

Subject Patho 3

موضوع الدرس

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التاريخ

Malformations and developmental disorder:

Are for unknown etiopathogenesis

and studies show a relation with

1. Genetic
2. Toxic compounds
3. infectious agent.

① Neural tube defect

- defect in the central along Midline related to CNS

involve :
1. neural tissue
2. meninges
3. Bone and soft tissue

- The most common CNS Malformation

Pathogenesis : 2 types

① failure of neural tube closure
with 2ry mesenchymal tissue defect

↓
(bone + soft tissue)

Anencephaly (No brain formation)
وهو منقوص

+ Myelomeningocele

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(B) Iry bony defects with 2ry neural tube failure to close.

صنف في هادي الحالة

1] encephalocele (outpouching of brain and membranes out side skull)

2] Meningocele (outpouching of meninges)

3] spina bifida (split spine)

Morphology → Anencephaly (No brain and replaced

by ~~neurovasculosa~~ neurovasculosa)
remnant of nerve tissue + Blood vessel + meningo cells

في بعض الأحيان

ال cerebellum كبر موجود

Myelomeningocele: in lumbosacral region

Motor and sensory deficit of Lower limbs +

← Incontinence + infection supplied.

في

opening في

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encephalocele: outpouching of the meninges (often in the occipit area) and may in sphenoid area

Spina bifida: failure closure of the spine

The most NTD to occur

(مغنى محمّد مدنى بلا فقه ولا صحت المراجعة ويعيش حياة طبيعية) May be occult

Cerebral edema.

one of the most common brain condition

Lead to $\uparrow$ ICP (CRB limited Place with no elasticity)
 (أبوابه صيد خفي)

cerebral edema.

$\uparrow$ CSF volume

Expanding Mass

Subject _____ The 2 pathways are _____ موضوع الدرس

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Cerebral edema
has 2 pathways

① Vasogenic

② Cytotoxic

- اسبابه
- A. ↑ Extracellular fluid
 - B. Disruption of the BBB
 - C. paucity of Lymphatic

and it May be
Localized or generalized

↓
around
inflammation
or

↓
Global
Ischemic
injury

Neoplasm

① increase Intracellular
fluid due to cell
injury

Cell membrane
permeability
بترير

② Generalized hypoxia
or Ischemia

Swollen
neurons
filled
by

③ Metabolic
derangement

↑ amount of water

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hydrocephalus

due to $\uparrow$ CSF

CSF circulation : Choroid plexus



Lar ventricles



3rd ventricle



4th ventricle

by 2 foramen of Luschka
and Magendie foramin



Subarachnoid space



absorption by Arachnoid granulation



Sup sagittal sinus.

Causes : $\uparrow$ CSF production (rare & by tumors)

most common • $\downarrow$ CSF absorption of choroid plexus

• obstruction (lead to CSF accumulation)

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In childhood (when skull sutures are not closed yet)



Lead to enlarged head

in adult lead to ↑ IOP

• most cases are related to impaired flow and resorption

Hydrocephalus result → dilation of ventricles

↓
لو استمر لفترة طويـلة

↓
brain atrophy

hydrocephalus 2 types: (of ↓ absorption)

Obstructive

Communicating

↓
(non communicating)

↓
without obstruction

التي لا تتـوقف عنـه
absorption

↓
due to foramen obstruction

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Intracranial herniation :-

Displacement of brain tissue through the falx, tentorium and openings of the skull (هرول نقاط العنق)

- Mostly caused by mass effect

if in falx → Subfalcine.

" " tentorium → transtentorial

- ↑ Icp may compress the vasculature exacerbating edema.

(edema → Icp → Compression on venous return)

exacerbating edema ← ↑ veins hydrostatic pressure

Subfalcine :-

transtentorial



= uncal

• under falx cerebri

Cingulate Gyrus

• in Medial aspect of the temporal lobe

- unilateral expansion

(بؤفرة لا يكون في tumor & غير محيطة)

• Compression of 3rd CN

- May lead to compression of the ACA

تابع

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• Compression of 3rd CN → affects eye movement

• Compression of PCA

• Cerebral peduncle may be compressed

• and Duret hemorrhages occur

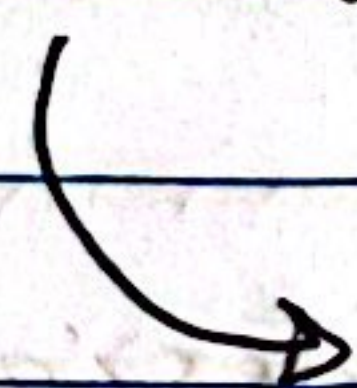
Tonsillar herniation



foramen magnum

Compression of the brain stem

Brain stem



will affect CVS and respiratory.

neurodegenerative diseases

progressive loss of particular group of neurons with shared functions

The main pathogenic mechanism:

Accumulation of protein aggregates



So neurodegenerative disease = proteinopathy



① due to mutations that alter the conformation or processing of clearance

② جمع

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↓ clearance^{or}

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② Due to imbalance between synthesis and clearance

(Related to genetic & environmental or stochastic factors)

- Accumulated protein typically resistant to degradation and show aberrant localization in the neuron.

- Sequestration of protein into

Large aggregates

Small aggregates

not toxic

toxic

لم يزل له معجزات كثيرة
 ينقل مكانها لله
 لو صغر يستقل
 ويدور لأمانه ثانية
 ينقل من
 neuron to other

Additional accumulation due to:

loss of protein function

• contribute to cell injury

May behave like a prion

↳ May be taken up by another cell and provoke additional aggregations.

The disease spread from one side to another but Not transmissible between individuals.

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Protein aggregations by microscope - shown
(as inclusions - Diagnostic)

→ "So it's Recognized as inclusions"

Alzheimer disease

- The most common cause of dementia in older adults.

- Impairment of higher cognitive functions

- Deficits in (over a course of 5-10 years)

↓

Memory & visuospatial orientation & personality
Language

and Then becomes profoundly disabled, mute and immobile

- 40% for 85-89

- 5-10% are familial

Pathogenesis

A accumulation of 2 proteins

AB

Tau

يسبب له خلل في
microtubels
cytoskeletons

Critical initiating
event

Insufficient to
induce AB deposition

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Accumulation of protein occur in specific brain regions as a result of excessive production and defective removal.

➤

Morphology

① Amyloid plaques.

Deposits of aggregated A β peptides in the neuropils.

neuropil : مَصْنَعُ دِماغ

↑ background cells

cell processes + neuroglial cells

② neurofibrillary tangles.

• Aggregates of microtubule binding protein tau

• persists after neuron death.

Both are contribute to neuron dysfunction.

Very thin gyrus
+ sulcus with
space

← ③ Cortical atrophy

Med temporal lobe

(hippocampus & amygdala & entorhinal cortex
is involved early in the course.

result to

← ④ compensatory ventricular
enlargement

③

⑤ neuritic (senile) plaques:

- Focal spherical collections of dilated tortuous, axonal or dendritic processes (dystrophic neurites) of around the central amyloid core (mainly of A β)
forms as a result of cleavage of App
- May be surrounded by clear halo
- contain tau aggregates

⑥ neurofibrillary tangles. (تَوَلُّج)

تَوَلُّج

- Microglia and astrocytes at the periphery

- Iy motor + sensory cortices are relatively spared

- All deposition of A β in brain parenchyma is considered a form of AD

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⑥ neurofibrillary tangles -

- 1 Tau containing bundles.
- 2 Displace and encircle the nucleus.
- 3 Flame shape in pyramidal neurons.
- 4 in rounder cells globose tangles
- 5 Basophilic fibrillary structures.
- 6 More clear by silver staining or IHC
- 7 Insoluble
- 8 resistant to clearance in vivo
- 9 Remain visible after neuron death
ghost or tombstone tangles.
- 10 in contrast to neuritic plaques
tangles are found in other
neurodegenerative disease.

(Non specific for Alzheimer disease)

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Parkinson disease (PD)

• neurodegenerative disease

• hypokinetic movement disorder (bradykinesia)

• Caused by loss of dopaminergic neurons from the substantia nigra



and thus, reduction of dopamine content in the striatum

Clinical: Masked faces

Stooped posture

Festinating gait (حركة سريعة بينها فترات طويلة)

Rigidity

"Pill-rolling" tremor

The diagnosis is based on the triad +

Tremor rigidity Bradykinesia.

1. absence of toxic etiologies

(المريض حتى حازم Drug able)

2. Response to L-Dopa replacement therapy

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12/7/2023

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Morphology

• pallor of the substantia nigra

• Lewy bodies.



Single or multiple cytoplasmic eosinophilic round to elongated inclusion.

found in some of the remaining neurons.

The end

12/7/2023

10:40 Am.