Brain abscesses: magnetic resonance imaging findings, diffusion weighted MR imaging and MR spectroscopy at 1.5 T and 3T MR imaging scanners

Poster No.: C-0711
Congress: ECR 2012
Type: Scientific Exhibit
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DOI: 10.1594/ecr2012/C-0711

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Purpose

Show the imaging findings of the cerebral abscesses at the conventional magnetic resonance imaging, diffusion-weighted imaging (DWI) and $^1$H- MR spectroscopy (MRS).

Methods and Materials

We retrospectively reviewed clinical notes, conventional MR sequences, DWI and MRS in fourteen patients referred to our tertiary care hospital for 10 years with brain abscesses.

There were 7 male and 7 female patients with an age range between 12 and 79 years old, mean age, 55 years.

10 patients were imaged on a 1.5 T superconducting magnet and 4 patients on a 3 T superconducting magnet.

Results

A total of 21 brain abscesses were evaluated for this study in 14 patients. Multiple abscesses were seen in 4 (29 %) of the 14 patients.

All lesions appeared hyperintense on T2-weighted image (WI) and hypointense on T1-WI, with variable perifocal edema, and showed ring enhancement on postcontrast T1-WI. They usually present a capsule, which was iso.-to midly hyperintense relative to brain on short TR/ short TE scans and iso.-to hypointense relative to white matter on long TR/ intermediate to long TE scans. On the long TR scans the relative hypointensity of the rim allowed for the typical morphologic features of the capsule, which in turn aided in differentiation of abscesses from other lesions.

The abscesses were located in the right frontal lobe in 6, the left frontal lobe in 2, the thalamus in 2, the right occipital lobe in 2, the left occipital lobe in 2, the right parietal lobe in 1, the right parieto-occipital lobe in 1, the left temporal lobe in 1, the right temporal lobe in 1, the right temporo-occipital lobe in 1, and the right cerebellum in 1 patient.

8 patients were treated with open drainage. 6 patients were treated with antibiotics.
Twelve patients were cured after surgery or antibiotic treatment. Two patients died due to complications.

In 7 patients the organisms were identified: Peptostreptococcus, Nocardia, Naegleria fowleri, Listeria, Sptreptococcus gallolyticus, Klebsiella pneumoniae and Corynebacterium.

The clinical information is summarized in the table 1.

Tables 2 y 3 summarizes clinical features, the location, laboratory results, treatment, types of MR studies and the outcome of the patients.

Brain abscesses evolve through for stages.

The initial stage of abscess formation is **early cerebritis** occurring during the first 4 to 5 days of an infection: Capillary endothelial swelling resulting in opening of the blood-brain barrier with edema and sometimes microhemorrhages occurs.

MRI demonstrate prolonged T1 and T2 signal without significant mass effect or contrast enhancement.

By the end of the first week: **late cerebritis stage** begins. Small zones of necrosis coalesce into one o two large foci filled with necrotic debris with a thin rim of vascular granulation tissue at the margin of the necrotic zone. Edema contiguous to the area of cerebritis develops and is sometimes associated with additional foci of inflammation, which can progress to satellite lesions. MR shows prolonged T1 and T2. However, local mass effect is usually present and minimal diffuse enhancement may be seen. At this stage if an abscess enlarges, it tends to extend within white matter where the inflammatory response is less due to the limited vascularity. In some cases, an abscess may rupture into the ventricular system.

Toward the end of the second week, **early capsule formation stage** begins during which a collagenous capsule forms, which then becomes progressively thicker over the next several weeks. Central necrotic material becomes liquefied. With maturation of the capsule, edema diminishes and gliotic reaction occurs at the outer margin of the capsule. The completed capsule has three layers: an inner inflammatory layer of granulation tissue and macrophages, a middle collagenous layer and an outer gliotic layer. MRI demonstrates a well-defined enhancing capsule of variable thickness surrounding the non-enhancing abscess. It is at this stage that surgical drainage of an abscess can be considered.
Late capsule formation stage: nearby edema decreases, cavity shrinks and the abscess capsule thickens and sometimes mineralizes. Although resorption of an abscess may occur either spontaneously or in response to systemic antibiotic therapy, most abscesses continue to grow until a catastrophic event occur, such as rupture into the subarachnoid space or the ventricular system. Surgery is best delayed until the capsular stage because attempts at aspiration before this time will not yield pus and can cause hemorrhage, from vascular granulation tissue.

If an abscess undergoes resorption, the hypointense ring on T2-WI disappear, ADC values increased and finally there isn’t enhancement.

Axial T2-WI (fig. 1), Axial T1 WI before and after contrast injection (figs. 2 and 3) and Coronal contrast-enhanced T1-WI (fig. 4) show an early cerebritis stage: Left frontal lesion, poorly marginated with prolonged T1 and T2 signal, without significant mass effect or contrast enhancement. (Case 1, patient number 5)

Axial T2 WI (fig. 5), Axial T1 WI before and after contrast injection (figs. 6 and 7): show a hypointense ring with thin-walled enhancing ring: early capsule formation stage. (Case 13, patient number 2)

Case 1: A 53-year-old man, HIV positive. Bilobulated right frontal brain abscess.

Sagittal T1-WI (fig. 8) and Axial T2 WI (fig. 9) show the mildly hyperintense ring on T1-WI and hypointense on T2-WI with perifocal edema. The signal of ring is related to collagen, blood products, or paramagnetic free radicles.

Axial T1-WI before and after contrast injection (figs. 10 and 11) and contrast-enhanced T1-WI (fig. 12) show ring-enhancing abscess with ventricular extension. Rim enhancement along the right lateral ventricular wall is consistent with ependymitis and ventriculitis.

Case 2: A 72-year-old man with a right frontal abscess.

Axial T2-WI (fig. 13) and Coronal FLAIR image (fig14) show the characteristic hypointense rim at T2-WI and hyperintense edema.

The abscess shows hypointense on T1 WI (fig. 15) with a slightly hyperintense wall. Axial and Coronal postcontrast T1-WI (figs. 16 and 17) reveal ring enhancement.

Axial DWI (fig. 18) shows strong hyperintensity within the abscess cavity. Axial ADC map from DWI (fig. 19) reveals low values, indicating restricted diffusion. This phenomenon
might be related to the high viscosity and cellularity of pus, which causes restriction of water proton mobility.

MRS: $^1$H MR Spectra with a TE of 36 ms (fig. 20) and with a TE of 144 ms (fig. 21) show resonances of amino acids (AA), 0.9 ppm, lipids (Lip), 0.8-1.2 ppm, lactate (Lac), 1.3 ppm and acetate (Ac), 1.92 ppm. With TE of 144 ms the resonances of AA (i.e., valine, isoleucine and leucine), Lac and alanine showed phase reversal, whereas the Lip resonances remained unchanged.

The normal brain metabolites such as N-acetylaspartate (NAA), Choline (Cho) and Creatine (Cr) are absent.

The presence of AA and Lip/Lac, along with AC and/or succinato (Suc), is considered specific for a brain abscess.

Lac and Lip are nonspecific metabolites resulting from glycolysis and necrotic brain tissue in the brain abscess. Both lactate and lipid peaks can also be observed in the necrotic tumor.

The $^1$H MR spectral patterns (figs. 22 and 23) from the center of brain abscesses are characteristic:

- Succinato (2.4 ppm) is a marker of anaerobic infection.
- Acetate (1.92 ppm) is an important marker of infection.
- Lactate (1.3 ppm) is generated from anaerobic energy generation and can be found in both brain abscess and necrotic tumor and therefore is not useful marker of abscess.
- Lipids (0.8-1.2 ppm) can be found in both brain abscess and necrotic tumor.
- Amino acids (0.9 ppm) are a key marker of brain abscess. By using echo time of 135 ms, the resonance peaks of Lac and AA would invert due to J coupling whereas the Lip resonance peak do not invert due to uncoupled spins. This is important, because lipids can be found in necrotic tumor.

The presence of AA on vivo $^1$H MR spectroscopy is a sensitive marker of pyogenic abscess, its absence does not rule out an abscess.

**Case 3:** A 59 year-old man with necrotizing pneumonia that was treated with antibiotics. He presented with headache.

Sagittal T1-WI (fig. 24), Axial T2-WI (figs. 25 and 26) and coronal FLAIR image (fig. 27) (3T scans) show three abscess: right frontal, parietal and thalamic with a right frontal subdural empyema.
Axial DWI (figs. 28, 29 and 30) shows strong hyperintensity within the abscesses, which is consistent with restricted water diffusion. Note that subdural empyema is also hyperintense.

Axial ADC maps (figs. 31 and 32) show hypointensity, consistent restricted water diffusion. ADC value of thalamic abscess was $0.488 \times 10^3 \text{ mm}^2 /\text{s}$ and of the subdural empyema of $0.42 \times 10^3 \text{ mm}^2 /\text{s}$.

Coronal postcontrast T1 WI (fig. 33) shows a subdural empyema.

MRS (fig. 34) from the center of the lesion shows predominant lipid peak (1.3 ppm).

**Case 4:** A 60-year-old man with three nocardia brain abscesses.

Axial T2- WI (figs 35, 36 and 37) show a right frontal, a left occipital and a right cerebellum abscesses with perifocal edema.

Axial postcontrast T1-WI (figs. 38 and 39) show ring enhancement.

Nocardia infection is a bacterial infection that usually starts in the lungs. It may spread to other organ systems, most often the brain and the skin.

Axial DWI (figs 40, 41 and 42) shows hyperintensity and Axial ADC maps (figs. 43, 44 and 45) show low ADC.

MRS with a TE of 35 ms (Fig 46) and 144 ms (fig. 47) showing the presence of lipid and lactate.

**Case 5:** A 51-year-old man that was found unresponsive.

Axial T2-WI (fig. 48), axial DWI (fig. 49) and axial postcontrast T1-WI (fig. 50) show low signal lesion in the left frontal lobe on T1-WI and high signal on T2-WI and on DWI without enhancement, related to early cerebritis stage.

Although brain abscesses are potentially fatal, with mortality up to 40 %, they are treatable lesions. It is thus important to suggest infection in such cases.

There is a parafalcine and left fronto-parietal subdural empyemas. Axial DWI may help differentiate empyemas from other subdural collections, including chronic subdural hematoma, subdural effusion, and hygroma. DWI is very important to demonstrate the presence, extent, and complications of a subdural empyema.
Neurosurgery was performed and histopathologic examination confirmed the diagnosis an Amoeba: Naegleria Fowleri was isolated.

**Case 7:** A 58-year-old woman with colorectal cancer, with liver metastasis and peritoneal carcinomatosis was found to have fever and headache.

Axial T2 WI (fig. 51) shows three abscesses: left frontal, right parieto-occipital and in the corpus callosum. The abscesses are hyperintense with a hypointense wall, massive edema, and mass effect.

Axial T1-WI (Fig. 52) shows hypointense lesion with an isointense wall.

Axial postcontrast T1-WI (fig. 53) shows strong ring enhancement.

Axial DWI and ADC map (figs. 54 and 55) show hyperintensity in the cavity with low ADC. These features suggesting the diagnosis of brain abscesses.

Craniotomy was performed and pus was aspirated from the lesions. Pus culture showed Listeria.

In this case the differential diagnosis is with metastasis. Most necrotic brain tumors and cystic metastases showed low signal intensity on DWI, the value of the apparent diffusion coefficient is much higher than that of brain parenchyma. In necrotic tumor or cystic metastases, the cystic contents are less viscous, containing less inflammatory cells and more serous fluid, so the value of ADC is high. However the pus is a viscous fluid consists of inflammatory cells, mucoid protein, bacteria and necrotic tissue, so the value of ADC is low.

Recent studies have shown that restricted diffusion value is an important sign but is not specific for cerebral abscess. Restricted diffusion has been found in metastatic squamous cell carcinoma and radiation necrosis. The underlying pathophysiology is not known but probably due to sterile liquefaction necrosis. In sterile liquefaction necrosis, it may contain creamy pus like material with polymorphonuclear leukocytes.

**Case 9:** A 12-year-old woman with right otomastoiditis and fever.

Axial T2-WI (fig. 56), axial postcontrast T1-WI (fig. 57) and coronal postcontrast T1-WI (fig. 58) show a right temporal abscess surrounded by edema, there is a ring enhancement.

MR venography (fig. 59) demonstrates absent flow in the right transverse sinus and sigmoid sinus, related with a right transverse and sigmoid venous sinus thrombosis.
**Case 10:** A 43-year-old man with headache and facial palsy.

**Axial T2-WI** (fig. 60) shows a right frontal abscess with the characteristic hypointense rim, perifocal edema, and mass effect.

The abscess shows mixed intensity with a slightly hyperintense wall on axial T1-WI (fig. 61a). Axial postcontrast T1-WI (fig. 61b) shows ring enhancement.

Neurosurgery was performed and the abscess was confirmed. The brain abscess has an odontogenic origin.

**Case 11:** A 79-year-old woman with left hemiparesis.

Axial T2-WI (fig 62) shows a hyperintense lesion with a hypointense wall and perifocal edema.

Axial and coronal postcontrast T1-WI (figs. 63 and 64) show ring enhancement.

Axial DWI and axial ADC map (figs. 65 and 66) show a hyperintense lesion with a low ADC, indicating restricted diffusion.

**Case 12:** A 51-year-old woman asymptomatic with a history of treated arteriovenous malformation. A routine CT scan shows a left occipital lesion.

Axial T2-WI and axial FLAIR image (figs. 67 and 68) show a well-defined hyperintense lesion with a hypointense wall and perifocal edema.

Axial T1-WI (fig. 69) shows the lesion hyperintense with a hypointense wall.

Axial postcontrast T1-WI (fig. 70) shows ring enhancement.

Axial DWI and the ADC map (figs. 71 and 72) show restricted diffusion within the abscess cavity.

Multivoxel-short-echo MR proton spectra (figs. 73 and 74) show depression of the NAA and CR peaks, and elevation of the lactate and lipid peaks.

**Case 14:** A 74-year-old woman presented with fever, cough and chills.

Axial T2-WI (fig. 75) shows a right occipital abscess.
Coronal postcontrast T1-WI (fig. 76) show the two abscesses with ring enhancement.

Axial DWI (fig. 77) show restricted diffusion.

The abscesses were drained. Cultures of pus grew Klebsiella Pneumoniae. Despite the intervention, the patient died.

**Differential diagnosis:**

**Glioblastoma multiforme:**

- Thick and nodular wall.
- Wall isointense or hyperintense on T2-WI.
- Low signal on DWI and high ADC.
- Hemorrhage common.

A 43-year-old man with glioblastoma multiforme. It can be confused with an abscess on conventional MR sequences.

Axial T2-WI (fig. 78), axial T1-WI before and after contrast injection (figs. 79 and 80), coronal contrast-enhanced T1-WI (fig. 81) show a hyperintense mass on T2-WI, hypointense on T1-WI. Postcontrast T1-WI shows ring enhancement.

Axial DWI and ADC map (figs. 82 and 83) show low signal on DWI and high signal on ADC map, which is consistent with no diffusion restriction. The DWI may allow the differentiation of brain abscess from necrotic or cystic brain tumor.

A 63-year-old man with surgical proven left temporal glioblastoma multiforme.

Axial T2-WI (fig. 84), Axial and Coronal contrast-enhanced T1-WI (figs. 85 and 86), axial DWI and ADC map (fig. 87 and 88) show a left temporal glioblastoma multiforme with a thick and nodular wall in the inferior part, and with low signal on DWI and high ADC, so DWI is more useful than conventional MRI in providing a greater degree of confidence in distinguishing brain abscesses from cystic or necrotic brain tumors.

**Parenchymal metastases:**

- Thick-walled, centrally necrotic mass.
- Often multiple with marked edema.
- Low signal on DWI, rarely high signal and mimics abscess.

A 60-years-old woman with a metastatic adenocarcinoma. She had a history of treated lung adenocarcinoma, presented with left hemiparesis.

Axial T2 WI (fig. 89), axial FLAIR (fig. 90) show a lesion hyperintense with a hypointense wall and perifocal edema.

Axial and coronal after contrast injection (figs. 91 and 92) showing ring enhancement.

Axial DWI and ADC map (figs. 93 and 94) show high signal on DWI and low signal in the periphery and a small central area of high signal, which mimics an abscess, surgery and histopathologic examination, demonstrated a metastatic adenocarcinoma, underlying pathophysiology is not known but probably due to sterile liquefaction necrosis, it may contain creamy pus like material with polymorphonuclear leukocytes.

**Subacute cerebral infarction:**
- History of stroke.
- Vascular distribution.
- Gyriform enhancement.

**Resolving intracerebral hematoma:**
- History of trauma or vascular lesion.
- Blood products present on MR.

**Demyelinating disease:**
- Multiple sclerosis, ADEM.
- Ring enhancement often incomplete. Characteristic lesions elsewhere in brain.
- Masss effect small for size of lesion.

**Images for this section:**
**Table 1:**

- Vomiting
- Headache
- Disturbed consciousness
- Fever
- Focal neurologic deficits
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<th>Nº</th>
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<th>Clinical Findings</th>
<th>Location</th>
<th>Organism Treatment</th>
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**Table 2:** Table 2
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Fig. 86: Coronal postcontrast T1-WI
Fig. 87: Axial DWI
Fig. 88: Axial ADC map
Fig. 89: Axial T2-WI
Fig. 90: Axial FLAIR image.
Fig. 91: Axial postcontrast T1-WI
Fig. 92: Coronal postcontrast T1-WI
Fig. 93: Axial DWI.
Fig. 94: Axial ADC map
Conclusion

Cerebral abscess may sometimes mimic necrotic tumor and cystic metastasis. A definitive diagnosis is difficult just base on the conventional MR imaging. When DWI is coupled with MR spectroscopy, a correct diagnosis can be established quickly and noninvasively.

References


• Restricted Diffusion within ring enhancement is not pathognomonic for brain abcess. Marius Hartmann et al. AJNR 2001; 22: 1738-1742.


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Poster previously presented at SERAM 2010