THE NERVOUS SYSTEM

. MAYOR COMMUNICATION (BODY).
• INTEGRITY-METABOLIC PROCESSES.
• DISORDERS (NS) \(\rightarrow\) COMPLEX.
• ISOLATED MIN. LESION ORGAN \(\rightarrow\) NO EFFECTED NORMAL FUNCTION.
• INFARCT POSTERIOR LIMB (INTERN-CAPSULE OF THE BRAIN \(\rightarrow\) PARALYSIS CONTRA LATERAL.
BRAIN PARENCHYMA

- NEURONS → GLIAL CELLS (AST’CYTE, OLIGODENDROCYTES & EPENDYMA)
- BLOOD VESSELS.
- MICROGLIA.
- NEUROPIIL (FIBRILAR BACKGROUND)
NEURONS

• SMALL ROUND CELLS.
• GRANULE CELLS → CEREBELLUM.
• DESTRUCTION (COAGULATION NECROSIS ← HYPOXIC-ISCHEMIA)
  - LOSS OF CYTOPLASMIC RIBONUCLEO PROTEINS & DENATURATION OF CYTOSKLETAL PROTEIN → CYTOPLASMIC EOSINOPHILIA (“RED NEURON”) H&E.
- NUCLEAR CHANGES
  * PYKNONIS
  * KARYORHEXIS
  * KARYOLYSIS

- APOPTOSIS

- CHROMATOLYSIS → AXON INJURY
  (DISPERSION NISSL SUBSTANCE AND SWELLING OF THE NEURONAL CELL BODY)

- EXPL : - ALZHEIMER & PARKINSON
GLIAL CELLS

• NEUROGLIAL
  - ASTROCYTES (SUPPORTIVE FNC)
  - OLIGODENDROCYTES (NUTRITIVE)
• MICROGLIAL
ASTROCYTES (SUPPORTIVE)

- MAYOR SUPPORTING CELLS (FIBROUS)
- DO NOT PRODUCTION COLLAGEN
- INJURY → SWELLING CYTOPLASMIC (INCREASED SYNTHETIS GFAP)
- GEMISTOCYTIC ASTROCYTES
- ROSENTHAL FIBRE
- CORPOR A AMYLACEA
OLIGODENDROCYTES

• NUTRITIVE FNC.
• SMALL CELLS WITH SHORT PROCESSES
• NEURONE & MYELINE (+)
• NEURONES DAMAGED → SATELITOSIS
MICROGLIAL CELLS

- MEMBER OF THE MN-PHAGOCYTIC
- NEAR BLOOD VESSELS
- LIKE MACROPHAGES
- ECCENTRIC NUCLEUS AND FOAMY CYTOPLASM.
- AROUND INFARCT (+)
DIS. INTRA CRANIAL VAULT.

- EDEMA
- HERNIATION
- HYDROCEPHALUS
CEREBRAL EDEMA.

• INCREASED WATER CONTENT WITHIN THE BRAIN PARENCHYMA.
* VASOGENIC EDEMA → EXTRA VASASI FLUID TO INTERSTITIAL SPACE (INFLAMRATION, NEOPLASMS)
* CYTOTOXIS EDEMA → INCREASED IN TRACELLULAR FLUID (INTRA CELULAR EDEMA)
CEREBRAL EDEMA

• MORPHOLOGY :
  - SOFTER THAN NORMAL
  - OVERFULL CRANIAL VAULT
  - FLATTENED GYRI
  - INTERVENING SULCI NARROWED
  - COMPRESSED VENTRICULAR CAVITY
  - HERNIATION MAY ACCURED
HERNIATION.

- INCR. INTRA CRANIAL PRESSURE (INFANTS, CHILDREN, ADULT)
- SURFACE EDEMATOUS BRAIN.
- TRANSTENTORIAL HERNIATION.
- SUBFALCINE HERNIATION.
- TONSILAR HERNIATION.
HYDROCEPHALUS.

- CEREBRO SPINAL FLUID \(\rightarrow\) (VENTR)
- CHOROID PLEXUS \(\rightarrow\) CSF (LATERAL AND FOURTH VENTRICLE)
- \(\rightarrow\) INTRA CRANIAL PRESSURE \(\rightarrow\)
- PATH- DECREASED RESORPTION.
  (TUMOR CHOROID)
  - OVERPRODUCTION
- TYPE - NONCOM’TION HDC (OBSTR)
  - COMMUNICATION HDC (MENINGITIS OR SUBARACHNOID HEM’GE)
HYDROCEPHALUS EX VACUO

• DILATATION VENTRICULAR SYSTEM
• COMPENSATORY INCREASE CHF VOLUME SECONDARY (LOSS OF BRAIN PARENCHYMA)
• ASSOCIATED WITH ATROPHY PARENCHYMA (THINNING OF CORTICAL GYRI AND WIDENING OF SULCI)
VASCULAR DISEASES

• 15 % CARDIAC OUTPUT (BRAIN)
• 20 % OXYGEN BODY (BRAIN)
• INTERUPTION B. FLOW (MINUTES) → IRREVERSIBLE INJURY (BRAIN)
• - GEN.REDUCT.BLOOD FLOW
• - INFARCT (LOCAL OBSTRUCTION)
• - HEMORRHAGE.
GLOBAL HYPOXIC

• = ISCHEMIC ENCEPHALOPATHY.
• SYSTOLIC < 50 MMHG /→ AUTOREGULATORY MACHANISM <
• HYPOXIA  → OXYGEN <
•  → DAMAGE OF NEURONE.
GLOBAL HYPOXIC

• MORPH:
  - ACUTE (CAR. ARREST) → Mac/Mic (N) 24-48 hr → SOFTENED & IRR.EDEMA, DISCOLORATIONOM, HEM’RRHAGE (+/-) (LAMINAR CORTICAL NECROSIS)
  - DEMARCATION (GRAY/WHITE MTR) BLURRED & EDEMA.
  - CER.PAR’CHYM → SOFT & NECR’TIC → BRAIN DEATH (SWOLLEN, DUSKY)
GLOBAL HYPOXIC

• MICROS (12 -24 HR) :
  - NEURONAL (SHRINKAGE, SWELLING, EOSINOP.CYTOPL, PYKNSIS, NECR.)
  - PARENCH (VACUOLATED, EDEMA & ENDOTHELIAL CELL SWELLING)
  - INFLAMATORY, NEUTONS(-) → CYST AND GLIOTIC.
GLOBAL HYPOXIC

• CLINIC:
  - CARD.DYSRITH., SHOCK, INCR.I.C.P
  - RESPONS → YOUNG < OLDER
  - TIME & TEMP. → HYPO < HYPER
  - → ELECTRICAL ACTIVITY (-)
INFARCTS.

- BLOOD FLOW (-)
- 70 – 80 % (CER.VASC.DIS/STROKE)
- SEVENTH DECADE >>
- MALE > FEMALE
- CER.VASC.ATH’SIS → BRAIN INF’CTS RISK : HYPERTENSION/DM/SMOKING OCCL’TION/EMBOLI
- LOC : I.C.ART, PROX.MIDLE C.ART AND BASILAR ART)
INFARCTS.

• MORPHOLOGY:
  - 4 – 12 HR NORMAL (BRAIN=MACR) → ISCH. NEURAL CHANGES + INFL.
  - 36 – 48 HR → NECROTIC, BLURRED, EDEMA, HEMORRHAGE.
  - THIRD DAY → MACROPHAGE, NECR. PARENCHYMA → SHARP DEM’TION.
  - 1 MTH → PHAG >>, LIQ’ION, CAVITY.
  - 6 MTH → COMPLET LIQUEFACTION
INFARCTS

- CLINICAL:
  - TRANSIENT EPISODE= TIAs (UP TO 24 HR = 1/3 PTS) \(\rightarrow\) SUDDEN

- LOC:
  - \# BRANCH. MCA (\(>>\)) \(\rightarrow\) (EMBOLI).
  - \(\rightarrow\) CONTRA.LAT. HEMIPARESIS & SPASTIS, LOSS OF SENSATION, VISUAL FIELD ABNOM, APHASIA
INFARCTS.

# OCL.INT.CART.ART (THROMB) → <<
  → IPASI LAT. → MONO OCC.BLIND.
# BRANCH OF VERTEBROBASILAR (ATHERO SCL & THROMB EMBOLI)
  → FATAL (BRAIN STEM)
  → SMALL → CLINICAL SILENT INFC.
I. CRANIAL HEMORRHAGE

• PRIMARY BRAIN PARENCHYMAL HEMORRHAGE.
• SACCULAR ANEURYSM & SUBARACHNOID HEMORRHAGE.
• VASCULAR MALFORMATION.
  * ART. VENOUS MALFORMATION.
  * CAPILARY TELENGLIECTASES.
  * VENOUS ANGIOMAS.
  * CAVERNOUS ANGIOMAS.
INTR.CRANIAL HEMORRH.

- ANTICOAGULANT THERAPY.
- VENOUS OCCLUSION.
- TRAUMA (EPI OR SUBDURAL SPACE)
PRIM.BRAIN PARENCH.HEM.

• NONTRAUMATIC → MIDDLE TO LATE ADULT LIFE (60 OLD → >>).  
• HYPERTENSION >> 50%  
• 15 % → DEATH.  
• CHRONIC HYPERTENSION → MICRO ANEURISM (CHARCOT BOUCHARD M.A) <300UM (BASAL GANGLIA)  
• COAGULATION DISORDERS, O.H.SURG, NEIPLASM, AMYLOID DEPOSITS, VASCULITIS, SAC.ANEURYSM & VASC.MALFM.
MORPHOLOGY.

• BASAL GANGLIA (PUTAMEN & EXTER. CAPSULE) → THALAMUS, WHITE MAT TER, PONS & CEREBELLMUM.

• EFFECT : - HERNIATION
  - HEMATOMAS
  - INFARCTS
  - DURET HEMORRHAGES

• HEMOTOMAS → RESORB (CHRONIC)
CLINICAL FEATURES.

• ALMOUST $\Rightarrow$ ABRUPT $+$ I.ICR.PRESS. (SEVERE HEADACHE, VOMITING & RAPID LOSS OF CONSCIOUSNESS.

• LOCAL SIGNS (UNDETECT).

• BRAIN STEM COMPRESSION $\Rightarrow$ DEEP COMA, IRRG.RESP $+$ APNEA (CHENE STOKES RESPIRATION), DILATED PPL, AND SPASTICITY.
SACC.ANEURYSMS (BERRY)

• SPONTANEOUS NON TR. SUBARC.H. (RUPTURE OF SACC.ANRM)
• +/- 1 %
• >> (POLYCYSTIC KIDNEY), FIBR.MUSC. DYSPL, COARCTATION AORTA & ARTERIOVENOUS MALFORMATION (BRAIN)
• 80 % (A.BIFURCATIO), 15 – 20 % (POST. CEREBROBACILAR)
• MIDDLE A.CEREBRAL
SACC. ANEURYSMS

• CONGENITAL DEFECTS.
• BUT, INFANT AND CHILDHOOD <<<.
• 6 – 10 MM DMTRS → RUPTURE.
• 25 MM (GIANT ANEURYSM)
MORPHOLOGY

- SMALL $\rightarrow$ ASYMPTOMATIC (3 MM)
- ROUND FORM.
- $>30\% \rightarrow$ MULTIPLE ANEURYSMS.
- LAMINATED THROMBUS ($+$)
- COMPRESSION ($+$) $\rightarrow$ LOCAL EFFECT.
- RUPTURE $\rightarrow$ BRAIN INFARCT
CLINICAL FEATURES.

- LESS COMMON.
- WOMEN > MEN.
- BEFORE AGE 50 YEARS
- SEVERE HEADACHE, VOMITING, LOSS OF CONSCIOUSNESS.
- MENINGEAL SIGN: NECK RIGIDITY, BLOODY CFS.
- 50 % CAUSED RUPTURE.
CLINICAL FEATURES

• 4 – 9 DAYS $\rightarrow$ ACUTE HYDROCEPHALUS AND HERNIATION.

• CHRONIC $\rightarrow$ HYDROCEPHALUS, GRANULATION $\rightarrow$ OBSTR. CFS FLOW.
VASCULAR MALFORMATION

• ARTERIOVENOUS MALFORMATION.
• CAPILARY TELENGETASIASES.
• VENOUS ANGIOMAS.
• CAVERNOUS ANGIOMAS.
A.V. MALFORMATION.

• >> CONGENITAL.
• → CLINICAL HEMORRHAGE.(CER. HEMISPHERES, BRANCHES OF MDL.C.ART.
• GROSS → CONGLOMERATE.
• MICROS → COLLECTION HAPHAZARDLY ARRANGED VESSELS.
  → REMOTE HEMORRHAGE, CALCIFICATION, GLIOSIS.
• CLINIC : - SPONTAN HEMORRHAGE.
CAVERNOUS ANGIOIMAS.

- SPONTAN I.CR. HEMORRHAGE & SEIZUR.
- THICK-WALLED, VENOUS CHANELS SEPARATED BY FIBROUS STROMA.
CAPILLARY TELENGIECTASIS.

- SMALL.
- PUNCTATE.
- LOC. → PONS & CER. WHITE MATTER.
- CAPILLARY CHANNELS
VENOUS ANGIOMAS

• MENINGEN & SPINAL CORD >>
• DILATATED B. VESSELS.
• THIN WALLET VEINS.
• ASYMPTOMATIC.
CNS TRAUMA

• HEAD TRAUMA INJURIES $\rightarrow$ DEATH $\frac{1}{4}$ X ALL ACCIDENTAL DEATHS & 20 % $\rightarrow$ LONG TERM DISABILITY, 5 % $\rightarrow$ PERMANENT VEGETATIVE STATE (WEST).

• MOST FATAL (BLUNT TRAUMA) + BULLET INJURIES. $\rightarrow$ 1. EPIDURAL H.
  $\rightarrow$ 2. SUBDURAL H.
  $\rightarrow$ 3. PARENCHYMAL INJ.
missile

**Scalp** — laceration ± haematoma

**Skull** — fracture comminuted ± depressed fragments

**Brain** — contusion, laceration or haematoma
A. Epidural hematoma

B. Subdural hematoma
**Fig. 21.17** Extradural hemorrhage.

**Fig. 21.18** Subdural hemorrhage.
EPIDURAL HEMATOMA.

- ET.>> RUP. MEN’EAL ART.(SKULL FR).
- LOC. → BRANCH M.M.ART. → BETWEEN DURA MATER & SQUAM.PORTION TEM PORAL BONE.
- → COMPRESS & GYRAL FLATEN.
- LATE → UNCAL GYRAL & HERNIATION, BRAIN STEM COMPRESSION & DEATH.
- IMMEDIATELY → LUCID INTERVAL → PROGRESSIVE UNCONSCIOUS.
SUBDURAL HEMATOMA

• LOC: INT. D. MATER – ARA’NOID MATER
• ET: DISRUPTION BRIGING VEIN.
• TYPE: - ACUTE SD.H
  - CHRONIC SD.H
ACUTE SD. HEMATOMA.

• ET : - TRAUMA (UNI/BILAT, INFANT)
  - CONTAIN CLOTTED BLOOD

• LOC : - FRONTOPARIETAL. → SWELLING OF CEREBELLUM ON THE SIDE OF HEMATOMA.
CHRONIC SD. HEMATOMA.

• LESS FREQUEN.
• ET : - BRAIN ATROPHY (BRAIN LABIL) → TEARING OF BLOOD VESSELS.
• >> BILATERAL
• LIQUEFIED BLOOD OR YELLOW-TINGED FLUID → GRANULATION & COLLAGEN.
• CLINIC : MENTAL STATUS, FOCAL NEUROLOGIC DEFICIT, DEMENTIA (ALZ ?)
TRAUMATIC PARENCHYMAL INJURY.

- CONCUSSION.
- CONTUSION.
- LACERATION.
- DIFFUSE AXONAL INJURY.
- TRAUMATIC INTRA CER. HEM’AGE.
- GENERALIZED BRAIN SWELLING.
CONCUSSION.

• → TRANSIENT LOSS
• → PARALYSIS, SEIZURE (+/-) → RECOVERY (HOUR TO DAYS), SQUELAE (-)
• → MINIMAL LESSION, TRANSIENT INJ.
DIFFUSE AXONAL INJURY

- ET $\rightarrow$ POST TR. DEMENTIA.
  $\rightarrow$ HYPOXIC-ISCHEMIC INJ.
- $\rightarrow$ MINIMAL CHANGES.
- SEVERE CASES $\rightarrow$ HEMORRHAGES CORP. CALLOSUM & DORSAL AREAS OF BRAIN STEM.
CONTUSION

- ET: BLUNT TRAUMA → HEMORRHAGE (SUPERFICIAL BRAIN PARENCHYMAL)
- LOC: ANY PLACE → >> FRONTAL LOBE, TEMPORAL POLES, POST.CEREB’LLUM.
- FRACTURE SKULL (+) → OFTEN INTACT.
- COUP CONTUSION → FORCE & SMALL.
- CONTRECOUP CONTUSION → FRONTAL + TEMPORAL.
- → HEMORRHAGE SUPERFICIAL GRAY MATTER & +/- WHITE MATTER.
TRAU.I.CEREB.HEMOR’AGE.

- USUALLY MULTIPLE & >> FRONT.LOBE, TEMPORAL LOBE AND DEEP G.MATTER
- >>  ➔ CONTUSIO.
- + DIFFUSE AXONAL INJURY.
- +/- HEMORRHAGE  ➔ WITHOUT OTHER OBVIOUS EVIDENCE TRAUMA.
primary damage
- scalp laceration
- skull fracture
- cerebral contusions
- cerebral lacerations
- intracranial hemorrhage
- diffuse axonal injury

secondary damage
- ischemia
- hypoxia
- cerebral swelling
- infection
Skull fractures

(i) Skull damage

Sudden deceleration due to impact against a hard flat surface.

Fractures running into middle ear

Linear fractures vertex and base

Continuing into skull base
Acceleration/deceleration injury

Brain damage

Frontal contusion
Temporal contusion

brain continues to move forward and is contused by rough surface middle fossa and orbital plate

surface of gyri, the sulci being spared
Rotation

Rotary movement of cerebral emispheres

(i) variable diffuse damage to the cerebral hemispheres as minute haemorrhages

(ii) shearing of the corpus callosum: axonal bulbs from where axons have been sheared.

Sudden torsion of the midbrain (± vascular damage) may be fatal, but decerebrate survival sometimes occurs.
CONG’TAL MALFORMATIONS & PERINATAL BRAIN INURY.

• +/- 3 % (NEW BORN).
• 1/3 → INFANT MORTALITY AND LONG TERM DISABILITY.
• CNS DEVELOPMENT DIPEND :
  → GENETIC.
  → EXOGEN.
  → COMBINED.
• >> CNS MALFORMATION → ONKNOWN
CONG.MALFORMATION N.S.

- NEURAL TUBE DEFECTS.
- ASS.WITH HYDROCEPHALUS.
- PRIMARY FOREBRAIN MALFORMATION.
- NEUROCUTANEOUS SYNDROME.
  - NEUROFIBROMATOSIS → TYPE I/II.
  - TUBEROUS SCLEROSIS.
  - VON HIPPEL-LINDAU DISEASE.
  - STURGE-WEBER DISEASE.
NEURAL TUBE DEFECTS.

• → ABNORMAL CLOSURE OF THE NEURAL TUBE.
• → INVOLVE THE BRAIN OR SP.CORD.
• EXAMP : - ANECEPHALY.
  - CRANIAL MENINGOCELE.
  - ENCEPHALOCELE.
  - SPINA BIFIDA → L.SACRAL
ASS. WITH HYDROCEPHALUS.

• ARNOLD-CHIARI MALFORM.
  → EXTENSION MED.OBLONGATA & PORTIONS CEREBLAR VERMIS → F.MAGNUM
  → LOWER B.STEM ELONATED & COMPR.
  + HERNIATED, STENOSIS AQUEDUCT & MALFORM.DORSAL PART MIDBRAIN.

• DANDY-WALKER MALFORM.
  → CERB.VERMIS APLASIA/HYPOPLASIA.
  → DILATATION VTR.IV/FOSSA POST.>>
FOREBRAIN DISORDERS.

• HOLOPROSENCEPHALY.
  - CEREBRAL HEMISPHERES ABNORMAL.
    (TRISOMI 13 & 15) +/- SP. CHROMOSOME

• CEREBRAL CORTICAL MALFORMATION.
  - POLYMICROGYRIA / ABSENCE GYRI
    (LISSENCEPHALY).
  → EPILEPSY.
NEUROCUTANEOUS SYNDROME.

- PHAKOMATOSIS.
- NON/NEOPLASTIK PROLIFERATION.
  - NERVOUS SYSTEM
  - EYES.
  - OTHER ORGAN SYSTEM.
PERINATAL INJURY.

- EXOGEN.
- → EARLY GESTATION.
- → DESTROYED BRAIN (GLIOSIS/INFL).
  - DD : PRIMARY MALFORMATION.
- → HYPOXIC-ISCHEMIC INJURY
- → INFECTION, INTRAUTERINE TOXIN, BIRTH TRAUMA → CNS INJURY.
PERINATAL BRAIN INJURY.

• GERMINAL MATRIX HEMORRHAGE.
   → INTRAVENTRICULAR HEMORRHAGE (PREMATURE INFANT)

• WHITE MATTER INJURY ⇐ PERIVENTRICULAR LEUKOMALACIA → FATAL.

• GRAY MATTER INJURY ← HYPOXI-ISCH, INFECTION.

• → CEREBRAL PALSY.

• → GLIOSIS → MULTICYSTIC ENCEPH’THY.
INFECTIONS OF N.SYSTEK.

• HEMATOGENOUS.
• DIRECT IMPLANTATION (TAUMA, CONGENITAL MALFORMATION, MIDDLE EAR & SINUS INFECTION, VIA PERIFERAL NVS = RABIES)
• RAPIDLY INFECTION (LEPTOMENINGES)
• LOCAL INFECTION \(\rightarrow\) ABSCESSES \(\rightarrow\) PYOGENIC BACTERIA.
EPI/SUBDURAL INFECTION.

• E.D. ABSCESS & S.D. EMPYEMAS <<<
  → HIGH MORTALITY.

• COMPLICATION IN PARANASAL SINUS OR MASTOID TRAUMA.

• AGENT: - STAPHYLOCOCCI
  - STREPTOCOCCI

• SPINAL EPIDURAL INFECTION >>>.
LEPTOMENINGITIS.

• = MENINGITIS.
• INFECTION / CHEMICAL AGENT → M’ITIS.
• - ACUTE PURULENT MENINGITIS (BACT)
  - ACUTE LYMPHOCYTIC ME’IT IS (VIRUS)
  - CHRONIC MENINGITIS (NUMBER OF DIFFERENT INFECTIOUS AGENTS)
ACUTE (PURULENT) LPT’M’IS.

- !! → MORBIDITY AND MORTALITY.
- CAUSED BY BACTERIA.(B.STREAM).
- CLINICAL FEATURES
  - NEONATAL PERIOD (FLORA MGT) BT. STREPT. AND ESCH. COLI.
  - CHILDREN OLDER THAN 6 Mth (H.INFL.)
  - YOUNG CHILDREN (STRP.PNEUMONIA)
CONT.

• OLDER CHILDREN, ADOLESCENT AND YOUNG ADULTS (NEISSERIA MENINGITIS).

• OLDER ADULTS (STR. PNEUMONIA).

• ELDERLY, NEONATUS (LISTERIA MONOCYTOGENES).

• HYDROCEPHALUS OPERATION (SHUNT) → STAPHYLOCOCCUS AUREUS
MORPHOLOGY

- MENINGES CONGESTED AND CONTAIN EXUDATE IN SUBARACHNOID SPACE.
- BRAIN AND SPINAL CORD CONGESTED AND EDEMATOUS.
- MICROS : LEPTOMENINGEN CONGEST AND CONTAIN NEUTROPHILS AND FIBRIN (ACUTE PHASE). SMEARS EXUDATE → BACTERIA (+)
- INFLAM PROCESS → VENTRICULAR CAVITY, LYMPHOCYTE & MN (+)
CLINICAL FEATURES.

• FEVER, HEADACHE, STIFF NECK & ALTERED MENTAL STATUS.
• CSF $\rightarrow$ NEUTROPHILS (+), PROTEIN $\gg$, $\rightarrow$ VASCULAR PERMEABILITY $\gg$.
• CSF GLUCOSE $\gg$.
• PROGNOSIS $\rightarrow$ DEPEND (THERAPY)
ACUTE LYMPHOCYTIC (VRL) MENINGITIS.

- CAUSED BY VIRUSES.
  (ECHO V, COXSACKIE V, MUMPS V, HIV.
- = ASEPTIC MENINGITIS.
- PROGNOSIS → (+).
- CLINICAL: - LESS SEVERE.
  - CSF (LYMPHOCYTES +)
  - CSF PROTEIN >>.
  - CSF GLUCOSE (N)
CHRONIC MENINGITIS.

- CAUSED BY BACTERIA AND FUNGI.
  - TB, CRYP.NEOFORMANS / BRUCELLA AND T.PALLIDUM (<<)
- AIDS ← CRYPTOCOCCAL MENINGITIS.
MORPHOLOGY.

• DURA MATER → THICKENED & EXUDATE (+) IN SUBARACHNOID SPACE.

• ON TB → EXUDATE AROUND THE BASE BRAIN.

• ARACH. ADHESIONS → OBSTR.H.CEPH.

• INFLITRATE → LYMPHOCYTES >>, PLASMA CELLS AND EPIT’OID HIST’CYTES.

• ON FUNGAL → OBLITERATIVE ENDARTE RITIS. → INFARCTS.
CLINICAL FEATURES.

• HEADACHE, STIFF NECK (+/-), MENINGEAL IRRITATION (+/-).

• CSF : - MN CELLS, PROTEIN >>, GLUCOSE <<.

• T. PALLIDUM → CONG/ACQUIRED.
  - MENINGES, VESSELS, CNS PERENCHY.
  → TERTIARY N.SYPHILIS.

• MENINGEAL THICK, MN INFILT, PLASMA

• TABES DORSALIS (+).
PARENCHYMAL INFECTIONS.

• = ENCEPHALITIS.
• LOCALIZED. → BACTERIAL ABSCESSES, TB AND TOXOPLASMOSIS.
• DIVIDED : - BRAIN ABSCESSES
  - TB & TOXO
  - VIRAL ENCEPH’TIS.
BRAIN ABSCESSES.

- $\rightarrow$ BACTERIA.(COCCI).
- HEMATOGEN (BAC.ENDOCARDITIS, LUNG ABSCESS,BR.ECTASIS) $\rightarrow$ BRAIN
- CONTIGUOUS $\leftarrow$ SINUSITIS, OMP.
- DIRECT IMPLNT (TRAUMA)
MORPHOLOGY.

• LOC → ANYWHERE. CEREBRAL HEMISPHERES (>>).

• SOLITARY (>>, MULTIPLE (+/-)

• M.EAR AND PARA NASAL SINUS INFC → TEMPORAL AND FRONTAL LOBE.

• LESION BEGINS → SOFTENING (CEREBRITIS) → LIQUEFIES → CAVITY + PUS.

• NEXT FEW WEEKS → PROL.FIBROBLS. AND COLLAGEN → PUS <<<
CLINICAL FEATURES.

• FEVER.
• INTRA CRANIAL PRESSURES >>
• FOCAL NEUROLOGIC DEFICIT.
• CSF : - SCANTY CELLS
  - PROTEIN >>
  - GLUCOSE (N)
• COMPLICATION : BRAIN HERNIATION,
  RUPTURE ABSCESES → VENTRICLE
  OR SUBARACHNOID SPACE.
TUBERCULOSIS

• INVOLVE BRAIN PARENCHYMA/MENING.
• HEMATOGEN ← TB PULMO
• MORPHOLOGY : - BACTERI
  - TUBERCOLOMA
  - CENTRAL NECROSIS
  - EPI’OID HIST’CYTES
  - GIANT CELL
  - LIMPPHOCYTE.

* SUBARACHNOID SPACE RUPTURE → MENINGITIS TUBERCULOSE.
TOXOPLASMOSIS.

• + OS AIDS.
• GRAY MATTER (MULTIPLE)
• MORPH : - AREA NECROSIS.
  - MONONUCLEAR CELL
  - PSEUDOCYST/TACHYZOID
  - DD : CYSTICERCOSIS
    ECHYNOCOCCO
    FREE LIVING AMEBA
VIRAL ENCEPHALITIS.

• MOST COMMON
• HSV (TEMPORAL), PML (MULTIFOCAL), CMV → HOST IMMUNOSUPPRESSION.
• MORPH: - PERIVASCULAR INF (MN, LIMPH, PLASMA & MPG)
  - AG’ATE M.GLIAL CELL
  - NEUROPHAGIA.
  - INCLUSION BODIES (NCL/CYTOPLASMIC).
DISEASES OF MYELIN

• MULTIPLE SCLEROSIS.

• OTHER ACQUIRED DEMYELINATING.

• LEUKODYSTROPHIES.
MULTIPLE SCLEROSIS.

- MOST COMMON.
- YOUNG ADULTS (18 – 40 YEARS).
- T-CELL MEDIATED INJURY TO MYELINE SHEATS AND/OR OLIGODENDRO-GLIAL CELLS.
- CD4 + CD8 CELLS (+) \(\rightarrow\) AUTOIMMUN.
- GENETIC \(\rightarrow\) (HLA-DR2 GENES \(\rightarrow\) TWINS)
MORPHOLOGY

• MACROS (EXTERNAL) → NORMAL

• ON CUT → MULTIPLE PLAQUES (DEMYELINATION)

• LOC. → ANYWHERE (BRAIN & SP.CORD)
  - PERIVENTRICULAR WHITE MATTER.
  - OPTIC NERVE.
  - WHITE MATTER SPINAL CORD.

• PLAQUE (WELL DEMARCATED)
CONTINUED.

• FEW MM TO SEVERAL CM DIAMETERS.
• ACUTE LESSION ➔ SOFT & SLIGHTLY PINK.
• OLD LESSION ➔ FIRM & PEARLY GRAY TO PINK.
MICROSCOPIC.

- AREA DEMYELINISATION (+) → PERIVENOUS DISTRIBUTION.
- PERIVASCULAR LYMPHOCYTIC INFILTRATE (VARIABLE).
- LIPID LADEN MAGROPHAGES (MYELIN BREAKDOWN)
- RESIDUAL AXONAL PROCESSES (+), BUT REDUCED IN NUMBER (CHR. LESSION) → PLAQUE (SHADOW PLAQUE)
OTHER ACQ.DEMYE.DSS.

• ACUTE DISSEMINATED ENCEPHALO-MYELITIS (IMMUNE-MEDIATED DEMYE LINATING DSS (MEASLES, CHICKENPOX, RUBELLA AND VACCINATION)

• CENTRAL PONTINE MYELINOLYSIS → DE MYELINATION IN BASIS PONTIS. (ALKCO HOLISM) → HYPONATREMIA.

• INFECTIONS (VIRAL ENCEPHALITIS, HER-PES ZOSTER AND CMV ENCEPHALITIS)
LEUKODYSTROPHIES.

- AUTOSOMAL RECESSIVE & X-LINKED.
- SPECIFIC LYSOSOMAL ENZYMATIC DEFECT (NOT ALL).
- INFANCY AND CHILDHOOD (PROGRES).
- PERIPHERAL NERVE (+)
MORPHOLOGY.

• WIDESPREAD, SYMMETRIC LOSS OF MYELIN (BRAIN AND SPINAL CORD).

• BRAIN ATROPHY (SHRUNKED CENTRUM OVALE AND OTHER CENTRAL WHITE MATTER) → GRAY AND TRANSLUCENT
MICROSCOPIC.

- LOSS MYELIN.
- MYELIN BREAKDOWN.
MENINGIOMA.

- ARACNOID GRANULATION.
- BENIGN LESSION.
- MICROS: - WHORLS FIBROUS TSS.
  - SPINDLE CELLS.
  - CALCIFIED PSAMMOMA BODIES
GANGLION CYST.

• SIRCUMSCRIBE.
• CYSTIC.
• WALL → SPINDEL WAIFE CELLS + FIBROSIS.
• INTRALUMINAL MATERIAL (GELLY LIKE)
NEOPLASMS O/T CNS.

- GLIOMAS.
  - ASTROCYTOMAS.
  - OLIGODENDROGLIOMAS.
  - EPENDYMOMAS.
- PRIMITIVE NEUROEPITHELIAL NEOPL.
- NEURONAL NEOPLASMS.
- OTH.PRIM.INT.PARENCH.NEOPLASMS.
- MENINGIOMA & MENINGEAL NEOPL.
- METASTATIC NEOPLASMA.
ASTROCYTOMAS.

• MOST COMMON.
• PRIMARY CNS TUMORS.
• CIRCUMSCRIBED.
• SLOW-GROWING.
• TYPE : - PILOCYTIC TO HIGHLY MLGT.
  - GLIOBLASTOMAMMULTIFORME.
• CLASS : - FIBRILLARY.
  - PILOCYTIC.
FIBRILLARY ASTROCYTOMAS

• = DIFFUSE ASTROCYTOMAS.
• INFILTRATIVE GROWTH.
• ADULTS >>.(ANY AGE).
• LOC. CEREB. HEMISPHERE (BUT ANY).
• GRADE: - WELL.D. (ASTROCYTOMA)
  - INT.MD. (ANAPLASTIC)
  - GLIOBLASTOMA MF(AGRESS)
• MICR : NCL PLEOMORF, MITOTIC, VASC, AND NECROSIS.
WELL-D ASTROCYTOMAS.

- MICR : - IRREG. ASTROCYTE >>.
  - INFILTRATIVE (+)
  - ATYPICAL CELLS (+)
  - FIBRILLAR PROCESSES (+)
ANAPLASTIC ASTR.

- CELLULARITY $\rightarrow$ >>>.

- NUCLEAR PLEOMORPHISM (+)

- MITOTIC ACTIVITY $\rightarrow$ >>>
GLIOBLASTOMA MULTIFORM

• IRREGULAR LESSION.
• EDEMA (PARENCHYMA).
• HEMORRHAGE.
• NECROSIS. (PALISADING)
• CYSTIC
PILOCYTIC ASTROCYTOMA

• CHILDREN (MORE COMMON) ⇒ ANY
• CEREBELLUM (>>), THIRD VENTRICLE, AND OPTIC NERVE.
• WELL DEFINED LESIONS, CYSTIC.
• ASTROCYTE ELONGATED (HAIRLIKE).
• BRIGHTLY EOSINOPHILIC “ROSENTEAL FIBRE”, PROTEIN-RICH DROPLETS (HYALINE GRANULAR BODIES)
• CELLULAR ATYPIA, MICR.VASC.PROL.
OLIGODENDROGLIOMA.

- ADULTHOOD (CEREB. HEMISPHERE).
- CYTOGENETIC ABNORM. → LOSS OF HETEROZYGOSITY (LONG ARM OF CHROMOSOM 19 AND SHORT ARM OF CHR. 1). MUTATION TP53 GENE (UNCOMMON), IN CONTRAST TO INFILTR. AST’ CYTIC.
MORPHOLOGY

• GROSS: - SOFT, GELATINOUS, CIRCUMSCRIBED.
• CALCIFICATION (+).
• MICROS: - ROUNDED CELL INFILTR.
  - UNIFORM NUCLEI + PERINUCL.HALO.
  - SATELLITITOSIS.
• NUCLEAR PLEOM, MITOTIC & NECROSIS → ANAPLASTIC CHANGES.
EPENDYMOMAS.

• ANY AGE.
• LOC. VENTR.CAVITY, CENTRAL CANAL SPINAL CORD.
• INTR.CRANIAL EP. (FOURTH VENTRICLE) → 2 DECADE OF LIFE. → OBSTR.CSF.
• INTR.SPINAL EP. → ADULTS.
MORPHOLOGY.

- WELL DEMARCATED.
- ARISING FROM VENTRICLE WALL OR INTRASPINAL CENTRAL CANAL.
- MICROSC: - ELONGATED CELLS. → RADIATING VESSELS (P.VSC. PSEUDO ROSET) OR EPENDYMAL ROSSET (LUMINA).
- MYXOPAPILLARY EPENDYMYOMA → FILUMTERMINALE OF SPINAL CORD.
PRIMITIVE NEUROEPITH. NPL.

• EMBRYONAL NEOPL. (SMALL CELLS)
• CHILDREN (MAY BE UNDIFF).
• INCLUDE : - MEDULLOBLASTOMAS
  - PINEOBLASTOMA
  - EPENDYMOBLASTOMA
  - MEDULLOEPITHELIOMA.
MORPHOLOGY.

• CEREBELAR VERMIS (FIRST 2 DECADES)
• CEREBELAR HEMISPHERE (OLDER)
• DISSEMINATED THROUGH CSF.
• MICR. : - SMALL AND PRIMITIVE CELLS.
  - SCANT CYTOPLASM.
  - SMALL ROSSETTS (HOMER WRIGHT ROSETTES)
THANK