

Subject _____

موضوع الدرس

Date : _____

الموافق

التاريخ

Cerebrovascular disease:-

CNS diseases

3rd leading Cause of death after Cancer

May be Ischemic or hemorrhagic
end up with infarction

Stroke: A group of neurologic signs
from Ischemic changes of a part
of brain or the entire brain
And the signs lasts More than 24 hours
neurological

↓
if less than 24 hours it's called
transient Ischemic event (TIE)

[Ischemia : deficient blood supply
[hypoxia : deficient O_2 Saturation

Ischemia is More harmful.

Ischemia or hypoxia May be Global or
focal ↓

→ affect certain region

• effect The entire CNS

• Generalized event.

as: generalized long lasting
hypotension or Respiratory system failure

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Date : / / الموافق التاريخ

Manifestations related to the region affected by Ischemia or hypoxia

if defect in Motor function → hemiplegia
if " " occipital lobe → vision disturbances

Thrombosis and embolism

↓
Part of Thrombus may disconnect travel

Through blood stream

كسر ما يتصلب في مكان آخر من سبب تجلط وتنتقل
obstruction

In CV diseases embolism is More Common than Thrombosis
cerebrovascular

Hemorrhagic Causes

due to HTN
or vascular anomalies

HTN

هو السبب الأساسي

CNS hemorrhage

or
May be Congenital

بالأطفال

as
rupture of aneurysm

rupture of abnormal BV

• mostly related to adults / elderly

• More common

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Date : / / الموافق التاريخ

• brain is 1-2% of body weight and consume 20% of O_2 in body

↓
So brain is very Susceptible to Ischemia or hypoxia

• brain or CNS has a constant Blood flow due to various Autoregulation Mechanisms related to → Blood vessels resistance and Blood flow

brain or CNS is strictly dependent on Aerobic Metabolism (Another Cause Make it Susceptible to Ischemia or hypoxia)

When Ischemia/hypoxia occur & The size of the lesion determined by:

1. Duration 2. Magnitude and rapidity of reduction

Abrupt cessation is worse than gradual one

3. Excitotoxicity

Collateral blood flow

بعض مريض لتكرار

Subject

brain

موضوع الدرس

Date:

/ /

الموافق

ICA / ECA
shunts as collaterals

يأخذ من

/ /

التاريخ

Collaterals (Anastomosis)

from: C. of willis And ECA (ophthalmic branch)

and Cortical branch And leptomeningeal of ICA from ECA

Deep parts^{as} (Basal ganglia)] Lack collaterals
(brain stem)

يعني في end arteries في اليا من غير ثاني.

Excitotoxicity: when Ischemia

↓
Neurons release glutamate

neurotransmitter

↓
Cause damage of neurons
by excessive influx of Ca^{++}

Penumbra: border of Ischemia

المنطقة التي حولها ال Ischemia ليس متأثرة بشكل كامل
و هي reservable (يمكن إنقاذها)

(Area of "at risk" brain tissue)

Subject _____

موضوع الدرس _____

Date : / / الموافق

التاريخ / /

Ischemia May produce apoptosis

→ so penumbra can be rescued by antiapoptotic interventions

نرجع ثاني لموضوع ال embolism في كادر بني صغرة

more frequent in CV conditions

Embolism: Sources

① heart chambers The most common source at conditions of (rheumatic fever)

stasis due to dilation

مصدر ال embolism في القلب في حالة R.F. كالتصلب في mitral stenosis وفتح
عند dilation of Lt. atrium زيادة في حجمه
لثقل سرعة الدم وبالتالي هاد صغرة
تكوين ال Thrombus و هاد من امراض

② Valvular disease.

(Thrombus formed at valve surface)

③ Carotid artery

(atherosclerosis في كادر)

Abdominal aorta

and in some cases

Site of bifurcation

atheroma plaque May cause some changes.

Subject ingeneral DVT is the most Common Cause of Thromboembolism
Date: / / الموافق التاريخ

④ Paradoxical : من الجهة اليسرى و هيكل يعني
Lung Embolus ال
رئتي صرّوع على الجهة اليمنى الشحالة لكنه لم يفي شحاف
BU Shunt Septal defect عنده
Heart صليبه من ال rt لا لا و ثم ل brain
Side

⑤ following Cardiac surgery

⑥ tumor , Fat and air

↓ إلى ال
أثناء جراحة بالعض
+ accidents
destruction
of Limbs
embolus
or bones.

(عند BM يفل
عبر ال vein و يفل كأي Embolus)

So The Most Common source of embolus
related to Cardia Thrombi (نول 3 أسباب)

embolus Most frequently in the
Middle cerebral artery
(استد ل ICA)

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Date : / / الموافق التاريخ

Focal cerebral ischemia: Causes

① Thrombotic occlusion: Most commonly

acute plaque

changes in the

bifurcation

لaminale flow
Laminar flow
bifurcation
thrombotic event in endothelial surface

tear
or
rupture

stable atheroma plaque (stable snuere)

→ Lead to Thrombus formation

Called Acute event of atheroma plaque

fragile plaque → occlusion

embolus → partial occlusion of BV.

② Sy Inflammatory process: due to

→ Syphilis & TB

→ vasculitis (immune mediated condition)

and occlusion of

BV May take place

Subject _____

موضوع الدرس _____

Date : / / الموافق

التاريخ / /

③ hypercoagulability → increase (التخثر كثره)

~~Dissemination~~

tendency to Thrombus

(formation)

④ Dissecting Aneurysm:

↓
tear of tunica intima

BV Layer Dissect يدخل فيها الدم ويقتل
كأنه ما يدخل ما فاتت معينه وصاد عادة يبدأ
في الـ Ascending aorta ويبدأ

⑤ Drug abuse

Iv drug abuser لأنه ما في تعقيم فيه تلات فحمله أنه

bacteremia وقله نفس شالك

في (Bloodvessels أو الـ Valves) وكثره

بكتيريا زي (S. aureus) أصغر

damaged

صحة تری

normal

B.v

and

valves.

Focal cerebral

Ischemia

• Starts as nonhemorrhagic
(pale & anemic)

Then • 2ry hemorrhage due to
reperfusion (Spontaneous or by drug)

and hemorrhage is petechial (spot^{dot} like)

Subject _____

موضوع الدرس

Date : / / الموافق

التاريخ

Focal cerebral hem Ischemia:-

- (A) Grossly:
- (1) first 3 hours : No changes.
 - (2) 3 - 48 hours → pale & soft swollen brain tissue + indiscriminate white and gray Matter
 - (3) 2 - 10 days : gelatinous & friable brain tissue related to fluid texture
- due to resolution of edema ← Distinguished normal area from affected ones
- (4) 10 days to 3 weeks :-
↓
Liquefaction necrosis.
then
formation of fluid filled cavity

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Date : / / الموافق التاريخ

③ Microscopic

Acute infarction:-

- 6-12 hours:**
- 1 Eosinophilic neural necrosis
 - 2 edema
بنتون متسربة عند مركزية زيادة
 - 3 Indistinct gray/white Matter spaces
الintercellular spaces
 - 4 Disintegration of glial cells fibers and endothelium

48 hours: neutrophilic emigration
(Leukocyte recruitment)

Subacute infarct:

48 - 72 hours:

- 1 phagocytosis starts
(phagocytes + Microglia)

- 2 Reactive and proliferative astrocytes (gliosis)

granulation tissue (newly formed B.V.)
+ inflammatory condition

- 3 New blood vessels.

- 4 Astrocytes at the edge develop a network of cytoplasmic extensions

Subject

موضوع الدرس

Date :

الموافق

التاريخ

Healed infarct

after several months:

1. Dense meshwork of glial fibers

2. new Capillaries.

3. perivascular C.T

New blood vessel ← 4. Chronologic Morphologic changes

from outside to inside

فمن الخارج إلى الداخل - من الأضلاع

Most central area

مرحلة مختلفة من ال repair في مكانه

Still necrotic

مرحلة tissue في وسطه في مرحلة مبكرة

لا يزال في الداخل

مرحلة repair خاصة

يعني حاليه في gradual transition في أماكن مختلفة

hemorrhagic infarction

The same with blood extravasation

(blood elements + خلايا في)

extensive if under anticoagulant.

Venous infarction (Thrombus of the brain sinuses)

خاصة ال Sagittal

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Date : / / الموافق التاريخ

Lacunar infarcts:-

- occlusion of small blood vessels as a result of atherosclerosis or hypertension

• حجم ال infarct لا يتجاوز 1cm و 1.5cm

- less than 15 mm

- Single or Multiple (ألز Multiple)

Types . Involves Putamen

Globus pallidus

Thalamus

internal Capsule

deep white Matter

Caudate nucleus

Pons

- Tissue loss surrounded by gliosis

Global cerebral hypoxia or Ischemia:-

Seen in 1 Cardiac arrest

2 Shock

3 Severe hypotension

4 Co poisoning (يرتبط مكانه 2)

5 Transient postischemic Confusional State with complete recovery
or

لغير على المرة
transient
لرطوبة
Irreversible

irreversible damage - (diffuse hypoxic/ischemic encephalopathy)

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Date : / / الموافق التاريخ

Border Zone (watershed) infarcts:-

• Border between arterial territories

يُعرف عادةً بنشوءه في نقاط الاتصال بين الشرايين Cerebral arteries
يُعرف في مكانه وحدد المناطق بكونه عرضة للإسchemia

عكس الأشخاص إلى عدم Global cerebral Ischemia ينتهي بباري الحالة

(Ant
Med
post)

- Result in wedge shaped infarct
- with 2ry hemorrhage.
- often bilateral
- Commonly after Cardiac arrest resuscitation

Global cerebral hypoxia/Ischemia: Morphology

- 1 edematous and swollen brain
- 2 Poor gray white demarcation
- 3 Irreversible injury

6-12 hour . dead red neuron + eosinophilic neuro

2 hours - 2 weeks . Subacute changes

necrosis , Macrophages

vascular proliferation + gliosis

After 2 weeks : removal of necrotic tissue and gliosis

Subject

موضوع الدرس

Date :

الموافق

التاريخ

Intracranial hemorrhage

~~Signs and symptoms~~ types : epidural and subdural

Mostly trauma related

but subarachnoid and intraparenchymal
related to cerebrovascular disease.
as (atherosclerosis) →

Intraparenchymal hemorrhages

① Sudden onset of symptoms.

عادة حدوث مفاجئ hypertensive

② Middle or late adult life

ملاحظة سريرية

• dendritic spikes (aggregates of proteins)
have a. (biological elasticity)

(A.P) Action potential يؤدي إلى

Morphological changes of proteins.

لبنية مسارات لا تقل لفيد تقي السلسلة على حقا
بعد فترة صا لو القتر الى بنفقا منه متى مصدر ولكن
خلص صا الاشى بعرا له certain A.p وتقي ار

circle بتكرر فبذكر الاشى .

في نهاية
axon
في
aggregation
of
ribosomes

وماد تقي
الفكرة

Subject _____

موضوع الدرس _____

Date : / / الموافق

التاريخ / /

③ Versus 2nd hemorrhage → (result in hematoma formation)

④ 2 types Maybe

Ganglionic or lobar hemorrhage

↓
aggregation
of gray matter
islands inside
the white
matter

↓
as a result of
large blood vessel
hemorrhage

↓
as a result
of small blood vessel
hemorrhage
and
hypertension → (Most common)

So - 60% of cases in putamen

• Cerebral amyloid angiopathy is the risk factor most commonly associated with lobar hemorrhage.

Small artery من راسه 2 ل. arteriole ج. Smooth muscle layers

مورفولوجيا Morphology:

↓
Concentric layering of arterial
(one of important effect of
HTN on small blood vessels)

Subject _____

موضوع الدرس _____

Date : _____

الموافق _____

التاريخ _____

Subarachnoidal hemorrhage:

- The most common cause is rupture of Saccular (berry) aneurysm in a cerebral artery

Saccular aneurysm

Fusiform (entire wall bulging)

1. One side of the wall is bulging
2. 2% of the population
3. Young age Sudden death ربيتر صلاية
4. 90% found near Major arterial branch points in the Ant. circulation



- ① 40% in Ant. cerebral artery when contact with the other side

لا ربيتر صلاية
بجانب الفص
لا ربيتر صلاية
لا ربيتر صلاية

Pathogenesis:-

1. A developmental anomaly

2. Absence of smooth muscle and elastic lamina

(anurysm ربيتر صلاية Thin wall ربيتر صلاية rupture ربيتر صلاية BP ربيتر صلاية)

Subject _____ موضوع الدرس

Date: / / الموافق التاريخ

3 The majority are sporadic ^{as cigarette} (HTN)

4 Genetic factors

Morphology

1 Atheroma, Thrombus

2 prior hemorrhage

3 Bleeding into the Subarachnoid or brain substance

4 Absence of smooth muscle and elastic lamina

(Thickened hyalinized intima and a covering of adventitia)

So very limited Mechanical Resistance So HTN is very serious Condition

Vascular Malformations 4 group

[1] AV (Arterio venous) Malformation

[2] Cavernous Malformation

[3] Capillary telangiectasias

[4] venous angiomas

Risk of hemorrhage

+ KRAs mutation

characterized by Cave like blood vessel

Proliferation of blood vessel

dilated capillary

(dilated and enlarged)

directly under skin
في الرطب خاصة بالاكبار / في الدم بالنس لا لرنهم قاع

Subject _____

موضوع الدرس

Date : _____

الموافق

التاريخ

Morphology

AV malformations:



1. warm like
2. pulsatile swelling
3. Subarachnoid or brain
4. prior hemorrhage.
5. Duplication and fragmentation of elastic lamina.
6. Thickening and replacement of T. Medra by hyalinized
7. blood vessels separated by gliosis

Cavernous Malformations:-

- No brain parenchyma
- Low flow + signs of old hemorrhage
- Most often in cerebellum + pons.

Vascular dementia - ^{as} results. Multiple infarcts over
↓ Caused by years

1. Atherosclerosis

~~Atherosclerosis~~ HTN

as results as of recurrent Micro infarctions.

2. Thrombus or embolus from Coronid or heart

Syptoms:
Dementia
gait abnormalities

3. Arteriosclerosis from Chronic HTN