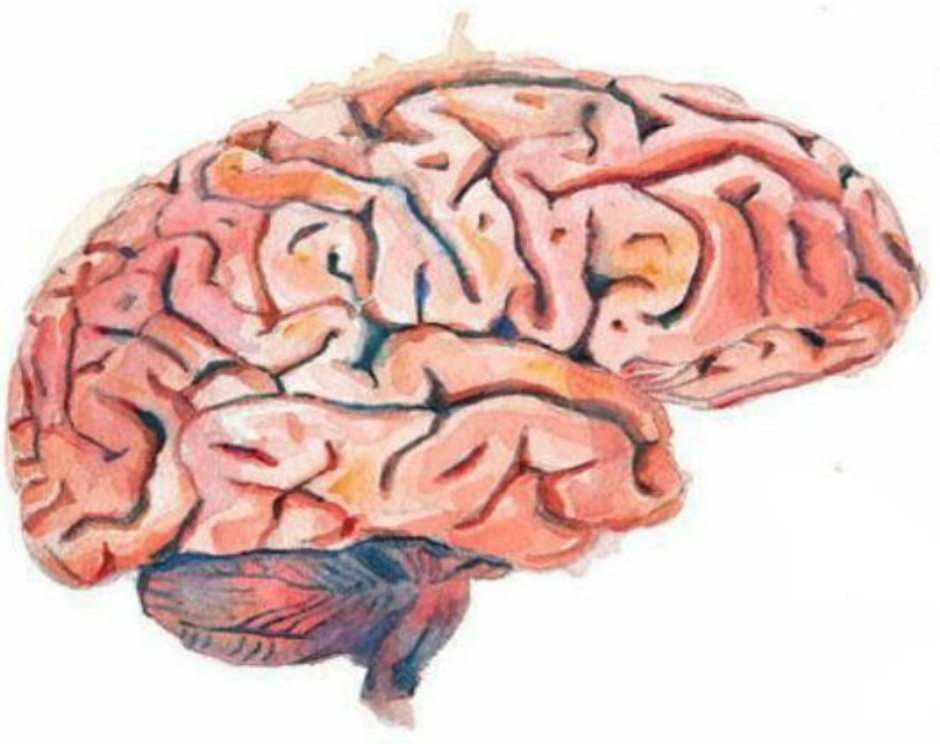
PATHOLOGY TEAM 436

MEDICINE
KING SAUD UNIVERSITY

Pathology Practical

Neuropsychiatry Block



PLEASE NOTE: Any grey arrows or circles are EXTRA just to help you understand
However, Colored arrows aren't extra !

Important Notes

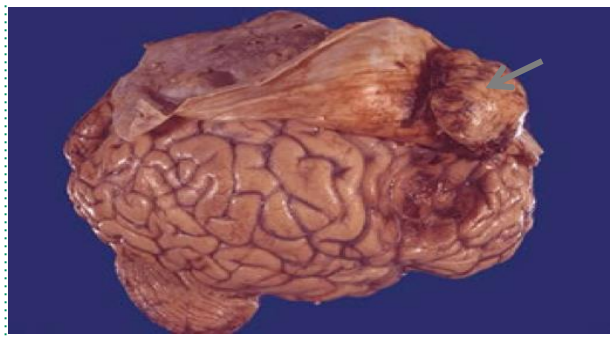
Extra Notes

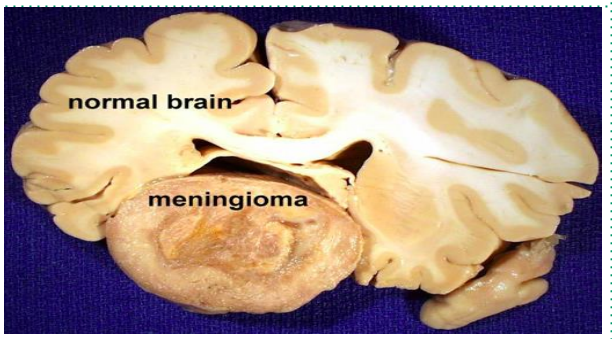
Doctors' Notes

Case 1: Meningioma

A 43-year old female complained of headache and **two attacks of seizure** in the past 4 months. Brain MRI revealed a **3 cm extra-axial mass** in the parietal region. It was dural-based with mild edema in the surrounding brain tissue.
What is your provisional diagnosis? Meningioma.
Meningioma is **benign tumor** (grade I) , they can **rarely** be aggressive and invade.

Gross

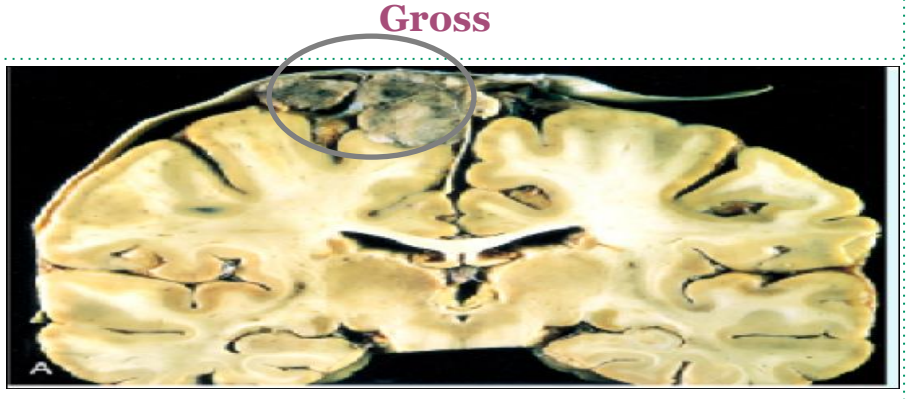




1. A meningioma beneath the dura compressing the underlying cerebral hemisphere.
2. These neoplasms are slow growing, but may reach a large size before symptoms lead to detection.

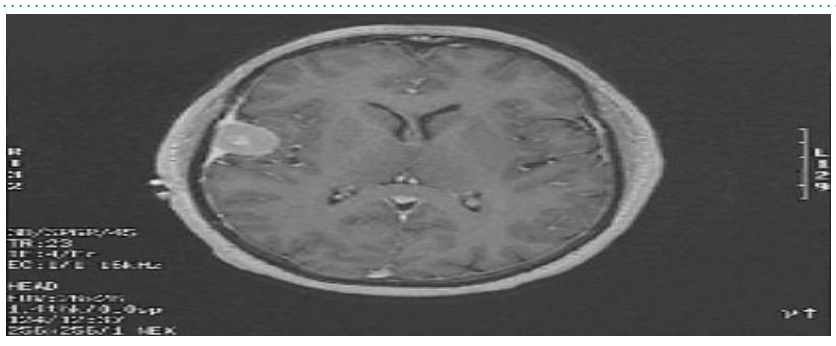
1. No haemorrhage nor necrosis.
2. compressing but it is well-defined, Not invading the cerebral cortex.

Gross



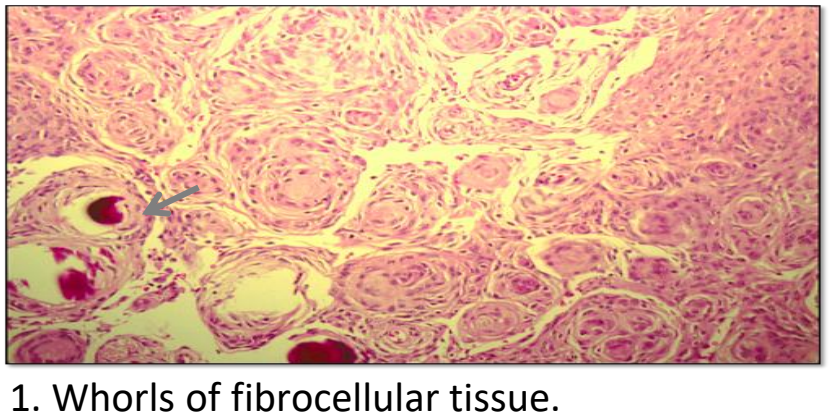
1. Parasagittal multinodular meningioma attached to the dura with compression of underlying brain.

MRI



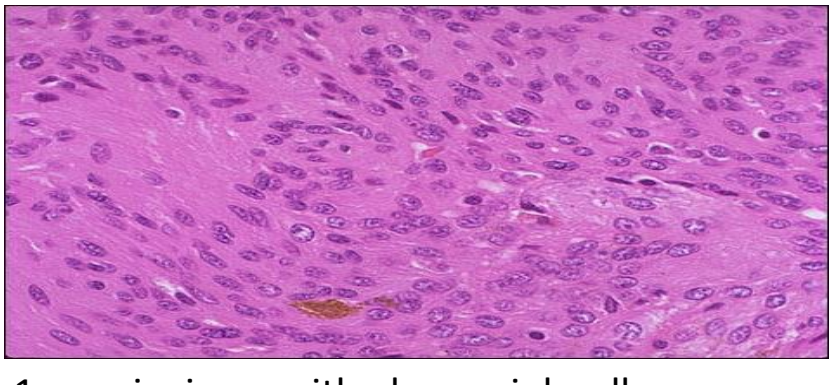
1. well-defined mass along the lateral convexity of the brain.
2. It is extending from the dural base impinging upon the cerebral hemisphere
3-This is consistent with a meningioma.

Microscopic view - LPF- (low- power- field)



1. Whorls of fibrocellular tissue.
2. Cells are oval, spindle shaped or elongated and lacking mitosis.
3. Psammoma bodies (spherical calcified particles).

Microscopic view - HPF- (high- power- field)



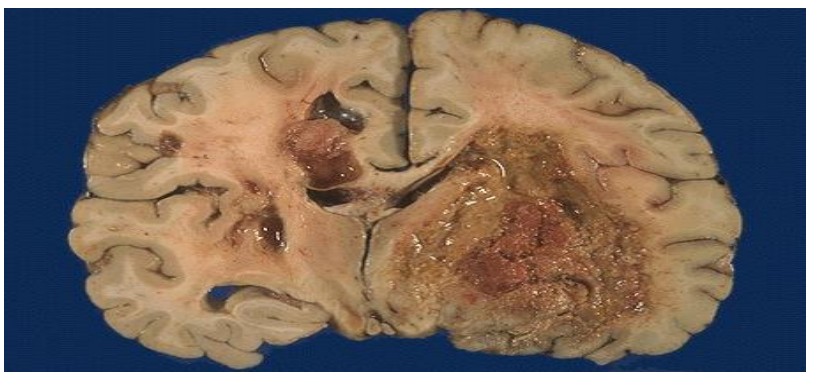
1. meningioma with plump pink cells.
2. A small amount of brown granular hemosiderin.
3. psammoma bodies (spherical calcified particles).

Case 2: Glioblastoma Multiform

A 55 years old man complained of headache for the last 2 months . Brain MRI reveals a 3 cm frontal **intra - parenchymal space** occupying lesion with rim enhancement on contrast studies.

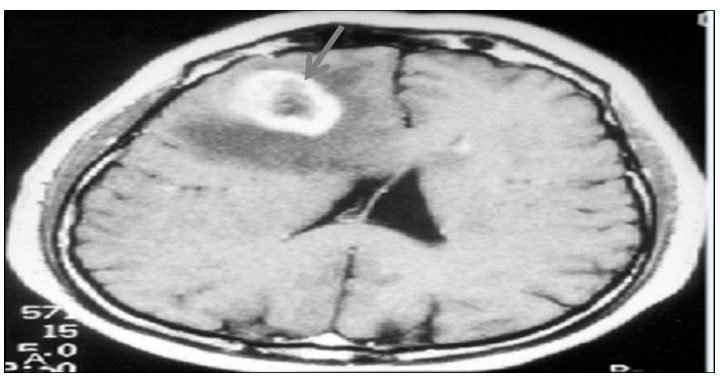
- What is your provisional diagnosis ? Glioblastoma Multiform.
- This is the **worst** possible form of Giloma , the cells of a GBM can infiltrate widely, particularly along the white matter tracts and even though the CSF.
- Gene mutated in low grade astrocytoma: **IDHI**

Gross



1. The neoplasms are highly vascular**.**
2. There is necrosis and haemorrhage.
3. It crossed the midline to the opposite side.

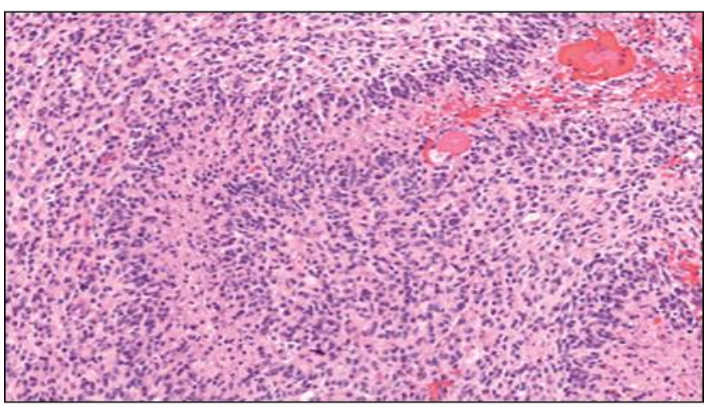
CT scan



1. CT scan of a large tumor in the cerebral hemisphere showing signal enhancement with contrast material.
2. pronounced peritumoral Edema.

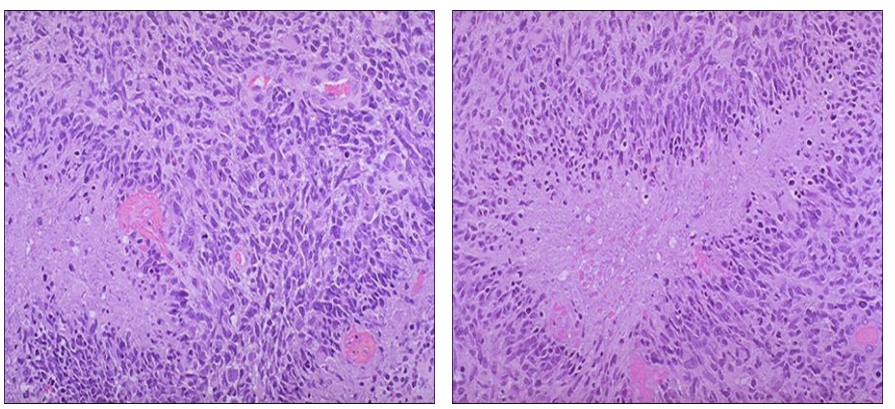
Note: When there is Edema that means this tumor is growing fast.

Microscopic- LPF (low- power- field)



1. There is foci of Necrosis
2. **Pseudopalisading** arrangement of malignant nuclei
3. Endothelial cell proliferation

Microscopic- HPF (high- power- field)



1. High cellularity
2. Marked hyperchromatism and pleomorphism
3. Prominent vascularity
4. Areas of **Pseudopalisading** necrosis with neoplastic cells palisading around it.

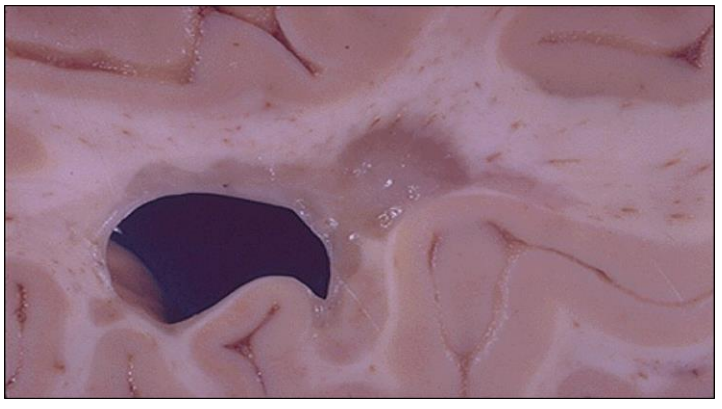
Case 3: Multiple Sclerosis

A 27 years old woman presents with a sudden onset of **right sided blindness** and weakness in her left leg. There is no history of trauma. However, **she experienced a similar episode 8 months ago** and was diagnosed as aseptic meningitis.

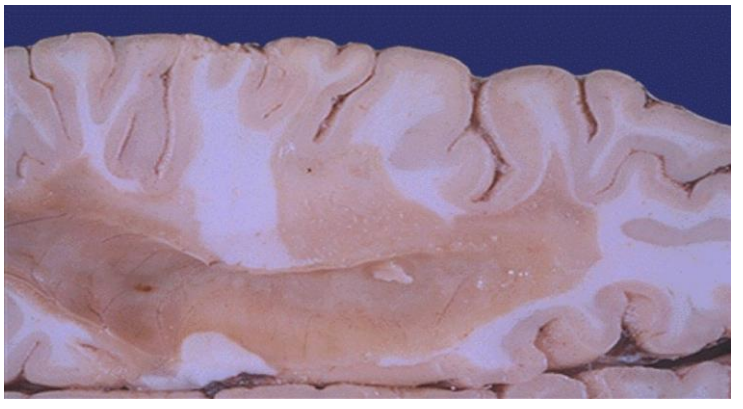
- What is your diagnosis ? Multiple sclerosis.
- Definiton: Ms is a demyelinating autoimmune disease .
- What are the microscopic features? 1.Perivenous mononuclear inflammation (lymphocytes, plasma cells and macrophages) . 2.Loss of myelin and variable loss of oligodendrocytes
  3. Relative preservation of axon 4. Relative astrogliosis (scelorosis)
- CSF findings: Increase IgG that demonstrate oligoclonal bands of electrophoresis.
- Possible Etiologies: 1. Genetic predisposition 2.immuno mediated (CD4) injury to myelin.
- Lesions can be seen with MRI scan but SCF samples are better to diagnose with.

Early (Acute) lesions	Chronic lesions
• Perivascular & parenchymal infiltration by inflammatory mononuclear cells. • myelin breakdown & phagocytosis by macrophages.	• fewer inflammatory cells • almost complete demyelination.
• No Astrogliosis.	• Severe Astrogliosis.
• Axons are relatively preserved.	• some secondary axonal loss in advanced cases. • There can be oligodendrocyte loss

Gross



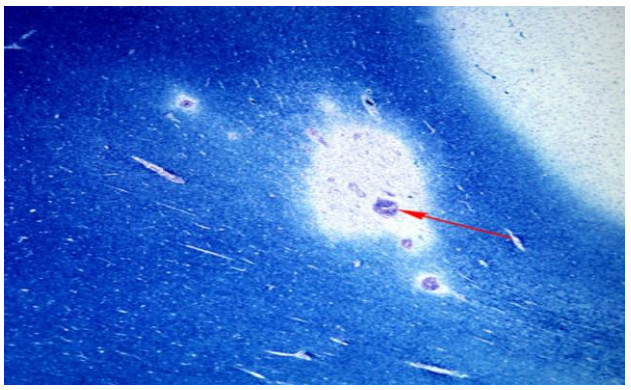
1. Demyelinating plaque lesion in white matter.



1. A large plaque of demyelination in the white matter .
2. Lead to the clinical appearance of loss of neurological function.
3. The disease is multifocal and the lesions appear over time.

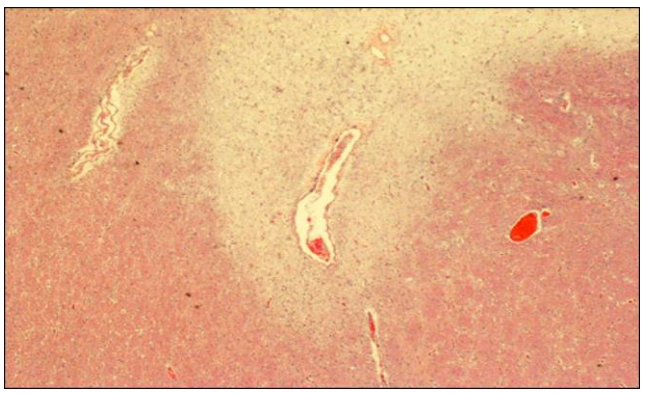
Case 3: Multiple Sclerosis

Microscopic- early MS plaque



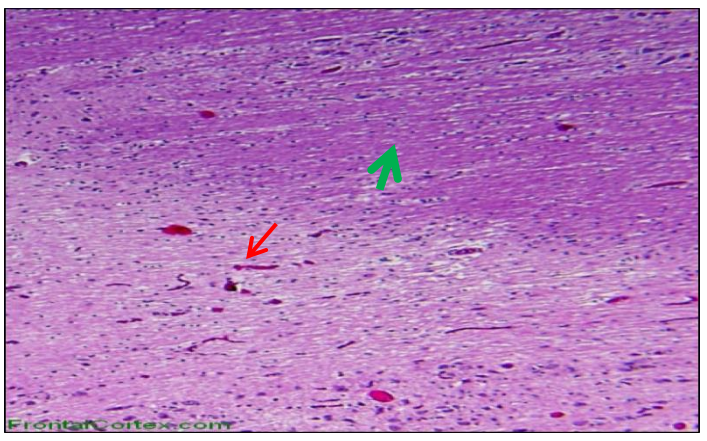
1. myelin stain (luxol fast blue/PAS).
2. The lesion is centred around a small vein (arrow) which is surrounded by inflammatory cells.

Microscopic - Long standing MS (old) plaque



1. H&E stained.
2. Lesion centred on a vein with little inflammation.
3. Myelin loss (lighter pink than the normal white matter surrounding it) .

Microscopic- Inactivated MS plaque

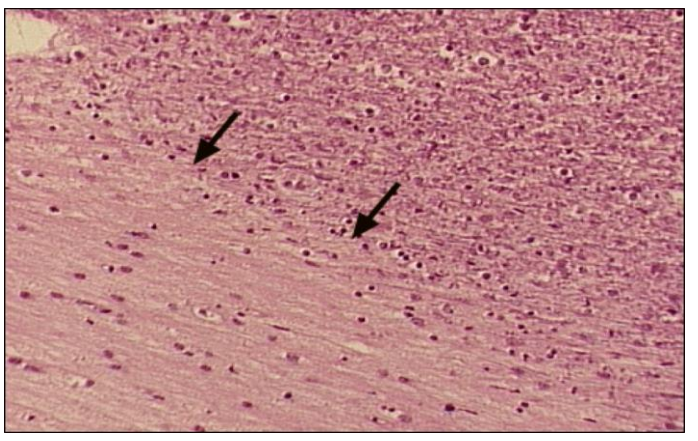


1. Inactive demyelinated plaque.

2.**Red arrow**: pale- plaque, indicates a lack of myelin.

3.**green arrow:** darker normal neuropil.

Microscopic - Older MS plaque



1. Pallor of plaque with almost no myelin.

2. Decreased oligodendroglial nuclei and increased astrocyte nuclei

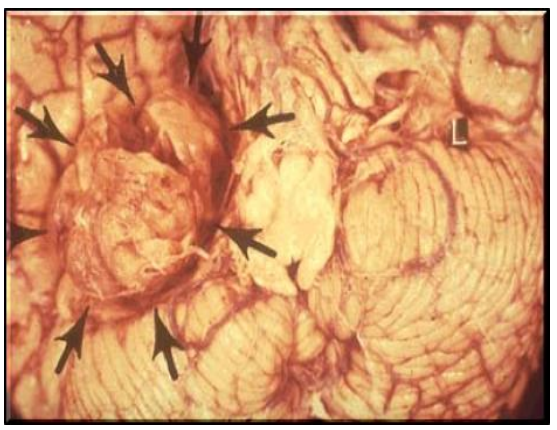
Case 4: Schwannoma

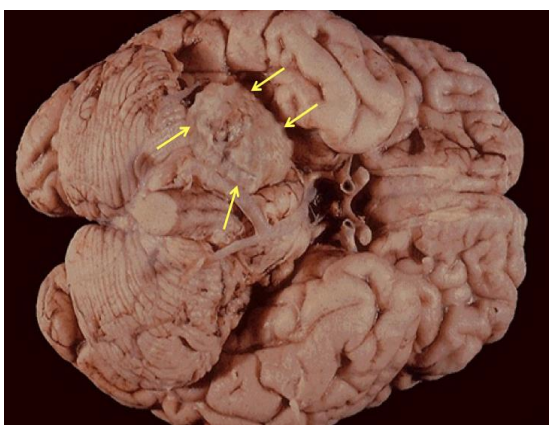
A 39 years old man complains that he had noticed a progressive **hearing loss** over a 2 years period. Except for occasional headache, he has no other complaints . Evaluation discloses severe sensorineural hearing loss of the left side . MRI shows 1.5 cm. **mass at the left cerebellopontine angle** .

What is your provisional diagnosis ? Schwannoma

- Acoustic tumors are benign tumors that can be removed, but usually **not without** damaging the eighth nerve and sometimes the facial nerve and brain stem .
- Bilateral Acoustic schwannoma is associated with NF2 (neurofibromatosis type 2)
- Acoustic Schwannoma : patients may present with hearing loss.

Gross



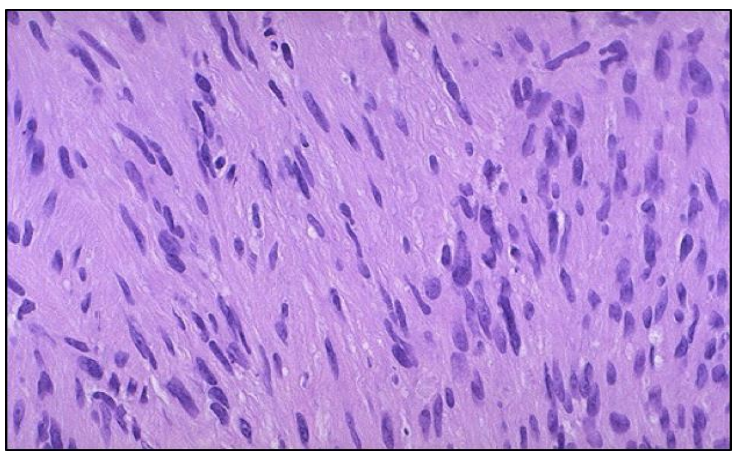


1. A nerve sheath tumor that seen most frequently on the eighth nerve (acoustic neuromas).
2. they occupy the cerebello- pontine angle (arrows)

Cut Section

1. "fish flesh" soft tan appearance. (well circumscribed)
2. similar to that of many mesenchymal neoplasms.

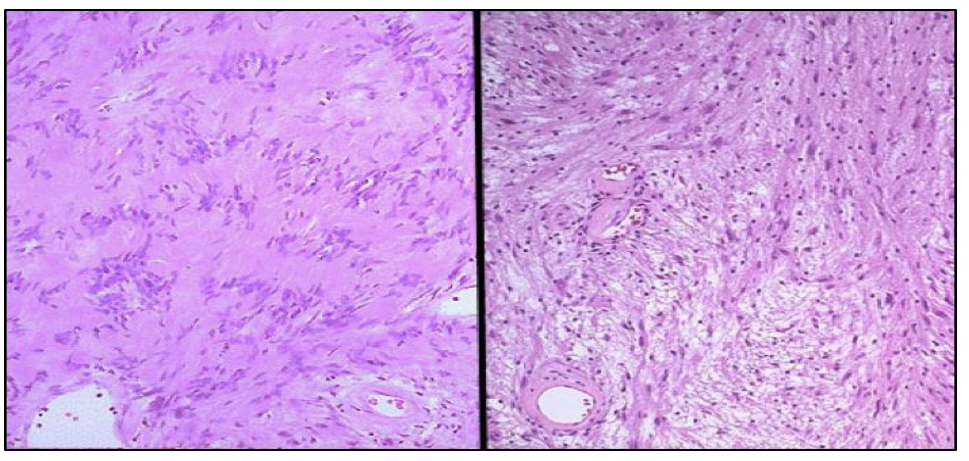
Microscopic- HPF



1. The schwannoma is seen here at higher magnification(antoni A).

(4:38) nice video if you want to understand what Antoni A and B means ☺

Microscopic- LPF



(Left) (Right)

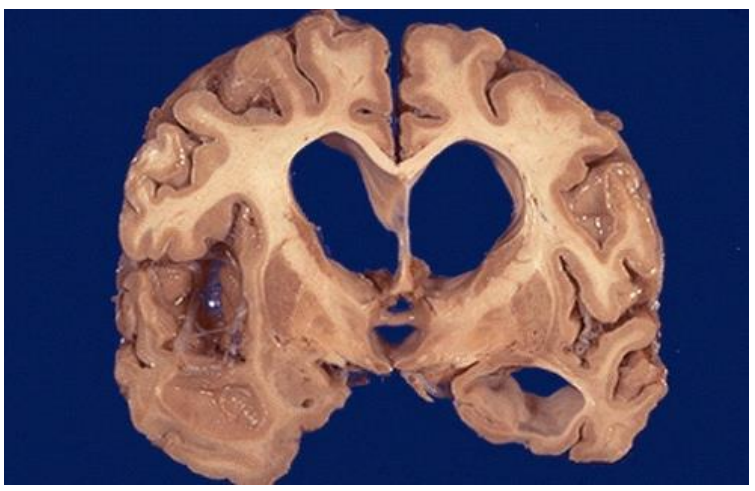
1. Cellular "Antoni A" pattern more on the left, with palisading nuclei .
2. Verocay bodies: Nuclear free zone between nuclear palisading.
3. Cellular "Antoni B“ pattern more on the right ,with a looser stroma, fewer cells, and myxoid change.

Case 5: Hydrocephalus

A 9 months infant was suffering from **enlarged head size** and admitted to hospital with convulsions, went into coma and died. Autopsy was done and the brain was **large with dilated ventricles .**

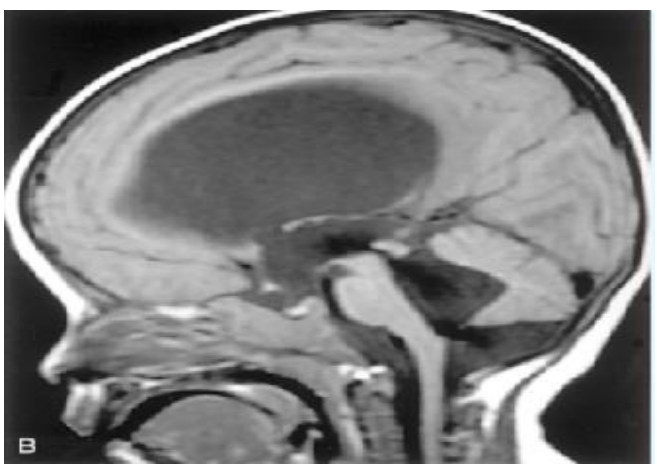
- What is your diagnosis ? Hydrocephalus.
- What are the causes of this disease? 1. Lack of absorption of CSF . 2.Obstruction to flow of CSF
- What are some clinical features of this disease? 1.increased intracranial pressure 2. dilatation of the ventricles.

Gross



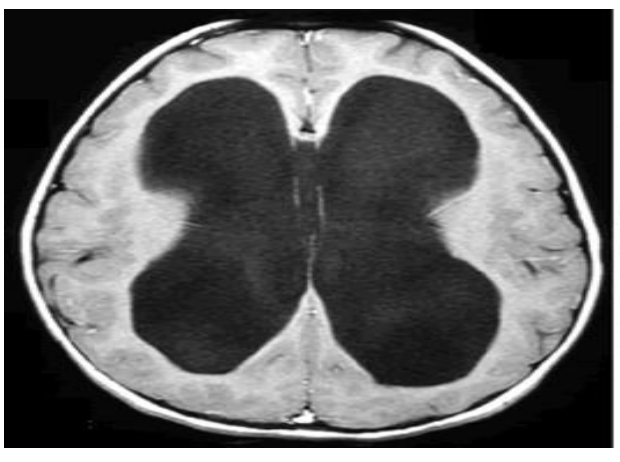
1. Marked dilations of cerebral ventricles

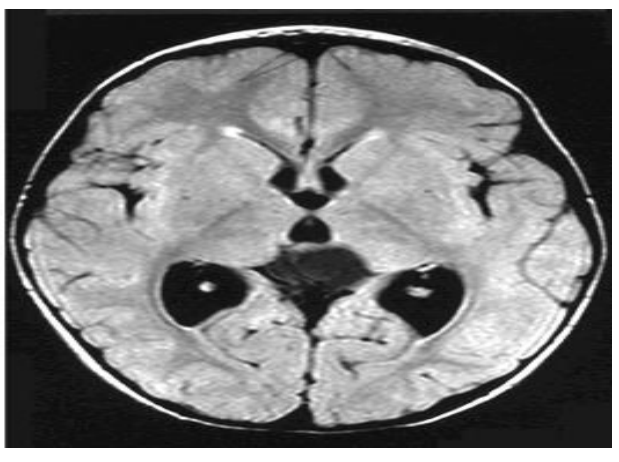
MRI- Mid Sagittal View



1. A chlid with communicating hydrocephalus , involving all ventricles (Dilated ventricles).

MRI scan of the Brain





(Left)
The large dark area is the dilated ventricles , made bigger because of the build-up of CSF .

(Right)
Normal brain

Case 6: Pyogenic (bacterial) Meningitis

4 years old child who was treated from otitis media and suddenly complained from headache, vomiting, fever and stiff neck. CSF was found to be **clouded** with abnormal **increase of neutrophils, increased protein** and **absence of sugar**. Gram stain of the CSF fluid showed meningococci .

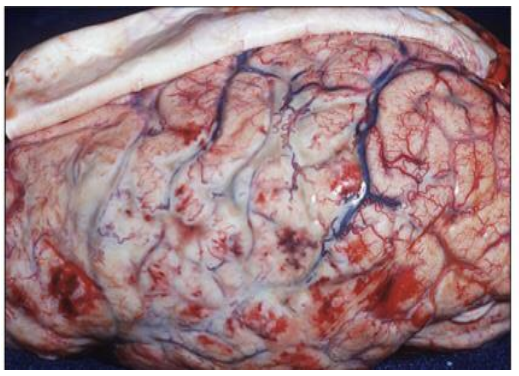
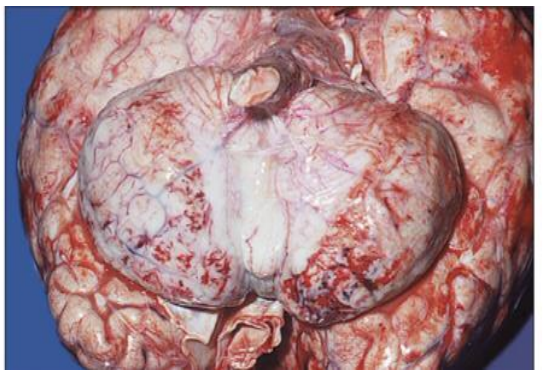
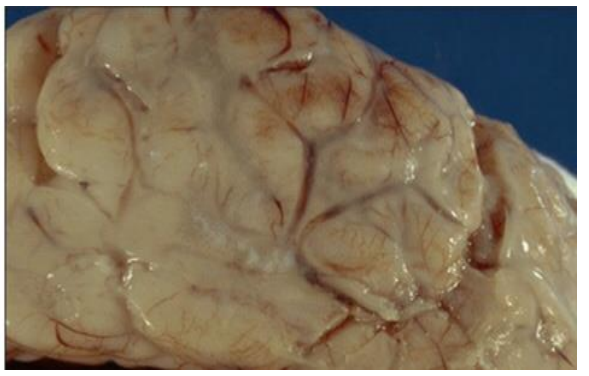
- What is your diagnosis ? Pyogenic (bacterial) Meningitis.
- What are the clinical features of this disease ? 1. headache 2.fever 3.stiff neck 4.photosensitivity.
- **What are the CSF finding ?** 1. increased neutrophils 2. increased protein level 3. decreased glucose

(to help you remember ☺ : bacteria eat glucose and secrete proteins)

- Possible Etiological agents:

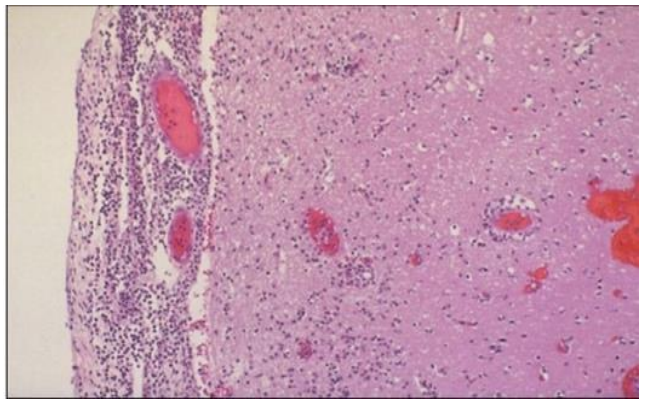
New born = Group B streptococcus , E.coli, Listeria monocytogenes.
Infants and children: Streptococcus Pneumoniae , Neisseria meningitidis , H.Influenzae.
Adults : Streptococcus Pneumoniae , Neisseria meningitidis.

Gross

1. Creamy purulent exudate covering the cerebral hemispheres and settles along the base of the brain, around cranial nerves and the openings of the fourth ventricle.

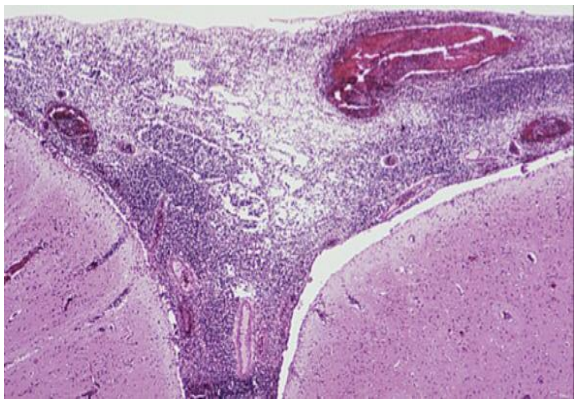
Microscopic (LPF)



1. Intense inflammatory infiltrate going into brain parenchyma.
2. Neutrophilic exudate involving the meninges at **the left.**
3. Prominent dilated vessels.
4. Edema and focal inflammation (extending down via the Virchow-Robin space) in the cortex to the right.

Virchow-Robin space is the space between arteries/veins in the pia matter.

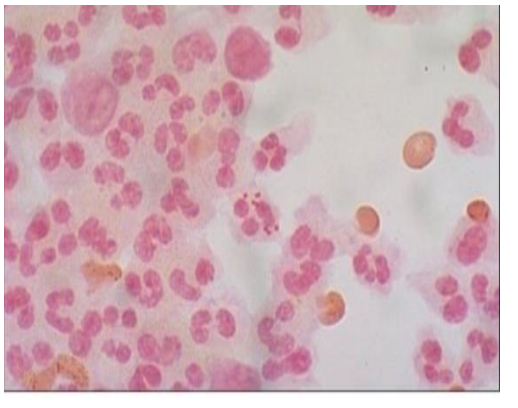
Microscopic (LPF)



Neutrophil infiltration in the subarachnoid space causing:

1.Cranial nerve damage resulting with cranial nerve deficits
2. invasion of leptomeningeal vessels.
3. Arteritis with thrombosis
4. Ischemic infarction
5. Phlebitis= (inflammation of vein)

CSF gram stain



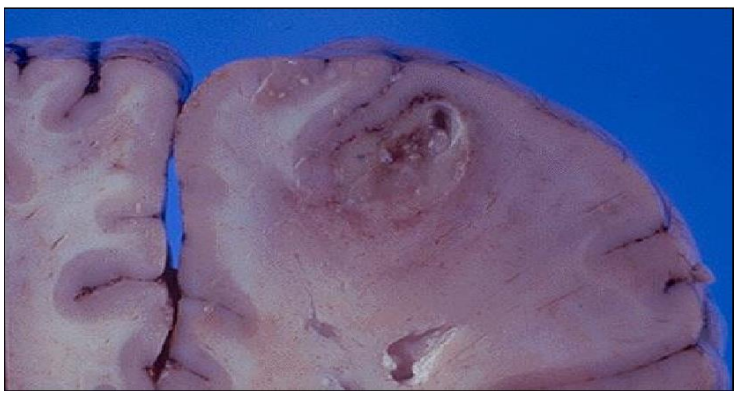
1. Gram negative diplococci within a neutrophil, typical for Neisseria meningitidis.

Case 7: Cerebral Abscess

A 35 years old lady complains from otitis media. Suddenly she suffers from headache and convulsions. Brain MRI reveals 5 cm. fluid filled cavity in the temporal lobe. examination of the CSF shows increased pressure with lymphocytes and **increased protein** but there is **no change of sugar content.**

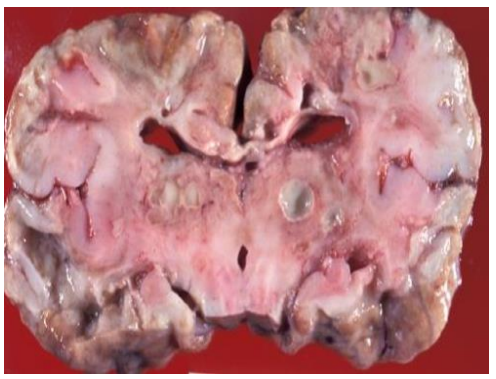
- What is your diagnosis ? Cerebral abscess .
- What are the clinical features ? 1.Progressive focal neurologic defect 2. headache 3 .increased ICP 4. vomiting 5. confusion 6. convulsion 7.coma.
- Etiology agents: in non-immunosuppressed patients: 1. streptococci 2. staphylcocci.
- Abscesses result from: 1- haematogenous spread of bacterial infection 2. direct penetrating trauma . 3. extension from adjacent infection in sinuses.

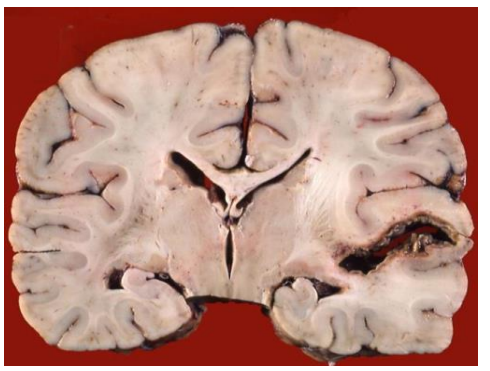
Gross



1. **liquefactive center** with yellow pus surrounded by a thin wall.

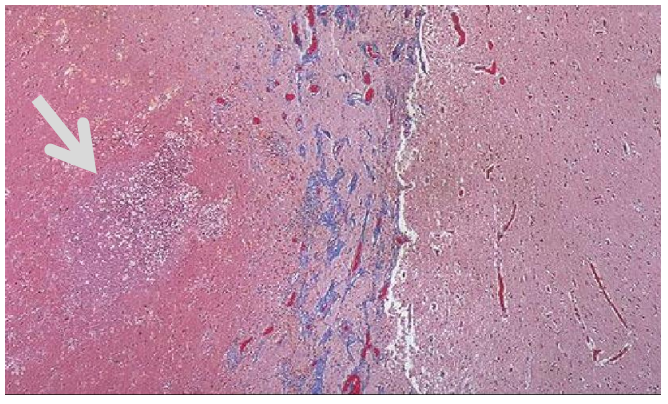
Gross





1. Chronic Abscess .

Microscopic



1. center of the abscess at the **left.**
2. Normal brain at the **right.**
3. trichrome stain demonstrates the light blue connective tissue in the wall of an organizing cerebral abscess.

CT scan

This CT scan of the head in transverse view demonstrates:

1. an abscess in the brain (red arrow) in a patient who had septicemia

MRI

This MRI scan of the head in transverse (axial) view demonstrates:

1. a small abscess in the brain (Red arrow) in a patient who had septicemia.

Case 8: Ruptured Berry Aneurysm causing subarachnoid hemorrhage

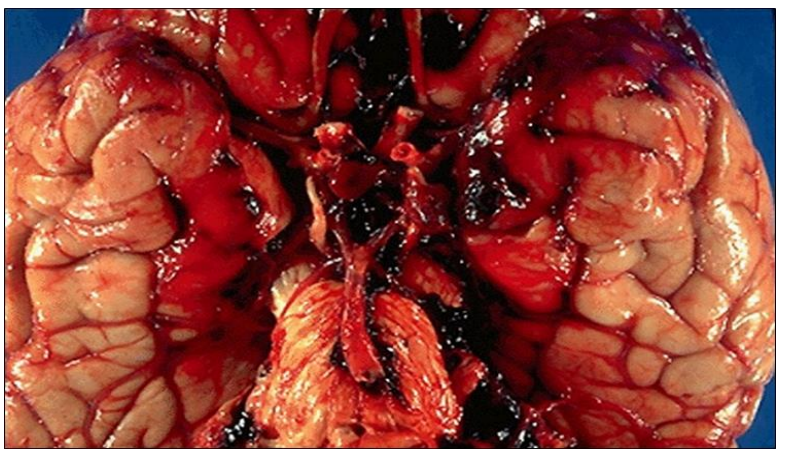
A previously healthy 31-year-old woman experiences a **severe headache** and **loses consciousness within an hour**. An emergent head CT scan reveals extensive **subarachnoid haemorrhage** at the base of the brain. She is a febrile. A lumbar puncture yields cerebrospinal fluid with many red blood cells, but no white blood cells. The CSF protein is slightly increased, but the glucose is normal.

- What is your provisional diagnosis ? Ruptured Berry Aneurysm causing subarachnoid haemorrhage.
- What is the most common site for berry aneurysm? 1. Anterior communicating artery 2. junction of internal communicating artery and posterior communicating artery.

Gross - Circle of Willis with berry aneurysms

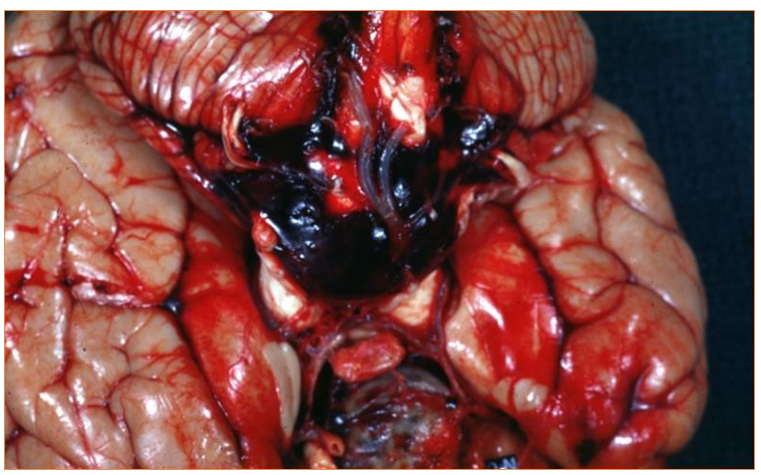
1. three berry aneurysms are seen.
2. Multiple aneurysms are seen in about 20-30% of cases of berry aneurysm.

Gross - Circle of Willis with berry aneurysms



1. The subarachnoid hemorrhage from a ruptured aneurysm is more of an irritant producing vasospasm than a mass lesion.

Gross - Circle of Willis with berry aneurysms



1. base of brain showing subarachnoid hemorrhage over anterior surface of pons.
2. a large aneurysm at top of photo which is located in the right internal carotid artery

Microscopic- LPF (low- power- field)

1. Aneurysmal dilatation with lack of medial structures in wall of aneurysm
2.This section is from basilar artery and adjacent portion of posterior inferior cerebellar artery.

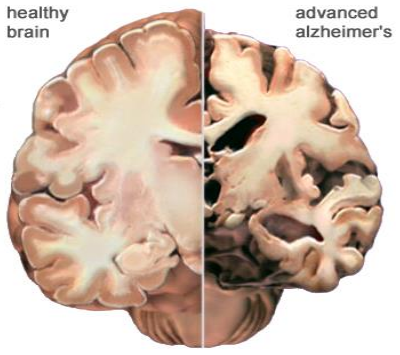
stain : H&E.

Case 9: Alzheimer's Disease

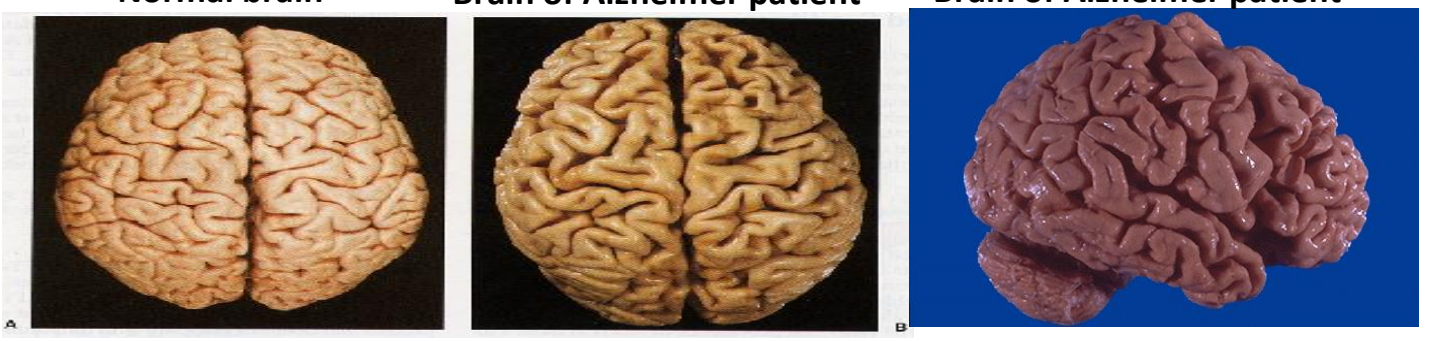
A 85 years old man complains of progressive **loss of memory**, **disorientation** and **alterations in mood** and behavior since 20 years. He was admitted to hospital because he was disabled and immobile and he died in hospital after one week of admission. Autopsy was done and the **brain cortex was found to be atrophied.**

- What is your diagnosis? Alzheimer's disease.
- The plaque can only be seen with silver stain.

Gross



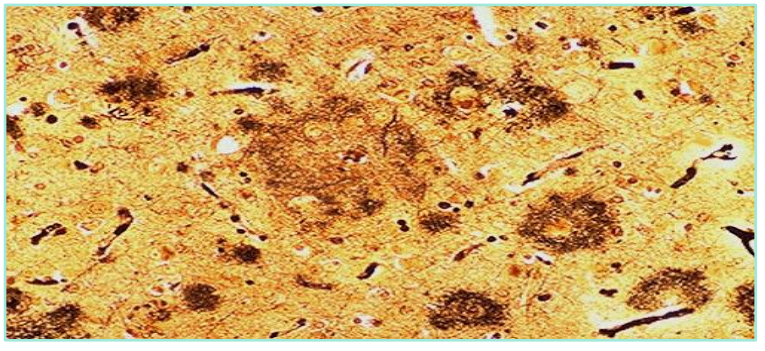
Normal brain | **Brain of Alzheimer patient** | **Brain of Alzheimer patient**



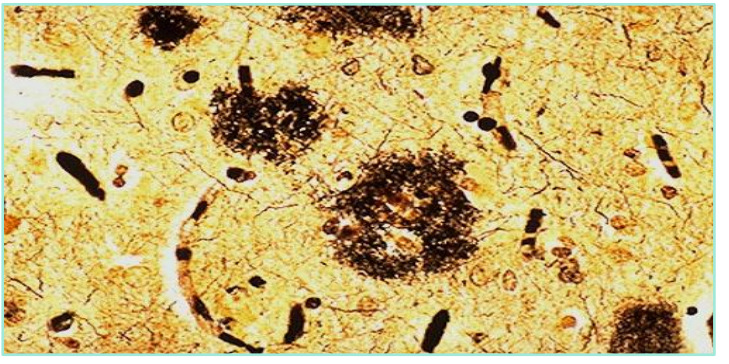
Brain of Alzheimer patient shows:

1. Cortical atrophy with **thin gyri** and **prominent sulci** in frontal and parietal lobes.
2. The atrophy is due to senile dementia of the Alzheimer's type.

Microscopic- Neuritic plaques LPF(low power field)

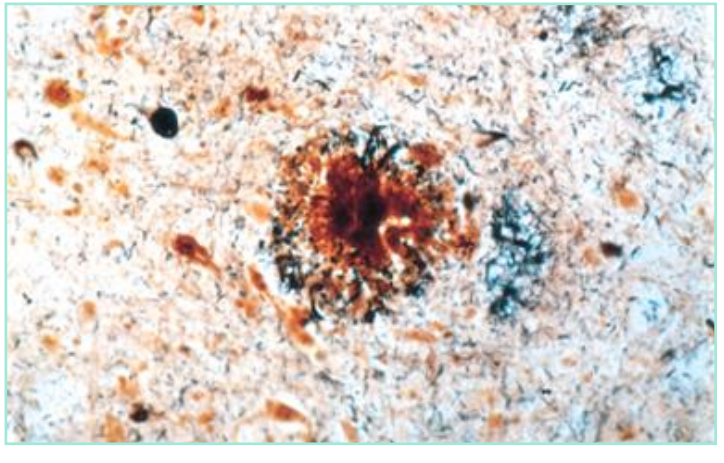


1. Many plaques of varying size.
2. "senile plaques" which are collections of degenerative presynaptic endings along with astrocytes and microglia



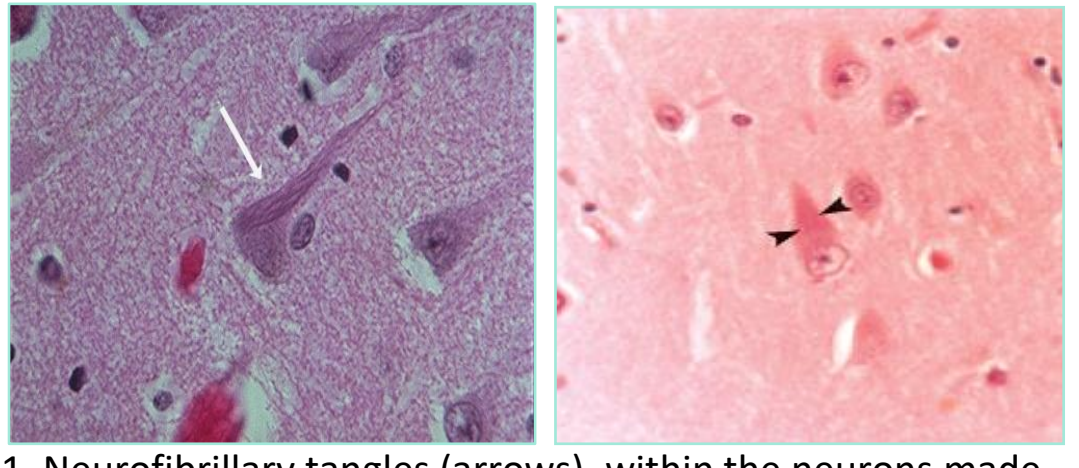
1. neuritic (senile) plaques are numerous in the cerebral cortex and hippocampus.
2. This dementia is marked mainly by progressive memory loss.

Microscopic-Neuritic plaques - LPF



1. A neuritic (senile) plaque with a rim of dystrophic neurites surrounding an amyloid core.

Microscopic- Neurofibrillary tangles – HPF (high- power-field)



1. Neurofibrillary tangles (arrows) within the neurons made up of: cytoskeletal intermediate filaments.

Thank you for checking our work & GOOD LUCK !

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