HIDAYAT, MD

Has attended as

SPEAKER

In The 37th Annual Scientific Meeting
Of Indonesian Ophthalmologist Association

Surabaya, July 5 – 7, 2012

Wimbo Sasono, MD
Chairman of The Organizing Committee



Prof. Nila F. Moeloek, MD, PhD.
President of Indonesian Ophthalmologist Association

SK PB IDI No. : 2228/PB/A.4/06/2012 :
Participant 12 SKP IDI, Speaker/Instructor 12 SKP IDI, Moderator 4 SKP IDI, Committee 2 SKP IDI