Patho 1

Disease	Feature	Cause	Sign & Symptoms	Histology & Microscopically	Other
Multiple Sclerosis (MS)	• Most common demyelinating disease • Distinct episodes of neurologic deficits that are separated in time and are attributable to patchy white matter lesions that are separated in space.	Autoimmune response directed against components of the myelin sheath. 1. Unknown 2. Genetic 3. Environmental : vitamin D	• Progressive / chronic / attacks • Motor/ Sensory / Visual • Relapsing and remitting episodes of variable duration marked by neurologic defects, followed by gradual and partial recovery of neurologic function • Over time there is usually a gradual accumulation of neurologic deficits . • Unilateral visual impairment due to *optic nerve* involvement • Brainstem involvement produces → cranial nerve signs ; ataxia & nystagmus • Spinal cord lesion give rise → to motor & sensory impairment. • CSF : mildly elevated protein level, moderate pleocytosis, increased immunoglobulin(Ig) with oligoclonal bands.	• Lesions → plaques: discrete, slightly depressed, glassy-appearing, and gray in color, and commonly near the ventricles. • *Active plaques*(ongoing myelin)breakdown 1. contain abundant foamy macrophages stuffed with myelin debris (lipid) , 2. Myelin is usually completely absent . 3. Axons are relatively preserved 4. perivascular cuffs of Lymphocytes. 5. centered on small veins • *Inactive plaques* (quiescent) 1. No macrophage-rich infiltrate 2. inflammation mostly disappears , 3. leaving little to no myelin , 4. Axons are usually greatly diminished in number 5. gliosis. 6. reduction in the number of oligodendrocyte nuclei	• Female(2:1) • > 50 • (20-40) • Rare in childhood & after the age of 50 • Diagnosis : 1. Clinical 2. MRI 3. CSF → support the diagnosis
Alzheimer Disease (AD)	• neurodegenerative diseases • Progressive loss of particular groups of neurons, which often have shared functions • The accumulation of protein aggregates "proteinopathy"	Aβ (amyloid β) and tau proteins accumulation • Amyloid precursor protein (APP) Starts processed with β-secretase (amyloidoigenic), Aβ generation	• Insidious onset of impaired higher intellectual function, memory impairment, & altered mood and behavior. • Over time, disorientation & aphasia . • In final stages they are disabled, mute & immobile. • Death →intercurrent pneumonia or other infections.	• Aβ is highly prone to aggregation and causing neural dysfunction & elicits a local inflammatory response that can result in further cell injury. 1. Cortical atrophy 2. widening of the cerebral sulci that is most pronounced in the frontal, temporal, and parietal lobes. 3. ventricular enlargement)hydrocephalus ex vacuo) 4. Amyloid plaques (extracellular – accumulation of Aβ amyloid . Neuritic plaques are focal, spherical collections of dilated, tortuous, processes of dystrophic neurites around a central amyloid (Aβ) core. (Diffuse plaques are Aβ deposition without neurites 5. neurofibrillary tangles (intracellular – Tau accumulation(Tau containing bundles of filaments in neurons cytoplasm (encircle the nucleus), flame shapes	• Most common cause of dementia in older adults • Rare before ,50 • incidence increases with age% 1 60 →to 64 reaching 47% in 85 and older • APP gene located on chr. (~ 21Down syndrome) • Extracellular – accumulation of Aβ amyloid • Intracellular –Tau accumulation