ROLE OF SPECIAL MR TECHNIQUES IN DISCRIMINATION BETWEEN CAPSULAR STAGE BRAIN ABSCESSES, NECROTIC AND CYSTIC BRAIN LESIONS

THESIS SUBMITTED FOR THE PARTIAL FULFILLMENT OF THE MD DEGREE IN RADIODIAGNOSIS

BY
KAREEM MOHSEN MOUSSA
M.B.B.CH., M.SC.
FACULTY OF MEDICINE, CAIRO UNIVERSITY

SUPERVISED BY

DR. IHAB ISMAIL ALI PROFESSOR OF RADIODIAGNOSIS FACULTY OF MEDICINE CAIRO UNIVERSITY

DR. AMR MAHMOUD SAFWAT PROFESSOR OF NEUROSURGERY FACULTY OF MEDICINE CAIRO UNIVERSITY

DR. MOHAMED ABD-ELFATTAH HASSAAN ASSISTANT PROFESSOR OF RADIODIAGNOSIS FACULTY OF MEDICINE CAIRO UNIVERSITY

FACULTY OF MEDICINE CAIRO UNIVERSITY 2012

ACKNOWLEDGEMENTS

First and foremost, thanks to Allah, the most beneficial and most merciful.

I am greatly indebted to Dr. Ihab Ismail, Professor of Radiology, Cairo University, I am honored and pleased to have had the opportunity to learn from his creative advices and expanded experience. His constant support, encouragement and willingness to teach and educate have pushed me forward throughout this work.

I wish to thank Dr. Amr Safwat, Professor of Neurosurgery, Cairo University, whose supervision, great assistance and precious advices have been of great help in presenting this work.

I am equally grateful to Dr. Mohamed Abd-Elfattah, Assistant Professor of Radiology, Cairo University, for his excellent supervision, sincere encouragement, valuable criticism, enlightening suggestion and kind guidance throughout the whole work.

To my fellow colleagues in the Radiology department, I would like to say "thank you all" for all the efforts you have put and for your support. Without you this work would have never come to life.

ABSTRACT

Brain abscesses and brain tumours may have similar clinical presentations. Also, the differential diagnosis of brain abscesses versus cystic or necrotic tumours may be difficult based on computed tomography or magnetic resonance (MR) imaging findings. However, the strategies of management for abscess and neoplasm are very different, and it is especially imperative to have a correct diagnosis before any surgical intervention of cystic brain lesions. The MR special techniques, e.g. diffusion-weighted imaging (DWI) and MR spectroscopy (MRS), are useful as additional diagnostic modalities for differentiating brain abscesses from cystic or necrotic brain tumours. DWI shows high signal intensity in most cases of cystic or necrotic tumours. MRS shows characteristic metabolites in pyogenic abscesses, distinct from those in cystic or necrotic tumours.

Key words

- Brian abscess
- Cystic or necrotic brain tumours
- Magnetic resonance imaging
- Diffusion weighted images
- Apparent diffusion coefficient
- Magnetic resonance spectroscopy

CONTENTS

	Page
Introduction	1
Review of Literature	
Basic physical principles of diffusion weighted images	4
Basic physical principles of magnetic resonance spectroscopy	20
In vivo MR spectroscopy	34
Role of diffusion weighted imaging and MR spectroscopy in differentiating brain abscess from necrotic and cystic brain tumours	58
Patients and Methods	81
Results	88
Case Presentation	98
Discussion	143
Summary and Conclusion	155
References	160

LIST OF FIGURES

Basic physical principles of diffusion weighted images

Figure 1. (A) Water molecules travel by "random walk" more freely than (B), as the freedom of this movement is reduced by barriers as cell membranes. The diffusion in (B) is restricted as compared with (A). Finally, ADC (B) is less than ADC (A) (de Figueiredo et al., 2011) 5
Figure 2. The bundle offers no resistance to water molecules in the diffusion direction parallel to the fibers but there is a severe restriction if perpendicular. In this case there is preferred water molecule direction due to anisotropy. Outside the bundle, the water molecules are in an isotropic environment and have no preferred direction (<i>de Figueiredo et al.</i> , 2011)
Figure 3. Stejskal-Tanner Scheme: 2 diffusion-sensitizing gradients inserted before and after 180° RF refocusing pulse using precisely controlled duration and distance. G, amplitude; δ , duration of the sensitizing gradient; Δ , time between the 2 sensitizing gradient lobes (<i>Le Bihan et al.</i> , 1968).
Figure 4. The diffusion sensitization gradients were applied in directions SI (B), RL (C), and AP (D). Image (A) is the average of (B), (C), (D), and is usually denominated combined or "isotropic" image. The white arrow points to splenium of corpus callosum that has restricted diffusion in the SI direction, is facilitated in the RL direction, and has mixed pattern in the AP direction. Combined image (A) minimizes the anisotropy effects of the individual images (de Figueiredo et al., 2011).
Figure 5. Example of T2 shine-through effect and correction using exponential and parametric ADC maps: White arrow indicates bright signal on T2 image (A), isointense signal on diffusion image (B). The exponential map (C) has the T2 shine-through artifact removed and the expected low signal of facilitated diffusion is present. The parametric ADC map (D) demonstrates high ADC value as a bright region and enables quantification of ADC (de Figueiredo et al., 2011).
Figure 6. T2 shine-through in a 35-year-old female with multiple sclerosis and weakness of the lower extremities. (A) T2-weighted image shows several hyperintense lesions, with the largest one in the right frontal lobe (arrow). (B) On T1-weighted image the lesion was hypointense (arrow). (C) On DW image the lesion is hyperintense (arrow). (D) ADC map also shows hyperintensity in the lesion $(1.2\times10^{-3} \text{ mm}^2/\text{s}; \text{ arrow})$. (E) Exponential image eliminates the T2 effect and shows the lesion to be hypointense (arrow). This confirms that the hyperintensity on DW image is due to a T2 shine-through (<i>Moritani et al.</i> , 2005)
Figure 7. T2 shine-through and restricted diffusion in a 56-year-old male with right-sided weakness due to acute infarction. MR imaging obtained 24 hours after the onset of symptoms. (A) FLAIR image shows a hyperintense lesion in the left middle cerebral artery territory. (B)

On T1-weighted image the lesion is hypointense. (C) On T2-weighted image (b=0) the lesion

is hyperintense. (D) DW image also shows hyperintensity in the lesion. (E) ADC map shows hypointensity in the lesion $(0.27-0.45\times10^{-3}\text{mm}^2/\text{s})$. (F) On the exponential image, which eliminates the T2 effect, the lesion remains hyperintense. This confirms that the DW
hyperintensity is due to both restricted diffusion and T2 prolongation (<i>Moritani et al.</i> , 2005).
Figure 8. T2 washout in a 45-year-old female with hypertension, seizures and posterior reversible encephalopathy syndrome. (A) FLAIR image shows hyperintense lesions in the bilateral occipital lobe (arrows). (B) T2-weighted image (b=0) also shows hyperintensity of the lesions (arrows). (C) DW image shows mild hyperintensity in the lesions. (D) ADC map shows hyperintensity of the lesions (1.18–1.38×10 ⁻³ mm ² /s; arrows). With the strong T2 prolongation one would expect more hyperintensity on the DW image, but the T2 shine-through effect is reduced by the hyperintensity on the ADC, resulting in a balance between increased diffusibility and hyperintensity on the T2-weighted image (T2 wash-out) (<i>Moritani et al.</i> , 2005)
Figure 9. T2 blackout in lung cancer metastasis in a 62-year-old male with adenocarcinoma of the lung. (A) T2-weighted image shows a hypointense mass (arrow) with surrounding edema in the left cerebellar hemisphere. (B) Gadolinium-enhanced T1-weighted image shows heterogeneous enhancement of the mass (arrow). (C) T2-weighted image (b=0) also shows hypointensity in the lesion with surrounding hyperintense edema (arrow). (D) ADC map shows central hyperintensity (1.63–2.35×10 ⁻³ mm ² /s; arrowhead) and peripheral hypointensity (1.13–1.38×10 ⁻³ mm ² /s; arrow) of the mass. There is also hyperintensity of the surrounding tissue, consistent with vasogenic edema. (E) DW image shows heterogeneous hypointensity of the mass (arrow) and isointensity of the surrounding edema. The DW hypointensity of the mass (arrow) is due to the increased diffusibility and hypointensity on T2-weighted image. The isointensity in the surrounding edema is due to the balance between the increased diffusibility and hyperintensity on T2-weighted image (T2 washout) (<i>Moritani et al.</i> , 2005).
Basic physical principles of magnetic resonance spectroscopy
Figure 10. Proton MR spectrum (Alger, 2004)
Figure 11. Common pulse sequences for single-voxel localization techniques. (A) Spatial localization is achieved by collecting signals from the intersection of three slice-selective RF pulses applied in orthogonal directions. (B) The STEAM sequence, consisting of three 90° slice selective pulses. (C) The PRESS sequence, consisting of a slice selective 90° excitation pulse and two 180° refocusing pulses (<i>Barker et al.</i> , 2009).
Figure 12. A 2D-PRESS-MRSI scan in the coronal plane of a patient with a lesion in the left hippocampus (visualized on T2 MRI) recorded at 1.5 T. The lesion can be seen to have a low NAA signal and elevated choline (and creatine) on both metabolic images and selected MRSI spectra from regions of interest in the left and right hippocampi, most likely consistent with a neoplastic process. The alternative diagnosis, mesial temporal sclerosis, rarely shows increased choline levels (Parken et al., 2000)
increased choline levels (Barker et al., 2009).

PRESS box is chosen that is angulated parallel to the long-axis of the temporal lobe, at the level of the hippocampus. Four saturation bands (indicated in blue) are used to suppress out of voxel signal. Phase-encoding is applied in the transverse plane (voxel size ~1 cm³). Spectra are recorded at 3 T (TR 2000, TE 140 msec). Note strong regional variations in pons, hippocampus, cerebellar vermis, and occipital white matter (<i>Barker et al.</i> , 2009)
Figure 14. Schematic representation of the location of the OVS pulses, forming an octagonal cone in order to conform to the contours of the skull. The four oblique axial MRSI slices are also represented on the sagittal schematic (<i>Duyn et al.</i> , 1993)
Figure 15. Example data from one slice (at the level of the lateral ventricles) of a multi-slice 2D-MRSI data from a normal human subject recorded at 1.5 T. In addition to the anatomical MRI scan, spectroscopic images of choline, creatine, N-acetylaspartate, and lactate are shown, and selected spectra (showing regional variations) from various white and gray matter regions within the brain (<i>Barker et al.</i> , 2009)
Figure 16. Multi-slice MRSI protocol covering the cerebellum and brain stem. Slice locations are chosen to avoid exciting unwanted signal in the clivus, oral cavity, and sinuses. Metabolic images show high levels of creatine and choline in the cerebellum (<i>Barker et al.</i> , 2009) 31
In vivo MR spectroscopy
Figure 17. Effects of metal artifacts and air—tissue interfaces on field homogeneity in the brain, in a 2-year-old female with prior resection for primitive neuroectodermal tumour (PNET). (A) Maps of magnetic field show inhomogeneities above the sphenoid sinus and auditory canals (open arrows), as well as major local field inhomogeneities caused by surgical staples applied during craniotomy (closed arrows). (B) Multi-voxel spectra (TR/TE 2300/280 msec) have poor quality in these regions compared to others (e.g. mid-brain) (<i>Barker et al.</i> , 2009)
Figure 18. Spectra obtained before and after the intravenous administration of contrast medium of an enhancing lesion (sub-acute infarct) at TE 30, 144, and 288. Spectra are dominated by lactate, as well other signals from Glu, creatine, and choline. Within the SNR of these scans, post-contrast spectra are not significantly different relative to pre-contrast (<i>Barker et al.</i> , 2009).
Role of diffusion weighted imaging and MR spectroscopy in
differentiating brain abscess from necrotic and cystic brain tumours
Figure 19. 61-Year-old patient with an abscess in the right frontal lobe. The abscess shows the typical image pattern with a large central cyst on conventional T2 weighted (A) and ring enhancement on post-contrast T1 weighted image (B). The abscess cavity is hyperintense on DWI scan (C) and shows restricted diffusion on ADC map (D) (<i>Reiche et al.</i> , 2010)
Figure 20. 68-Year-old patient suffering from glioblastoma on the left side. Conventional MR images (T2 weighted (A) and post-contrast T1 weighted (B)) show a tumour with a central cyst, ring enhancement, a small contrast enhancing satellite structure and perifocal edema.

Figure 13. 2D-PRESS MRSI for the bilateral evaluation of temporal lobe metabolism. A

The tumour cyst is hypointense in DWI sequence (C) and reveals no signs of diffusion restriction on ADC map (D) (<i>Reiche et al., 2010</i>)
Figure 21. Images obtained in a 50-year-old man with surgically proven brain abscess in the right basal ganglion. (A) Axial T1-weighted image (500/30) before administration of contrast material. (B) Axial T2-weighted image (4000/100). The 2x2x2 cm voxel (box) in the center of the lesion represents the MRS volume of interest. (C) Axial contrast-enhanced T1-weighted (500/30) MR image shows a ring-shaped cystic lesion and surrounding edema. (D) Axial diffusion-weighted (10,000/93; b=1000 s/mm²) image shows marked hyperintensity in the abscess cavity and slight iso- to hypointensity surrounding the edema. (E) ADC map reveals hypointensity in the abscess cavity, representing restricted diffusion, and hyperintense areas surrounding the edema. (F and G) In vivo spectra (2000/270 and 135) from the abscess cavity show resonances representing acetate (Ac), alanine (Ala), lactate (Lac), and amino acids (AA). At a TE of 135 (G), the phase reversal resonances are well depicted at 1.5, 1.3, and 0.9 ppm, which confirms the assignment to alanine, lactate, and amino acids, respectively (<i>Lai et al.</i> , 2002).
Figure 22. Images obtained in a 67-year-old man with a pathologically proven right cerebellar metastasis from primary lung adenocarcinoma. (A) Axial T1-weighted image (500/30) before administration of contrast material. (B) Axial T2-weighted image (4000/100). The 2x2x2 cm voxel (box) in the center of the lesion represents the MRS volume of interest. (C) Axial contrast-enhanced T1-weighted (500/30) MR image shows a ring-enhanced lesion in the right cerebellum. (D) Axial diffusion-weighted (10,000/93; b=1000 s/mm²) image shows markedly low signal intensity in the necrotic part of the tumour. (E) ADC map reveals high signal intensity in the necrotic part of the tumour that is similar to that of CSF, reflecting marked diffusion. (F and G) In vivo spectra (2000/270 and 135) from the necrotic center of the tumour show a lactate (Lac) peak at 1.3 ppm that is inverted at a TE of 135 (<i>Lai et al.</i> , 2002).
Figure 23. Representative in vivo MR images and spectra from patients with cerebral abscesses caused by <i>S aureus</i> and GBM. MR images and spectra were acquired with clinical 1.5-T MR imagers. (A-C) patients with <i>S aureus</i> brain abscess and (D) a patient with a cystic GBM. Transverse T2-weighted MR images (2200/80) are shown in the top row. MR spectra were acquired from the volume of interest centered within the lesions and outlined on the images. Middle row: stimulated-echo acquisition sequence (3000/20 or 30). Bottom row: point-resolved MRS sequence (3000/135). Volume of interest, 8–12 cm ³ . MR spectra from all staphylococcal abscesses lacked N-acetylaspartate and were dominated by resonances from lipids and lactate, similar to spectra from patients with GBM. The presence of lactate was confirmed with phase inversion in MR spectra acquired with an echo time of 135 msec. No resonances arising from acetate, succinate, or amino acid were detected (<i>Himmelreich et al.</i> , 2005)
Figure 24. Images obtained 35 days after the start of initial antibiotic treatment in a 45-year-old man with multiple pyogenic brain abscesses. (A) Axial T1-weighted image (500/30) before administration of contrast material. (B) Axial T2-weighted image (4000/100). The 2x2x2 cm voxel (box) represents MRS volume of interest. (C) Axial contrast-enhanced T1-weighted (500/30) MR image shows two ring-shaped enhanced lesions in the right basal ganglion and left frontal lobe. (D) Axial diffusion-weighted (10,000/93; b=1000 s/mm²)

Figure 28. A 67-year-old man with a pathologically proven left occipital metastasis from primary lung adenocarcinoma. (A) Axial T2-weighted image (4000/100). (B) Contrastenhanced T1-weighted MR image show a ring-enhanced lesion in left occipital lobe. The box in the center represents the volume of interest. (C) Diffusion-weighted imaging shows markedly low signal intensity in the necrotic part of the tumour. (D) Apparent diffusion coefficient map reveals high signal intensity in the necrotic part of the tumour, reflecting marked diffusion. The ADC value was 2.83×10^{-3} mm ² /s. (E and F) In vivo MRS spectra (2000/270 and 135) from the necrotic center of the tumour show a lactate (Lac) peak at 1.3 ppm that is inverted at a TE of 135 (<i>Lai et al.</i> , 2007).
Figure 29. A 63-year-old woman with a pathologically proven left occipital metastasis from primary colon adenocarcinoma. (A) Coronal T2-weighted image (4000/100). (B) Contrastenhanced T1-weighted MR image show a ring-shaped enhanced lesions in left occipital lobe. The box in the center represents the volume of interest. (C) Axial DWI shows markedly high signal intensity in the necrotic cavity and isointense surrounding edema. (D) Apparent diffusion coefficient map reveals low signal intensity in the necrotic cavity representing restricted diffusion and hyperintense surrounding edema. The ADC value was 0.54×10^{-3} mm ² /s. (E and F) In vivo MRS spectra (2000/270 and 135) from the necrotic cavity show a lactate (Lac) peak (1.3 ppm) that is inverted at a TE of 135 and a lipid (Lip) peak (0.9-1.2 ppm). Surgery revealed viscous creamy material similar to thick pus (<i>Lai et al.</i> , 2007) 80
<u>Results</u>
Figure 30. Distribution of the patients in the tumour group according to their final diagnosis.
Figure 31. Signal intensity in DWI
Figure 32. Line chart of the distribution of the ADC values of all patients in both groups 92
Figure 33. Line chart of the distribution of the Cho/Cr ratios of all patients in both groups 94
Figure 34. Line chart of the distribution of the Cho/NAA ratios of all patients in both groups.
Figure 35. Line chart of the distribution of the NAA/Cr ratios of all patients in both groups. 96
Figure 36. Sensitivity, specificity, PPV, and NPV of DWI and MRS
<u>Case Presentation</u>
<u>Case 1</u>
Figure 37. Axial T1 WIs showing right temporoparietal multiloculated cystic lesion 99
Figure 38. Axial T2 WIs showing right temporoparietal multiloculated cystic lesion 100
Figure 39. Contrast enhanced axial T1 WIs showing marginal contrast uptake and non-enhancing central cystic contents

Figure 40. Contrast enhanced coronal T1 WIs showing marginal contrast uptake and non- enhancing central cystic contents	102
Figure 41. DWI showing hypointense signal denoting facilitated diffusion	102
Figure 42. ADC showing hyperintense signal denoting facilitated diffusion	102
Figure 43. Short TE MRS showing choline and lipid peaks	103
Figure 44. Long TE MRS showing choline peak	103
Case 2	
Figure 45. Axial T1 WIs showing left parietal cystic lesion	105
Figure 46. Axial T2 WIs showing left parietal cystic lesion	105
Figure 47. Axial FLAIR WIs showing left parietal cystic lesion	105
Figure 48. Coronal T2 WIs showing left parietal cystic lesion	106
Figure 49. Contrast enhanced axial T1 WIs showing irregular marginal contrast uptake and non-enhancing central cystic contents	
Figure 50. Contrast enhanced coronal T1 WIs showing irregular marginal contrast uptake a non-enhancing central cystic contents	
Figure 51. DWI showing hypointense signal denoting facilitated diffusion	107
Figure 52. ADC showing hyperintense signal denoting facilitated diffusion	107
Figure 53. Short TE MRS showing choline and lactate peaks	107
Figure 54. Long TE MRS showing choline and lactate peaks	108
Case 3	
Figure 55. Axial T1 WIs showing multiple hypointense lesions	110
Figure 56. Axial T2 WIs showing multiple hyperintense lesions	110
Figure 57. Contrast enhanced axial T1 WIs showing irregular marginal contrast uptake and non-enhancing central necrotic part.	
Figure 58. Contrast enhanced coronal T1 WIs showing irregular marginal contrast uptake a non-enhancing central necrotic part.	
Figure 59. Contrast enhanced sagittal T1 WIs showing irregular marginal contrast uptake an non-enhancing central necrotic part.	
Figure 60. DWI showing hypointense signal denoting facilitated diffusion	112

Figure 61. ADC showing hyperintense signal denoting facilitated diffusion	. 113
Figure 62. Short TE MRS showing choline and lipid peaks.	. 113
Figure 63. Long TE MRS showing choline peak.	114
Case 4	
Figure 64. Axial T2 WIs showing left temporoparietal hyperintense lesion	. 116
Figure 65. Contrast enhanced axial T1 WIs showing irregular marginal contrast uptake ar non-enhancing central necrotic part.	
Figure 66. Contrast enhanced sagittal T1 WIs showing multiloculated cystic marginally enhancing lesion and non-enhancing central necrotic part	116
Figure 67. Contrast enhanced coronal T1 WIs showing multiloculated cystic marginally enhancing lesion and non-enhancing central necrotic part	117
Figure 68. Left: DWI showing hypointense signal and right: ADC showing hyperintense signal denoting facilitated diffusion.	117
Figure 69. Intermediate TE MRS showing choline peak.	117
Figure 70. Long TE MRS showing large choline and smaller lipid/lactate peaks	118
<u>Case 5</u>	
Figure 71. Left: axial T1 WIs showing right temporal hypointense lesion. Right: axial T2 showing right temporal hyperintense lesion.	
Figure 72. Contrast enhanced axial T1 WIs showing non-uniform marginal contrast uptak and non-enhancing central necrotic contents	
Figure 73. Contrast enhanced sagittal T1 WIs showing non-uniform marginal contrast up and non-enhancing central necrotic contents	
Figure 74. Contrast enhanced coronal T1 WIs showing non-uniform marginal contrast up and non-enhancing central necrotic contents	
Figure 75. Left: DWI showing hypointense signal and right: ADC showing hyperintense signal denoting facilitated diffusion.	121
Figure 76. Long TE MRS showing large choline peak.	121
Case 6	
Figure 77. Axial T1 WIs showing right posterior parietal hypointense cystic lesion, bilate subdural right parietal extradural and right parieto-occinital subcutaneous collections	

Figure 78. Axial T2 WIs showing right posterior parietal hypointense cystic lesion, bilateral subdural, right parietal extradural and right parieto-occipital subcutaneous collections 123
Figure 79. Coronal T2 WIs showing right posterior parietal hypointense cystic lesion, bilateral subdural, right parietal extradural and right parieto-occipital subcutaneous collections
Figure 80. Contrast enhanced axial, coronal and sagittal T1 WIs showing homogenous regular marginal contrast uptake and non-enhancing central cystic contents
Figure 81. DWI showing hyperintense signal denoting restricted diffusion
Figure 82. ADC showing hypointense signal denoting restricted diffusion 125
Figure 83. Short TE MRS showing amino acids and lactate peaks
Figure 84. Long TE MRS showing amino acids and lactate peaks
<u>Case 7</u>
Figure 85. Axial T1 WIs showing right frontoparietal hypointense cystic lesion 128
Figure 86. Sagittal T1 WIs showing right frontoparietal hypointense cystic lesion 128
Figure 87. Coronal T2 WIs showing right frontoparietal hyperintense cystic lesion 128
Figure 88. DWI showing hyperintense signal denoting restricted diffusion
Figure 89. ADC showing hypointense signal denoting restricted diffusion
Figure 90. Long TE MRS showing amino acid, succinate, lactate and choline peaks 129
Figure 91. Follow-up images showing disappearance of the previously seen abscess and the development of an area of encephalomalacia in its place
Case 8
Figure 92. Axial T1 WIs showing right posterior parietal hypointense lesion
Figure 93. Axial T2 WIs showing right posterior parietal iso to hyperintense lesion 132
Figure 94. Contrast enhanced axial T1 WIs showing marginal contrast uptake and non-enhancing central cystic contents
Figure 95. Contrast enhanced sagittal (right) and coronal (left) T1 WIs showing marginal contrast uptake and non-enhancing central cystic contents
Figure 96. DWI showing hyperintense signal denoting restricted diffusion
Figure 97. ADC showing hypointense signal denoting restricted diffusion
Figure 98. Short TE MRS shows amino acid, acetate and lactate peaks

Figure 99. Long TE MRS shows amino acid, acetate and lactate peaks
Case 9
Figure 100. Axial T2 WIs showing right frontal cystic space occupying lesion with hypointense rim. It is surrounded by hyperintense vasogenic brain oedema. Evidence of mass effect in the form of effacement of the overlying cortical sulci, midline shift and indentation of the right lateral ventricle.
Figure 101. DWI showing hyperintense signal denoting restricted diffusion
Figure 102. Long TE MRS showing small amino acid peak and a large lactate peak 137
<u>Case 10</u>
Figure 103. Axial T2 WIs showing left side otitis media and multiple left temporal cystic space occupying lesions surrounded by hyperintense vasogenic oedema
Figure 104. Coronal T2 WIs showing left side otitis media and left temporal cystic space occupying lesion surrounded by hyperintense vasogenic oedema
Figure 105. Axial FLAIR WIs showing left temporal cystic space occupying lesions surrounded by hyperintense vasogenic oedema
Figure 106. Contrast enhanced axial (top) and coronal (bottom) T1 WIs showing homogenous regular marginal contrast uptake and non-enhancing central cystic contents
Figure 107. DWI showing hyperintense signal denoting restricted diffusion
Figure 108. ADC showing hypointense signal denoting restricted diffusion
Figure 109. Short TE MRS showing amino acids, acetate, succinate and lactate peaks 141
Figure 110. Intermediate TE MRS showing amino acids, acetate, succinate and lactate peaks. Note the inversion of the amino acids and lactate peaks below base line
Figure 111. Long TE MRS showing amino acids, acetate, succinate and lactate peaks 142
Summary and Conclusion
Figure 112. Flowchart for the use of DWI and MRS to differentiate brain abscess from cystic or necrotic tumour
Figure 113. Flowchart of how to differentiate abscess from cystic or necrotic tumour by MRS.

LIST OF TABLES

Table 1. The 2 x 2 contingency table between the case number of the final diagnosis and the result according to the imaging modalities	
Table 2. Age distribution of the patients	8
Table 3. Sex distribution of the patients	9
Table 4. Distribution of the patients in the tumour group according to their final diagnosis 8	9
Table 5. Signal intensity in DWI	0
Table 6. ADC values in both groups	1
Table 7. Comparison Cho/Cr ratios in both groups	4
Table 8. Comparison Cho/NAA ratios in both groups	5
Table 9. Comparison NAA/Cr ratios in both groups	6
Table 10. Sensitivity, specificity, PPV, and NPV of DWI and MRS	7