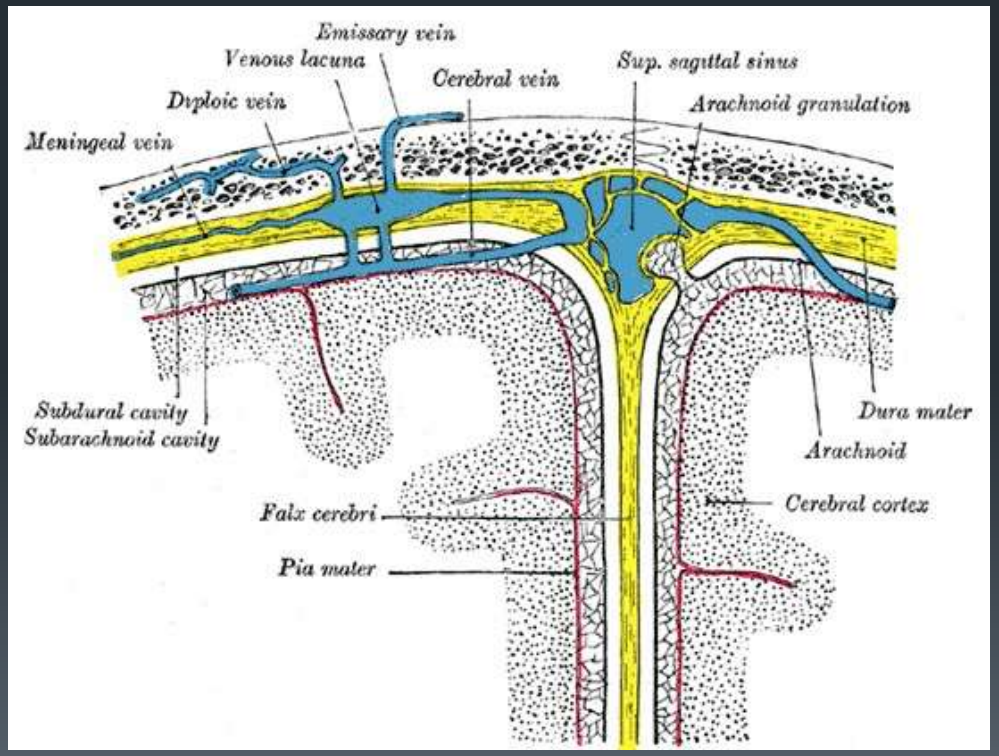
INFEKSI SUSUNAN SARAF PUSAT

dr. Risma Karlina Prabawati, Sp.S

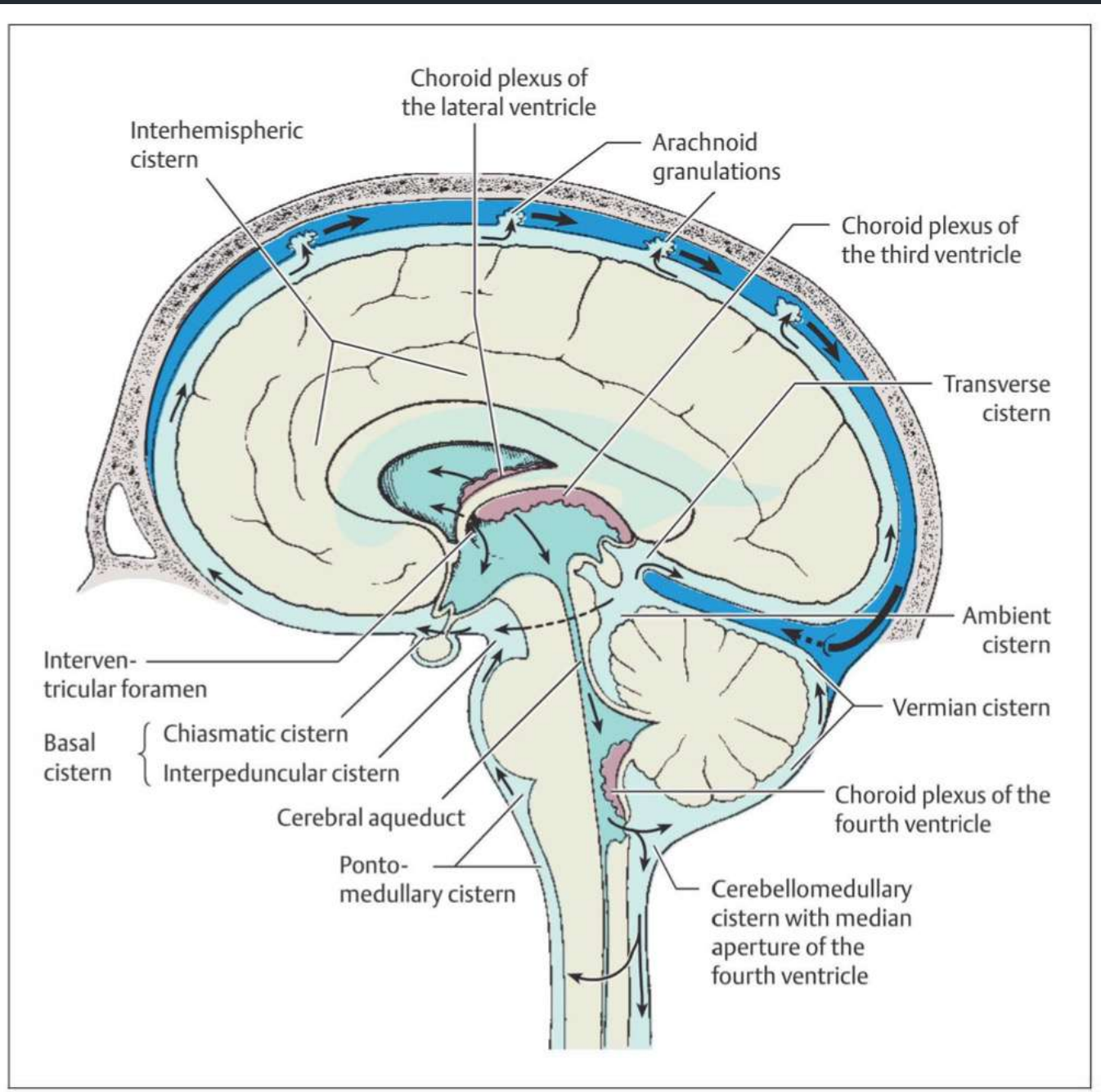
Pendahuluan

- Paling banyak mengenai meningen dan system ventrikel
- Bisa juga mengenai otak dan myelum
- Masuk dalam kegawatdaruratan neurologi
- Insiden meningkat → meningkatkan morbiditas dan mortalitas
- Penyebab: virus, bakteri, jamur, parasite, dan agen infeksius lain

Neuroanatomi Meningen dan Sistem Likuor Serebrospinal



Ghannam JY, Al Kharazi KA. Neuroanatomy, Cranial Meninges. [Updated 2023 Jul 24]. In: StatPearls [Internet]. Treasure Island (FL): StatPearls Publishing; 2024 Jan-. Available from: https://www.ncbi.nlm.nih.gov/books/NBK539882/


Choroid plexus of the lateral ventricle
Interhemispheric cistern
Arachnoid granulations
Choroid plexus of the third ventricle
Transverse cistern
Ambient cistern
Interven-tricular foramen
Vermian cistern
Basal cistern
Chiasmatic cistern
Interpeduncular cistern
Cerebral aqueduct
Choroid plexus of the fourth ventricle
Ponto-medullary cistern
Cerebellomedullary cistern with median aperture of the fourth ventricle

MENINGITIS BAKTERIAL AKUT
(meningitis purulenta)

DEFINISI

- LEPTOMENINGITIS
- RDG PIAMATER, ARAKHN, SUB ARAKH
- CSF : LEKOSIT ↑, EKSUDAT
- BAKTERI SPESIFIK
- TERJADI DALAM WAKTU <3 HARI

EPIDEMIOLOGI

- INSIDENS : AS 3- 5 / 100.000 PDDK/ TH
- FX : MALNUTRISI, IMMUN ↓

ETIOLOGI

TABEL ORGANISME ETIOLOGI MENINGITIS BAKTERI BERDASAR UMUR

Umur	ORGANISME PENYEBAB YANG UMUM
0 - 4 minggu	S. agalactiae, E. coli monocytogenes, K. Pneumonia
4 - 12 minggu	S. agalactiae, E. coli monocytogenes, H. influenzae, S. pneumonia, N. meningitidis
3 bl - 18 th	H. influenzae, S. pneumonia, N. meningitidis
18 - 50 th	S. pneumonia, N. meningitidis
> 50 th	S. pneumonia, N. meningitidis, L. monocytogenes, Aerobic gram negative bacii

PATOGENESA & PATOFISIOLOGI

MLL :

- **HEMATOGEN**
- **PERKONTINUITATUM**
- **FR. TERBUKA**

MEKANISME MENEMBUS BBB ?

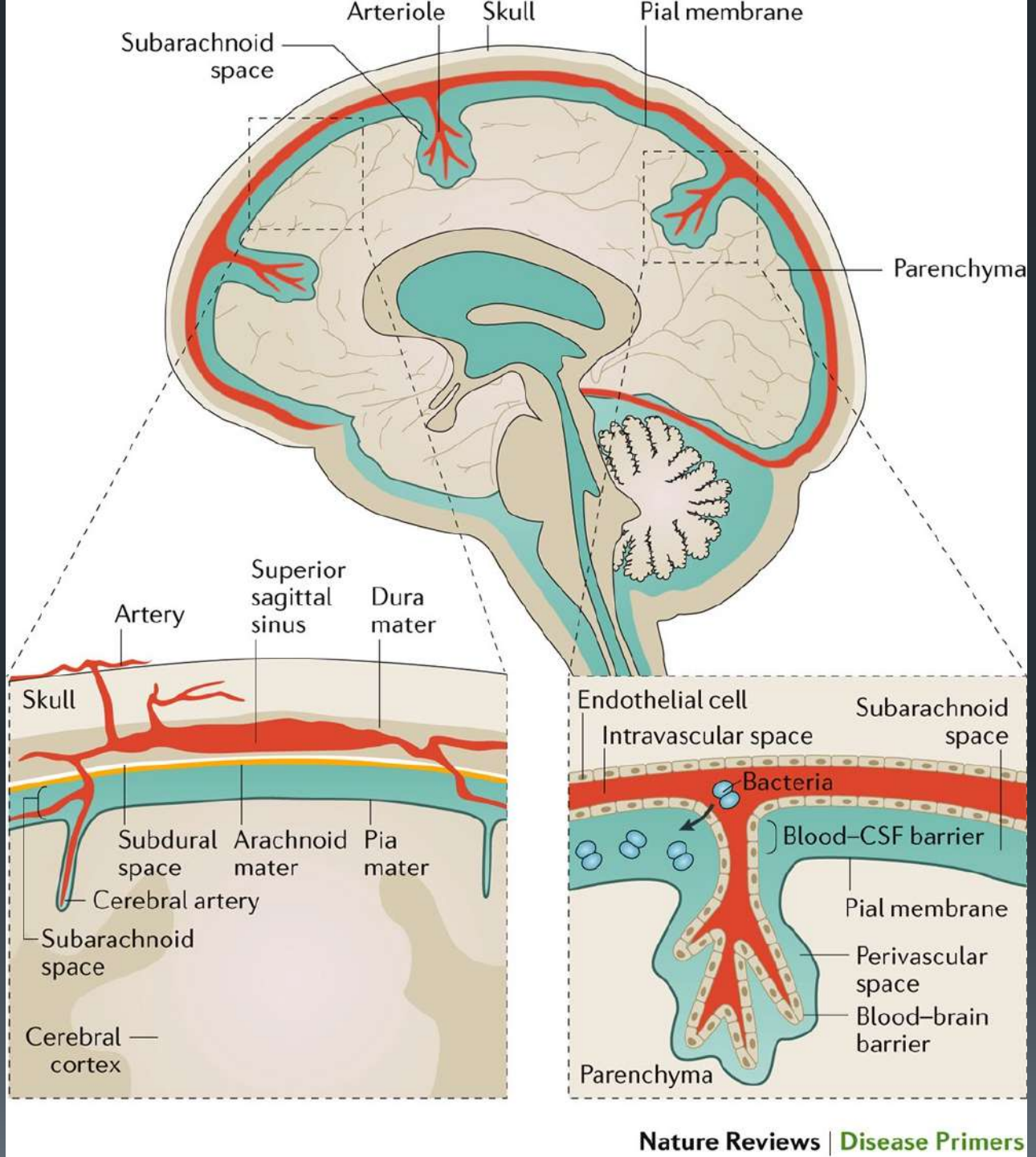
KOMP DDG SEL → CSS → IL 1, 6; PG; TNF

→ INFLAMASI, BBB RUSAK

→ EDEMA VASOGENIK

EDEMA SITOTOKSIK

EDEMA INTERSTITIIL


Arteriole
Skull
Pial membrane
Subarachnoid space
Parenchyma
Superior sagittal sinus
Artery
Dura mater
Skull
Subdural space
Arachnoid mater
Pia mater
Cerebral artery
Subarachnoid space
Cerebral cortex
Endothelial cell
Intravascular space
Subarachnoid space
Bacteria
Blood–CSF barrier
Pial membrane
Perivascular space
Blood–brain barrier
Parenchyma

GEJALA KLINIS

- AKUT
- PALING SERING: **NYERI KEPALA, PANAS BADAN, FOTOFOBIA**
- INFEKSI SALURAN NAFAS , NYERI OTOT, SYOK SEPTIK
- MUNTAH, KK, KERNIG, BRUDZINSKI
- FOKAL (KEJANG, KESADARAN ↓, HEMIPARESE, DLL)
- GEJALA HIDROCEFALUS (NYERI KEPALA HEBAT, MUNTAH, KEJANG, PAPILEDEMA)

DIAGNOSA

- TANDA DAN GX KX (**DEMAM, KAKU KUDUK, PENURUNAN KESADARAN**
- LUMBAL PUNGSI CSS : LEKO, GLUK, PROT, KULTUR, CRP, PCR
- LAIN-LAIN : X-FO, KULTUR DRH, CTSCAN, EEG

KRITERIA DIAGNOSA:

GEJALA & TANDA KLINIS
plus
PARAMETER LCS ABNORMAL
(predominasi PMN, rasio glc LCS:darah < 0,4)
plus
BAKTERI LCS (+) / KULTUR LCS (+) atau KULTUR LCS (-) dengan KULTUR DARAH (+) atau PCR LCS (+)

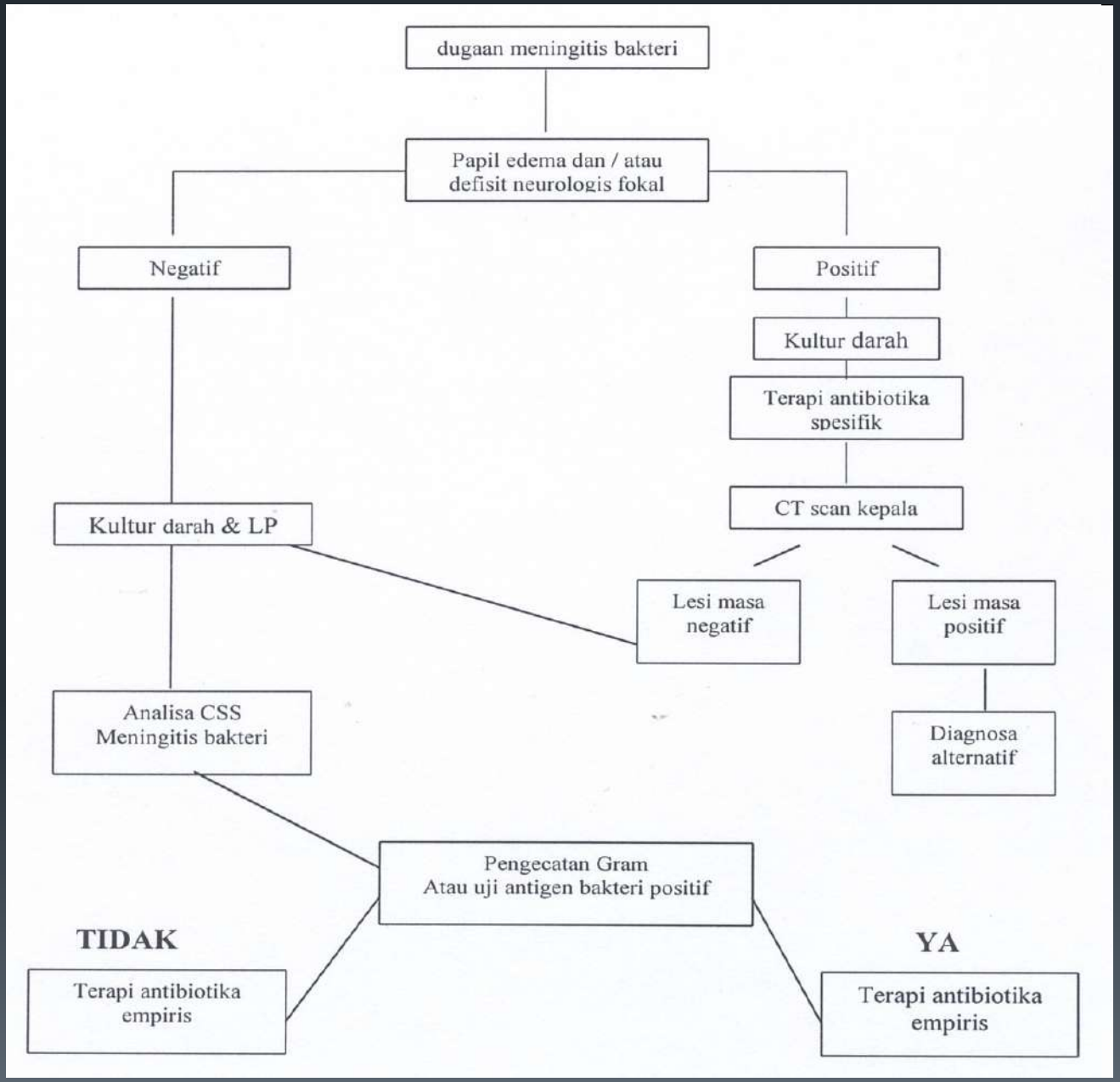
Characteristic	Normal	Bacterial meningitis	Viral meningitis	Tuberculous meningitis
C-reactive protein	<10 mg per l	40–400 mg per l	<10 mg per l	10–100 mg per l
Blood leukocytes	4–10 × 10^9 per µl	10–30 × 10^9 per µl	4–10 × 10^9 per µl	5–15 × 10^9 per µl
Opening pressure‡	6–20 cm H_2O	20–50 cm H_2O	6–30 cm H_2O	15–40 cm H_2O
CSF white cell count	<5 cells per µl	>1,000 cells per µl	10–1,000 cells per µl	10–1,000 cells per µl
CSF protein level	<0.60 g per l	>2 g per l	<0.60 g per l	1–4 g per l
CSF/blood glucose ratio	>0.60	<0.40	>0.60	<0.40

CSF, cerebrospinal fluid.

KOMPLIKASI

- **NON NEURO : MIOKARDITIS**
- **NEURO : EDEMA OTAK**
  EFUSI SUBDURAL
  INFARK, NEKROSIS OTAK
  HIDROSEPALUS
  PARESE N. KRANIAL

PENANGANAN


dugaan meningitis bakteri
Papil edema dan / atau defisit neurologis fokal
Negatif
Positif
Kultur darah
Terapi antibiotika spesifik
CT scan kepala
Kultur darah & LP
Lesi masa negatif
Lesi masa positif
Analisa CSS Meningitis bakteri
Diagnosa alternatif
Pengecatan Gram Atau uji antigen bakteri positif
TIDAK
YA
Terapi antibiotika empiris
Terapi antibiotika empiris

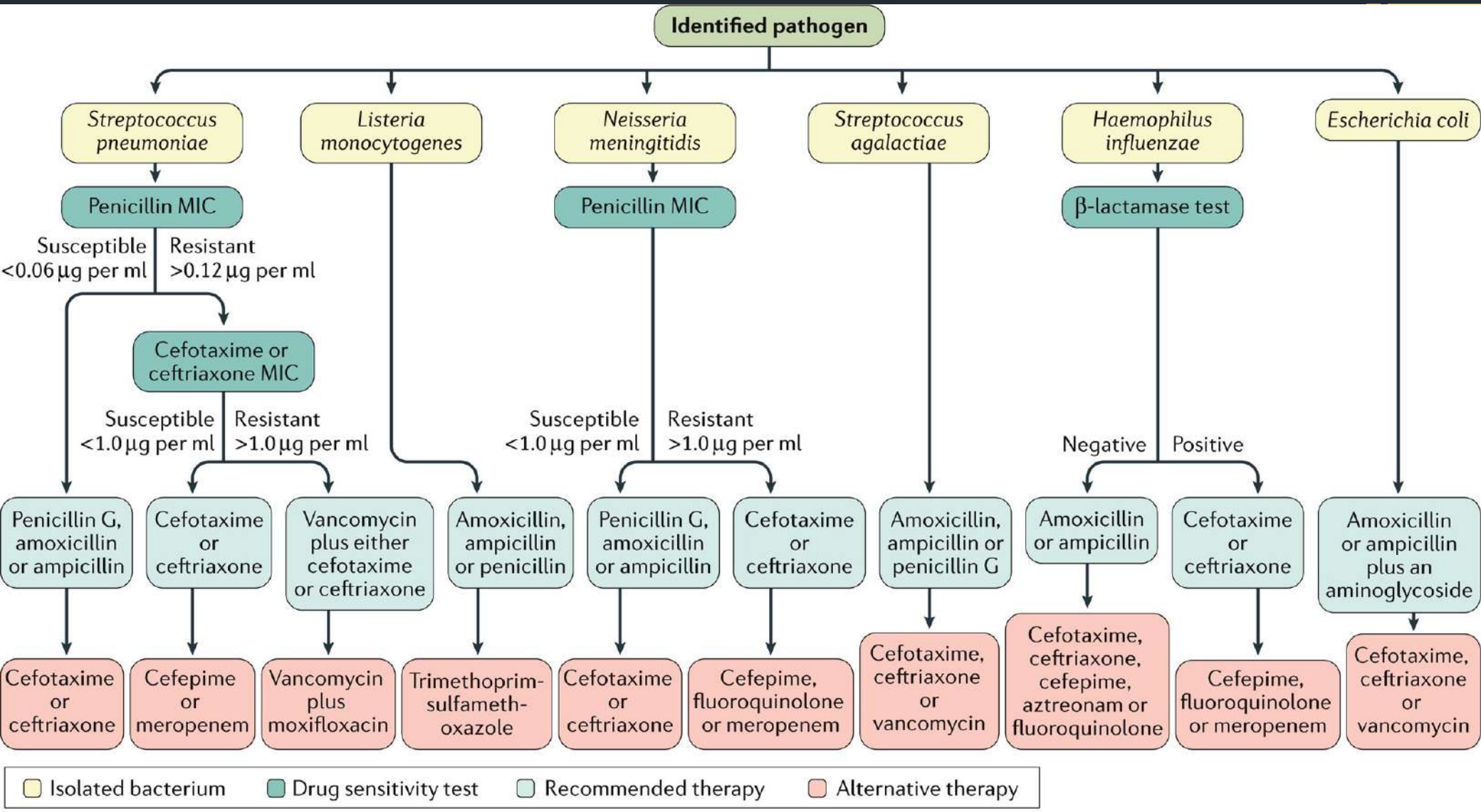
TABEL PILIHAN ANTIBIOTIKA BERDASAR UMUR

Patient age or risk factor	Common pathogens	Empirical therapy
<1 month	· *Streptococcus agalactiae* · *Escherichia coli* · *Listeria monocytogenes*	Amoxicillin or ampicillin plus cefotaxime, or amoxicillin or ampicillin plus an aminoglycoside
1–23 months	· *S. agalactiae* · *E. coli* · *Streptococcus pneumoniae* · *Neisseria meningitidis*	Vancomycin plus either cefotaxime or ceftriaxone[*,‡]
2–50 years	· *S. pneumoniae* · *N. meningitidis*	Vancomycin plus either cefotaxime or ceftriaxone[*,‡]
>50 years	· *S. pneumoniae* · *N. meningitidis* · *L. monocytogenes* · Aerobic Gram-negative bacilli	Vancomycin, ampicillin and either cefotaxime or ceftriaxone
Immunocompromised state[§]	· *S. pneumoniae* · *N. meningitidis* · *L. monocytogenes* · *Staphylococcus aureus* · *Salmonella spp.* · Aerobic Gram-negative bacilli	Vancomycin, ampicillin and either cefepime or meropenem

[*] Add amoxicillin or ampicillin if meningitis caused by *L. monocytogenes* is also suspected.
[‡] In countries where the prevalence of cephalosporin-resistant pneumococcus is <1%, cefotaxime or ceftriaxone alone is appropriate.
[§] For example, owing to HIV infection or immunosuppressive therapies (for example, post-transplantation). Adapted from Ref. 143.

TX TAMBAHAN

- DEKSAMETASON
- ↓ TIK


Identified pathogen
Streptococcus pneumoniae
Penicillin MIC
Susceptible <0.06 µg per ml
Resistant >0.12 µg per ml
Penicillin G, amoxicillin or ampicillin
Cefotaxime or ceftriaxone
Cefotaxime or ceftriaxone MIC
Susceptible <1.0 µg per ml
Resistant >1.0 µg per ml
Cefotaxime or ceftriaxone
Cefepime or meropenem
Vancomycin plus either cefotaxime or ceftriaxone
Vancomycin plus moxifloxacin
Listeria monocytogenes
Amoxicillin, ampicillin or penicillin
Trimethoprim-sulfameth-oxazole
Neisseria meningitidis
Penicillin MIC
Susceptible <1.0 µg per ml
Resistant >1.0 µg per ml
Penicillin G, amoxicillin or ampicillin
Cefotaxime or ceftriaxone
Cefotaxime or ceftriaxone
Cefepime, fluoroquinolone or meropenem
Streptococcus agalactiae
Amoxicillin, ampicillin or penicillin G
Cefotaxime, ceftriaxone or vancomycin
Haemophilus influenzae
β-lactamase test
Negative
Positive
Amoxicillin or ampicillin
Cefotaxime, ceftriaxone, cefepime, aztreonam or fluoroquinolone
Cefotaxime or ceftriaxone
Cefepime, fluoroquinolone or meropenem
Escherichia coli
Amoxicillin or ampicillin plus an aminoglycoside
Cefotaxime, ceftriaxone or vancomycin
Isolated bacterium
Drug sensitivity test
Recommended therapy
Alternative therapy

PROGNOSA

- UMUR PX
- JENIS BAKTERI CSS
- KESADARAN MRS
- DX DAN TX
- DEFISIT FOKAL

MENINGITIS TUBERKULOSA

- ONSET > 4 MGG
- EX : MIKOBAKTERIUM TB
- PROGRESIF
- FOKUS ORGAN LAIN → KELJ REGIONAL & DUKTUS TORASIKUS → SIRK DRH →SSP (tuberkel pecah yg masuk ke rg sub arakhnoid dan dasar tengkorak)

GEJALA KLINIS

STADIUM I: gx prodromal non spesifik, nyeri kepala, demam

STADIUM II: drowsy, perub mental, iritasi meningen, parese n. III, IV, VI

STADIUM III: koma, kejang, hemiparese

DIAGNOSA

- **TANDA DAN GX KX**
- **LCS : LEKOSIT 50 – 400 / MM³**
  GLUKOSE ↓ (40 MG / 100 ML)
  PROT ↑ (80 – 400 MG / 100 ML)

KOMPLIKASI

- BASIS KRANII (> SISTERNA)
- HIDROSEPALUS, HEMIPLEGI, AFASIA, KEJANG, PARESE N.KRANIAL (N.III, VI)

TERAPI

- MENEMBUS BBB
- KORTIKOSTEROID
- OBAT TUBERLULOSTATIKA :
  1. INH 400 MG / HR
  2. PIRAZINAMID 15 – 30 MG/KG BB/HR
  3. RIFAMPISIN 15 MG / KG BB / HR
  4. STREPTOMISIN 1 GR / HR (IM)

MENINGITIS ASEPTIK
(SELF LIMITED)

ETIOLOGI

- **ENTEROVIRUS**
- **MUMPS VIRUS**

PATOFISIOLOGI

INVASI MLL USUS → FESES & NASOPHARING, PENULARAN ORAL – FEKAL

GEJALA KLINIS

- AKUT
- DEMAM, MENGGIGIL, NYERI KEPALA, FOTOFOBIA, MUNTAH, KAKU KUDUK

DIAGNOSA

GX KX

LCS : JERNIH
PLEOSITOSIS 50 – 500/MM^3
(LIMPOSIT 90 %)
GLUKOSA N
PROTEIN ↑ (80 – 100 MG/ML)
VIRUS +
SEROLOGI : TITER AB ↑

KOMPLIKASI

- MENINGOENSEFALITIS
- SIADH

TX

- UMUM = MENINGITIS BAKTERIAL
- SPESIFIK : ACYCLOVIR

PROGNOSA

TIDAK BAIK

Bacterial Meningitis

Most common causative organisms

In neonates

- Gram-negative bacilli, 50% (*E coli*, *H influenzae*, etc)
- Streptococci, 20%
- Other (*S aureus*, etc)

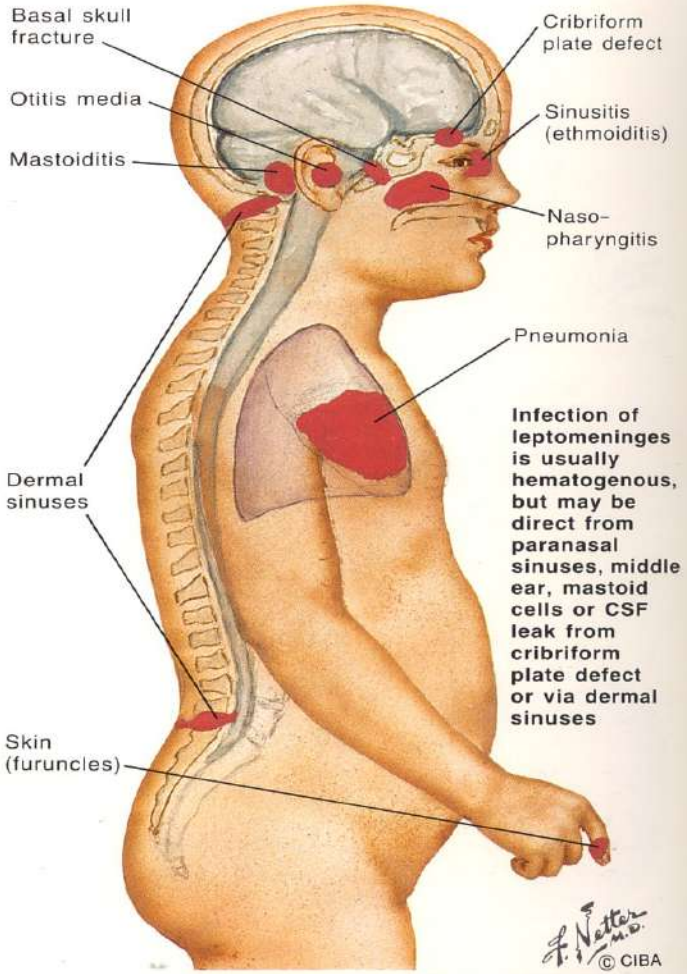
In children

- *H influenzae*, 50%
- *N meningitidis*, 25%
- Other (*Listeria*, etc)

In adults

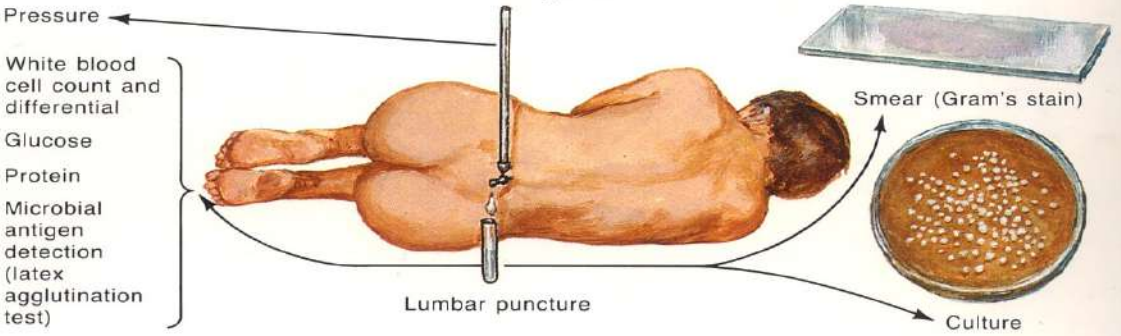
- *S pneumoniae*, 30%
- *N meningitidis*, 15%
- Gram-negative bacilli
- Other (*Listeria*, etc)

Sources of infection

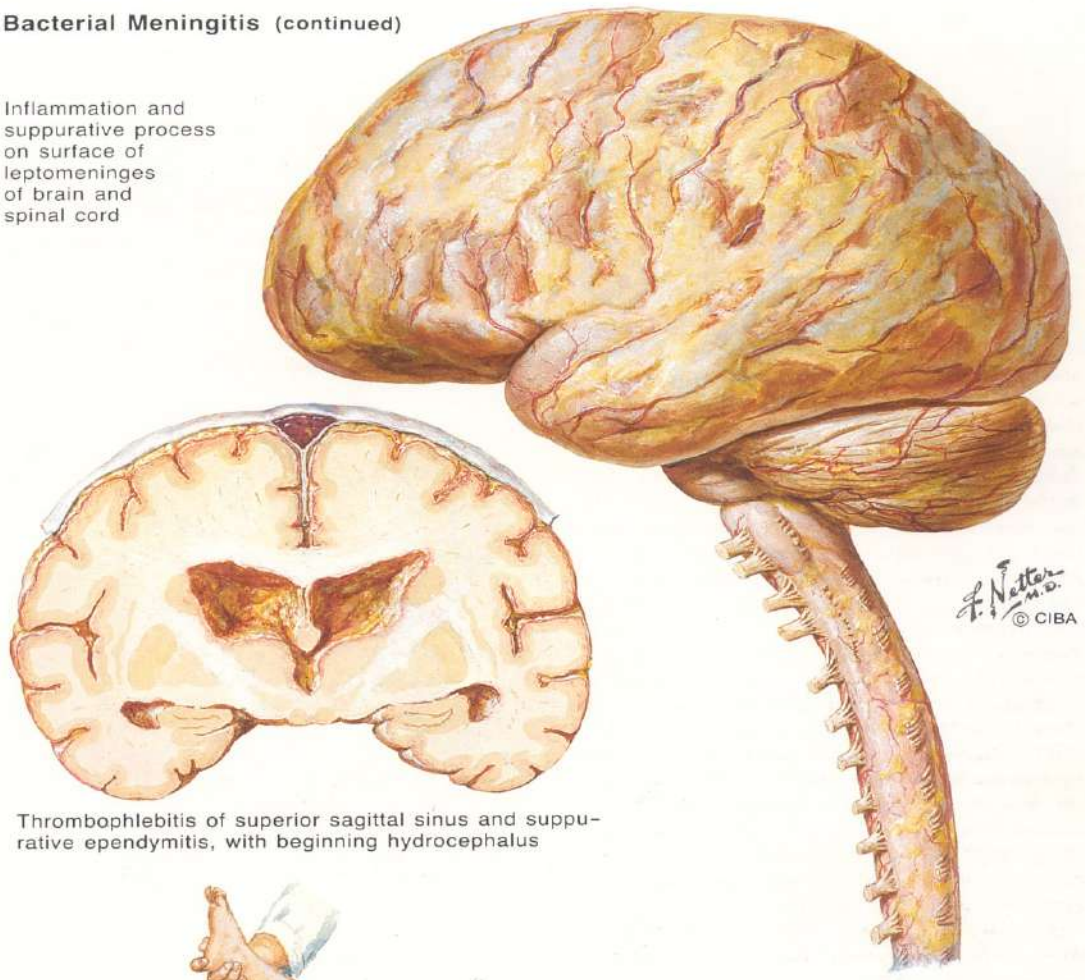


Infection of leptomeninges is usually hematogenous, but may be direct from paranasal sinuses, middle ear, mastoid cells or CSF leak from cribriform plate defect or via dermal sinuses

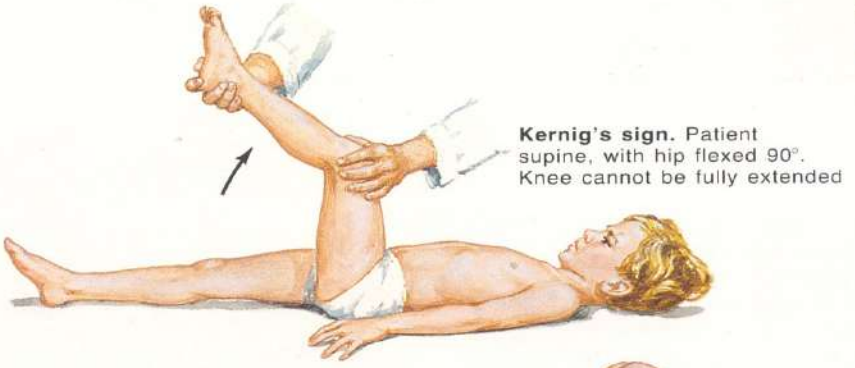
Diagnosis



Bacterial Meningitis (continued)



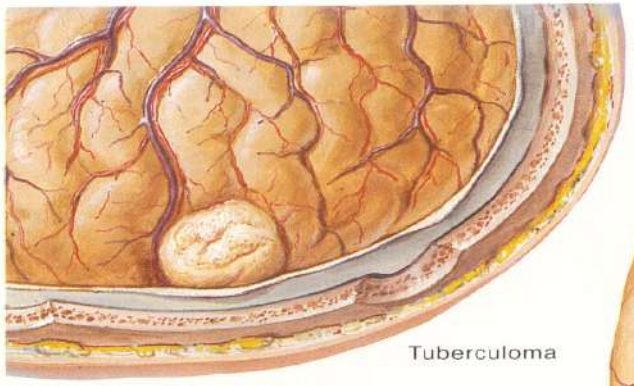
Thrombophlebitis of superior sagittal sinus and suppurative ependymitis, with beginning hydrocephalus



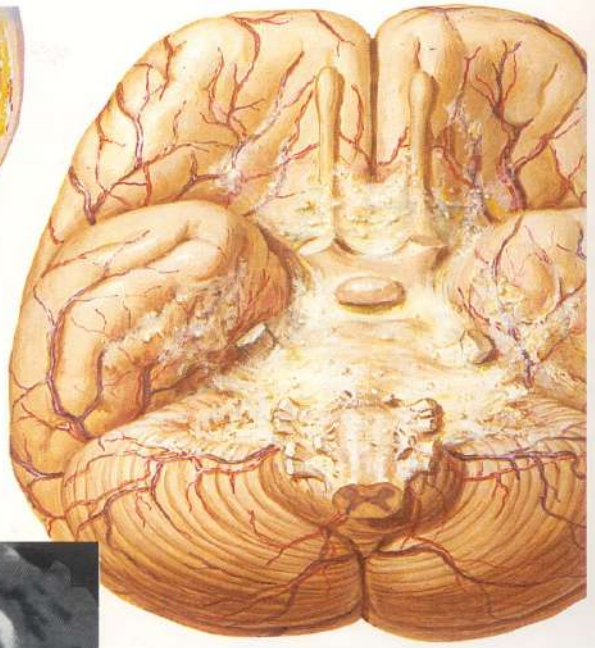
Kernig's sign. Patient supine, with hip flexed 90°. Knee cannot be fully extended

Neck rigidity (Brudzinski's neck sign). Passive flexion of neck causes flexion of both legs and thighs

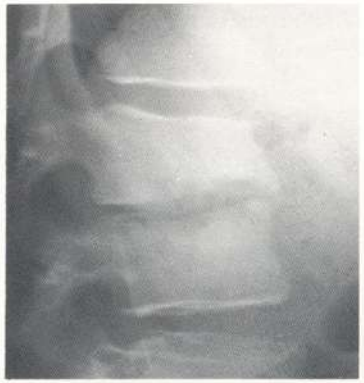
Tuberculosis of Brain and Spine



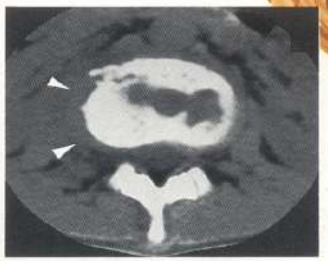
Tuberculoma



Tuberculous basilar meningitis



X-ray film: destruction of disc space and adjacent end plates of vertebrae



CT scan: paraspinous abscess in addition to bony destruction

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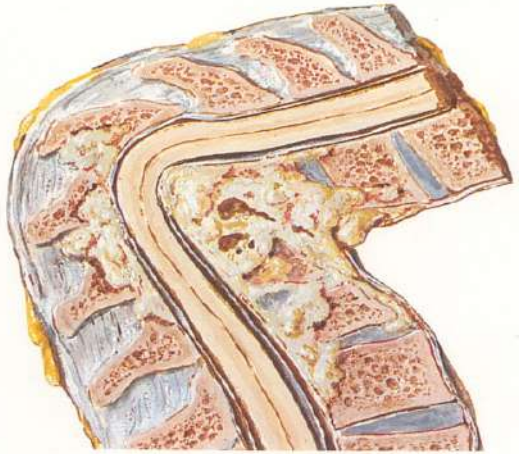
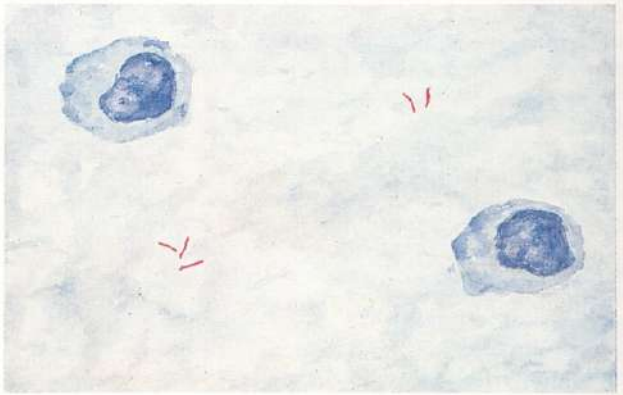


Tuberculosis of spine (Pott's disease) with marked kyphosis



Tubercle bacilli appearing as red rods in smear of CSF (Ziehl-Neelsen stain)

ENSEFALITIS

RADANG JARINGAN OTAK O. K.

- **BAKTERI**
- **RIKETSA**
- **PARASIT**
- **JAMUR**
- **VIRUS**

ENSEFALITIS SUPURATIVA

RADANG OTAK ➔ OTITIS MEDIA, MASTOIDITIS, SINUSITIS, OSTEOMILITIS, ABSES
RADANG LOKAL ➔ PROLIFERASI JAR.
IKAT & ASTROSIT ➔ KAPSUL ➔ JAR.
RUSAK ➔ CAIR ➔ ABSES

TANDA DAN GEJALA

- **TIK ↑**
- **GX INFEKSI UMUM**

PEMERIKSAAN + AN

LCS, EEG, FOTO KEPALA, CT SCAN, (LP -)

TX

- **AMPICILLIN 4 × 3 – 4 GR**
- **KHLOROMICETIN 4 × 1 GR**
  → SELAMA 10 HR
- **ABSES BESAR, OPERABEL → EKSISI**

PROGNOSA

† 50 %

MALARIA OTAK

- **DEFINISI:**

  ensefalopati akut, 3 kriteria: koma menetap > 30 menit setelah kejang, disertai adanya *Plasmodium falsiparum*, dan penyebab lain ensefalopati (-)

- **Terjadi gangguan pd Eritrosit → LIKAT & MELEKAT → SUMBATAN dan PERDARAHAN → NEKROSIS**

- **GEJALA KLINIS:**

  malaria tropika: panas ireguler, anemia, splenomegali, parasitemia.
  3-7 hari setlh panas → koma, bisa tjd cepat dan mendadak (1-2jam), disertai defisit neurologis, kejang, TIK ↑
  harus disingkirkan penyebab lain: hipoglikemia, asidosis, gagal ginjal, sepsis, meningoensefalitis

DIAGNOSIS:

- px dr daerah endemis/ berada di daerah endemis
- demam atau riwayat demam tinggi
- manifestasi serebral
- parasit malaria dlm sediaan darah tepi (+)
- kelainan CSF (-)

PENATALAKSANAAN:

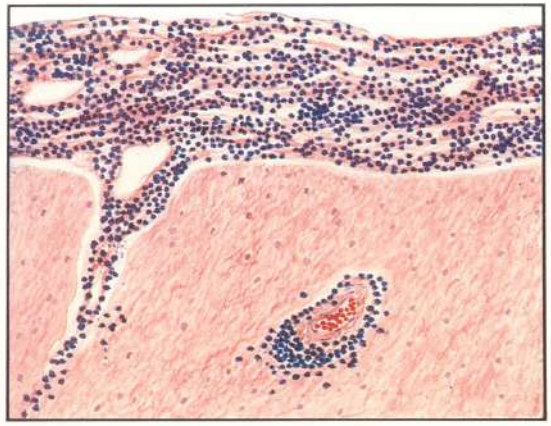
- rawat ICU/intensif
- terapi suportif umum
- pemberian cairan yg adekuat
- pemberian OAM (obat anti malaria)

KOMPLIKASI

kejang, hipoglc, hiperpireksi, ggn fungsi ginjal, hiperparasitemia

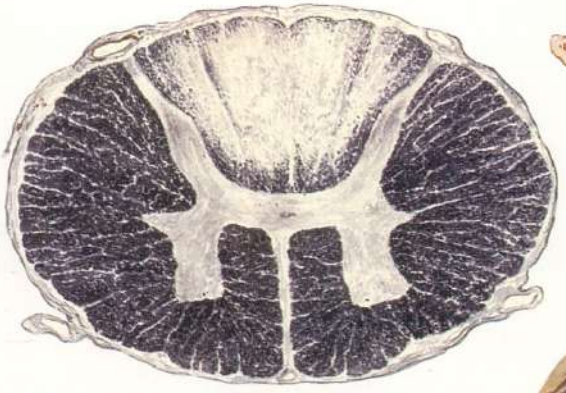
PROGNOSIS: tergantung kecepatan dx, tx, komplikasi

Neurosyphilis

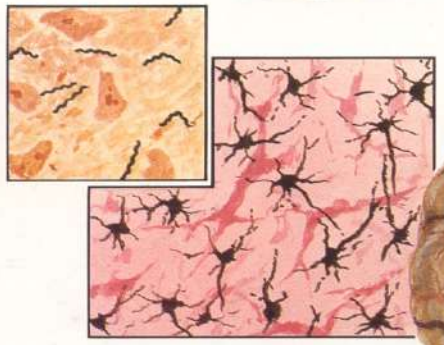


Syphilitic meningoencephalitis with perivascular infiltration

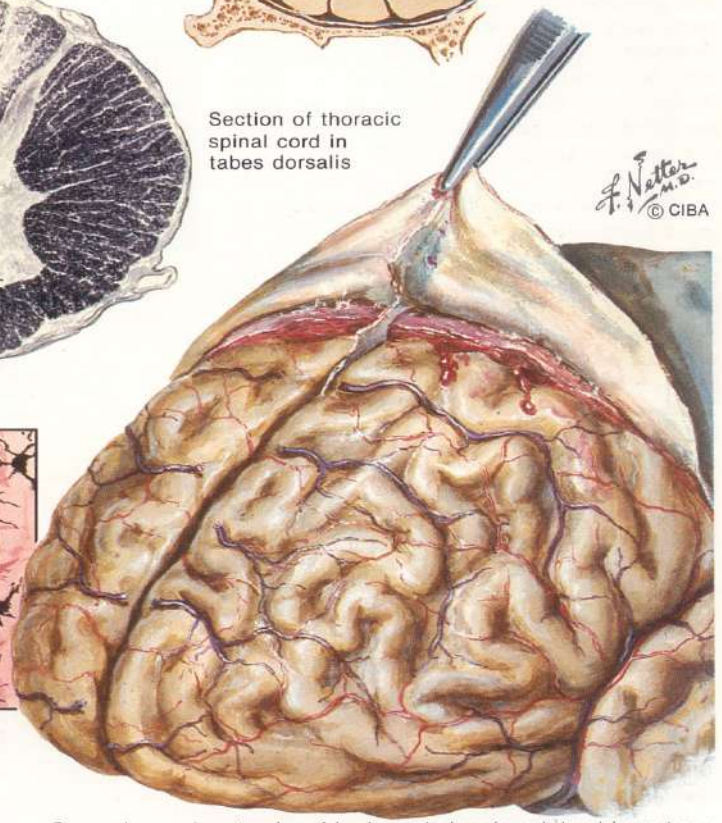
Gumma with beginning erosion of skull



Section of thoracic spinal cord in tabes dorsalis

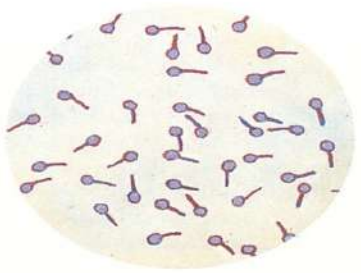


General paresis: astrocytosis in cortex in reaction to loss of nerve cells. Small inset shows spirochetes in brain



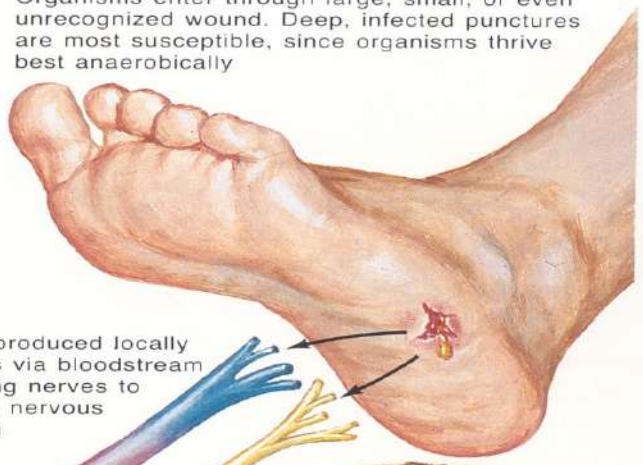
General paresis: atrophy of brain and chronic subdural hematoma

Tetanus

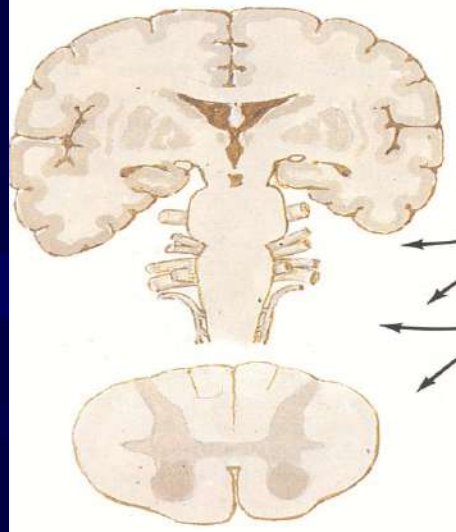


Clostridium tetani: gram-positive, spore-bearing rods

Organisms enter through large, small, or even unrecognized wound. Deep, infected punctures are most susceptible, since organisms thrive best anaerobically

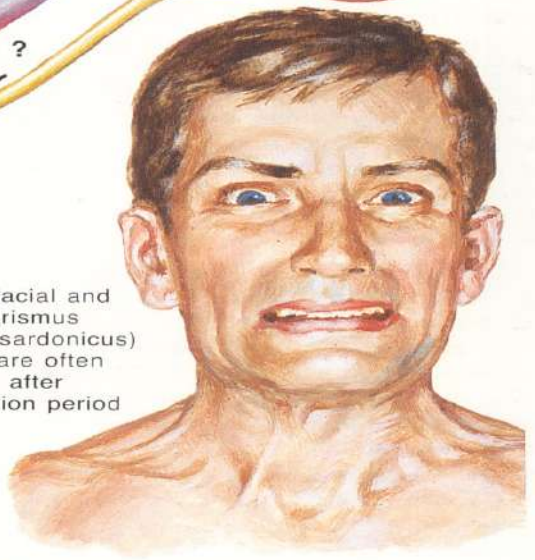


Toxin produced locally passes via bloodstream or along nerves to central nervous system



Motor neurons of spinal cord (anterior horn) and of brainstem become hyperactive because toxin specifically attacks inhibitory (Renshaw) cells

Spasm of jaw, facial and neck muscles (trismus [lockjaw], risus sardonicus) and dysphagia are often early symptoms after variable incubation period



Complete tetanic spasm in advanced disease. Patient rigid in moderate opisthotonos, with arms extended, abdomen boardlike. Respiratory arrest may occur

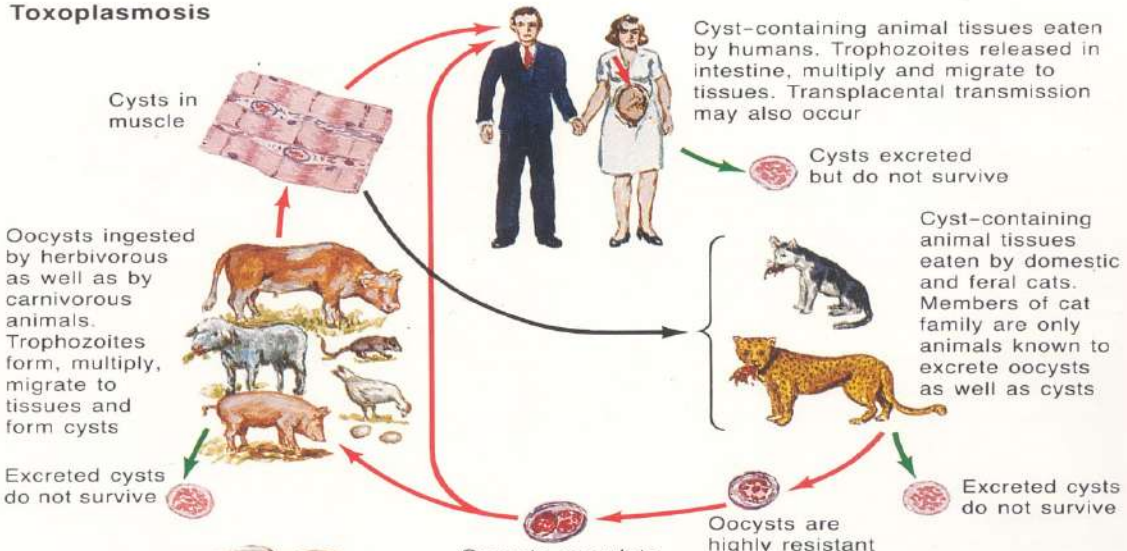
TOXOPLASMOSIS

- TOXOPLASMA GONDII GX (+) KEADAAN IMUN ↓
- DX : SEROLOGI DARAH
- LCS : LIMPOSIT ↑, PROTEIN ↑
- FOTO RONTGEN : KALSIFIKASI
- CT : PENGKAPURAN & HIDROSEFALUS

- TX : - SULFADIAZIN 100 MG/ KG BB
  - PIRIMETAZIN 1MG/ KgG BB
  - SPIRAMICIN 3 x 500 MG/ HR

} 1 bln p. o

Toxoplasmosis



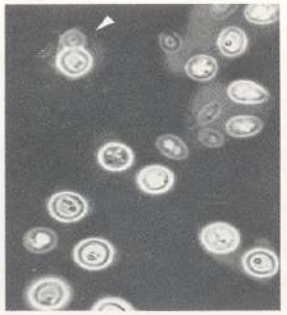
Brain section with nodule of *Toxoplasma gondii* in basal ganglia and necrotizing encephalitis in left frontal and temporal corticomedullary zones

CT scan showing enhancing lesions of toxoplasmosis in right frontal and left temporal lobes of immunocompromised patient

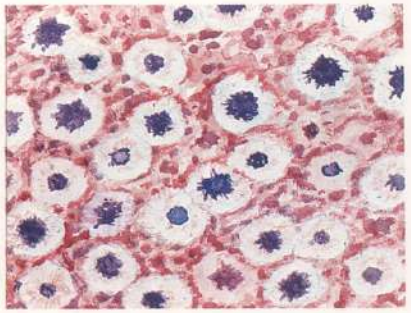
F. Netter M.D. © CIBA

Cryptococcosis

Infection is by respiratory route. Pigeon dung and air conditioners may be factors in dissemination



India ink preparation showing budding and capsule



Accumulation of encapsulated cryptococci in subarachnoid space (PAS or methenamine-silver stain)

ENSEFALITIS VIRUS

- **VIRUS → RADANG OTAK**

  V. RNA (V. PAROTITIS, V. MORBILI,

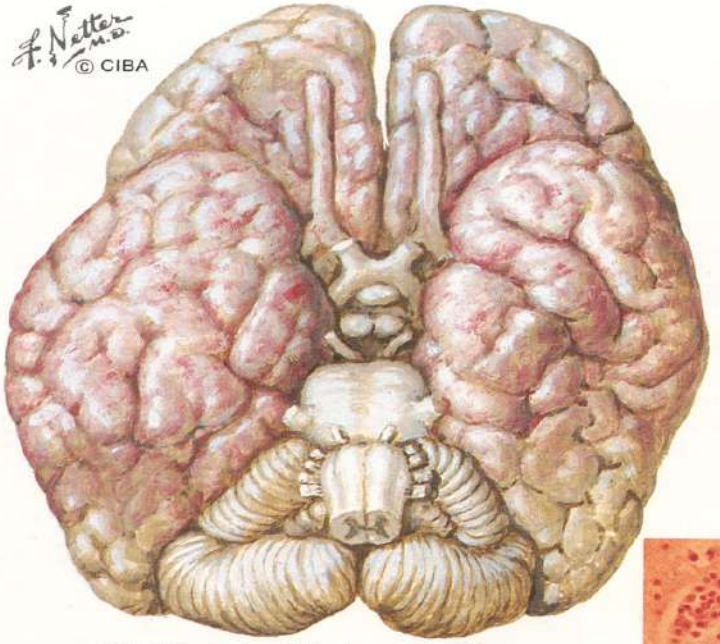
  V. RABIES, V. RUBELA)

  V. DNA (HERPES ZOSTER-VARISELA,

  V. EPSTEIN-BARR, VARIOLA, AIDS)

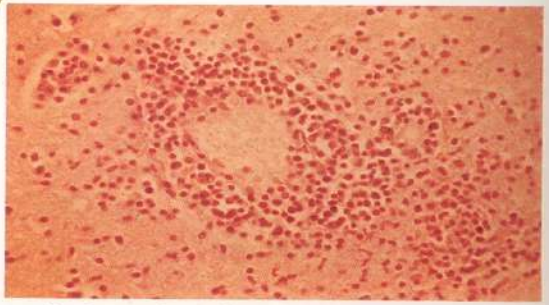
- ***GEJALA KLINIS***
  - ✓ **DEMAM, NYERI KEPALA, VERTIGO, NYERI BADAN, MUAL, KESAD ↓ → KEJANG**
  - ✓ **DEF. NEUROLOGI → TGT LOKASI**

Herpes Simplex Encephalitis

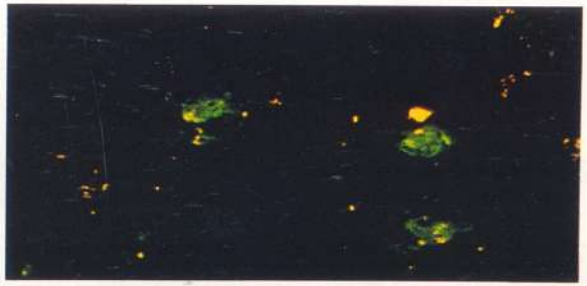


Swelling and patchy hemorrhagic areas, most marked in right temporal lobe

CT scan showing characteristic low absorption in temporal lobes

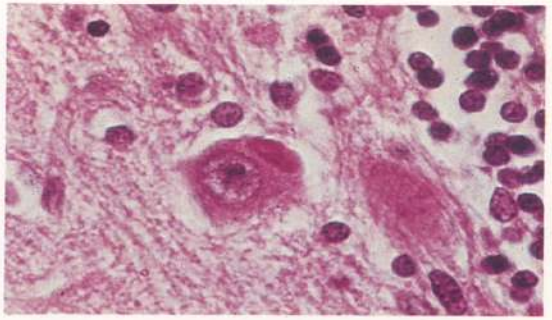


Perivascular infiltration with mononuclear cells in disrupted brain tissue



Immunofluorescent staining shows presence of herpesvirus antigen in neurons

Rabies



Negri inclusion body in Purkinje cell of brain

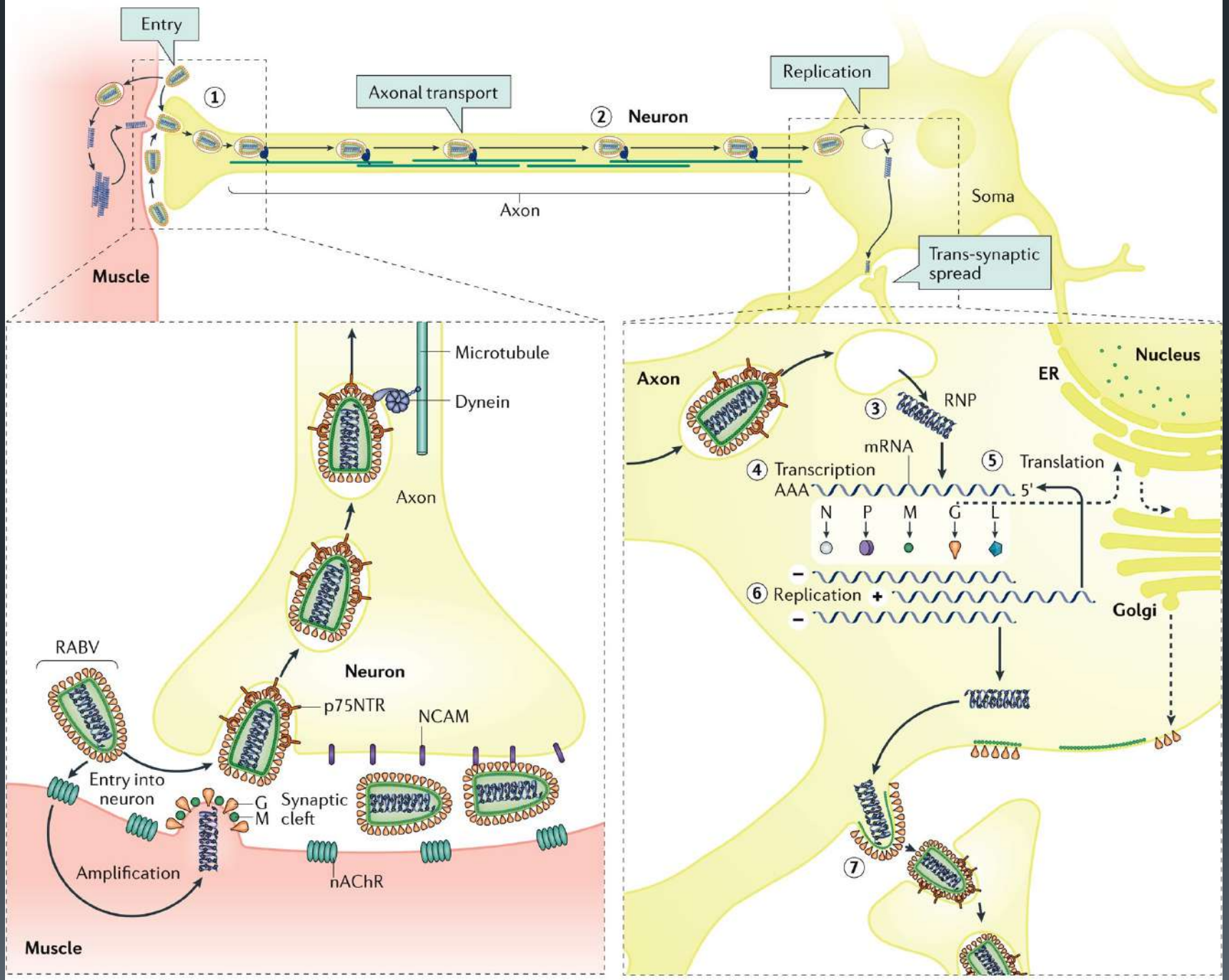
Common animal disseminators

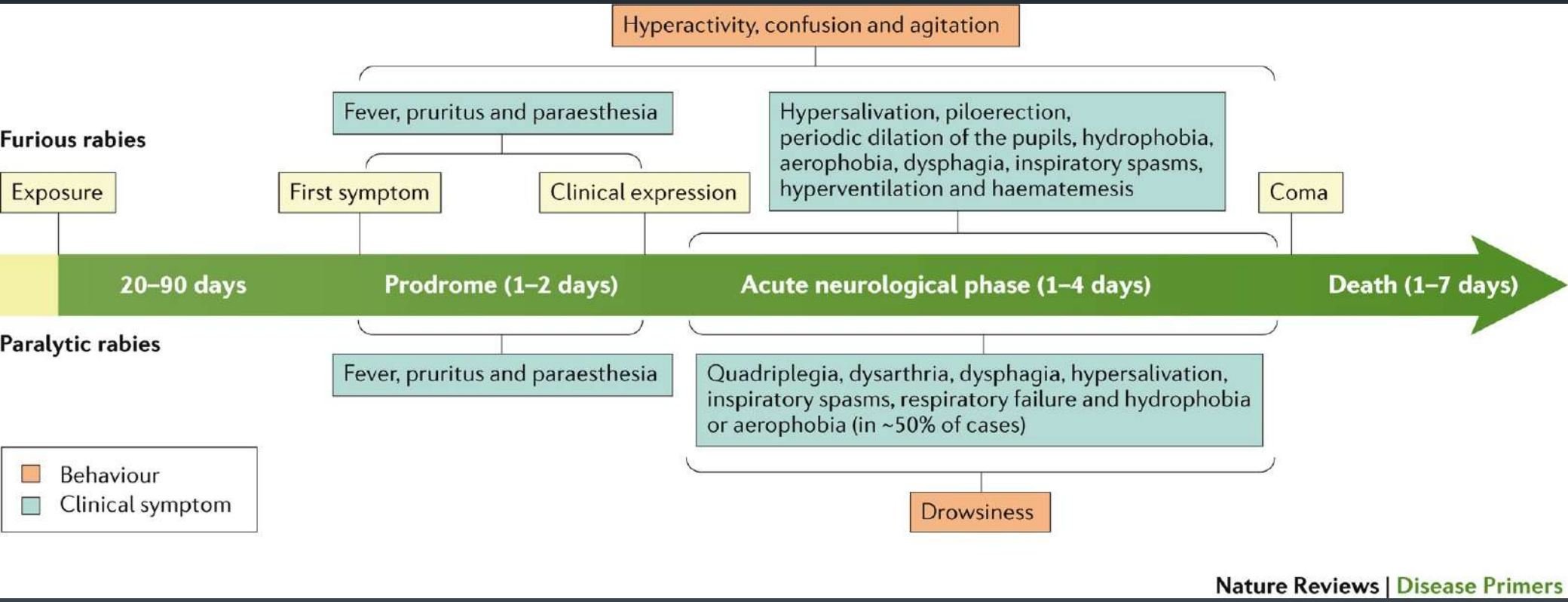
RABIES

- V. RABIES (GIGITAN HEWAN) → OTOT

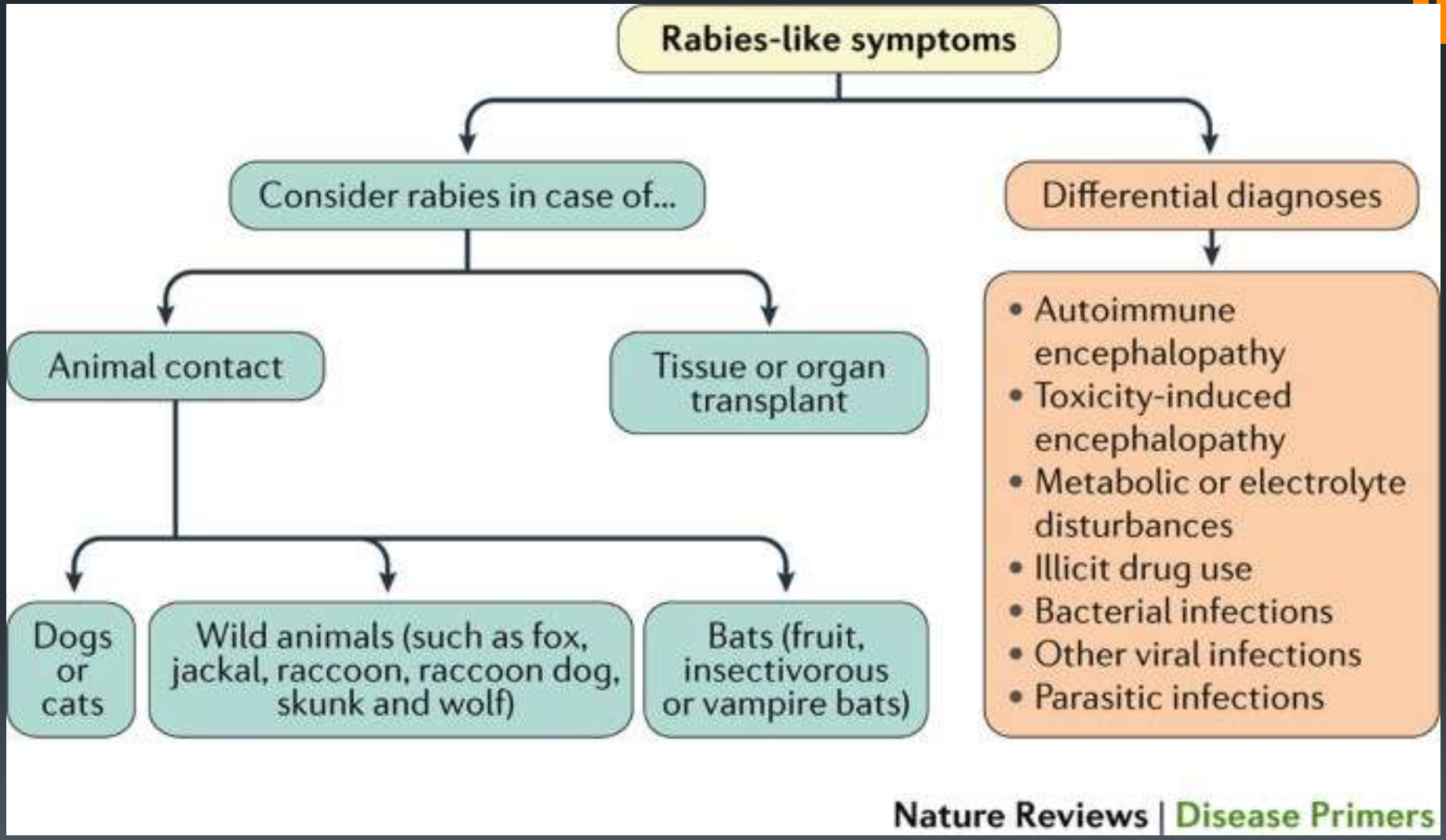
  → SARAF PERIFER → OTAK → BERKEMBANG DLM, SEL-SEL SARAF → HIDROFOBIA, NYERI DAN DISPNEA

- STLH SERANGAN BERHENTI → CIALOREA & HIPERHIDROSIS → PARESE N. KRANIAL & PARESE LENGAN DAN TUNGKAI


Entry
1
Axonal transport
2 Neuron
Replication
Soma
Axon
Trans-synaptic spread
Muscle
Microtubule
Dynein
Axon
RABV
Neuron
p75NTR
NCAM
Entry into neuron
G
M
Synaptic cleft
Amplification
nAChR
Muscle
Axon
3 RNP
mRNA
4 Transcription
AAA
5 Translation
5'
N
P
M
G
L
6 Replication
ER
Nucleus
Golgi
7



Nature Reviews | Disease Primers


Rabies-like symptoms
Consider rabies in case of...
Differential diagnoses
Animal contact
Tissue or organ transplant
• Autoimmune encephalopathy
• Toxicity-induced encephalopathy
• Metabolic or electrolyte disturbances
• Illicit drug use
• Bacterial infections
• Other viral infections
• Parasitic infections
Dogs or cats
Wild animals (such as fox, jackal, raccoon, raccoon dog, skunk and wolf)
Bats (fruit, insectivorous or vampire bats)
Nature Reviews | Disease Primers

Managemen Rabies

Category I exposure: touches or licks on intact skin

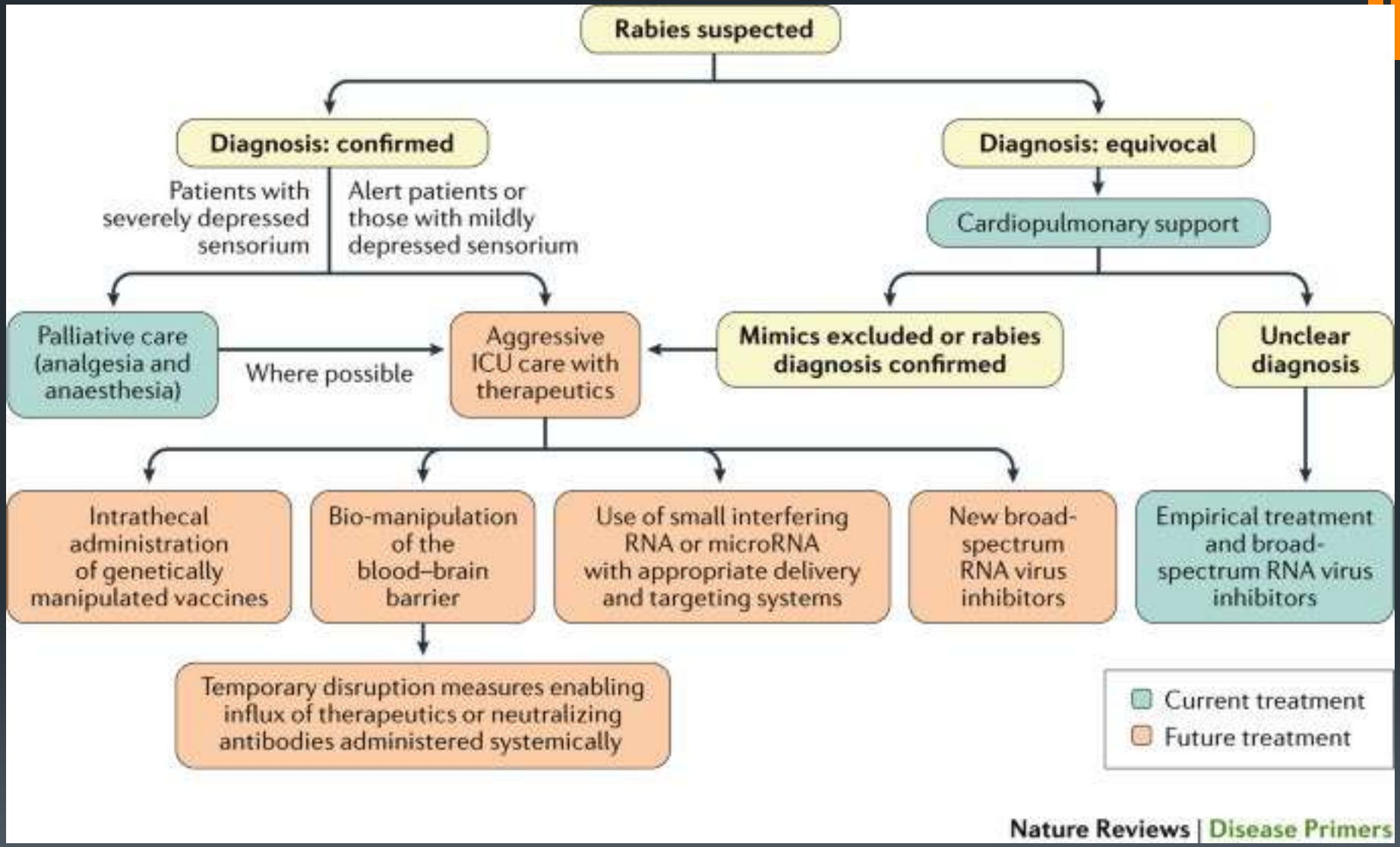
Contact with intact skin is not considered exposure; thus, post-exposure prophylaxis is not required. However, exposure assessment based on only the information provided by the individual involved might not be sufficient if the person is a minor, as contact with mucous membranes cannot be excluded. In such cases, it is indicated to test for the presence of broken skin with the application of an alcohol swab over the scratches (pain indicates broken skin). If the individual reports pain, the exposure risk is category II.

Category II exposure: nibbling over uncovered skin, minor scratches with broken skin

Rabies vaccination is required, but rabies immunoglobulin (RIG) is not. The exposed area should be vigorously cleansed and irrigated with detergent, alcohol or iodine.

Category III exposure: single or multiple transdermal bites or scratches or contamination by saliva from rabid animals on broken skin or mucous membranes

The exposed area should be vigorously cleansed and irrigated with detergent, alcohol or iodine. Human or equine RIG should be administered and infiltrated as much as possible into the wounds; if the calculated RIG dose is inadequate to infiltrate all wounds, RIG should be diluted with saline to infiltrate all wounds.


Rabies suspected
Diagnosis: confirmed
Diagnosis: equivocal
Patients with severely depressed sensorium
Alert patients or those with mildly depressed sensorium
Cardiopulmonary support
Palliative care (analgesia and anaesthesia)
Where possible
Aggressive ICU care with therapeutics
Mimics excluded or rabies diagnosis confirmed
Unclear diagnosis
Intrathecal administration of genetically manipulated vaccines
Bio-manipulation of the blood–brain barrier
Use of small interfering RNA or microRNA with appropriate delivery and targeting systems
New broad-spectrum RNA virus inhibitors
Empirical treatment and broad-spectrum RNA virus inhibitors
Temporary disruption measures enabling influx of therapeutics or neutralizing antibodies administered systemically
Current treatment
Future treatment
Nature Reviews | Disease Primers

AIDS

- AIDS (ACQUIRED IMMUNE DEFICIENCY SYNDROME) → RETROVIRUS HIV → LIMFOSIT T, MONOSIT, INDOTIL, NEURON DAN SEL GLIA
- STD. I : LIMPADENOPATI UMUM, HEPATOSPLENOMEGALI
- GX KOMPLEK : LELAH KRONIK, KERINGAT WAKTU MALAM, DIARE, HERPES SIMPLEK, KANDIDIASIS MULUT

STD LANJUT: DEMESIA, DISORIENTASI, GANGGUAN PENGLIHATAN & PERUB. KEPRIBADIAN

DAYA TAHAN ↓ → PENYAKIT-2 INFEKSI

PEMERIKSAAN :

D L
TITER A B THD VIRUS
LCS
KULTUR VIRUS
EEG, CT SCAN

TERAPI

SIMPTOMATIK
KORTIKOSTEROID→ EDEMA OTAK
ACYCLOVIR SELAMA 10 HR

Lumbal Pungsi

Lumbar puncture is used to obtain cerebrospinal fluid for analysis and to drain CSF and reduce intracranial pressure, for example in patients with idiopathic intracranial hypertension, communicating hydrocephalus or CSF fistula.

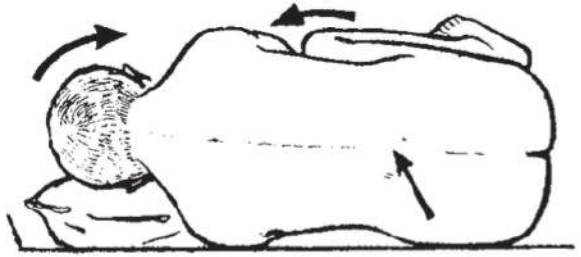
TECHNIQUE

Use the smallest gauge possible to reduce post LP headaches (PLPH), preferably 22G or 20G. Using 'atraumatic' needles rather than standard cutting needles reduces the frequency of PLPH, for 22G needles from ~20% to ~5%.

1. *Correct positioning of the patient is essential.* Open the vertebral laminae by drawing the knees up to the chest and flexing the neck. Ensure the back is parallel to the bed to avoid rotation of the spinal column.

2. Identify the site. Usually aim for the L3/4 space at iliac crest level, but since the spinal cord ends at L1 any space from L2/L3 to L5/S1 is safe.

3. Clean the area and insert a few millilitres of local anaesthetic.



4. Ensure the stylet of the LP needle is fully home and insert at a slight angle towards the head, so that it parallels the spinous processes. Some resistance is felt as the needle passes through the ligamentum flavum, the dura and arachnoid layers.

Lumbal Pungsi

Kontraindikasi

- Peningkatan TIK
- Trombosit dibawah 40.000/ PT < 50% control

Analisa

- Tekanan
- Bakteri/Kultur
- Biokimia (Protein, glukosa, dll)

Diagnosis	*Appearance*	*Pandy Reaction*	*Cell Count, Cytology*	*Biochemistry*	*Other Findings*
Normal lumbar CSF	Clear, colorless	–	Up to 4 cells/μl, mainly lymphocytes (85 %)	Lactate < 2.1 mmol/l. Albumin ratio: Adults over 40 years, < 8; under 40 years, < 7; children under 15 years, < 5	Glucose 50–60 % of blood level
Purulent (bacterial) meningitis	Turbid	+++	Several thousand/μl, mainly neutrophils	Lactate > 3.5 mmol/l; albumin ratio > 20×10^{-3}	Demonstration of bacteria
Brain abscess	Clear, occasionally turbid	+/-	A few hundred/μl, mononuclear cells and/or neutrophils	Albumin ratio normal or mildly elevated	Low glucose, bacteria sometimes demonstrable, local IgA synthesis
Encephalitis (herpes simplex)	Clear, colorless	+/-	Normal or mononuclear pleocytosis (lymphocytes)	Albumin ratio > 10×10^{-3}	IgG, IgM, IgA elevated; demonstration of specific Ab, PCR positive for HSV
Viral meningitis	Clear	+	Up to several hundred mononuclear cells, including activated B lymphocytes	Albumin ratio up to 20×10^{-3}; lactate < 3.5 mmol/l	
Tuberculous meningitis	Yellow-tinged	+++	Up to 1500/μl, mixed cellular picture, mostly mononuclear cells	Albumin ratio > 20×10^{-3}; glucose < 50 % of serum glucose	IgG and IgA elevated; mycobacteria demonstrated by culture and PCR
Neurosyphilis	Clear or turbid	+/-	Mononuclear pleocytosis		Immunoglobulins elevated, TPHA positive
Multiple sclerosis	Clear, colorless	+/-	Up to 40 mononuclear cells/μl	Albumin ratio < 20×10^{-3}	Oligoclonal bands revealed by isoelectric focusing
Acute neuroborreliosis (Lyme disease)	Clear		Up to a few hundred mononuclear cells/μl	Albumin ratio < 50×10^{-3}	Immunoglobulins elevated, demonstration of antibody
Fungal meningitis	Clear		Up to a few hundred mononuclear cells/μl		Immunoglobulins elevated, demonstration of fungi by culture and special stains
Polyradiculitis (Guillain–Barré syndrome)	Clear		No more than mild pleocytosis	Albumin ratio up to 50×10^{-3} ("albumino-cytological dissociation")	

Terima Kasih

MIELUM

ANATOMI:

- Mulai foramen magnum (C2) s/d vert L_1
- Pada manusia ascendens medulare (mielum lebih pendek daripada vertebre)
- Ptg dari mielum ada kornu anterior dan 3 traktus:
    1. Tr. Kortikospinalis
    2. Tr. Spinotalamikus
    3. Funikulus posterior
- Letak lesi harus dibawah C_2, gangguan bilateral

PEMERIKSAAN:

Pada penderita dengan kelumpuhan pada ke-2 tungkai (paraparese/plegi) harus ditetapkan beberapa hal:

1. Batas atas lesi: - Kelainan Sensoris
   - Kelainan Motoris
   - Kelainan SSO
2. Harus ditentukan adanya blok:
   - Tes queckenstedt
   - Ayala indeks
   - Kadar total protein dalam likuor
3. X-foto kolumna vertebralis

4. **Laboratorium:**
   - Hb, Eritrosit : Anemia
   - LED : Infeksi, karsinoma
   - Alkalid acid fostatase: keganasan
   - Spektrum protein

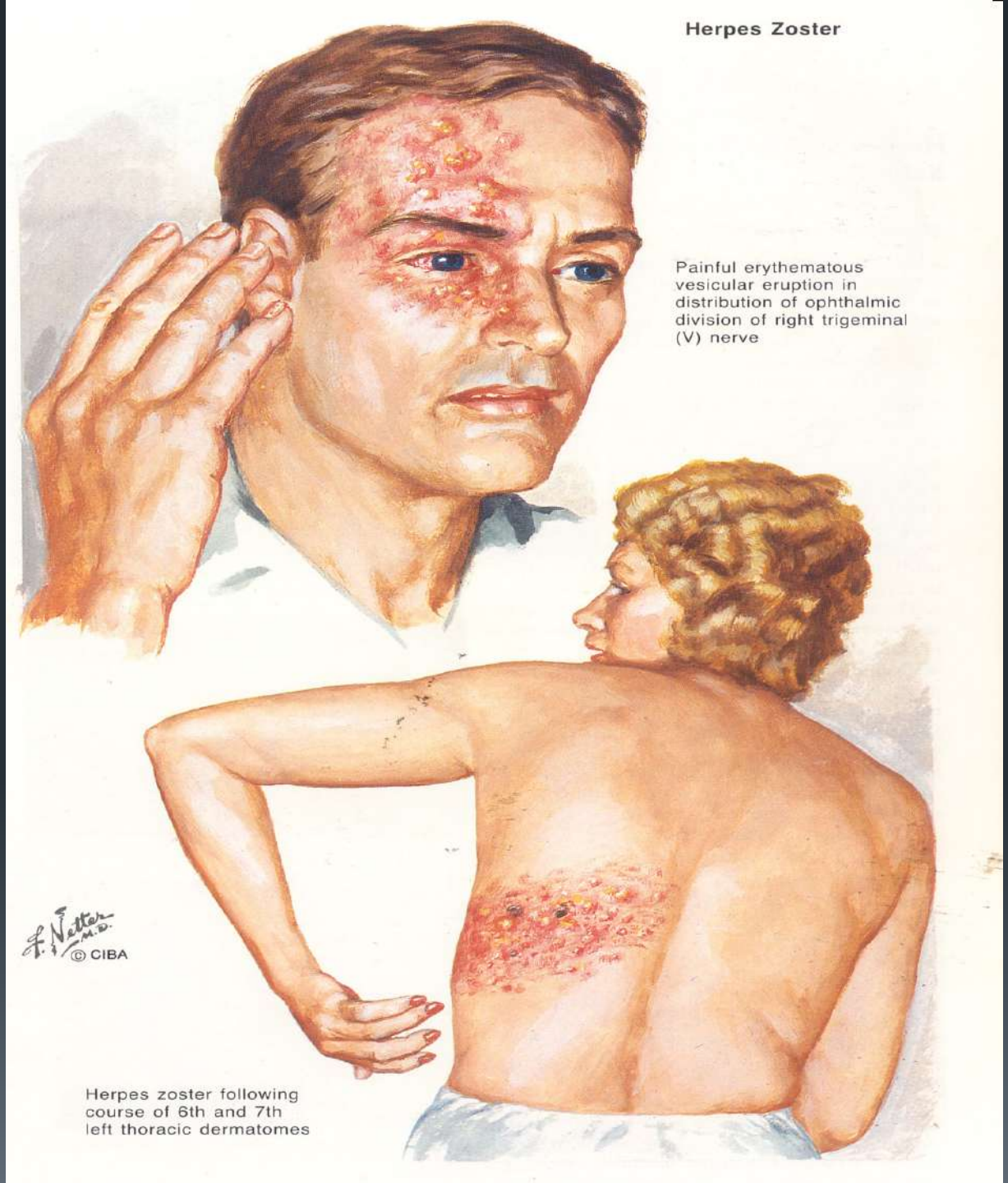
PENYAKIT MIELUM :

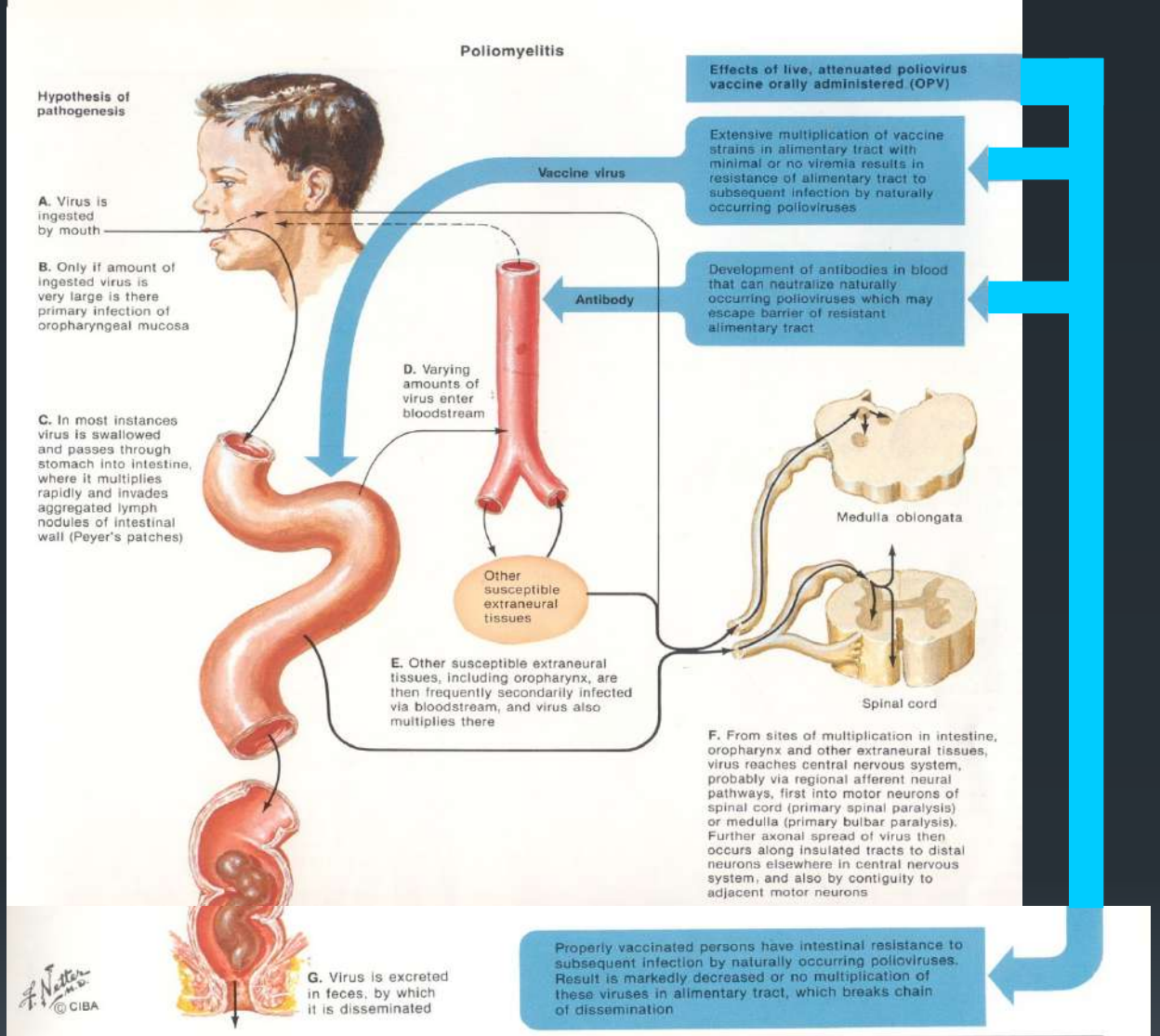
- Tumor
- Infeksi
- Trauma
- Lain-lain

MIELITIS

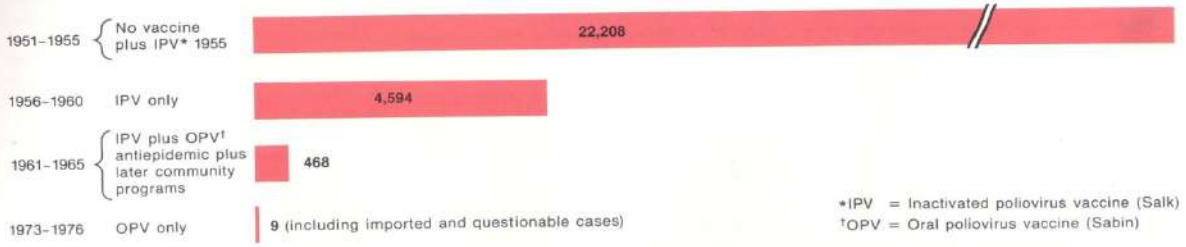
- Adalah suatu keradangan pada jaringan mielum
- Etiologi : Kokus
  Sifilis
  Virus
- Gejala :
  - Timbul mendadak
  - Rasa panas & nyeri pada pinggang (girdle pains)
  - Gangguan sensibilitas (rasa nyeri & raba) tidak komplit, batas tidak jelas
  - Gangguan motorik, flasid → spastik
  - Gangguan autonom : sulit miksi → overflow inkontinensia

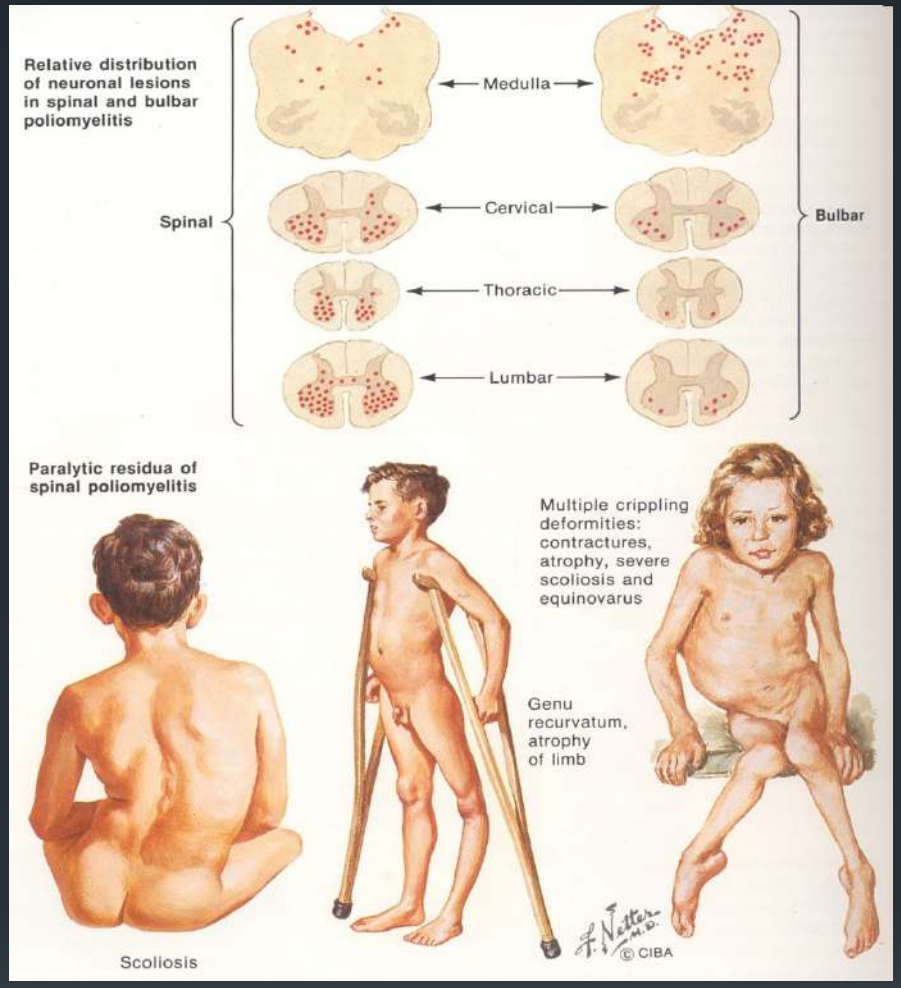
- **Pemeriksaan Likuor :**
  - Jumlah sel ↑
  - Kadar protein ↑
  - Kokus → sediaan hapus kokus (+)
  - Sifilis → WR / VDRL (+)
- **DD:** Defisiensi B12
  Siringomielia
  ALS
- **Terapi :**
  - Tergantung penyebab
  - Simptomatis

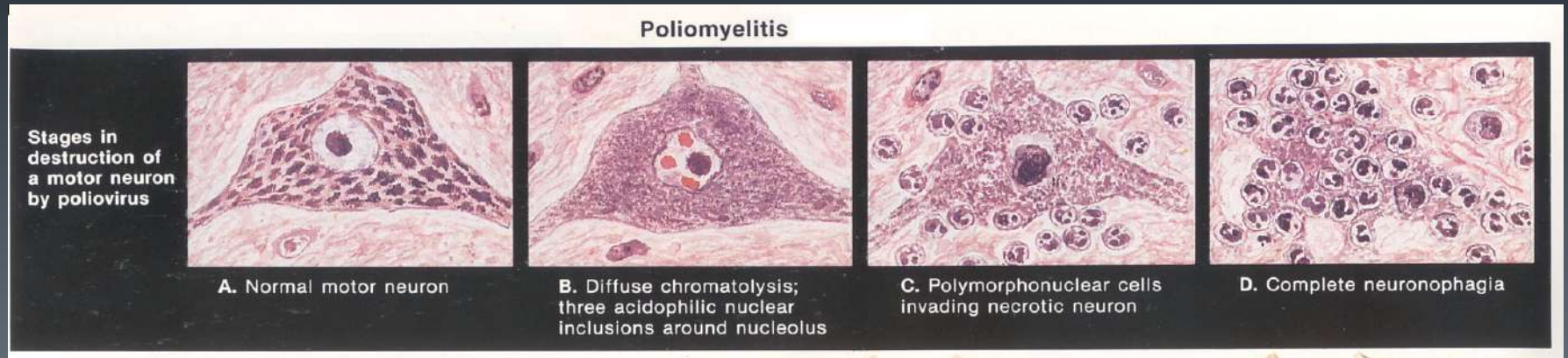

Herpes Zoster
Painful erythematous vesicular eruption in distribution of ophthalmic division of right trigeminal (V) nerve
F. Netter M.D.
© CIBA
Herpes zoster following course of 6th and 7th left thoracic dermatomes



Paralytic poliomyelitis in USA, 1951 to 1976 (average number of cases per year) and effect of vaccine



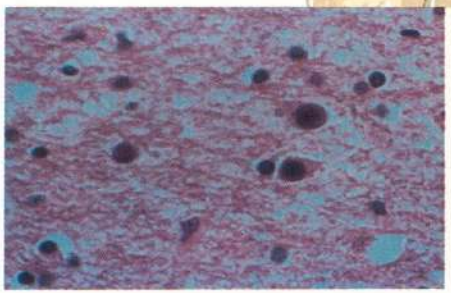

Relative distribution of neuronal lesions in spinal and bulbar poliomyelitis
Medulla
Spinal
Cervical
Thoracic
Lumbar
Bulbar
Paralytic residua of spinal poliomyelitis
Multiple crippling deformities: contractures, atrophy, severe scoliosis and equinovarus
Genu recurvatum, atrophy of limb
Scoliosis
F. Netter, M.D.
© CIBA


Poliomyelitis
Stages in destruction of a motor neuron by poliovirus
A. Normal motor neuron
B. Diffuse chromatolysis; three acidophilic nuclear inclusions around nucleolus
C. Polymorphonuclear cells invading necrotic neuron
D. Complete neuronophagia

Slow Virus Infections

Progressive multifocal leukoencephalopathy (PML)

Coronal section of brain showing many minute demyelinating lesions in white matter, which have coalesced in some areas to form irregular cavitations

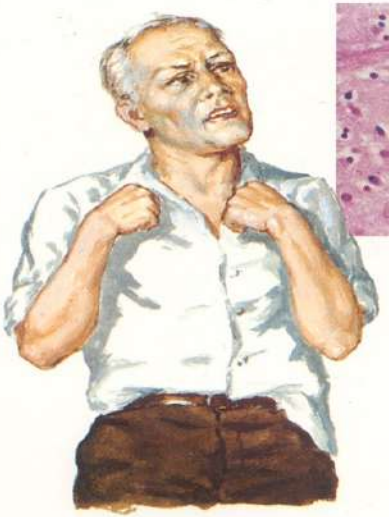


Section from edge of demyelinated focus showing abnormal oligodendrocytes with large hyperchromatic nuclei (H and E stain)

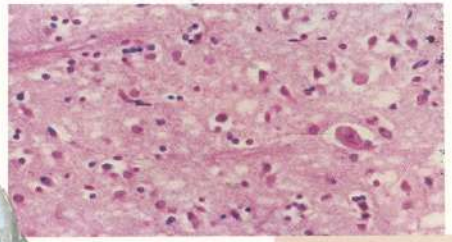
Electron micrograph showing giant glial nucleus with inclusion bodies

Electron micrograph showing papovavirus virions isolated from brain

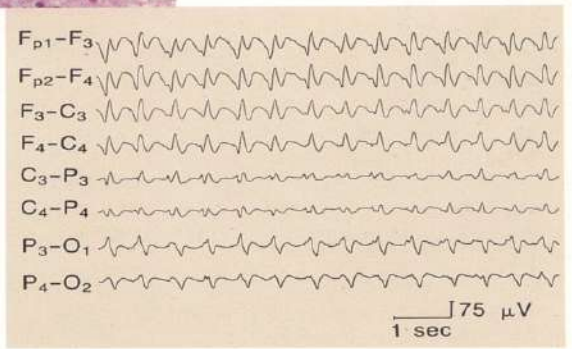
Creutzfeldt-Jakob disease



Demented patient exhibiting myoclonus



Section from putamen showing extensive loss of neurons and spongiform brain tissue. Spinal cord usually shows similar loss of motor neurons



EEG showing characteristic diffuse periodic wave pattern

F. Netter M.D. © CIBA