

Clinics in Diagnostic Imaging (51)

J F Liu, H S Lam, J R Jenkins

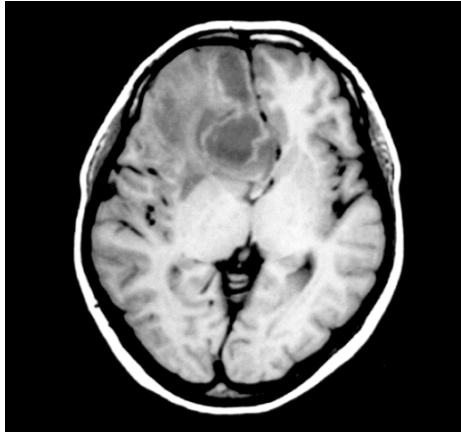


Fig. 1a Axial T1-weighted (TR/TE 570/14) MR image of the brain taken at the level of the corpus callosum.

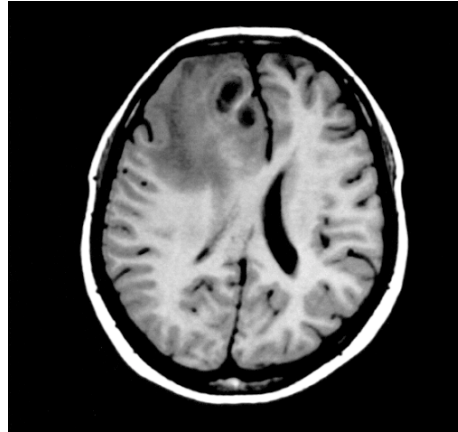


Fig. 1b Axial T1-weighted (TR/TE 570/14) MR image of the brain taken at the level of the lateral ventricles.

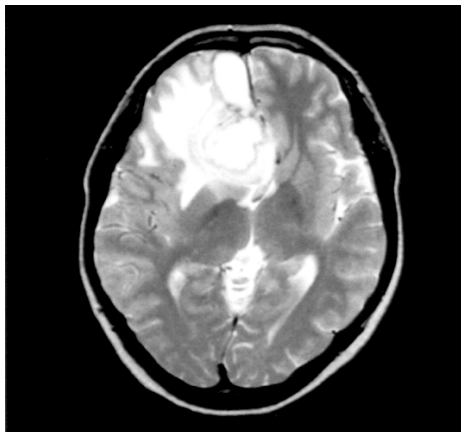


Fig. 1c Axial T2-weighted (TR/TE 3500/90) MR image of the brain taken at the same level as Fig. 1a.

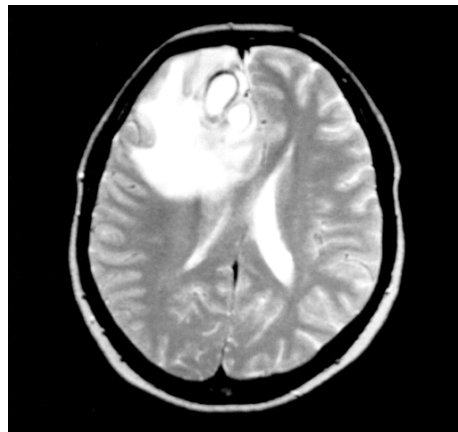


Fig. 1d Axial T2-weighted (TE/TE 3500/90) MR image of the brain taken at the same level as Fig. 1b.

CASE PRESENTATION

A 19-year-old woman was admitted for investigation of persistent headaches and intermittent fever. She was known to have Ebstein anomaly and a small ventricular septal defect. She first presented five weeks earlier with headache, mild fever and syncopal attack. A course of oral amoxicillin and phenytoin was given without improvement. Two weeks later, she was admitted to hospital with headache, fever of 38.4 degrees centigrade and shortness of breath. Apart from a mildly increased leucocyte count ($11.5 \times 10^9/L$) and a slightly elevated bilirubin level, the serum electrolytes and renal function

tests were normal. Blood and urine cultures were also negative. Her fever promptly subsided after a five-day course of intravenous ceftriaxone. The patient was discharged without further investigation. At the present hospital admission, the patient was found to be febrile (37.5 degrees centigrade) and cyanosed. Clubbing of the fingers was present. No focal neurological deficit was detected. Full blood count revealed elevated leucocyte levels ($16.9 \times 10^9/L$). Liver and renal function tests were normal.

What does magnetic resonance (MR) imaging of the brain (Figs. 1a-d) show? What is the diagnosis?

Department of
Diagnostic
Radiology
Kwong Wah Hospital
22 Waterloo Road
Hong Kong

J F Liu, MBBS,
FRCR, FHKCR
Senior Medical Officer

H S Lam, MBBS,
FRCR, FHKCR
Chief of Service

Department of
Radiology
University of
Nebraska
Omaha, USA

J R Jenkins, MD,
FACR
Professor and Director
of Neuroradiology

Correspondence to:
Dr J F Liu
Tel: (852) 278-15484
Fax: (852) 278-15199

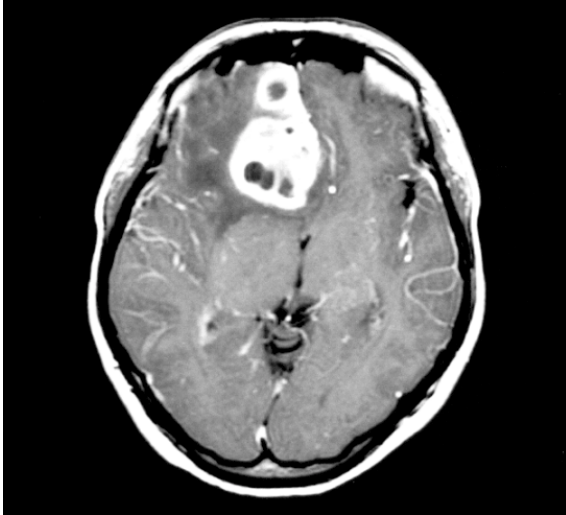


Fig. 2a Axial enhanced T1-weighted (TR/TE 570/14) MR image taken at the same level as Figs. 1a & 1c show multiple ring-enhancing lesions in the right frontal lobe. The multiloculated lesion is located anterior to the corpus callosum.

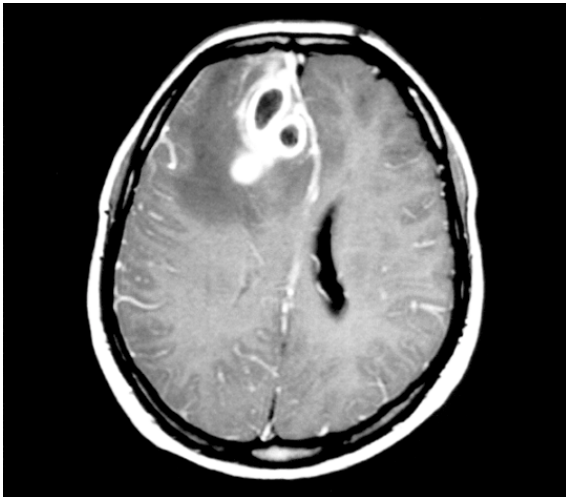


Fig. 2b Axial enhanced T1-weighted (TR/TE 570/14) MR image taken at the same level as Figs. 1b & 1d show the multiloculated abscess causing mass effect and subfalcine herniation.

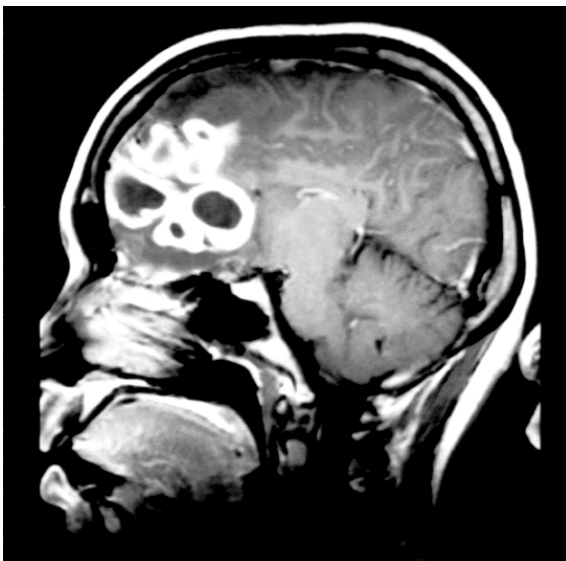


Fig. 3 Right para-sagittal enhanced T1-weighted (TR/TE 570/14) MR image demonstrates the multiloculated nature of the frontal lobe lesion and multiple adjacent satellite lesions.

IMAGE INTERPRETATION

The axial T1-weighted MR scans (Figs. 1a-b) showed a multiloculated hypointense lesion, with a slightly hyperintense rim, in the right frontal lobe. There was associated mass effect, with white matter oedema and subfalcine herniation. The oedema was better seen on the axial T2-weighted images (Figs. 1c-d) as hyperintense regions which were separated by a hypointense rim from the lesion centres. These also appeared hyperintense in signal intensity. In addition, a T2-hypointense concentric ring was noted in the centre of the lesion. Multiple ring-like areas of enhancement were seen on the axial enhanced MR scans (Figs. 2a-b). The multiloculated nature of the lesion and adjacent satellite lesions were better appreciated on the sagittal enhanced MR scan (Fig. 3). The imaging features are consistent with multiple multiloculated right frontal lobe abscesses.

DIAGNOSIS

Multiloculated cerebral abscesses due to paradoxical cardiac emboli.

CLINICAL COURSE

A semi-urgent craniotomy was performed two days following admission and purulent pus was drained. No micro-organism was cultured, probably due to the previous antibiotic therapy. Post-operatively, the patient was recovering well on triple antibiotic therapy that included ceftriaxone, penicillin and metronidazole. However 10 days later, her fever recurred and blood studies showed a decrease in the leucocyte count ($1.8 \times 10^9/L$). Drug-induced neutropenia was diagnosed. Phenytoin was discontinued and the antibiotic regimen was changed to rifampicin and ciprofloxacin. Repeat blood studies a week after the antibiotic alteration showed a normal leucocyte level ($4 \times 10^9/L$). The patient made a complete clinical recovery and was discharged five weeks after admission. No recurrence of the abscess was seen on follow-up computed tomography (CT) performed three months later (Figs. 4a-b). The CT scan only showed changes of encephalomalacia in the right frontal lobe.

DISCUSSION

Brain abscess is due to advanced focal intraparenchymal infection. Infectious agents may reach the central nervous system (CNS) via a number of pathways. These include direct extension from local infections of the paranasal sinuses, retromastoid air cells and middle ear cavities, or via a track due to a breach of the dura mater such as penetrating skull vault injury, surgery or a dermal sinus track. Alternatively, a brain abscess can result from systemic dissemination with bacteraemia due to

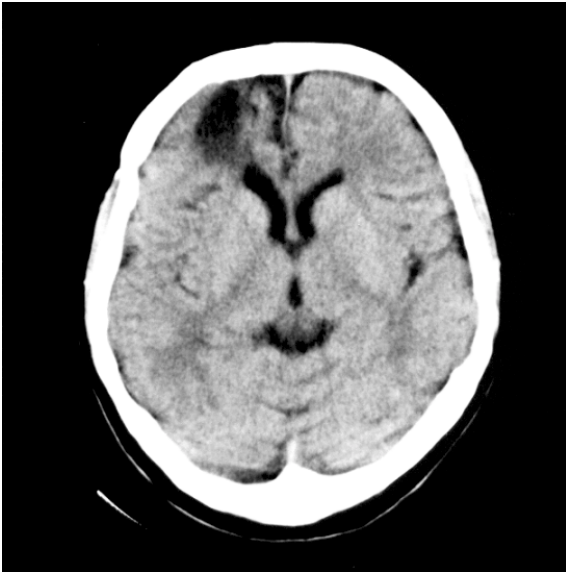


Fig. 4a Axial unenhanced CT scan of the brain at 3 months follow-up shows only a hypodense area in the right frontal lobe.



Fig. 4b Axial enhanced CT scan of the brain taken at the same level as Fig. 4a shows no contrast enhancement at the previous site of the abscess. Changes are that of encephalomalacia.

peripheral non-CNS infections, or from septic emboli in patients with peripheral arteriovenous shunts or right-to-left cardiac shunts (eg. congenital cyanotic heart diseases)⁽¹⁻³⁾. In about 20% of cases, no obvious source or focus of infection is found⁽⁴⁾.

The common causative organisms include *Staphylococcus*, *Streptococcus* and anaerobic bacteria such as *Bacteroides*. Following initial infection of the brain, there is resultant cerebritis with acute inflammatory response and oedema. As the infection progresses, formation of a capsule, with central liquefactive necrosis and moderate to marked adjacent cerebral oedema, occurs.

The capsule becomes more distinct as the abscess matures⁽⁵⁾. Clinically, patients with brain abscesses often present with non-specific symptoms such as headache, nausea and vomiting. Changes of consciousness and focal neurological deficits are less common. Leucocyte counts and C-reactive protein levels may be elevated. However, only up to 50% of patients are febrile at presentation⁽⁶⁾. Therefore, imaging plays a vital role in the early diagnosis and management of brain abscesses.

On CT scans, the early cerebritis stage of parenchymal CNS infection may be seen as areas of hypodensity with patchy or gyriform types of enhancement. Later, the capsule appears on unenhanced CT as a rim which is of higher density than the central hypodense cavity⁽⁷⁾. The abscess capsule usually has a thin smooth margin except for the deep wall which may appear thinner. This is attributed to the relatively-poor vascularity of the deep white matter and is seen in up to 50% of cases⁽⁸⁾. There may also be adjacent satellite or daughter abscesses.

The early cerebritis stage appears on MR imaging as an area of T1-hypointense and T2-hyperintense signal intensity, with patchy or gyriform enhancement, similar to CT findings. In the capsular stage, part or all of the abscess wall may demonstrate both T1 and T2 shortening. As a result, the capsule may have a hyperintense rim on T1-weighted images and a hypointense rim on T2-weighted images. The proposed mechanism for this phenomenon include the presence of blood products in the capsule wall or alternatively, activated macrophages present in the capsule that may produce free radicals, causing this paramagnetic susceptibility effect⁽⁹⁾.

Following contrast administration, MR imaging demonstrates regular thickness ring enhancement of the capsule surrounding a central necrotic core which is T1-hypointense and T2-hyperintense. The extent of the cerebral oedema is better seen on the T2-weighted scans. There may also be concentric rings of variable signal intensity and thickness within the abscess cavity. This feature is usually not seen in necrotic or cystic tumours⁽⁹⁾. The exact mechanism producing this MR appearance is unknown at the present time.

The imaging features of ring enhancement with central necrosis can also be seen in necrotic or cystic tumours. However, with brain abscesses, the capsule is usually smooth and thin, and of nearly-uniform thickness. The presence of medial (ie. deep) wall thinning is highly suggestive of a brain abscess⁽⁷⁾. Furthermore, the wall of necrotic or cystic tumours is seldom hyperintense on T1 and hypointense on T2-weighted MR images. In fact, the presence of a hypointense rim on T2-weighted images is regarded as

being characteristic of brain abscess⁽⁹⁾. Another characteristic feature of abscess is the presence of satellite or daughter abscesses adjacent to the primary lesion. In addition, the presence of multiple concentric rings within the abscess cavity is indicative of brain abscess⁽⁹⁾.

To further enhance the differentiation between brain abscesses, and necrotic or cystic tumours, H-1 MR spectroscopy and diffusion MR scans have shown promising preliminary results. With the use of H-1 MR spectroscopy, metabolic constituents of body tissues can be analysed. Kim et al and Remy et al have shown differences in the spectroscopic patterns between brain abscesses, and necrotic or cystic tumours^(10,11). With brain abscesses, there are resonant peaks due to lactate (1.3 ppm), alanine (1.5 ppm), acetate (1.92 ppm), the amino acids, valine and leucine (0.9 ppm), and succinate (2.4 ppm). In necrotic or cystic tumours, only increased lactate (1.92 ppm) and lipid (0.9 ppm) peaks are present. In diffusion MR imaging, brain abscesses appear hyperintense in signal intensity centrally (ie. have decreased diffusion) whereas necrotic or cystic tumours are hypointense (ie. have increased diffusion)⁽¹²⁾.

With early diagnosis and prompt treatment of brain abscesses, the mortality rates have dropped dramatically from 40-50% to less than 5%⁽⁷⁾. The treatment usually consists of a combination of antibiotic therapy, and surgical drainage or excision. However, antibiotics combined with close image monitoring may be sufficient for successful treatment of small lesions. The use of corticosteroids to decrease oedema is a controversial therapeutic adjunct. Corticosteroids may decrease the inflammatory and glial response of the brain and alter the amount of ring enhancement by re-sealing the blood-brain barrier⁽⁶⁾. A cured (ie. sterile) brain abscess may continue to exhibit contrast enhancement on CT or MR images for up to nine months⁽¹³⁾. Eventually, there may be no sequelae, mild encephalomalacia or rarely, calcification at the healed abscess site. In conclusion, patients with brain abscesses may present with typical clinical features in only 50% of cases. Therefore, the use of CT and MR imaging have and will continue to play an important role in the early diagnosis and monitoring of the treatment progress in patients with brain abscesses.

REFERENCES

- Jenkins JR, Leite CD. Differential diagnosis of solitary, peripherally enhancing supratentorial intracranial lesions. In: Jenkins JR, Leite CD, editors. Neurodiagnostic imaging: pattern analysis and differential diagnosis. Philadelphia: Lippincott-Raven. 1998: 233-45.
- Sze G, Lee SW. Infectious diseases. In: Lee SW, Rao KCVG, Zimmerman RA, editors. Cranial MRI and CT. New York: McGraw-Hill. 1992; 546-52.
- Desal SP, Rees C, Jenkins JR. Paradoxical cerebral emboli associated with pulmonary arteriovenous shunts: report of three cases. AJNR 1991; 12:355-9.
- Kerr FWL, King RB, Mearher JN. Brain abscess: A study of 47 consecutive cases. JAMA 1958; 168:868-72.
- Magalhaes ACDA, Bacheschi LA. Viral and nonviral infections of the nervous system. In: Greenberg JO, eds. Neuroimaging: A companion to Adams and Victor's Principles of Neurology. New York: McGraw-Hill. 1995: 393-8.
- Scheld WM. Bacterial meningitis, brain abscess and other suppurative intracranial infections. In: Fauci AS, Braunwald E, Isselbacher KJ, Wilson JD, Martin JB, Kasper DL et al, editors. Harrison's Principles of Internal Medicine. New York: McGraw-Hill Inc. 1998: 2419-34.
- Whelan MA, Hilal SK. Computed tomography as a guide in the diagnosis and follow-up of brain abscesses. Radiology 1980; 135:663-71.
- Stevens EA, Norman D, Kramer RA, Messina AB, Newton TH. Computed tomographic brain scanning in intraparenchymal pyogenic abscesses. AJR 1978; 130:111-4.
- Haimes AB, Zimmerman RD, Morgello S, et al. MR imaging of brain abscesses. AJNR 1989; 10:279-91.
- Kim SH, Chang KH, Song IH, et al. Brain abscess and brain tumor: Discrimination with in vivo H-1 MR spectroscopy. Radiology 1997; 204:239-45.
- Remy C, Grand S, Lai ES, et al. 1H MRS of human brain abscesses in vivo and in vitro. Magn Reson Med 1995; 34:508-14.
- Kim YJ, Chang KH, Song IC, et al. Brain abscess and necrotic or cystic brain tumors: discrimination with signal intensity on diffusion weighted MR imaging. AJR 1998; 171:1487-90.
- Zimmerman RD, Weigarten K. Neuroimaging of cerebral abscesses. Neuroimaging Clinics North America. 1991; 1:1-16.

ABSTRACT

A 19-year-old woman with congenital cyanotic heart disease (Epstein anomaly and ventricular septal defect) had persistent headaches and intermittent fever for five weeks. Physical examination revealed central cyanosis, finger clubbing and fever. The leucocyte count was elevated. Cerebral MR imaging showed the characteristic features of brain abscesses in the right frontal lobe, including multiloculation with adjacent satellite lesions, ring enhancement, T1-hyperintense and T2-hypointense signal areas within the abscess rim, as well as hypointense internal concentric rings on T2-weighted images. The diagnosis of brain abscesses was confirmed by craniotomy and pus drainage. She made a good recovery with a combination of antibiotics and surgery. Follow-up CT scans showed only changes of encephalomalacia at the healed abscess site. The aetiology, clinical features, and the role of CT and MR imaging in the diagnosis and management of brain abscess are discussed.

Keywords: Brain abscess, Central nervous system, Infections, Computed tomography, Magnetic resonance imaging, Paradoxical cardiac embolus

Singapore Med J 2000 Vol 41(7):363-366