

# BRAIN ABSCESS MANAGEMENT

# Definition



- Focal, intracerebral infection that begins as a localized area of cerebritis and develops into a collection of pus surrounded by a well vascularized capsule.

**Table I.** Age and sex distribution of brain abscess patients

Age group (years)	Males	Females
< 1	1	2
3-10	13	2
11-20	12	2
21-30	5	0
31-40	3	2
41-50	4	1
51-60	3	0
Total	41	9

LAKSHMI V, R. R. RAO RR, DINAKAR I: Bacteriology of brain abscess - observations on 50 cases. J. Med. Microbiol. -Vol. 38 (1993), 187-190

# INCIDENCE

- VARIES ACCORDING TO GEOGRAPHY
- CONSTITUTES 8 % OF ALL INTRACRANIAL LESIONS IN INDIA TO 1-2% IN DEVELOPED NATIONS.\*

\*Osenbach RK, Loftus CM: Diagnosis and management of brain abscess.  
Neurosurg Clin N Am 1992; 3: 403-420.

Bhatia R, Tandon PN, Banerji AK: Brain abscess - an analysis of 55 cases. Int Surg 1973, 58: 565-568

# ETIOLOGICAL AGENTS

- In about one third of patients, more than one organism found.
- Organisms most frequently isolated include streptococci (both aerobic and anaerobic), anaerobes and staphylococci
- Microbiological profile is also changing
- Gram-negative organisms/ anaerobes - increasing cause of cerebral abscess.
- In neonates, more common organisms include *Citrobacter*, *Proteus*, *Pseudomonas*, and *Serratia* species
- These abscesses are often large with poorly formed capsules

- Organisms other than pyogenic bacteria include

*Mycobacterium tuberculosis*

Nontuberculous mycobacteria

Fungi

Parasites

*Actinomyces* and *Nocardia* species

rarely *salmonella*

# Predisposing condition and Microbiology

<b>Predisposing Condition</b>	<b>Usual Microbial Isolates</b>
Otitis media mastoiditis	Streptococci <i>Bacteroides</i> and <i>Prevotella</i> <i>Enterobacteriaceae</i>
Sinusitis	Streptococci, <i>Bacteroides</i> <i>Enterobacteriaceae</i> , <i>S. aureus</i> , <i>Haemophilus</i>
Dental sepsis	Mixed <i>Fusobacterium</i> , <i>Prevotella</i> <i>Bacteroides</i> streptococci
Trauma or postneurosurgical	<i>S. aureus</i> , streptococci, <i>Enterobacteriaceae</i> , <i>Clostridium</i>

Lung abscess empyema bronchiectasis	<i>Fusobacterium, Actinomyces, Bacteroides</i> and <i>Prevotella</i> <i>streptococci, Nocardia</i>
Bacterial endocarditis	<i>S. aureus</i> , <i>streptococci</i>
Congenital heart disease	<i>Streptococci, Haemophilus</i>
Neutropenia	gram-negative bacilli, <i>Aspergillus, Mucorales, Candida</i>
Transplant	<i>Aspergillus, Candida, Mucorales, Enterobacteriaceae, Nocardia T. gondii</i>
HIV	<i>T. gondii, Nocardia Mycobacterium Listeria monocytogenes, Cryptococcus neoformans</i>

# ETIOLOGICAL AGENTS

- Most abscesses are monomicrobial
- 1/3 of all abscesses are polymicrobial  
(especially otogenic)

Incidence of negative cultures is 25-30%

# location

**Table 4** Location of abscesses in the 49 patients with brain abscess

Location	No. (%) of cases <sup>a</sup>
Frontal	18 (35)
Temporal	9 (17)
Parietal	13 (25)
Posterior fossa	5 (10)
Deep-seated	3 (6)
Subdural	4 (8)

<sup>a</sup> Six patients had multiple abscesses

# Location

• OTOGENIC	24(48%)
• CONGENITAL HEART	09(18%)
DISEASE	
• PULMONARY DISEASE	06(12%)
• POST TRAUMATIC	01
TOTAL	50

LAKSHMI V, R. R. RAO RR, DINAKAR I: Bacteriology of brain abscess - observations on 50 cases. J. Med. Microbiol. -Vol. 38 (1993), 187-190

# Pathogenesis

- 1 contiguous focus of infection
  - most common
  - middle ear, mastoid cells, or paranasal sinuses.
  - otitis media : temporal lobe , cerebellum
  - Paranasal sinusitis : frontal lobe
  - sphenoid sinusitis: temporal lobe , sella turcica
  - molar teeth : frontal lobe
  - MECHANISM:RETROGRADE VENOUS THROMBO PHLEBITIS

- 2 hematogenous from a distant focus of infection.
  - usually multiple
  - higher mortality
  - chronic pyogenic lung diseases
    - lung abscess, bronchiectasis, empyema, and cystic fibrosis
  - wound and skin, osteomyelitis,
  - pelvic infection
  - cholecystitis, intra-abdominal infections

- cyanotic congenital heart disease
  - TOF or TGA
- bacterial endocarditis
- Hereditary hemorrhagic telangiectasia
- pulmonary arteriovenous malformations
- after esophageal dilation
- after sclerosing therapy for esophageal varices.

• TOF → CHRONIC HYPOXEMIA → POLYCYTHEMIA/ INCREASED VISCOSITY → MULTIPLE INFARCTS AT GREYWHITE JUNCTION  
→ MILIEU FOR BACTERIAL GROWTH

### 3. TRAUMA

- Open cranial fracture with dural breach
- Neurosurgery or foreign body

### 4. CRYPTOGENIC

# Natural History of Infection

- A canine model after inoculation of  $\alpha$ -hemolytic streptococci
- four stages of brain abscess evolution
  - early cerebritis (days 1 to 3)
  - late cerebritis (days 4 to 9)
  - early capsule formation (days 10 to 13)
  - late capsule formation (day 14 and later)

Britt RH, Enzmann DR, Yeager AS: *J Neurosurg* 1981; 55:590-603

- early cerebritis stage
  - acute inflammatory infiltrate with visible bacteria on Gram stain and marked edema surrounding the lesion.
  - No contrast enhancement



- late cerebritis stage
  - The center of the lesion becomes necrotic,
  - macrophages and fibroblasts invade the periphery.
  - proliferate blood vessel surrounding lesion

- early capsule formation
  - necrotic center decrease in size
  - development of a collagenous capsule that is less prominent on the ventricular side of the lesion
  - cerebral edema starts to regress during this stage.

- late capsule formation
  - the collagen capsule was complete circumferentially
  - increased in density and thickness
  - The wall of abscess consists
    - inner inflammatory layer
    - middle collagenous layer
    - outer gliotic layer.

- Similar findings in anaerobic
- capsule formation could not be divided into early and late stages because of delayed encapsulation.

*Britt RH, Enzmann DR, Placone Jr RC,. J Neurosurg 1984; 60:1148-1159.*

- Capsule formation was less prominent on the ventricular than on the cortical surface
- because differences in vascularity between cortical gray and white matter allowed greater fibroblast proliferation on the cortical side of the abscess.
- tendency to rupture into ventricular system rather subarachnoid space

## CAPSULE formation influenced by:

- Type of organism
- Metastatic abscesses poorly capsulated
- Use of corticosteroids

## Host Defense Mechanisms

- early cerebritis stage,
  - border around area of inoculation, composed of acute inflammatory cells, < neutrophils, plasma cells, and mononuclear cells.>
  - production proinflammatory cytokines (IL)-1 $\alpha$ , IL-1 $\beta$ , IL-6, and TNF- $\alpha$  occurred 1 to 6 hours after exposure

- late cerebritis stage
  - macrophages
  - fibroblasts
  - reticulin formation surrounds necrotic center.
- capsule formation
  - increased fibroblasts , macrophages
  - mature collagen deposited
  - then, necrotic center decrease in size
  - while gliosis develops outside the capsule

## CLINICAL PRESENTATION

Depends on:

- Location of the lesion
- Size of the lesion
- Multiplicity
- virulence of the organism
- Host response
- Severity of edema

# Clinical presentation

<i>Symptom or Sign</i>	<i>Frequency (%)</i>
Headache	~70
Mental status changes	≤70
Focal neurologic deficits	>60
Fever	45–50

Mandell, Bennett, & Dolin: Principles and Practice of Infectious Diseases, 6th ed.

Seizures	25-35
Nausea and vomiting	25-50
Nuchal rigidity	25
Papilledema	25

**Classical triad of headache, neurol deficits,  
fever seen in 25%\***

\*Loftus CM, Osenbach RK, Biller J: Diagnosis and management of brain abscess. In: Neurosurgery, second edition, Vol. III, Wilkins RH, Rengachary SS (eds) McGraw-Hill, New York 1996; 3285-3298

## HEADACHE

- indolent to fulminant
- due to size and location of a space-occupying lesion
- The headache may be moderate to severe
- hemicranial or generalized
- Sudden worsening headache, with a new onset of meningismus, may rupture abscess into ventricular space

- nocardial brain abscess
  - presence of pulmonary, skin, or muscle lesions .



- *Aspergillus* brain abscess
  - manifest signs of a stroke syndrome
  - secondary to ischemia or intracerebral hemorrhage, or both
  - signs of meningeal irritation are rare
- immunocompromised usually present with nonspecific findings i.e., alteration in mental status, or seizures, or both
- evidence of aspergillosis involving other organ

- CNS toxoplasmosis
  - immunocompromised
  - variable,
    - insidious process over several weeks
    - acute onset with a confusional state
  - initial symptoms and signs focal or nonfocal
  - basal ganglia and brain stem,
    - extrapyramidal symptoms resembling Parkinson's disease
  - some develop a diffuse, rapidly fatal encephalopathy

# Differential diagnosis of ring-enhancing cerebral lesions

- Brain abscess
- Primary or metastasis brain tumor
- Subacute ischaemic infarction
  - CYSTIC GLIOMAS
  - RESOLVING HEMATOMAS
  - DEMYELINTING DISEASE

# LAB INVESTIGATIONS

- NOT MUCH HELPFUL
- TLC > 20000 SEEN IN ONLY 10%
- ESR IS INCREASED IN > 90%
- CRP levels - Help in following response to treatment
- L.P. IS INCONCLUSIVE AND DANGEROUS – TO BE AVOIDED

# Plain X-Rays

- Radiographic findings usually limited to paranasal or mastoid sinus opacification
- Gas bubbles or air-fluid levels within cranium indicate gas-producing organism or communication with paranasal sinuses
- Occasionally, foreign bodies or osteomyelitis of maxillary bone indicate source
- Bone destruction of roof, floor, or lateral wall of sinuses indicates aggressive osteomyelitis

## CT brain

- hypodense center surrounded by smooth, regular thin-walled capsules with areas of ring enhancement.
- surrounded by variable hypodense area of brain edema
- areas of low attenuation without enhancement, during the early cerebritis

# CT Brain

- a 'ring' or 'doughnut' representing spherical wall or capsule of abscess
- contrast enhancement being result of breakdown of blood–brain barrier and hypervascularity of the granulation tissue
- nonenhancing abscess centre is pus or nonviable debris
- commonly extensive edema of vasogenic type in surrounding white matter.

- Differentiation between pseudo-capsule and true capsule may be achieved by delayed scanning after IV contrast medium, in cerebritis stage the centre of the lesion will fill in with contrast medium, whereas with mature abscess, there is never enhancement of the pus-filled centre.
- thick or markedly irregular wall suggests a tumor rather than an infective lesion

# CT Brain

- capsule is usually less well developed on ventricular side than on cortical side.
- useful for following the course of brain abscess
- although after aspiration, improvement in the CT appearance may not be seen for up to 5 weeks or longer.

# MRI

- more sensitive than CT
- variable & change with the stage of abscess
- therapy with corticosteroids can decrease enhancement seen with both CT and MRI

# MRI In Early Cerebritis

- T<sub>1</sub>-weighted images
  - poorly delineated enhancing areas within the isointense to mildly hypointense edematous region.
- T<sub>2</sub>-weighted images
  - ill-defined subcortical hyperintense zone

# MRI In Late Cerebritis

- T<sub>1</sub>-weighted images
  - thick somewhat irregularly marginated rim appears isointense to mildly hyperintense
- T<sub>2</sub>-weighted images
  - central necrotic area is hyperintense to brain tissue and rim isointense to relatively hypointense

# MRI In Early & Late Capsule Phase

- collagenous abscess capsule is visible prior to contrast
- thin-walled isointense to slightly hyperintense ring that becomes hypointense on T2-weighted MRIs.

# CT & MRI in Fungal Brain Abscess

- sensitive but not specific
- Aspergillosis : finding of a cerebral infarct in a patient with risk factors for invasive aspergillosis should suggest that diagnosis.
- Rhinocerebral mucormycosis : characteristic changes, including sinus opacification, erosion of bone, and obliteration of deep fascial planes

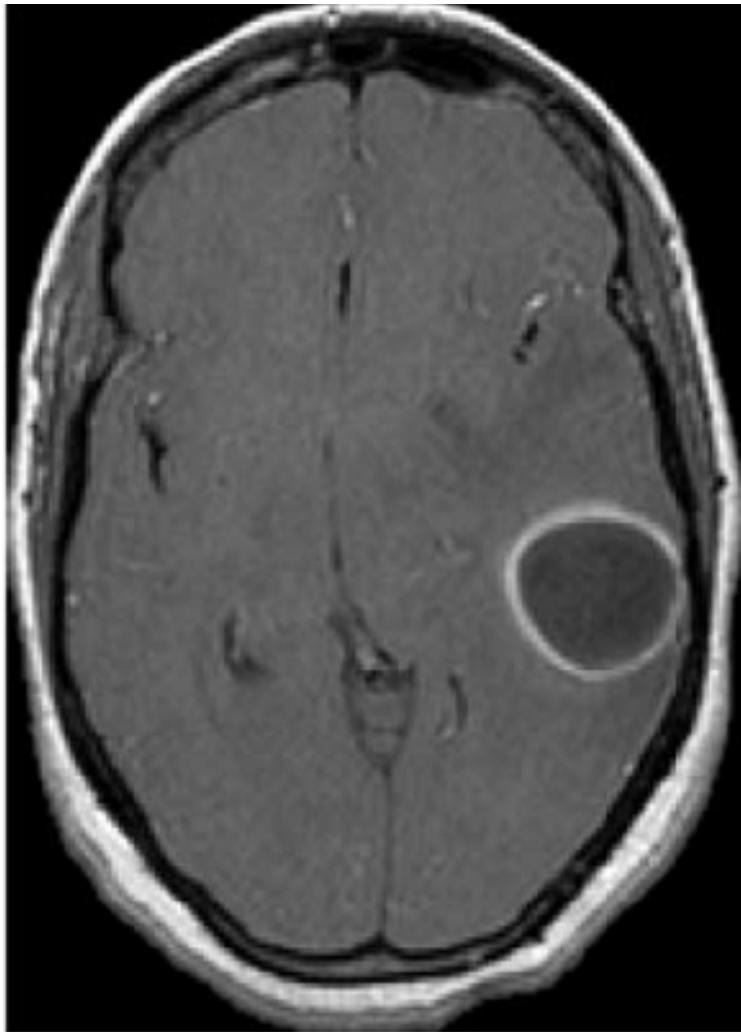
- brain abscesses may mimic necrotic tumours and cystic metastases
- both clinically and radiologically, and imaging findings may be indistinguishable on conventional MRI

(However, fever, meningism, raised ESR, multilocularity, leptomeningeal or ependymal enhancement, reduction of ring enhancement in delayed scan and finding of gas within the lesion favour a diagnosis of abscess.)

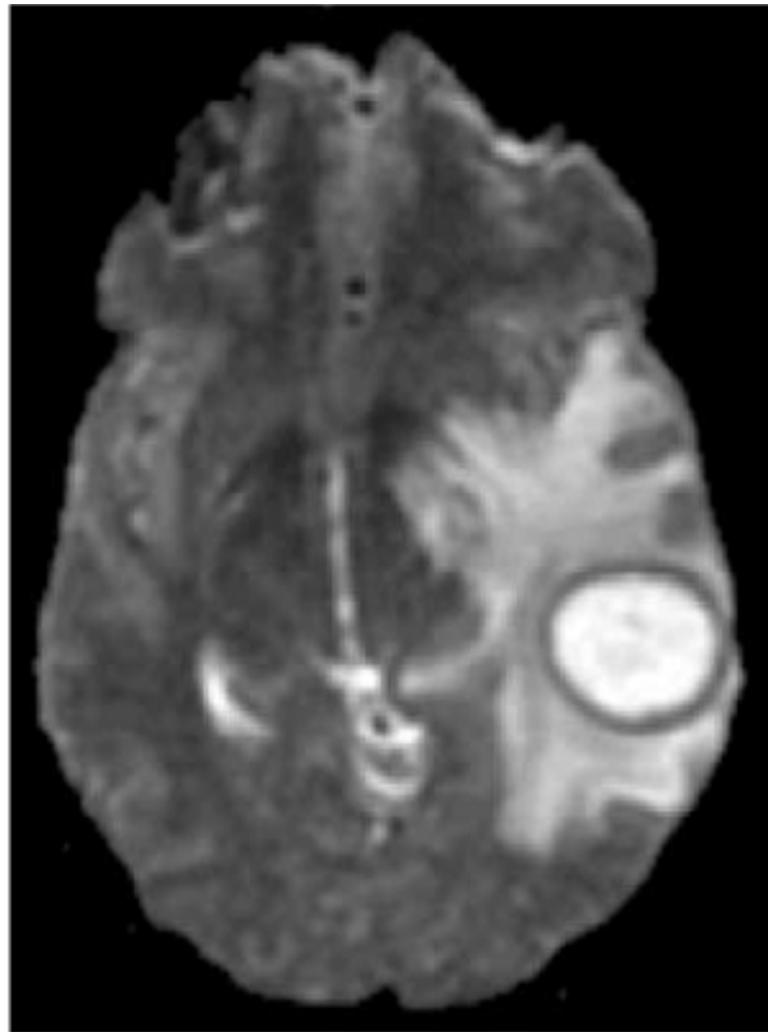
Diffusion-weighted MRI features of brain abscess and cystic  
or necrotic brain tumors  
Comparison with conventional MRI

Shih-Chin Chang<sup>a</sup>, Ping-Hong Lai<sup>a,\*</sup>, Wei-Liang Chen<sup>a</sup>, Hsu-Huei Weng<sup>b</sup>, Jih-Tsun Ho<sup>c</sup>,  
Jyh-Seng Wang<sup>d</sup>, Chuan-Yu Chang<sup>e</sup>, Huay-Ben Pan<sup>a</sup>, Chien-Fang Yang<sup>a</sup>

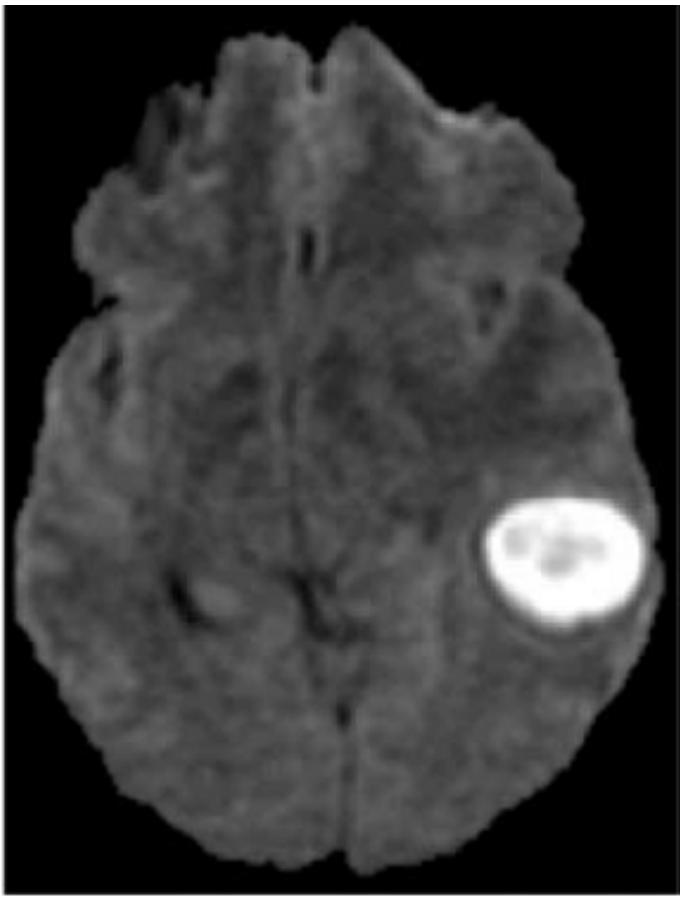
- **11 brain abscesses and 15 cystic or necrotic brain tumors**
- In all **pyogenic abscesses** : Increased signal in DWI and ADC maps showed restricted diffusion in the abscess cavity
- All **cystic or necrotic tumors** but one showed low signal intensity on DWI and high ADC values



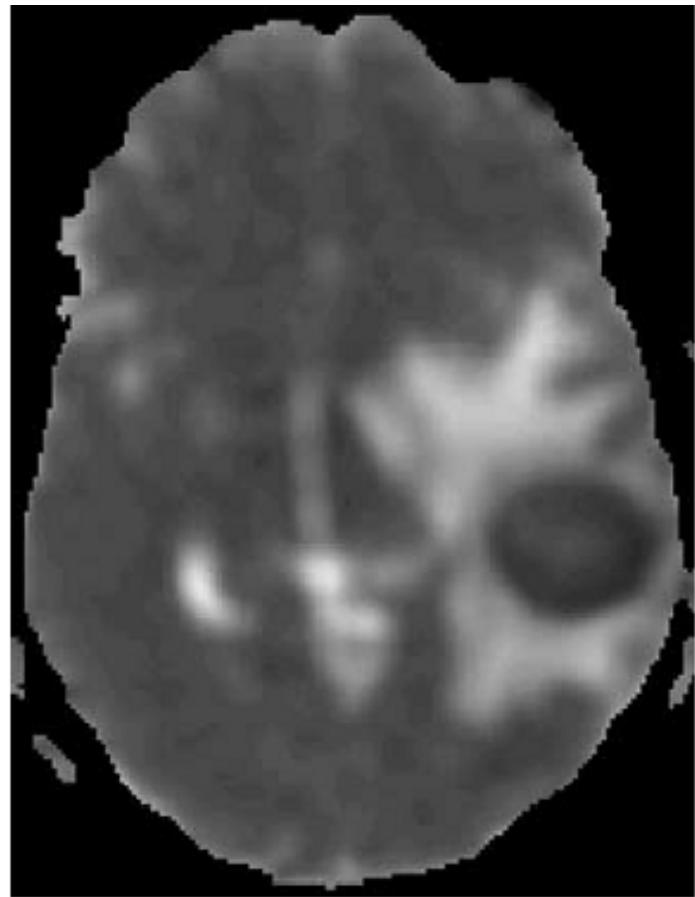
T<sub>1</sub>-weighted axial MR image



T<sub>2</sub>-weighted axial MR image



Diffusion-weighted MR image



ADC map

Surgical Neurology 66 (2006) 246–251

## **DWI yielded**

- a sensitivity of 93.33%
- a specificity of 90.91%
- a PPV of 93.33%
- a NPV of 90.91%

# Role of diffusion-weighted MR in differential diagnosis of intracranial cystic lesions

Y. Bükte<sup>a</sup>, Y. Paksoy<sup>b,\*</sup>, E. Genç<sup>c</sup>, A.U. Uca<sup>d</sup>

Table 3 Signal characteristics of the lesions on diffusion-weighted imaging

Lesion	Hypointense		Hyperintense		Isointense	
	Number	%	Number	%	Number	%
Abscess	0	0	22	100	0	0
All malignant lesions	25	83	3	10	2	7
Primary tumour	17	90	0	0	2	10
Metastasis	8	73	3	27	0	0

Table 4 Sensitivity and specificity of diffusion weighted imaging in the differential diagnosis of lesions (numbers)

	True positive	False positive	False negative	True negative	Sensitivity	Specificity	PPV	NPV
Abscess/malignancy	22	3	0	27	100	90	88	100
Abscess/primary tumour	22	0	0	19	100	100	100	100
Abscess/metastasis	22	3	0	8	100	73	88	100

PPV, positive predictive value; NPV, negative predictive value. (Odds ratio: 0.880; 95% confidence intervals: 0.761; 1.017).

# MR Spectroscopy

- Metabolic information about healthy and pathologic tissues can be obtained **Noninvasively by localized in vivo** MR spectroscopy to supplement morphologic information obtained with imaging modalities.

## MR Spectroscopic Findings in Patients with Brain Abscess or Tumor

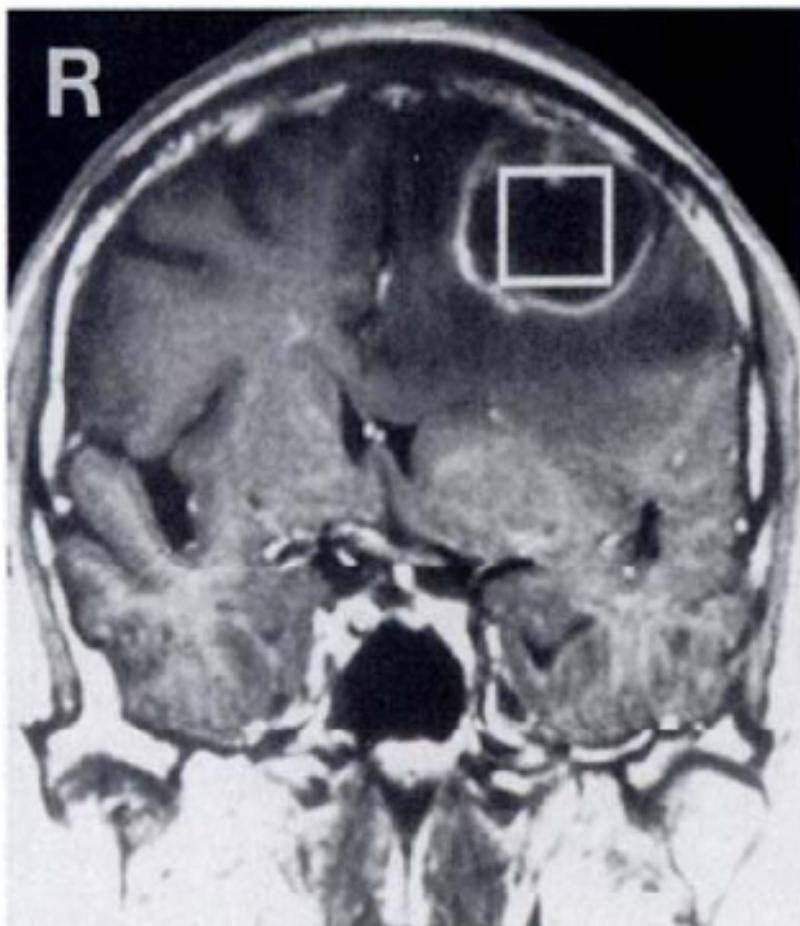
Patient No.	Final Diagnosis	Metabolites Detected*
1	Brain abscess	Lactate (++), acetate (+++), succinate (++), valine or leucine (++), alanine (+), unknown metabolite A (2.2 ppm), unknown metabolite E (3.8 ppm)
1†	Brain abscess	Lactate (+++), acetate (++), succinate (++), valine or leucine (++), alanine (+), unknown metabolite A (2.2 ppm)
2	Brain abscess	Lactate (+++), amino acid or lipid (+++), leucine (+++), unknown metabolite B (2.9 ppm), unknown metabolite C (3.2 ppm), unknown metabolite D (3.4 ppm)
3	Brain abscess	Lactate (+++), acetate (++), amino acid or lipid (+++), alanine (++), leucine (++)
4	Brain abscess	Lactate (+++), valine or leucine (++)
5	Brain abscess	Lactate (+++), acetate (+), amino acid or lipid (++)
6	Brain abscess	Lactate or lipid (+), succinate (+), alanine (+), N-acetyl aspartate, creatine-phosphocreatine complex
7	Brain abscess	Lactate (+++), acetate (+), succinate (+), amino acid or lipid (+), unknown metabolite C (3.2 ppm)
8	Glioblastoma multiforme	Lactate (++)
9	Recurrent glioblastoma multiforme	Lactate (+)
10	Glioblastoma multiforme	Lactate (+++)
11	Glioblastoma multiforme	Lactate (+++)
12	Pilocytic astrocytoma	Lactate (+)
13	Glioblastoma multiforme	Lactate (+)
14	Metastasis from non-small cell lung cancer	Lactate (++) , amino acid or lipid (+)

\* Data in parentheses indicate peak size. + = small peak, ++ = moderate peak, +++ = large peak.

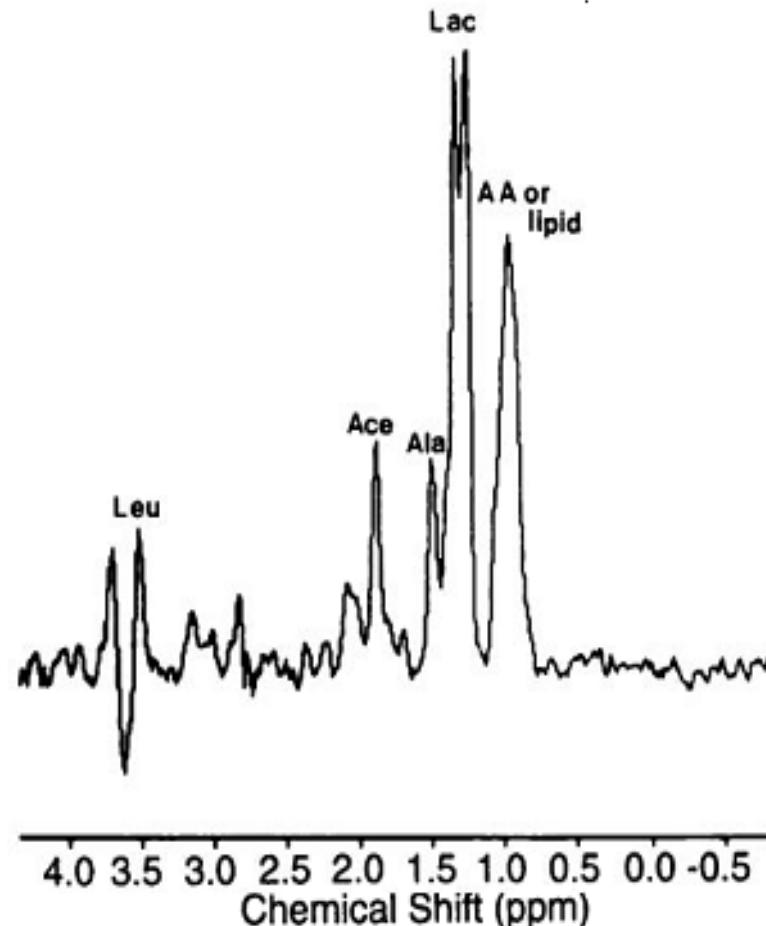
† Two spectra were obtained in patient 1, who had two brain abscesses. **Radiology** 1997; 204

# **Brain Abscess and Brain Tumor: Discrimination with in Vivo H-1 MR Spectroscopy<sup>1</sup>**

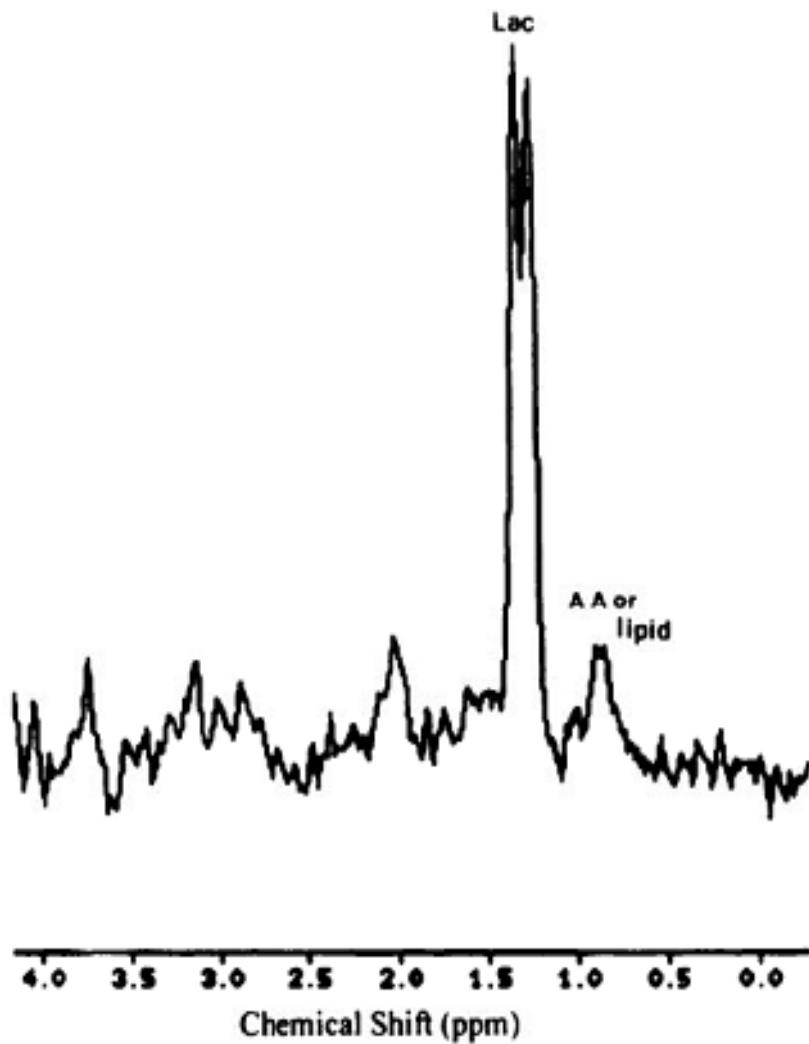
Sung Hyun Kim, MD • Kee-Hyun Chang, MD • In Chan Song, PhD • Moon Hee Han, MD  
Hee Chan Kim, PhD • Heung Sik Kang, MD • Man Chung Han, MD



Pyogenic brain abscess



**Radiology** 1997; 204:239–245



Metastasis brain tumor

Radiology 1997; 204:239–245

# MR Spectroscopy

- In 6/7 pts with abscess, there were various resonances attributed to lactate, valine, alanine, leucine, acetate, succinate, and unidentified metabolites
- In 6/7 pts with tumor, there was only a resonance attributed to lactate.

## CONCLUSION:

- Spectral patterns from in vivo MR spectroscopy may permit differentiation of brain abscess from necrotic or cystic tumor.

# LEUKOCYTE LABELLED SCANS

- Finding of uptake of labeled leukocytes exceeding the normal physiological uptake of the base of skull\*
- Sensitivity approaches 100%, Specificity 94%
- Non invasive
- However , optimal images takes 24 hrs, can be used only in pts. with good neurological condition.

\* Rehncrona S, Brismar J, Holtas S: Diagnosis of *brain abscesses* with indium-111-labeled leukocytes. Neurosurgery 16:23-26, 1985.

- imaging studies alone cannot differentiate between brain abscess caused by different pathogens.
- pathologic confirmation and/or microbiologic studies are needed to ensure proper management

# Management

- In general, the basic principles of treatment are
  - appropriate antibiotics with or without aspiration,
  - treatment of sequelae i.e. hydrocephalus, seizures etc.
  - eradication of primary source.\*
- The choice between conservative vs operative treatment is influenced by :  
age, neurological status, location, number, size and stage of abscess formation.
- Treatment ,even if medical ,should be under the guidance of neurosurgeon as prompt decision to operate has to be taken if pt fails medical management.
- Each case must be individualized and treated on its own merits.

\*Rosenblum ML, Mampalam TJ, Pons VG: Controversies in management of brain abscesses. Clin Neurosurg 1986;33: 603-632.

# MANAGEMENT

Current concepts in the management of  
pyogenic brain abscess

Sharma BS, Gupta SK, Khosla VK: Neurol India  
48:105–111, 2000

# Medical management

- Antibiotic therapy during early stage prevents progress from cerebritis to abscess.
- Patients who have symptoms for < 1 week have a more favorable response to medical therapy.
- Empirical treatment is advocated only when clinical and radiological improvement continues.
- **Rosenblum et al found that mean diameter of abscess that resolved with a/b was 1.7cm( no abscess> 2.5cm resolved with a/b alone)**

- Patients demonstrate clinical improvement before significant changes in CT scan.
- Improvement on CT scans generally observed within 1-4 weeks (average, 2.5 wk) and complete resolution in 1-11 months (average, 3.5 mo).
- Antimicrobial treatment generally long (6-8 wk) because of prolonged time needed for brain to repair and close abscess space
- If medical therapy is chosen , ct scans to be done weekly during t/t and mthly after discontinuing t/t till enhancement disappears

## EFFICACY OF ANTIBIOTICS DEPENDS ON:

- Bactericidal / bacteriostatic nature
- Route and duration of therapy
- Host response to infection
- Concentration of drug at the site of abscess
- Depth of the lesion

- Opportunistic organisms which generally are not pathogenic to humans, cause brain abscess in immunocompromised patients.
- Antibiotics are given for 3-12 months.

1. reduced lymphocytic function	<i>N. asteroides</i> / <i>T. gondii</i>	sulfonamide and pyrimethanium
2. T-lymphocytic defect,	<i>Candida</i>	5 flucytosine and amphotericin-B .
3. renal transplant recipients patients with blood cancer /steroids therapy	<i>Listeria</i>	ampicillin .
4. Leukaemia / lymphoma	<i>Pseudomonas</i>	aminoglycosides.

# ANTICONVULSANT THERAPY

- Legg\* advocated anticonvulsant therapy to all pts of cerebral abscess for 5 yrs.
- To be discontinued only when pt is seizure free for 2 yrs or EEG shows no epileptic activity

\*Legg NJ, Gupta PC, Scott DF: Epilepsy following cerebral abscess: A clinical and EEG study of 70 pts. Brain 1973;96:259-68

# SURGICAL MANAGEMENT

- Surgical drainage provides most optimal therapy
- **Aspiration** is most common procedure and is often performed using a stereotactic procedure
- CAN BE USED IN ALL STAGES OF ABSCESS
- In case of multiple abscesses or in abscesses in essential brain areas, repeated aspirations preferred to complete excision
- Aspiration can be guided by real time usg and stereotaxy.

At the time of aspiration,  
specimens should be sent for

- Gram stain
- aerobic and anaerobic cultures
- cultures for mycobacteria and fungi
- acid-fast stains for mycobacteria
- modified acid-fast stains for *Nocardia*
- special stains (e.g., mucicarmine, methenamine silver) for fungi

- Recently, encouraging results with **endoscopic stereotactic** evacuation of brain abscess.
- Neuroendoscopic treatment, when compared to stereotactic aspiration, has additional advantage of more complete drainage and lavage.\*

\* Hellwig D, Benes L, Bertalanffy H et al: Endoscopic stereotaxy - an [eight] years experience. Stereotact Funct Neurosurg 1997; 68: 90-97.  
Fritsch M, Manwaring KH: Endoscopic treatment of brain abscess in children. Minim Invasive Neurosurg 1997; 40: 103-106

# Role of Corticosteroids

- may be beneficial in
  - pts with increased intracranial pressure
  - potentially lifethreatening complications, such as impending cerebral herniation
- retard immune responses and encapsulation

# Excision

## Indications

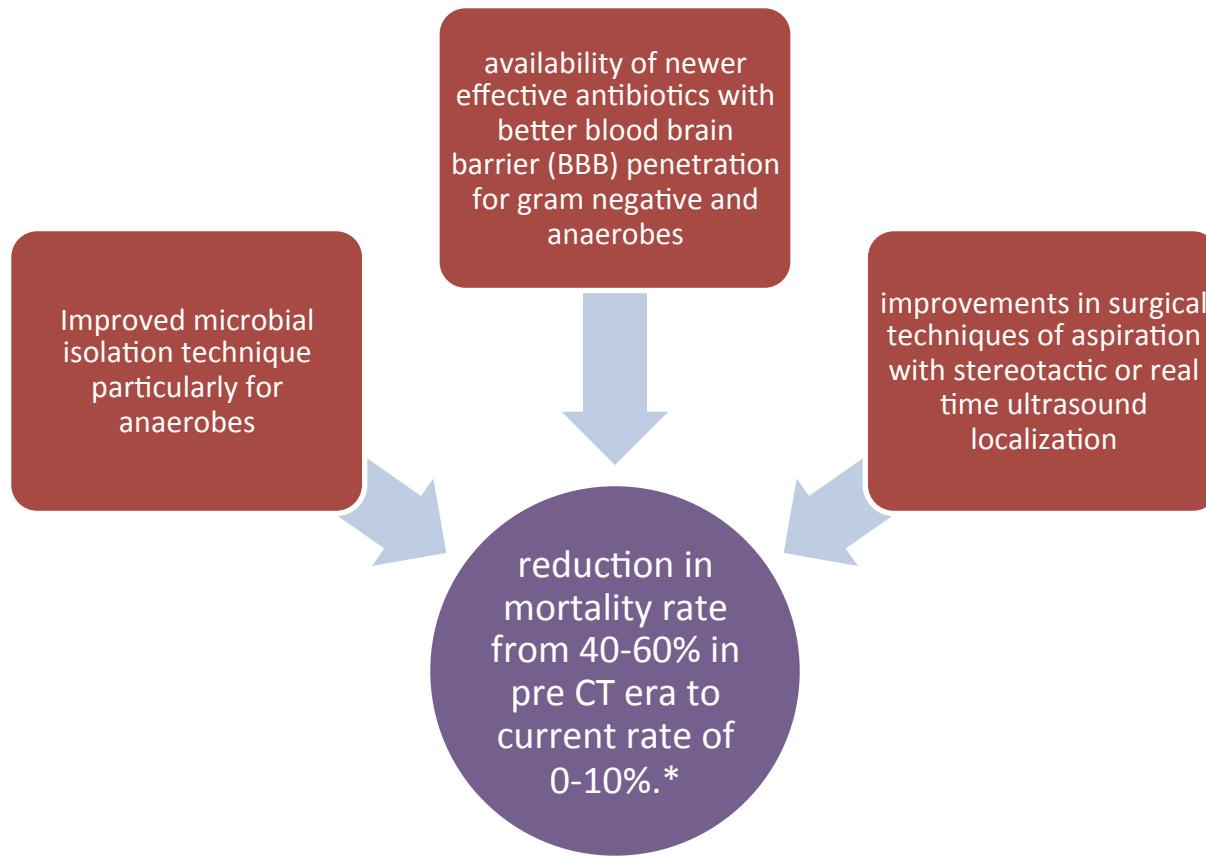
- Multiloculated abscesses
- Abscesses caused by more resistant pathogens
- Abscesses containing gas
- Post-traumatic abscesses containing foreign bodies or contaminated retained bone fragments
- Abscesses resulting from fistulous communication

# Excision

## Contraindications

- Abscesses in the cerebritis stages
- Deep-seated abscesses in eloquent areas
- Multiple abscesses

# PROGNOSIS



\* Yang SY: Brain abscess: A review of 400 cases. J Neurosurg 1981; 55: 794-799.

## Prognosis is worse for patients with

- intraventricular rupture
- associated meningitis, ependymitis or empyema
- an unknown primary source
- sterile pus or culture
- large abscess
- presence of hydrocephalus
- metastatic abscess
- multiple deep-seated abscesses
- inaccurate diagnosis
- congenital cyanotic heart disease

# SEQUELAE

- 30-50% of survivors are found to have neurological sequelae.
- The incidence of residual neural deficits - hemiparesis, cognitive and learning deficits in children, is less with aspiration than excision.
- About 72% of patients can have epileptic seizures upto five years of diagnosis. This incidence is less with aspiration than excision.
- 5 to 10% abscesses recur due to inadequate or inappropriate antibiotics, failure of removal of foreign body, dural fistula or failure of eradication of primary source.
- Hydrocephalus may also develop.

Koszewski M: Epilepsy following brain abscess. The evaluation of possible risk factors with emphasis on new concepts of epileptic focus formation. Acta Neurochir (Wien) 1991; 113: 117-120.

# SPECIAL TYPES

- **MULTIPLE ABSCESS**

- 5% to 50% of all brain abscess patients harbor multiple abscesses
- detection rate has increased since the advent of CT.
- Result from Hematogenous spread from systemic infections and the organisms may be found in peripheral source.
- Streptococcus and Staphylococcus are common organisms.
- More often in immunocompromised patients .
- The frequency of intraventricular rupture is also more in these cases.

Sharma BS, Khosla VK, Kak VK et al: Multiple pyogenic brain abscesses. Acta Neurochir (Wein) 1995; 133: 36-43.

# SPECIAL TYPES

- CEREBELLAR ABSCESS:
- Cerebellar abscesses comprise 6-35% of all brain abscesses.
- They are often ominously silent and carry significant mortality.
- can cause sudden total occlusion of CSF pathways early in the course of disease.
- Presence of periventricular lucency is an absolute indication of immediate ventricular drainage regardless of level of consciousness i.e. even if patient is fully conscious.
- burr hole aspiration has emerged as a satisfactory method.

Brydon HL, Hardwidge C: The management of cerebellar abscess since the introduction of CT scanning. Br J Neurosurg 1994; 8: 447-455.

# SPECIAL TYPES

- **FUNGAL ABSCESS:**

- Affects Immunocompromised hosts
- Aspergillus/ candida more common
- Infection either by direct extension or by hematogenous spread
- Definitive diagnosis only by biopsy but prompt exam. possible by KOH wet mount prep.
- Immunodiagnostic assays helpful in diagnosis
- Definitive therapy is antifungal chemotherapy + surgical excision

\* infection of central nervous system : By W.M. Michael scheld, Richard . J. whiley

# SPECIAL TYPES

- **Fungal abscess:**
  - i.v. AMB is the mainstay of therapy
  - Lipid formulation is preferred.
  - Can cause nephrotoxicity, hypokalemia, hypomagnesaemia, hepatotoxicity, leucopenia, thrombopenia, cardiac arrhythmias , cardiac failure.
  - 5-10mg given initially as a test dose.
  - Started initially at 0.3mg/kg at a rate of 0.1mg /ml . Dose escalated gradually to 0.7mg/kg
  - Candida infection requires :
    - AMB 0.7mg/kg/day+5-FU at 100mg/kg/day for 2wks f/b
    - Fluconazole 400-800mg/day for 6 wks
  - Aspergillosis : voriconazole 300mg/day for 6mths -1yr
  - Therapy is continued till lesions resolve or sustained stabilizations occur.

# SPECIAL TYPES

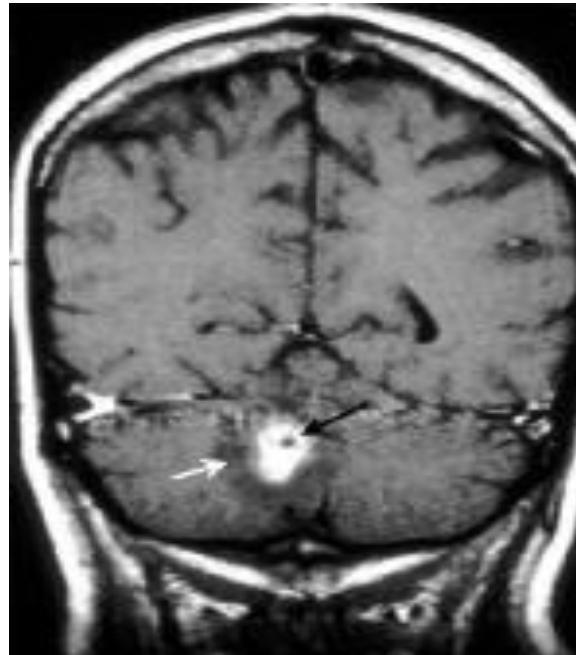
## NOCARDIAL ABSCESESSES

Nocardia are aerobic, gram-positive, partially acid-fast, filamentous bacteria.

Nocardia asteroides -most common pathogen

Affects immunocompromised hosts

Pulmonary infection is the most common initial infection, with hematogenous spread to other organs



Coronal T1-weighted spin-echo gadolinium-enhanced MRI demonstrates zone of enhancement , with a zone of decreased brightness (edema, white arrow). Nocardia organisms were cultured from within the abscess cavity.

# SPECIAL TYPES

- Tubercular abscess
- Very rare
- Caseation →→ liquefaction →→ necrosis
- Granulomatous changes absent
- Pus contains viable AFB bacilli
- Differentiated from tubercular cyst which contains clear yellowish fluid + granulomatous changes in the wall
- ATT mainstay of therapy
- Surgical drainage of the abscess required for diagnosis & Improving the neurological condition

**THANK YOU**