

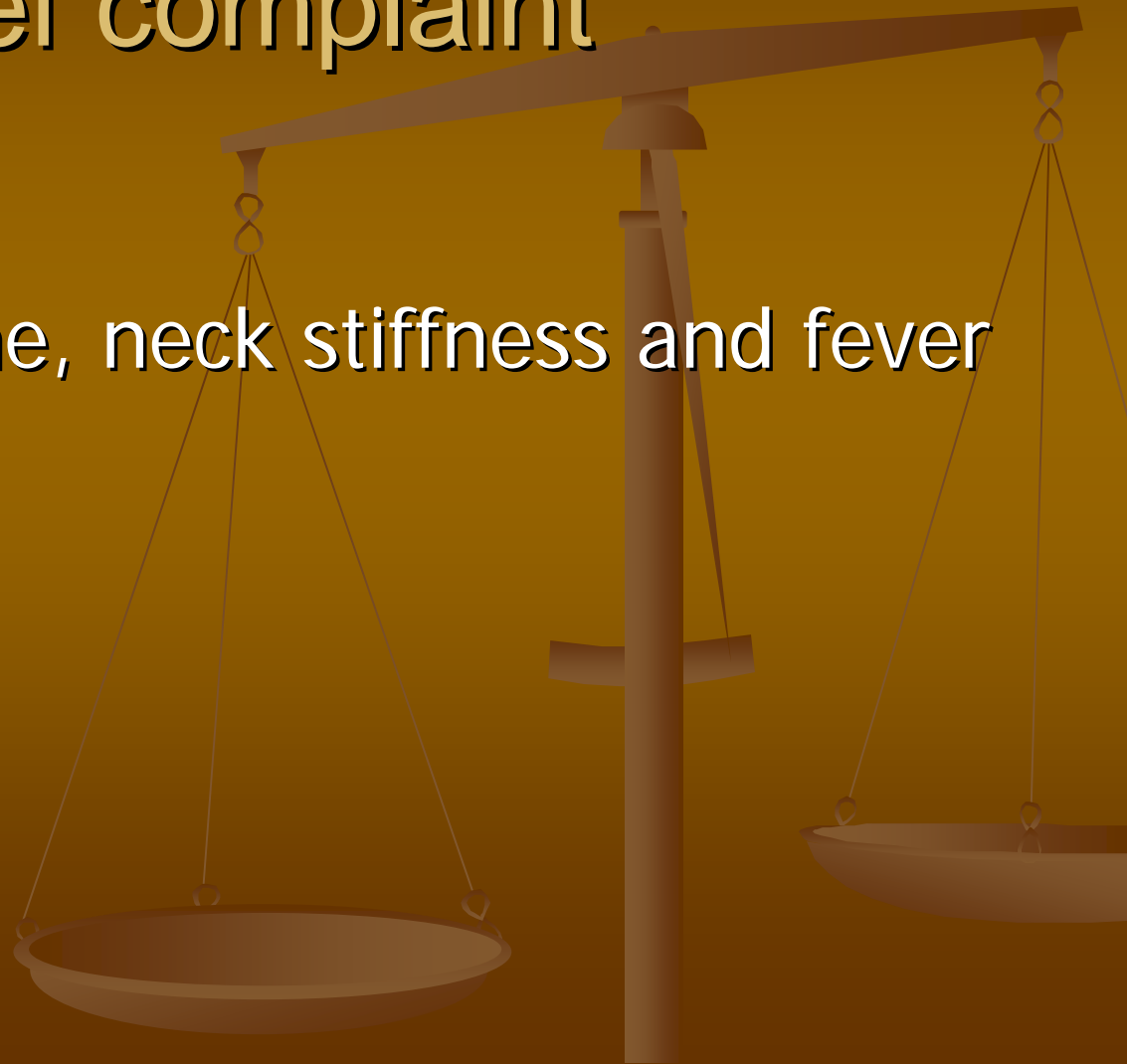
General Data

- Gender: female
- Age: 47
- Occupation: 環保局



Chief complaint

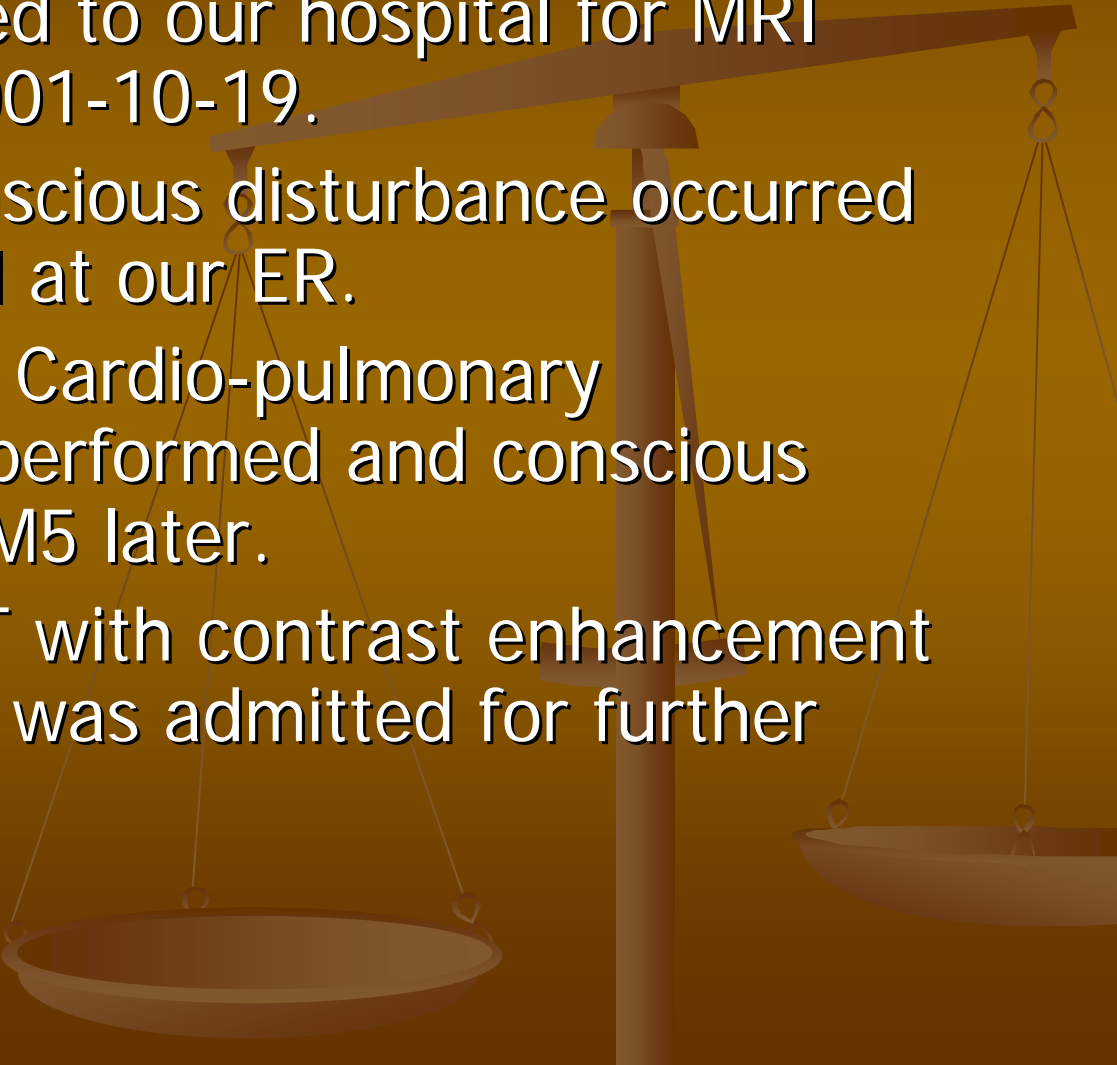
- Severe headache, neck stiffness and fever for 10 days.



Present illness (I)

- This 47-year-old female patient was a case of asthma for years. She suffered from progressive headache with neck stiffness for 2 weeks. Fever and conscious disturbance were also noted. So she was admitted to 忠孝 H. on 2001-10-18.
- At 忠孝 H, GCS was E3V5M6, fever up to 38.5°C and leukocytosis were noted.
- Brain CT showed a low density lesion at right frontal lobe with mild hydrocephalus. Right frontal tumor, infarction and brain abscess were suspected.

Present illness (II)

- She was transferred to our hospital for MRI examination on 2001-10-19.
 - Unfortunately, conscious disturbance occurred right before arrival at our ER.
 - GCS was E1V1M1. Cardio-pulmonary resuscitation was performed and conscious recovered to E3VtM5 later.
 - Emergent brain CT with contrast enhancement was done and she was admitted for further management.
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Past history

■ Medical history

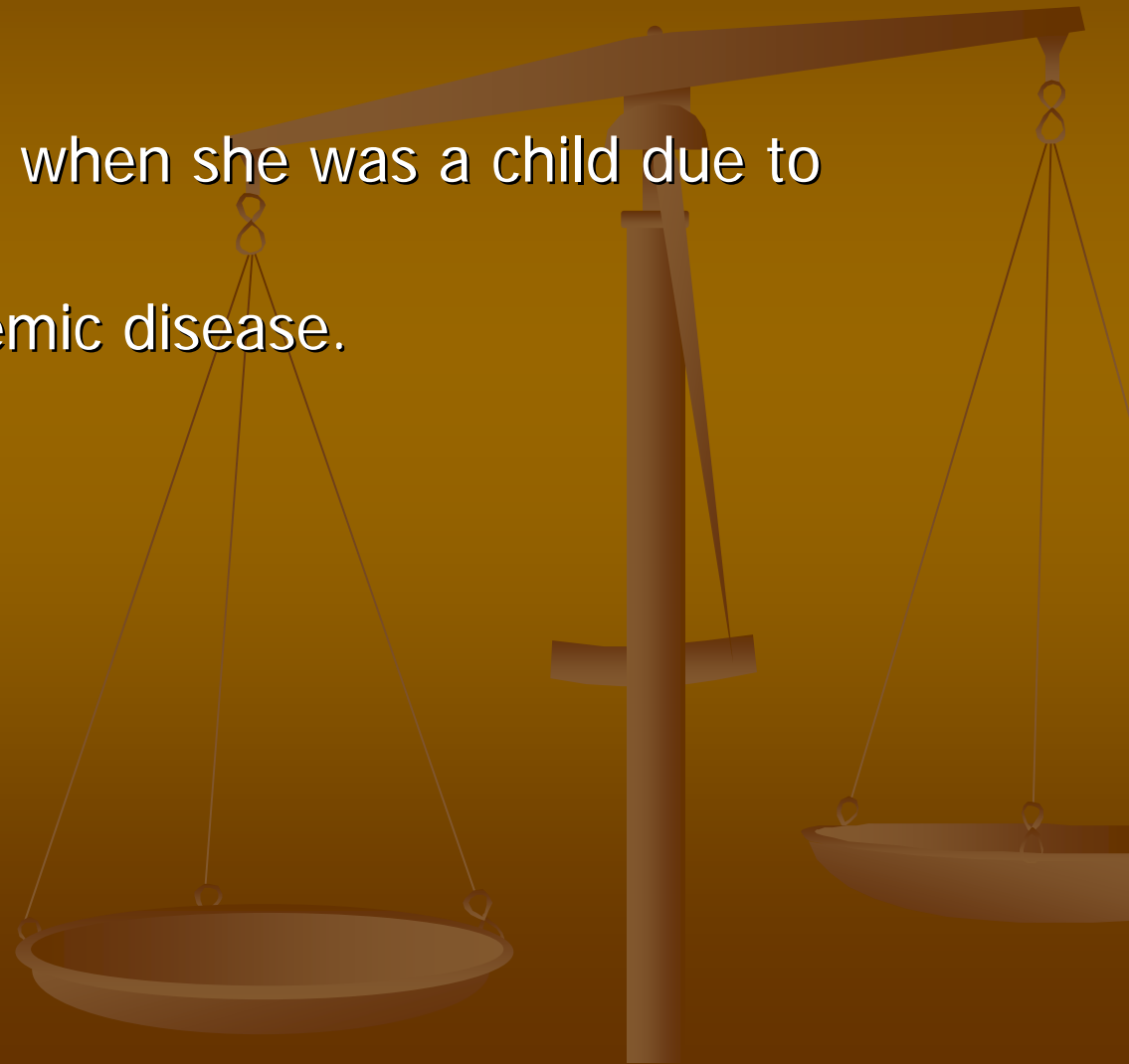
- Asthma for years.
- Left lung resection when she was a child due to trauma.
- Denied other systemic disease.

■ Personal history

- Smoking: denied
- Alcohol: denied
- Allergy: penicillin

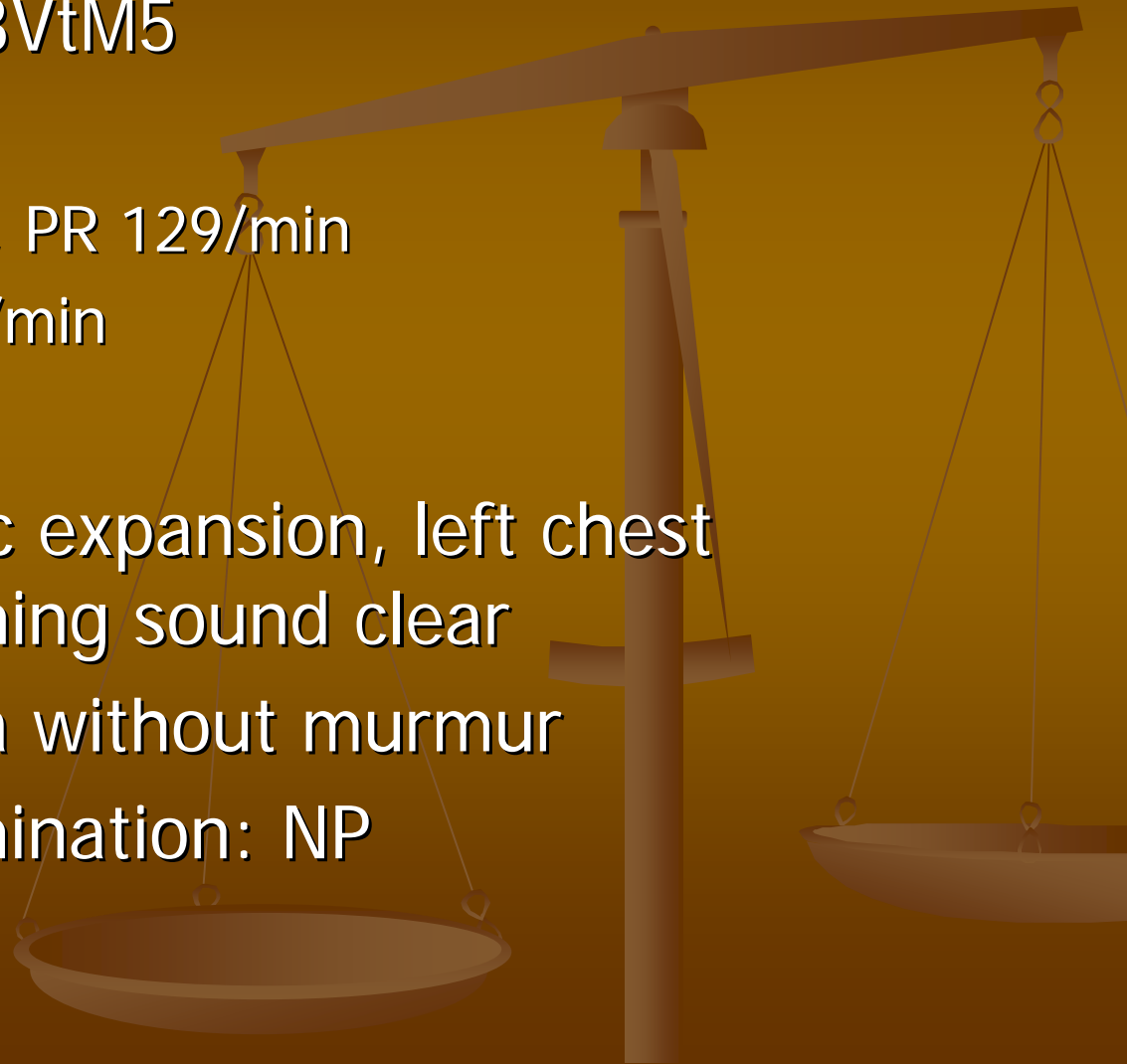
■ Family history

- Not contributory



Physical examination

- Consciousness: E3VtM5
- Vital sign
 - BP 159/94 mmHg, PR 129/min
 - BT 37.6°C, RR 20/min
- Neck: stiffness
- Chest: asymmetric expansion, left chest immobilize, breathing sound clear
- Heart: tachycardia without murmur
- Neurological examination: NP

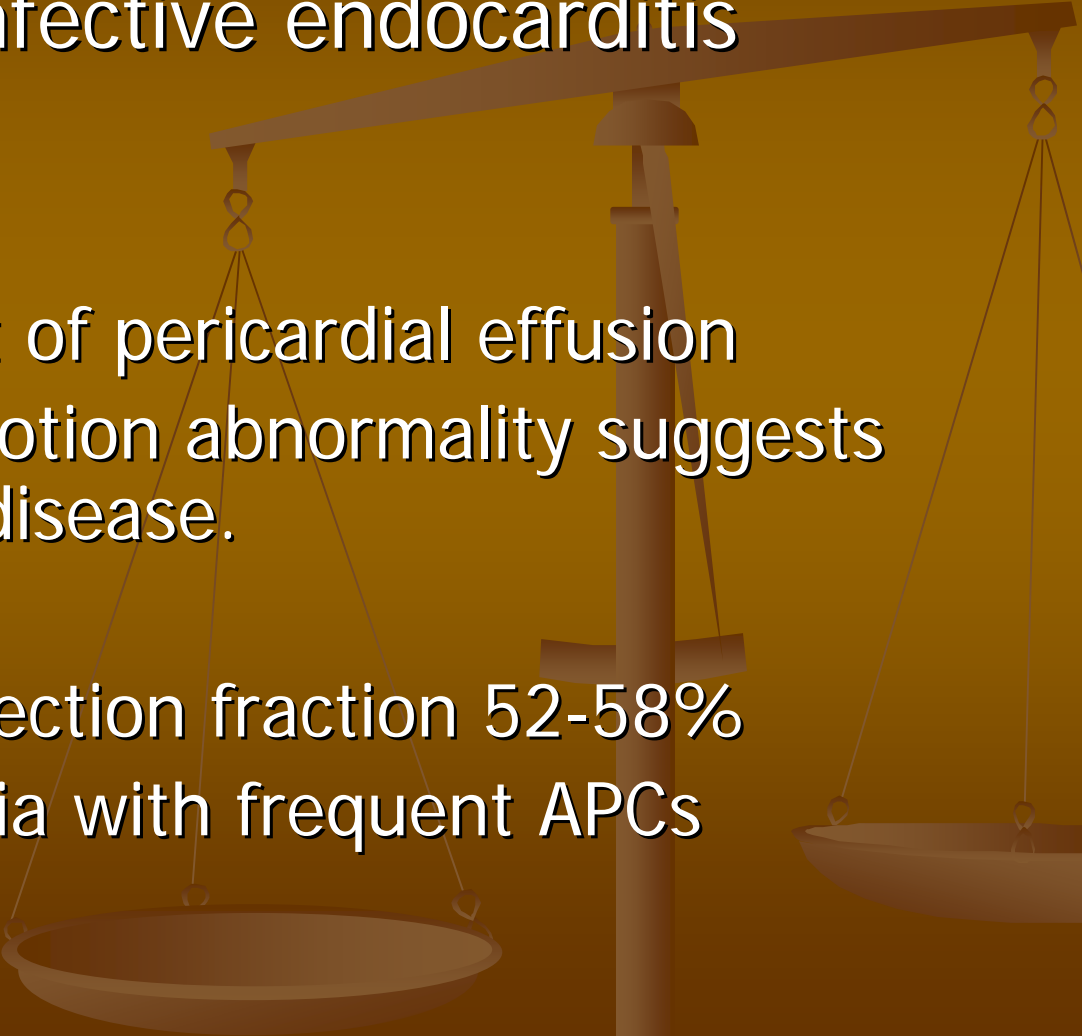


Lab. data

- WBC: 21460/uL
- Neutrophil: 90.1%
- Glucose 191 mg/dL
- K 3.3 mEq/L
- liver function: WNL
- Renal function: WNL



Cardiac sonography

- For suspected infective endocarditis
 - 2001-10-20
 - Mild LA dilation
 - Minimal amount of pericardial effusion
 - Regional wall motion abnormality suggests ischemic heart disease.
 - Mild MR
 - Estimated LV ejection fraction 52-58%
 - Sinus tachycardia with frequent APCs
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X-ray

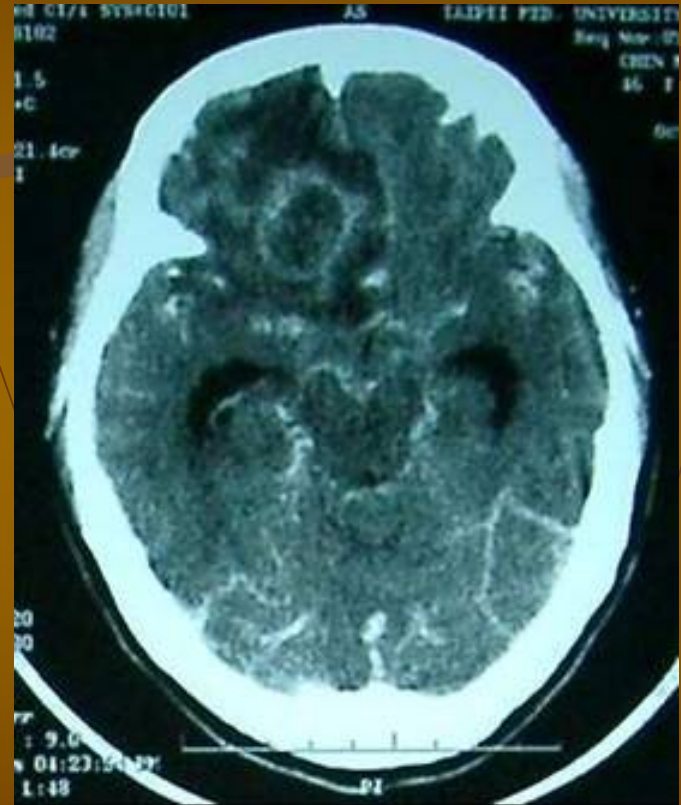
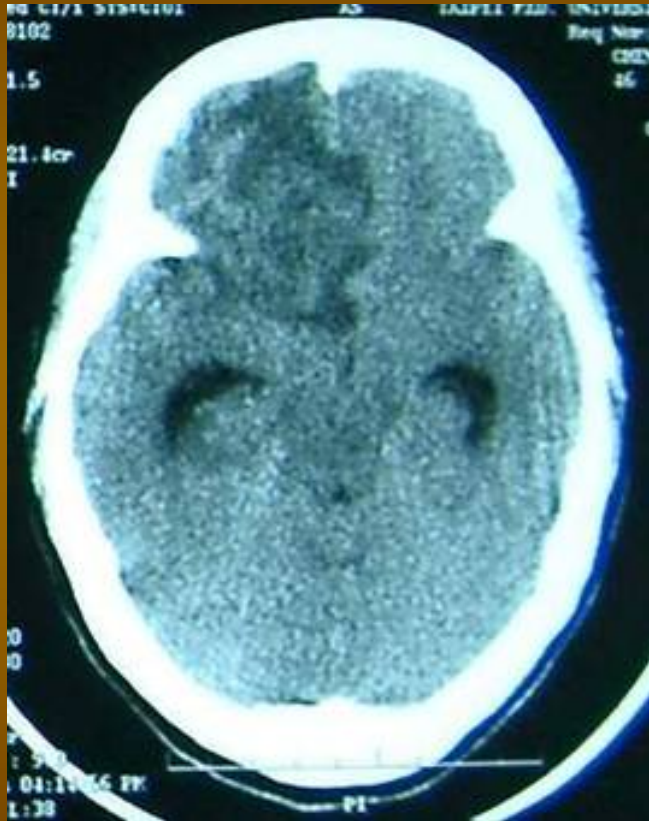


- 2001-10-20
- Total opacification of the left chest without mediastinal shifting.
- Amputation of left main bronchus.

Brain CT (1)

pre-enhanced

post-enhanced

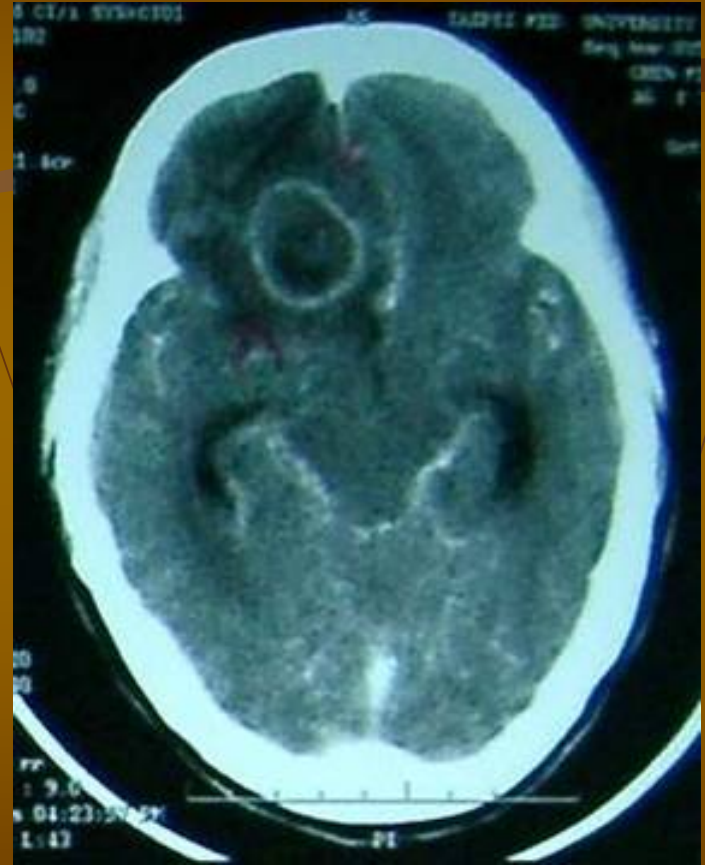
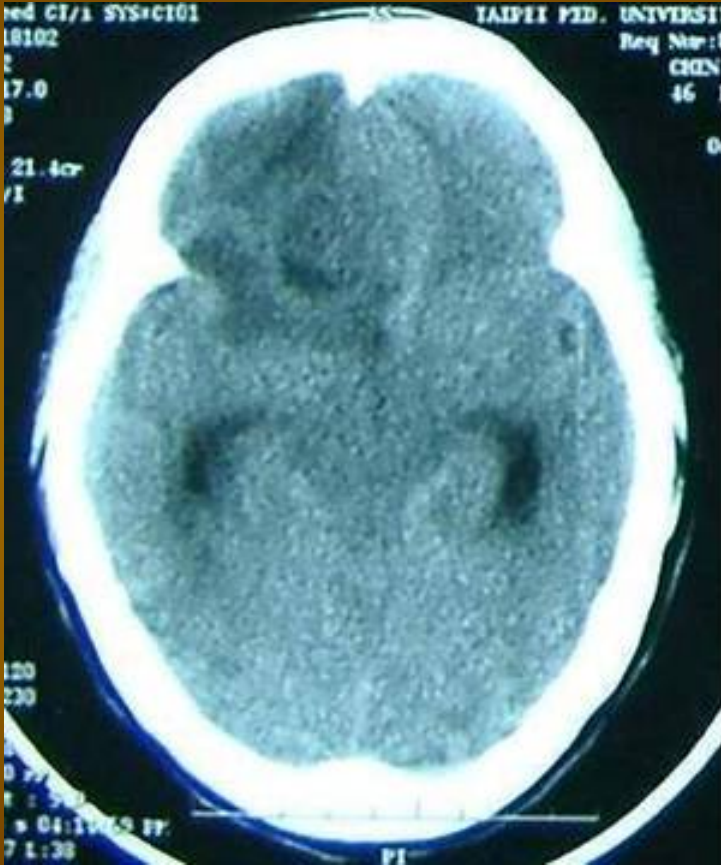


A hypodensity lesion over R't frontal lobe

Brain CT (2)

pre-enhanced

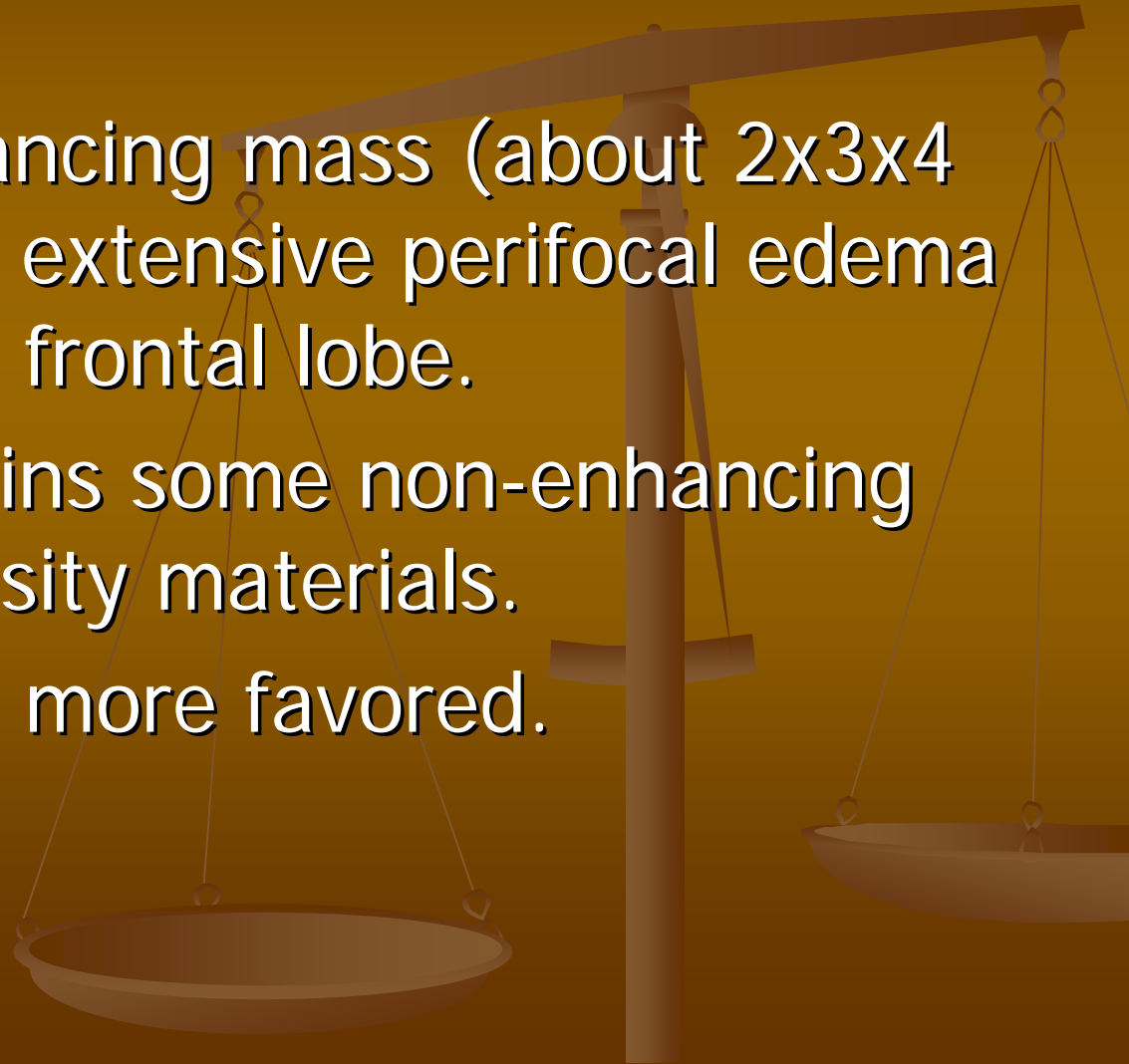
post-enhanced



A thin ring-enhancing mass with extensive perifocal edema

Brain CT findings

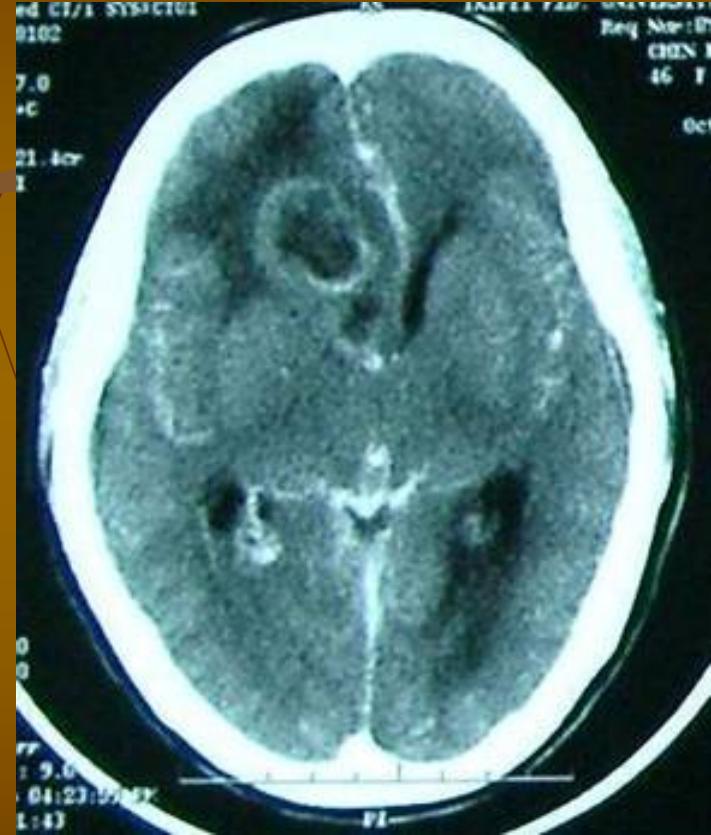
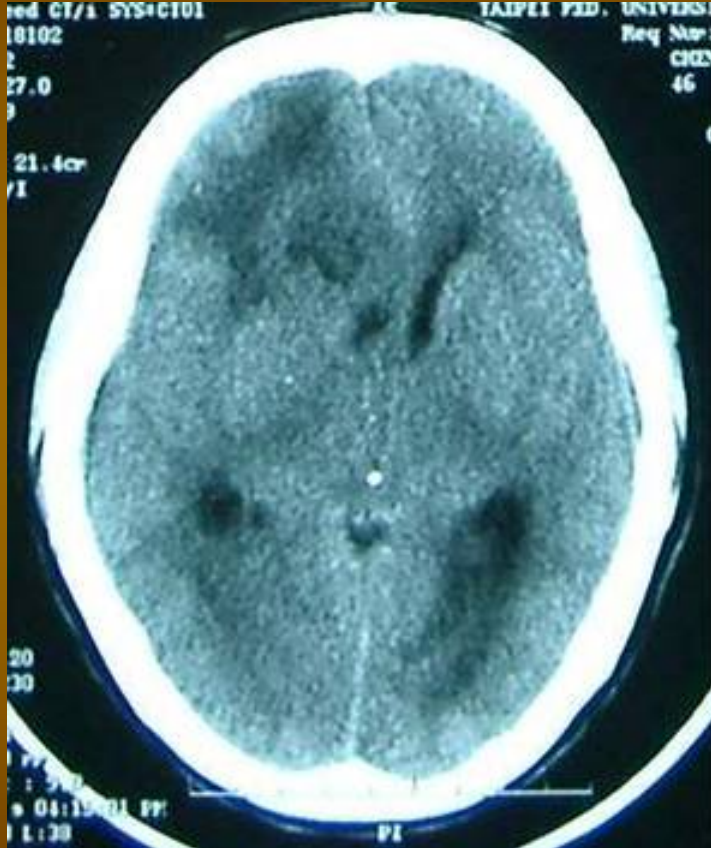
- 2001-10-19
- A thin ring-enhancing mass (about 2x3x4 cm in size) with extensive perifocal edema is noted in right frontal lobe.
- The mass contains some non-enhancing low and iso-density materials.
- Brain abscess is more favored.



Brain CT (3)

pre-enhanced

post-enhanced



- Diffuse brain edema with midline shift
- Subfalcine herniation

Brain CT (4)

pre-enhanced

post-enhanced



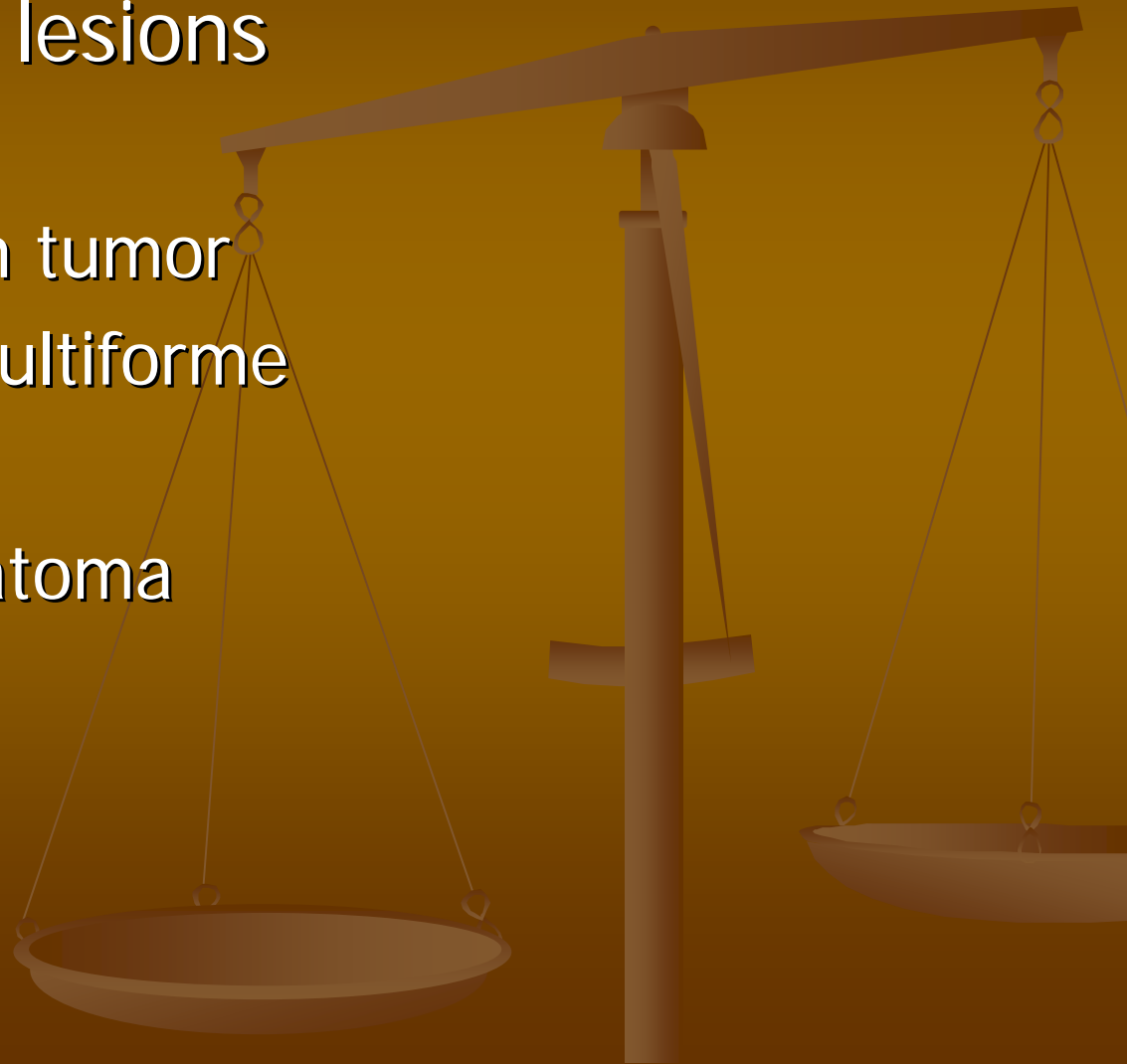
- There is ventricular wall enhancement in frontal horn of right lateral ventricle.
- Asymmetrical lateral ventricular dilatation.

Brain CT findings

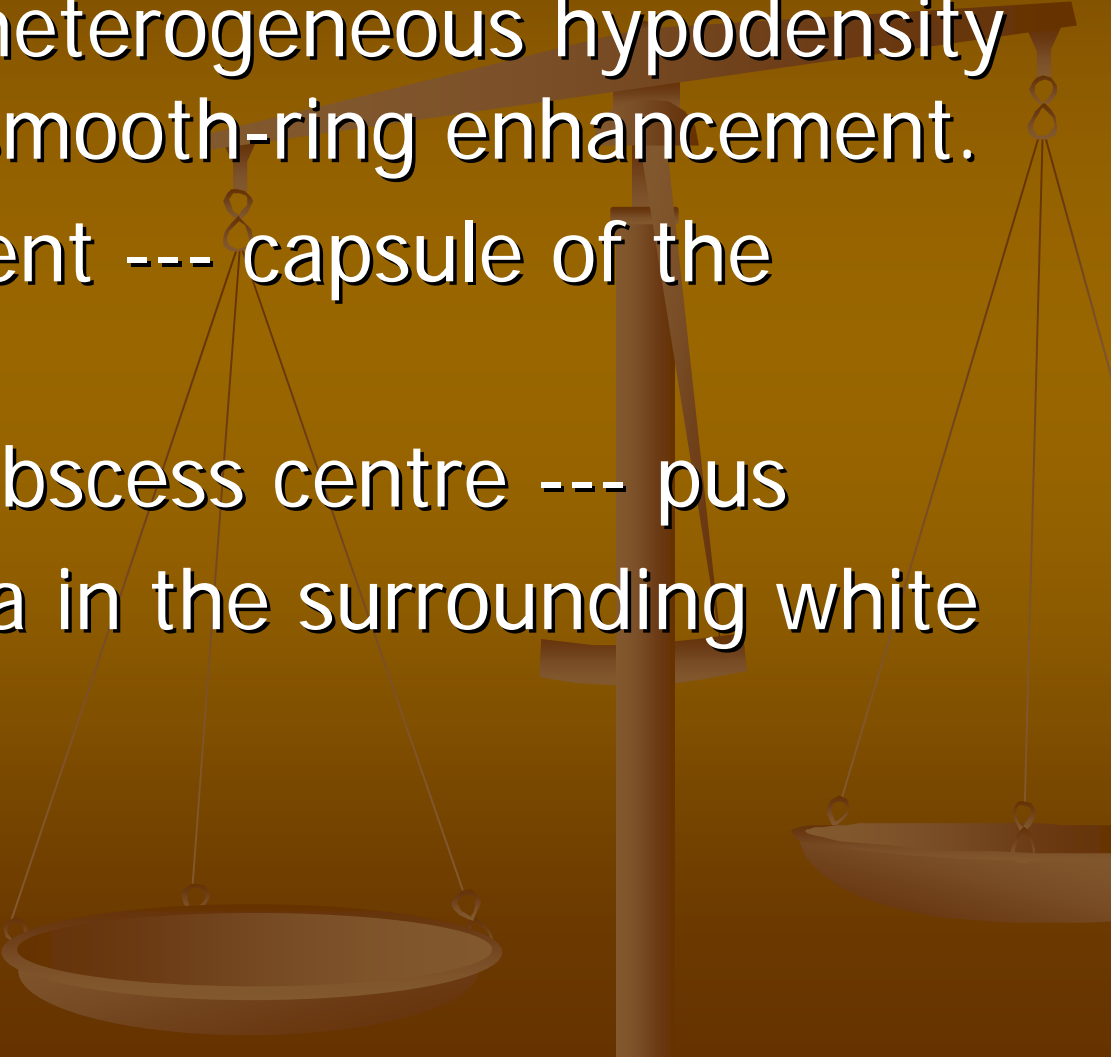
- There is ventricular wall enhancement in frontal horn of right lateral ventricle.
- Abscess rupture into ventricle, resulting in ventriculitis are considered.
- Asymmetrical lateral ventricular dilatation is noted, obstructive hydrocephalus due to compression by the abscess is suspected.
- Diffuse brain edema with midline shift and subfalcine herniation are also seen.

Differential diagnosis

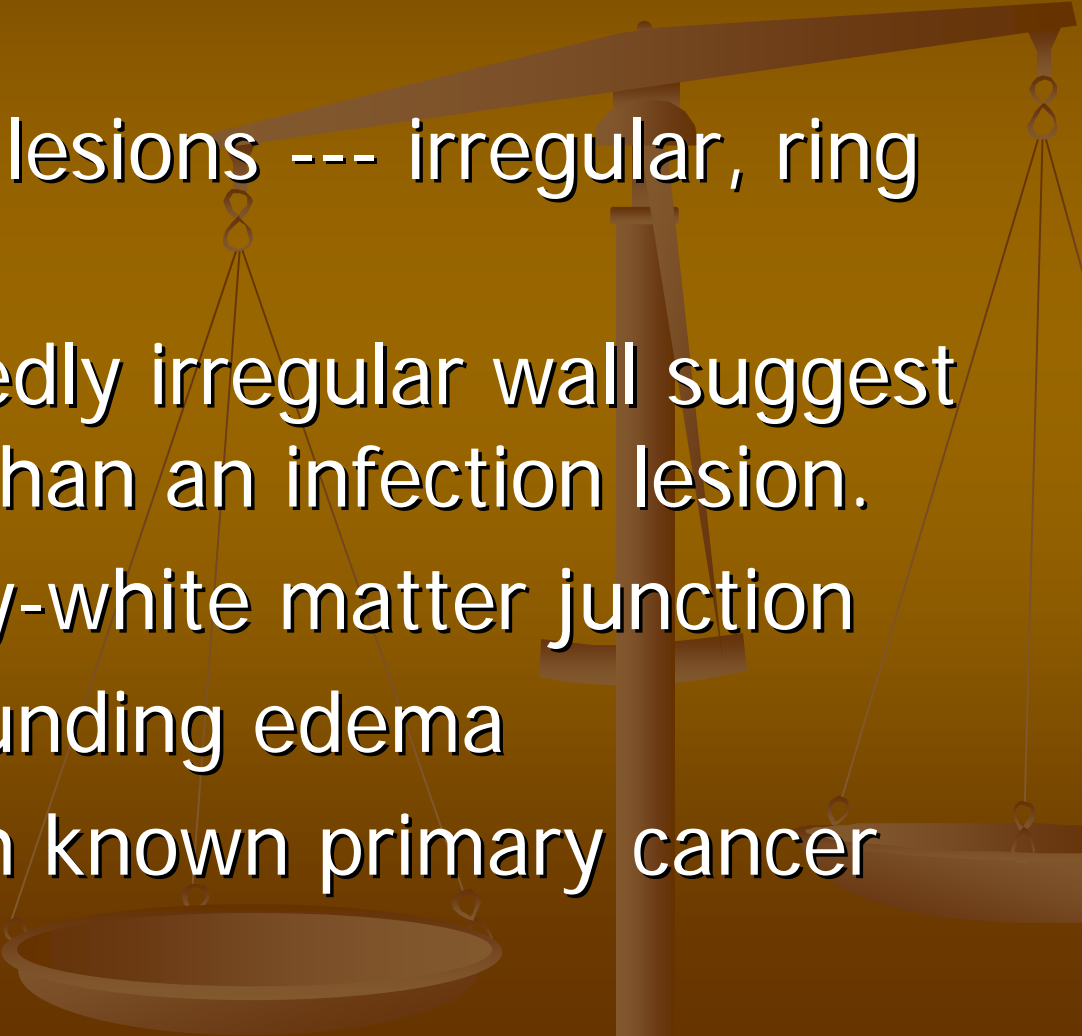
- Ring-enhancing lesions
 - Brain abscess
 - Metastatic brain tumor
 - Glioblastoma multiforme
 - Cerebral infarct
 - Resolving hematoma



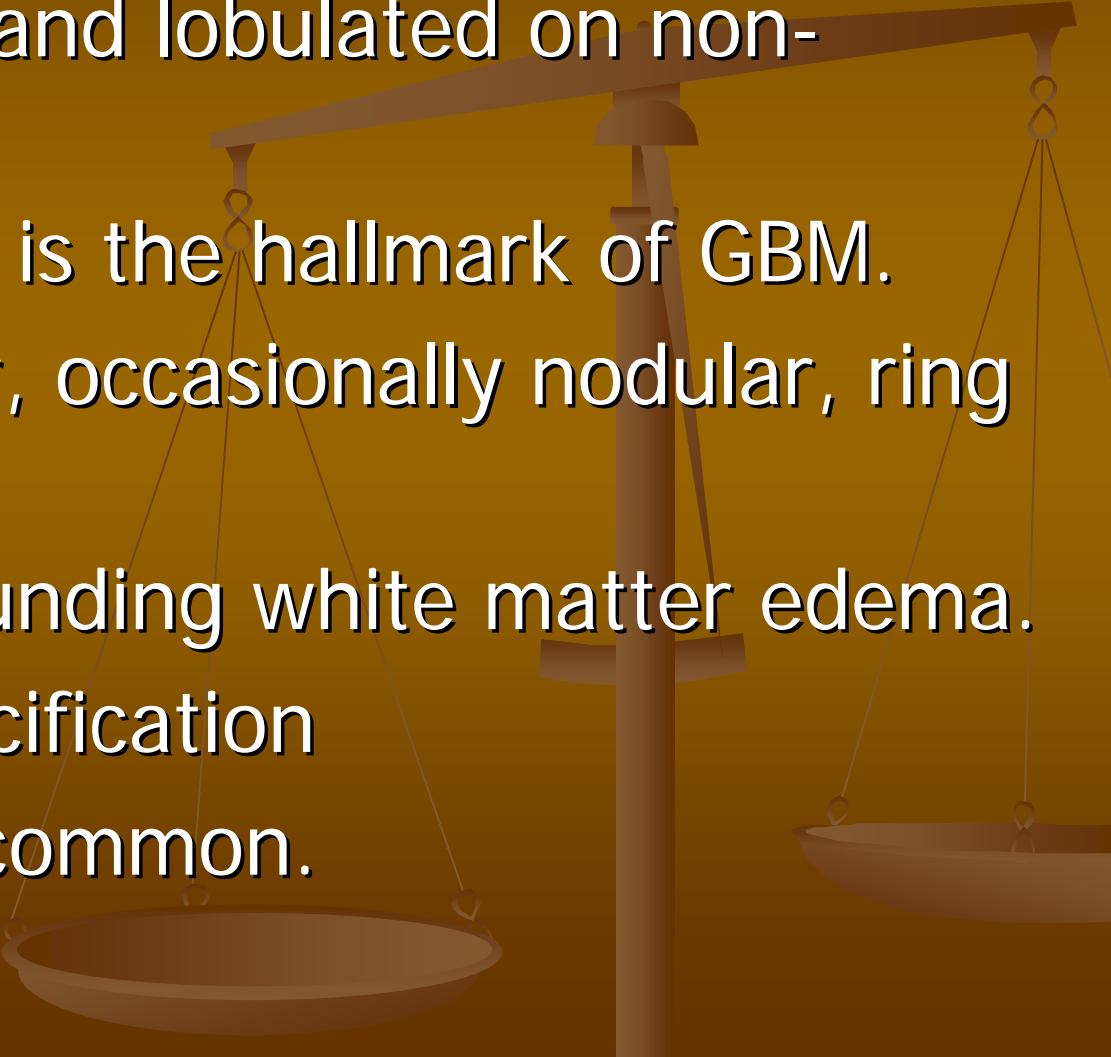
Brain abscess

- A focal area of heterogeneous hypodensity surrounded by smooth-ring enhancement.
 - Ring enhancement --- capsule of the abscess
 - Nonenhancing abscess centre --- pus
 - Extensive edema in the surrounding white matter.
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Metastatic brain tumor

- Multiple
 - Enhancing solid lesions --- irregular, ring pattern
 - A thick or markedly irregular wall suggest a tumor rather than an infection lesion.
 - Seen at the gray-white matter junction
 - Extensive surrounding edema
 - In a patient with known primary cancer
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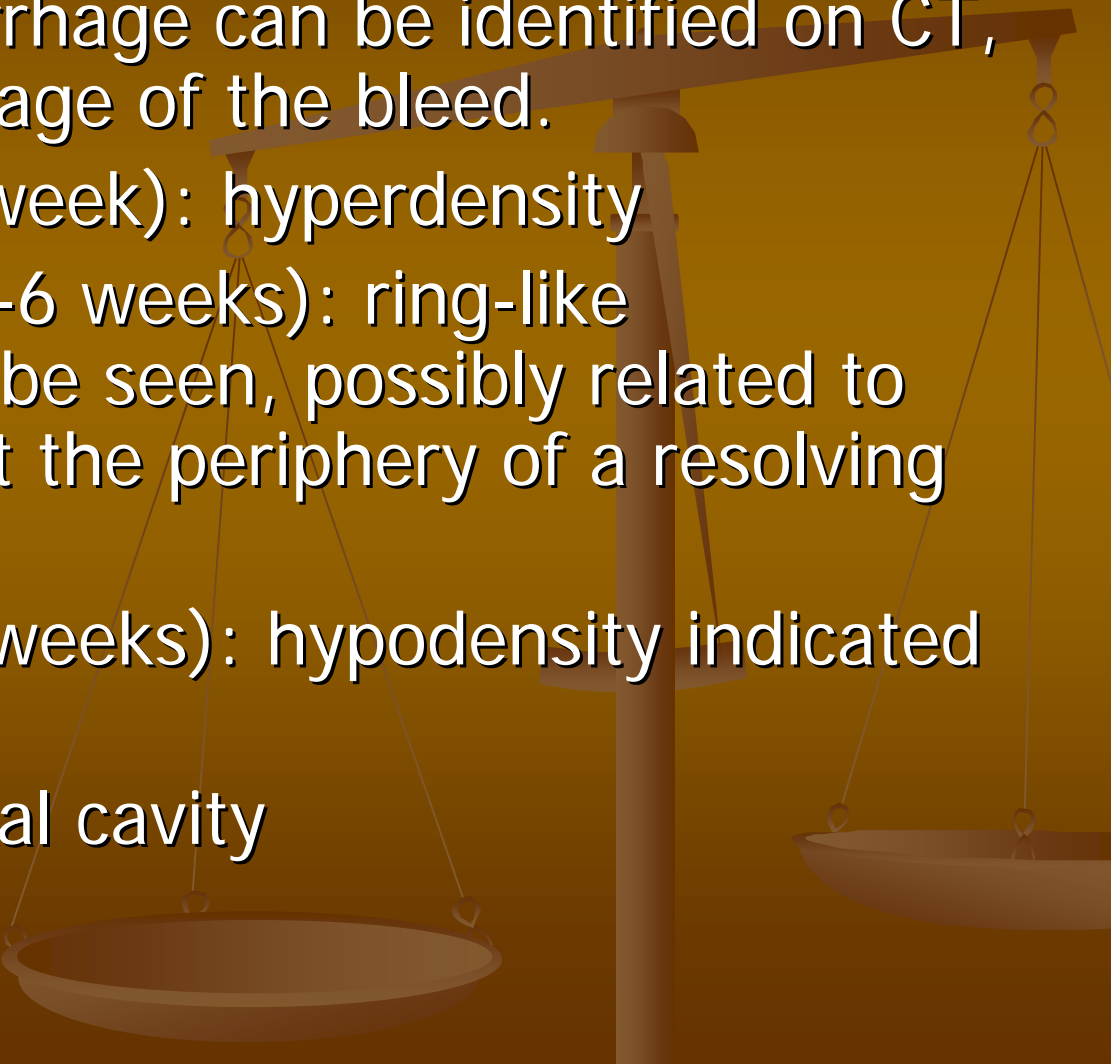
Glioblastoma multiforme

- Heterogeneous and lobulated on non-contrast CT.
 - Central necrosis is the hallmark of GBM.
 - Usually irregular, occasionally nodular, ring enhancement.
 - Extensive surrounding white matter edema.
 - Occasionally calcification
 - Hemorrhage is common.
- 

Cerebral infarct

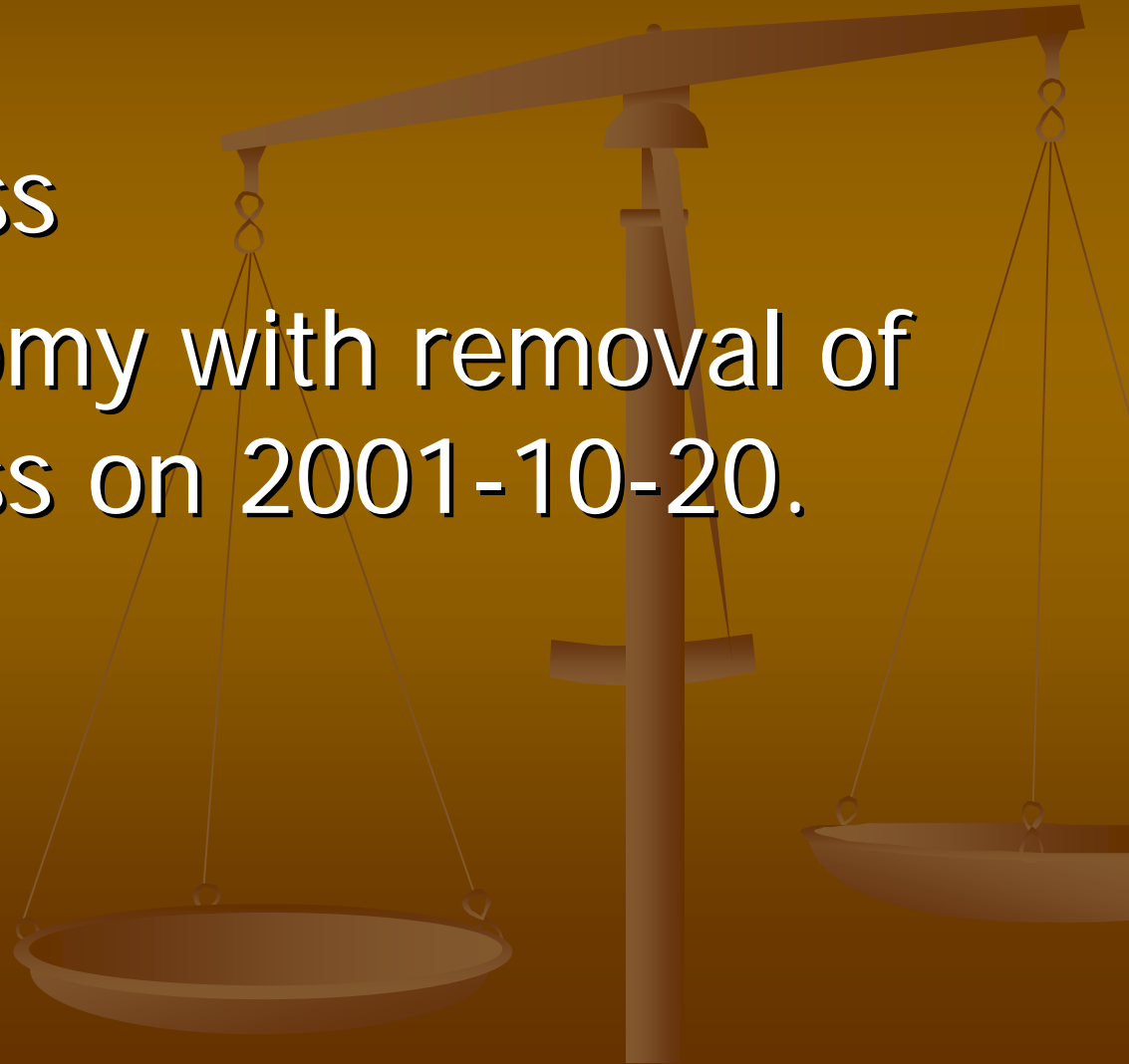
- CT showed hypodensity involving both gray and white matter.
- Cortical involvement is an important differential diagnostic criteria.
- On contrast-enhancement CT
 - Variable pattern: cortical, central, patchy and dense enhancement.

Resolving hematoma

- Intracranial hemorrhage can be identified on CT, depending on the age of the bleed.
 - Acute stage (< 1 week): hyperdensity
 - Subacute stage (1-6 weeks): ring-like enhancement can be seen, possibly related to hypervascularity at the periphery of a resolving hematoma
 - Chronic stage (>6 weeks): hypodensity indicated cavity formation.
 - Final stage: residual cavity
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Final diagnosis

- Brain abscess
- OP: craniotomy with removal of brain abscess on 2001-10-20.



Discussion

Brain abscess

■ Definition

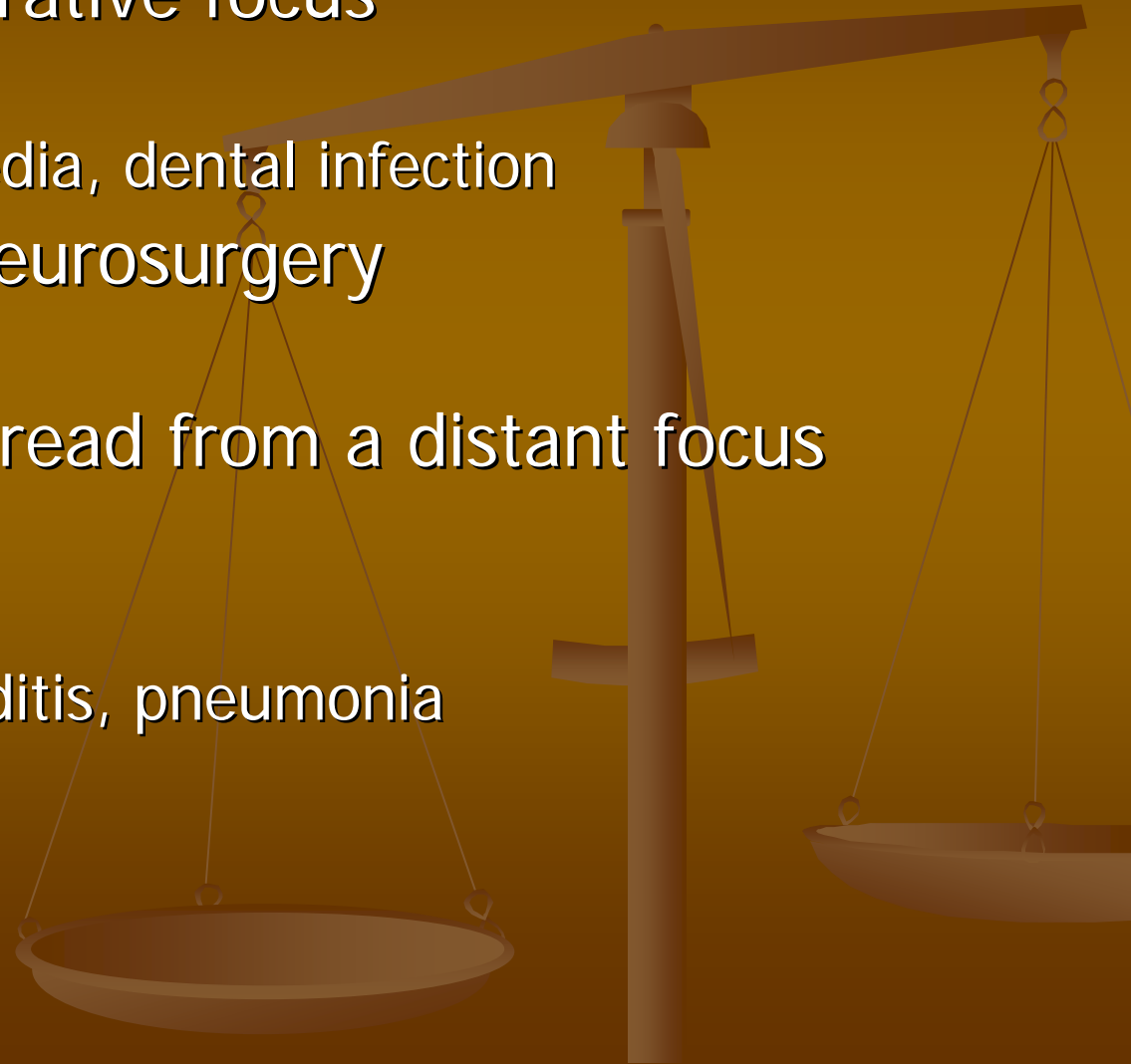
- A focal suppurative infection involving brain parenchyma with subsequent abscess formation .

■ Common etiologic agents

- Mixed flora
- Staphylococcus aureus
- Aerobic or microaerophilic Streptococci
- Aerobic gram-negative bacilli
- Other anaerobes

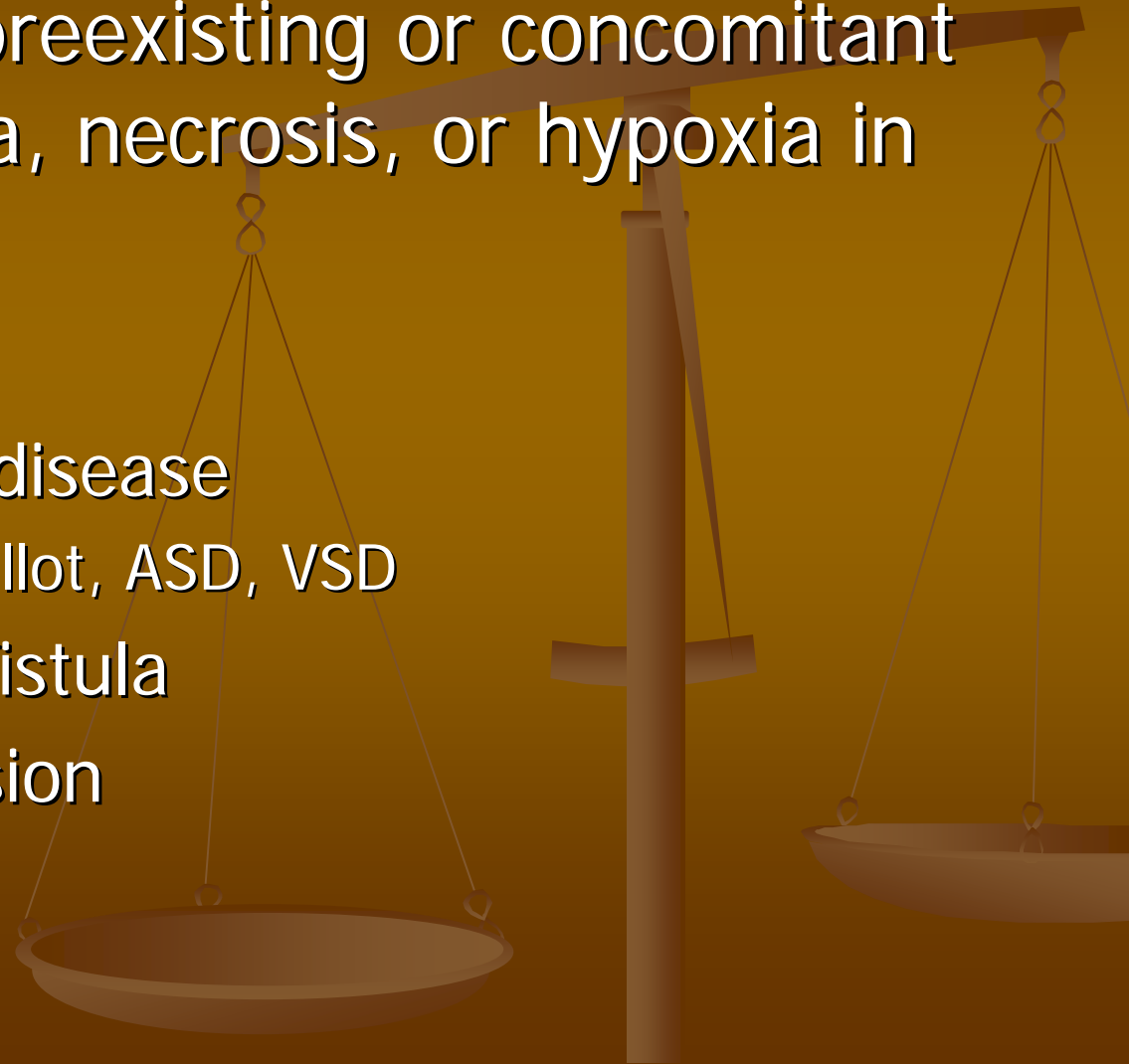
Etiology

- Contiguous suppurative focus
 - 45-50% of cases
 - Sinusitis, otitis media, dental infection
- Head trauma or neurosurgery
 - 10% of cases
- Hematogenous spread from a distant focus
 - 25% of cases
 - Multiple lesions
 - Infective endocarditis, pneumonia
- Cryptogenic
 - 15% of cases



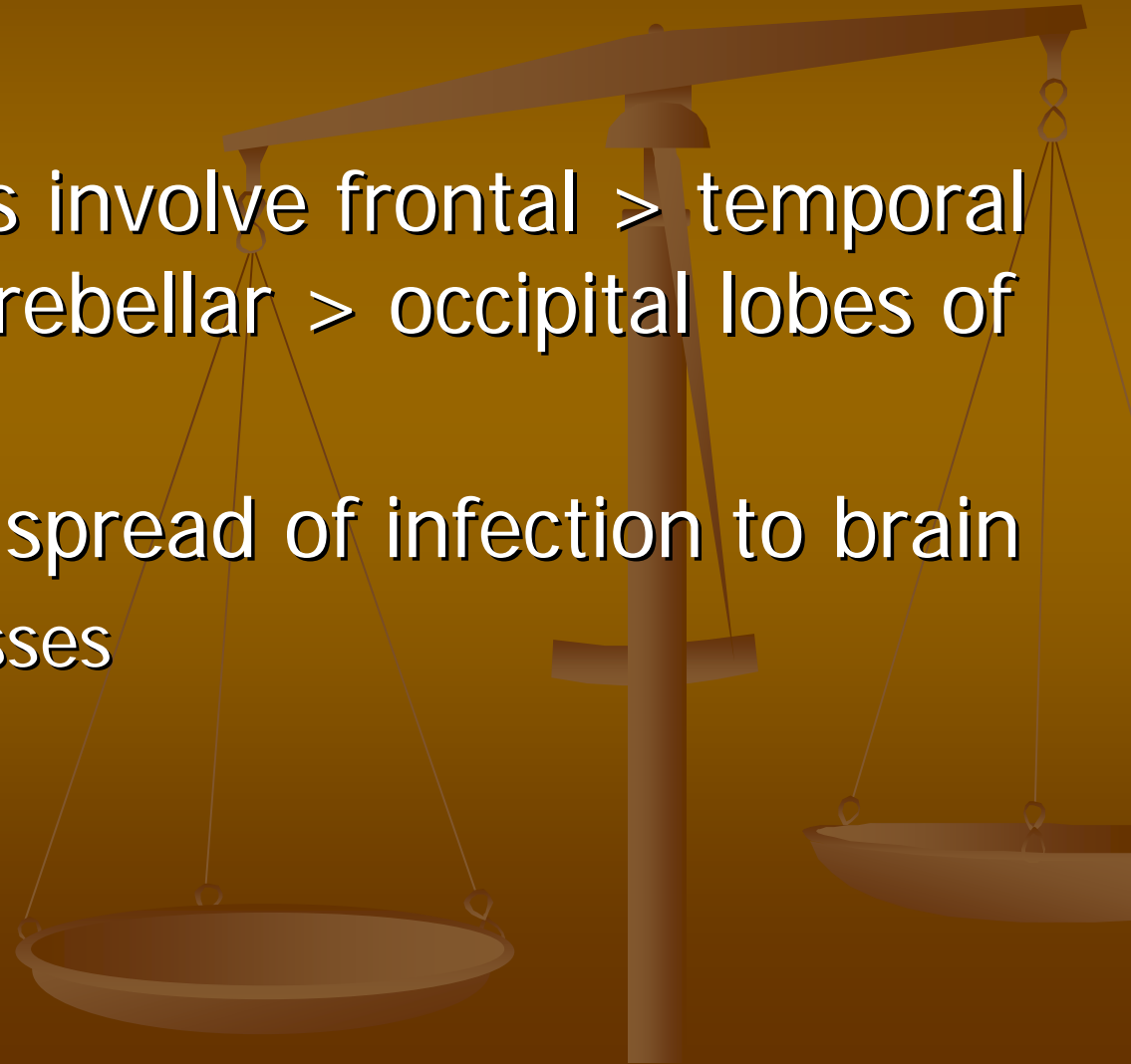
Results of experimental models of brain abscess formation

- there must be preexisting or concomitant area of ischemia, necrosis, or hypoxia in brain tissue.
- Risk factor
 - Cyanotic heart disease
 - Tetralogy of Fallot, ASD, VSD
 - Pulmonary AV fistula
 - immunosuppression



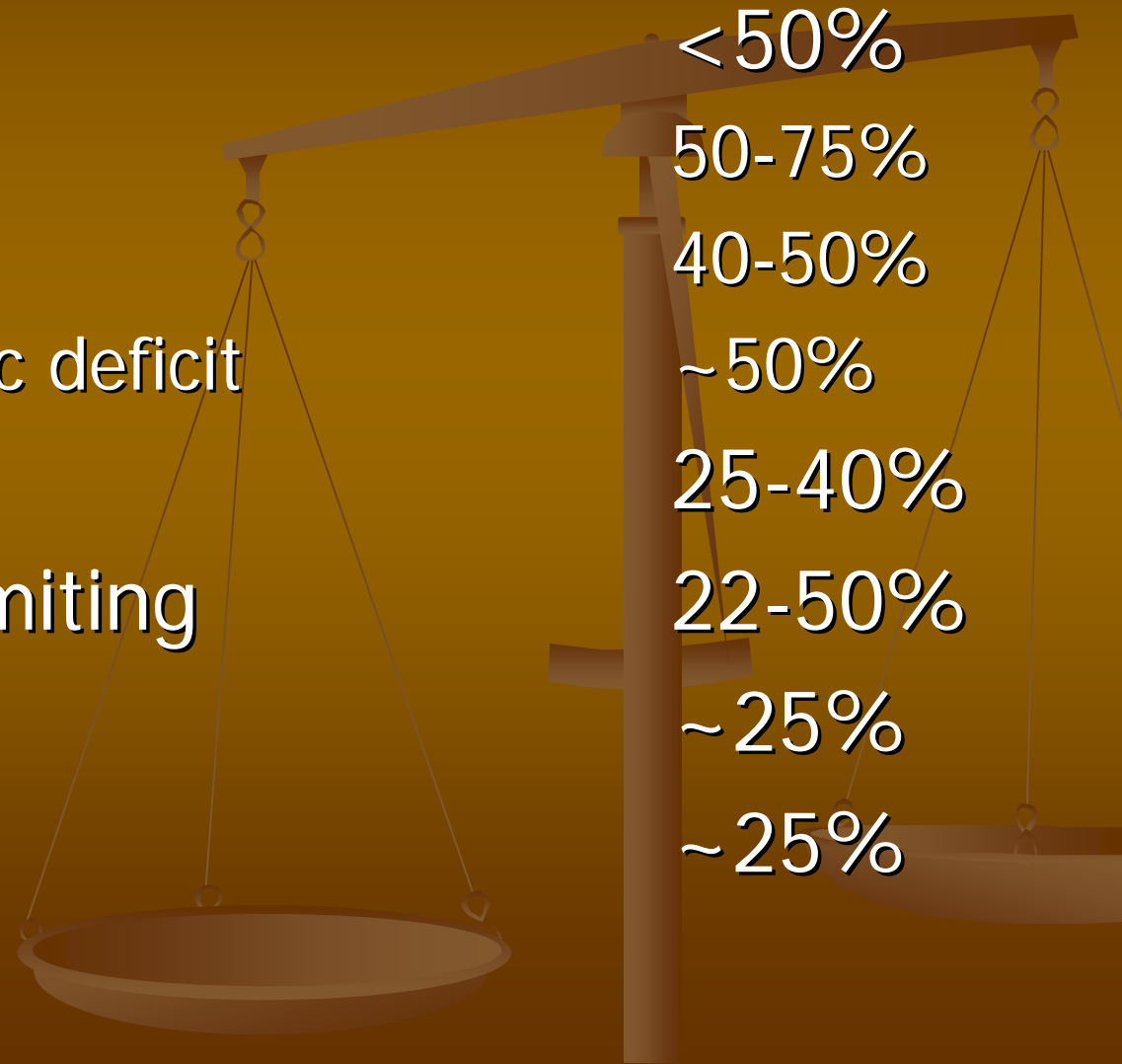
Clinical features

- Solitary abscess involve frontal > temporal > parietal > cerebellar > occipital lobes of the brain.
- hematogenous spread of infection to brain
 - Multiple abscesses



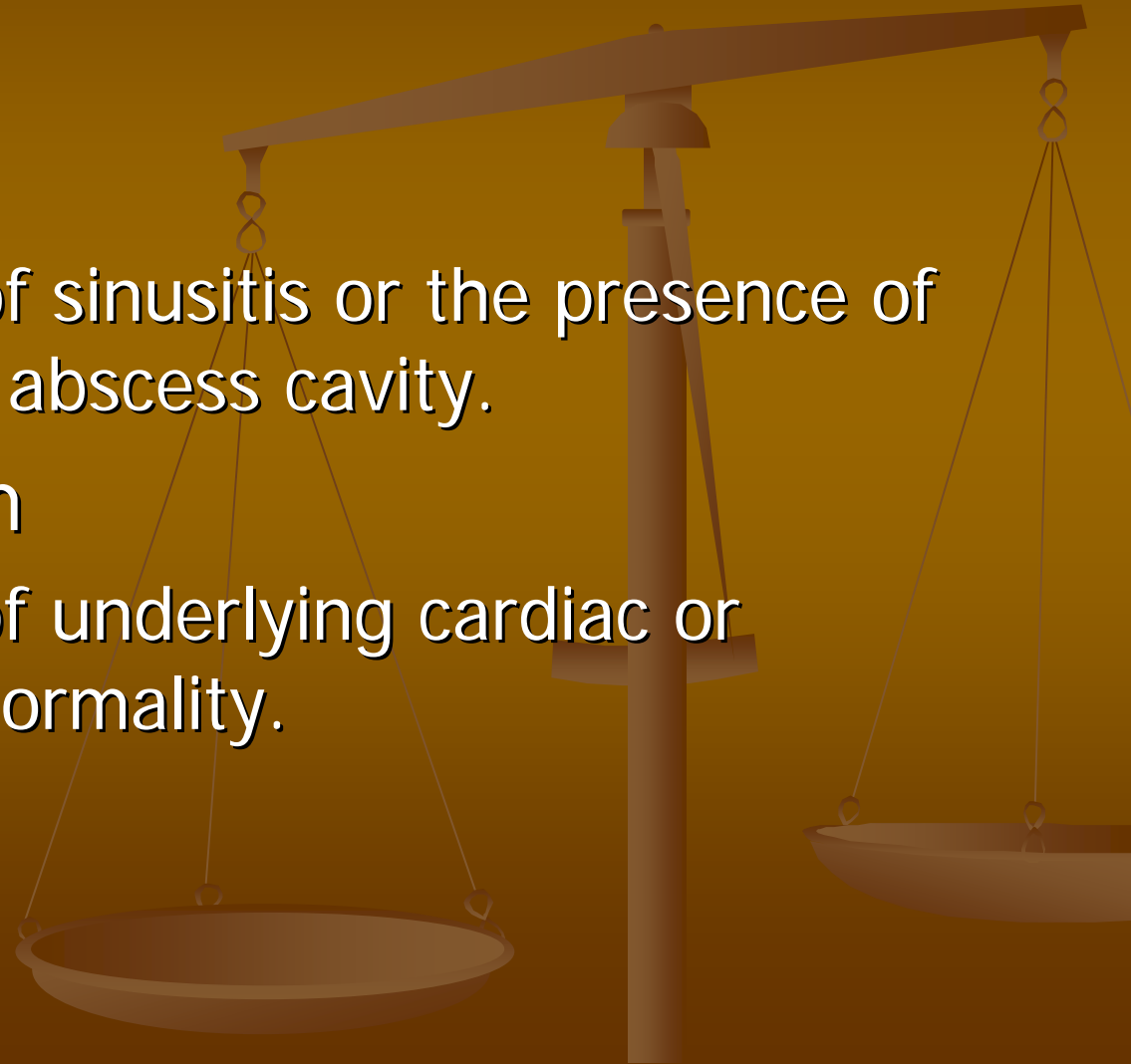
Symptom and sign

- Triad
 - Headache
 - Fever
 - Focal neurologic deficit
- Seizure
- Nausea and vomiting
- Neck stiffness
- papilledema



Radiological examination

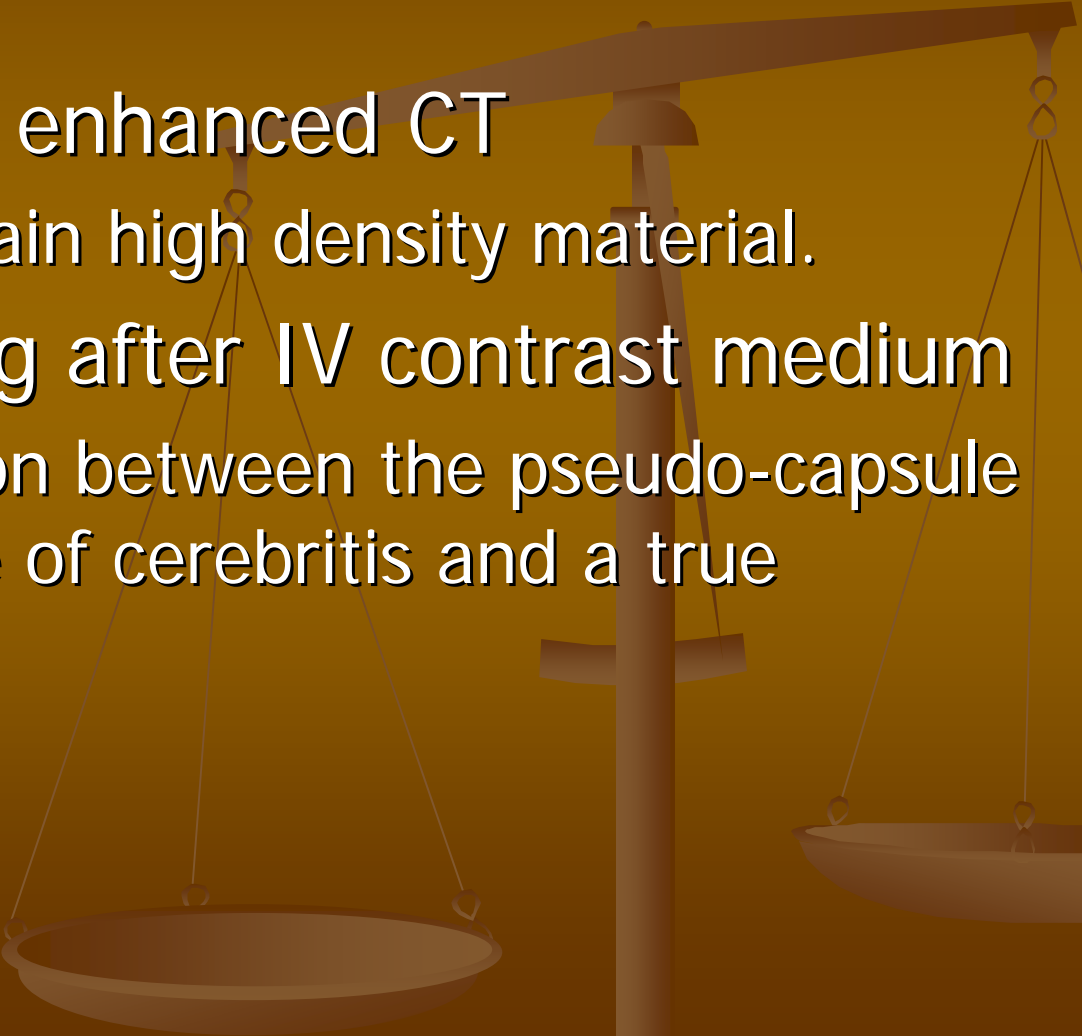
- Skull films
 - For diagnosis of sinusitis or the presence of free gas in the abscess cavity.
- Chest X-ray film
 - For detection of underlying cardiac or pulmonary abnormality.



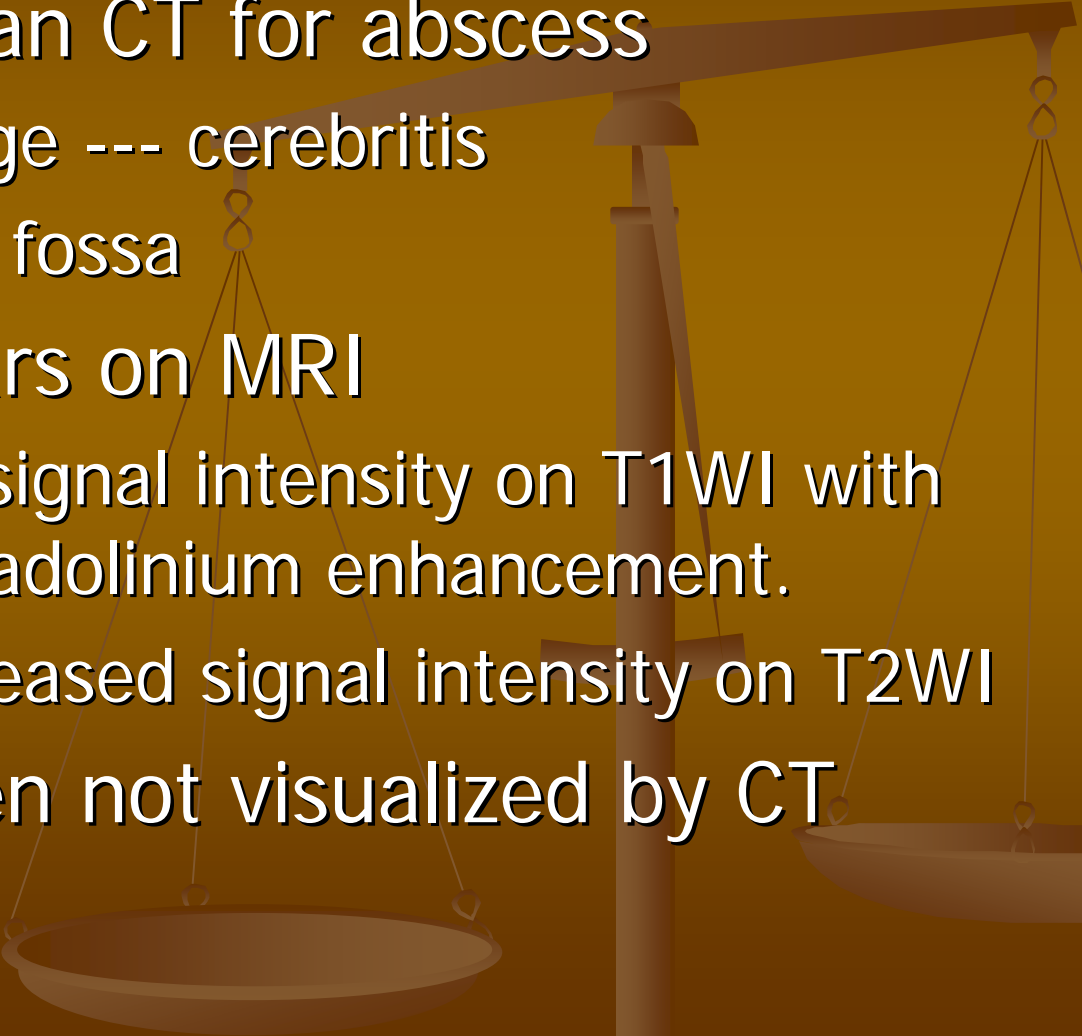
CT

- On contrast enhanced CT
 - An uniformly ring or doughnut representing the spherical wall or capsule of the abscess.
 - Breakdown of the blood-brain barrier
 - Hypervascularity of the granulation tissue.
 - The nonenhancing hypodensity abscess centre is pus.
 - There is commonly extensive hypodense-appearing edema in the surrounding white matter.

CT

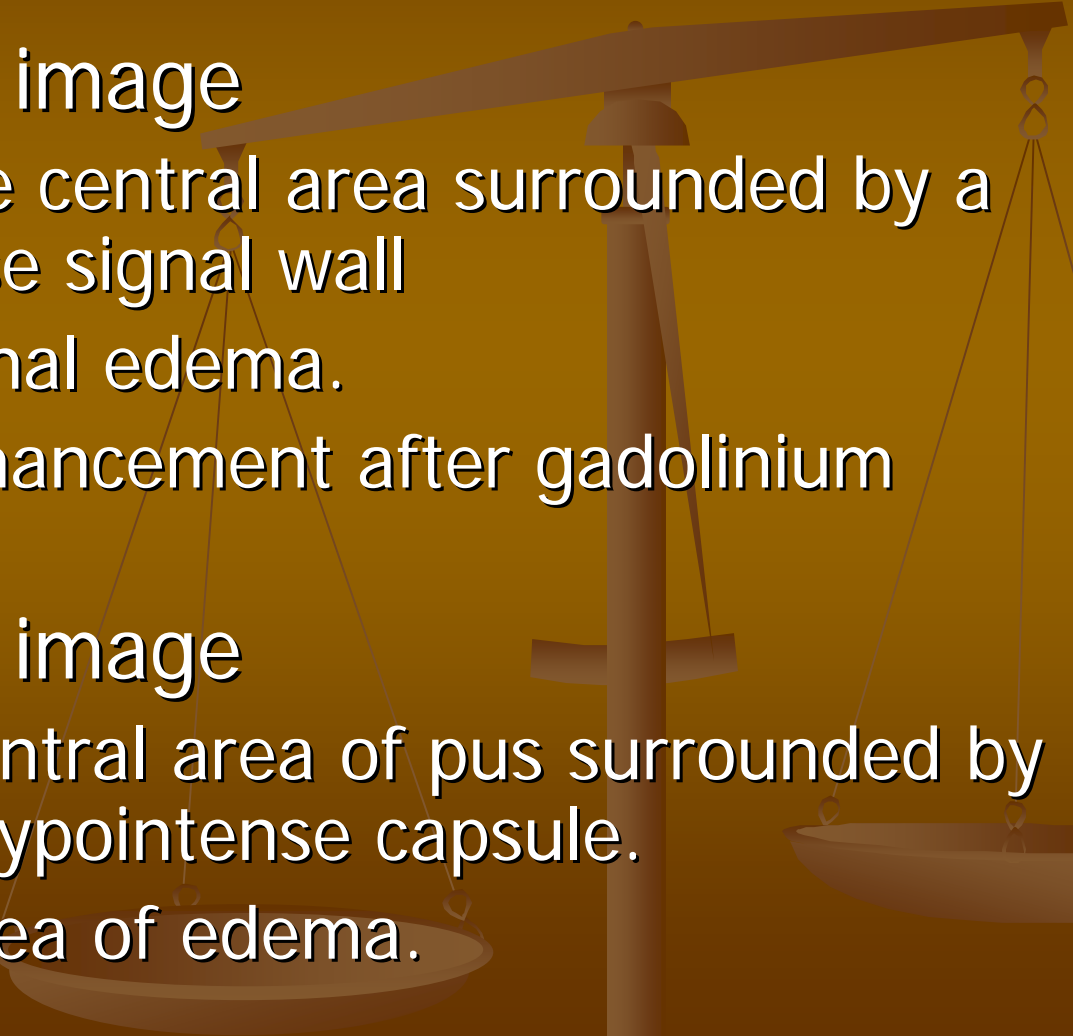
- On non-contrast enhanced CT
 - usually not contain high density material.
 - Delayed scanning after IV contrast medium
 - For differentiation between the pseudo-capsule of the late stage of cerebritis and a true capsule.
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MRI cerebritis

- MRI is better than CT for abscess
 - In the early stage --- cerebritis
 - In the posterior fossa
 - Cerebritis appears on MRI
 - An area of low-signal intensity on T1WI with irregular post-gadolinium enhancement.
 - An area of increased signal intensity on T2WI
 - Cerebritis is often not visualized by CT
- 

MRI

mature brain abscess

- On T1 weighted image
 - The hypointense central area surrounded by a thin hyperintense signal wall
 - hypointense signal edema.
 - Marked ring-enhancement after gadolinium administration
 - On T2 weighted image
 - Hyperintense central area of pus surrounded by a well-defined hypointense capsule.
 - Hyperintense area of edema.
- 

Treatment

- Surgical drainage
 - For encapsulated abscess
- Empirical antibiotic therapy
 - based on abscess culture
 - At least 6-8 weeks
- Prophylactic anticonvulsant therapy
 - At least 3 months
- Short-term steroid therapy
 - Used only in patients with proven or suspected elevation in ICP

