Singapore Med J 2007; 48(8) : 783

Magnetic resonance spectroscopy in pituitary tuberculoma

Saini K S, Patel A L, Shaikh W A, Magar L N, Pungaonkar S A

ABSTRACT

Magnetic resonance spectroscopy (MRS) is a new, noninvasive method of diagnosing a lesion in cases where magnetic resonance (MR) imaging cannot reliably differentiate between two or more possible aetiologies. This case report describes a 20-year-old pregnant woman who developed sudden onset of left-sided hemiparesis. MR imaging of the brain revealed an infarct of the right middle cerebral artery and a suprasellar mass. The endocrine workup was normal. As she was 20 weeks pregnant, the option of a transsphenoidal biopsy of the pituitary lesion was rejected in favour of MRS. It demonstrated features characteristic of a tuberculoma. She showed marked clinical improvement after she was started on anti-tuberculous drugs. MRS is a rapidly-developing diagnostic modality, and may be a useful and safe option for investigating intracranial lesions in patients who cannot undergo invasive procedures.

Keywords: intracranial tuberculoma, magnetic resonance spectroscopy, pituitary diseases, tuberculosis

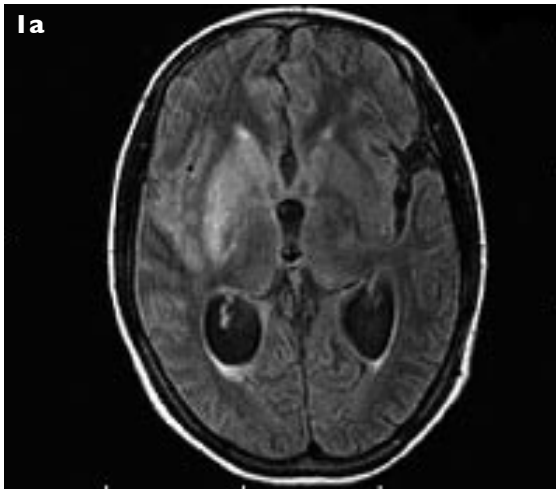
Singapore Med J 2007;48(8):783–786

INTRODUCTION

A pituitary mass is commonly encountered in clinical practice. The usual cause is an adenoma, and this may sometimes be unmasked during pregnancy. In rare cases, the underlying cause may be tuberculous granuloma. Imaging methods like computed tomography or magnetic resonance (MR) imaging may not reliably differentiate between the various possible causes of a pituitary mass. Traditionally, transsphenoidal biopsy has been the gold standard for determining the cause of a pituitary mass. The evolution and refinement of magnetic resonance spectroscopy (MRS) as a diagnostic modality, coupled with its increasing availability, offers a new, non-invasive method for diagnosing this intracranial lesion.

CASE REPORT

A 20-year-old-woman presented with sudden onset depressed sensorium and left-sided weakness. She was 20 weeks pregnant, and had a six-month history of low grade fever and a two-month history of intractable vomiting. She had no past history of gastric or oesophageal disorder, tuberculosis, diabetes mellitus, or any other major illness. On examination, she had terminal neck stiffness, sluggishly-reacting right pupil, right-sided optic atrophy, left upper motor neuron facial palsy, and left-sided hemiparesis. The respiratory and cardiovascular system examinations were normal.



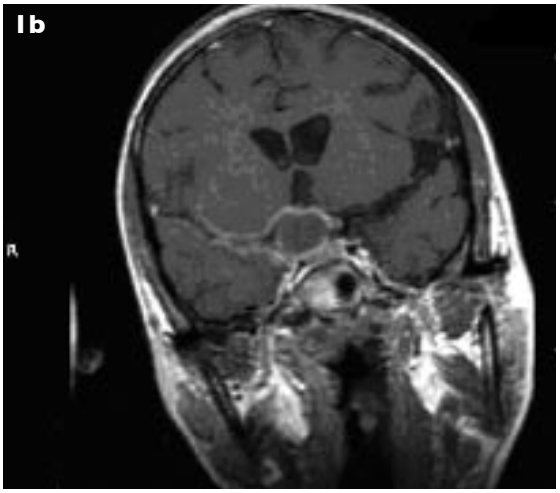


Fig. 1 (a) Enhanced axial T1-W MR image shows an infarct in the right middle cerebral artery territory. (b) Enhanced coronal T1-W MR image shows a suprasellar mass with enhancing exudates in the right sylvian fissure.

Department of Medicine, Grant Medical College, Sir JJ Group of Hospitals, Byculla, Mumbai 400008, India

Saini KS, MBBS, MD
Former Chief Resident

Patel AL, MBBS, MD
Professor

Shaikh WA, MBBS, MD
Assistant Professor

Department of Radiodiagnosis

Magar LN, MBBS, DNB
Former Chief Resident

Department of Imaging, Balabhai Nanavati Hospital, Mumbai 400056, India

Pungaonkar SA, MBBS, MD
Consultant

Correspondence to:
Dr Kamal S Saini
PO Box 3015,
Bangalore 560030,
India
Tel: (91) 80 2210 7371
Fax: (91) 80 2657 6570
Email: kamalsaini@gmail.com

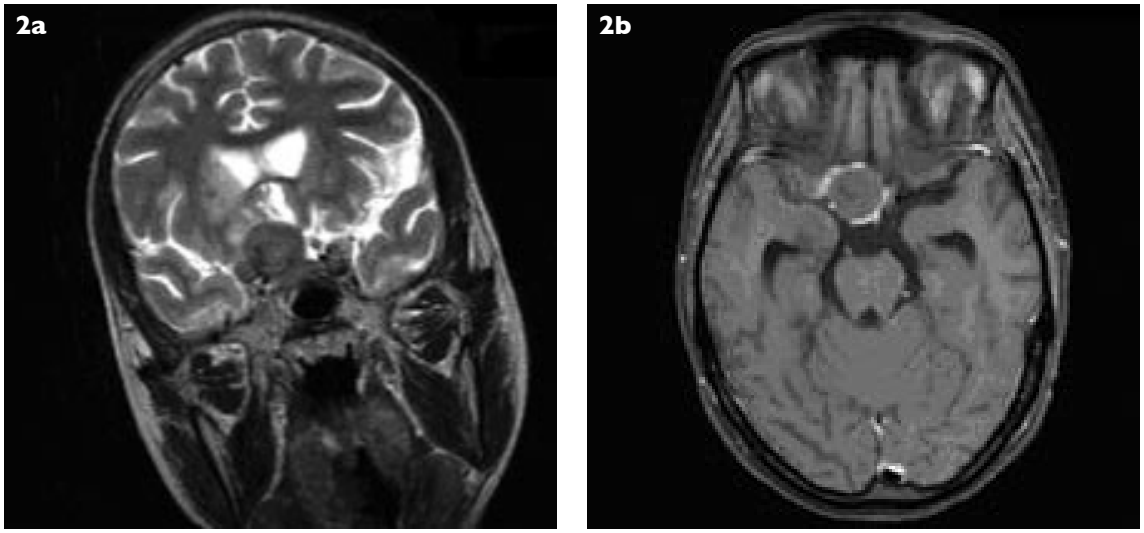


Fig. 2 (a) Coronal T2-W MR image shows a hypointense, suprasellar mass and an infarct. (b) Enhanced axial T1-W image shows a pituitary mass with peripheral enhancement.

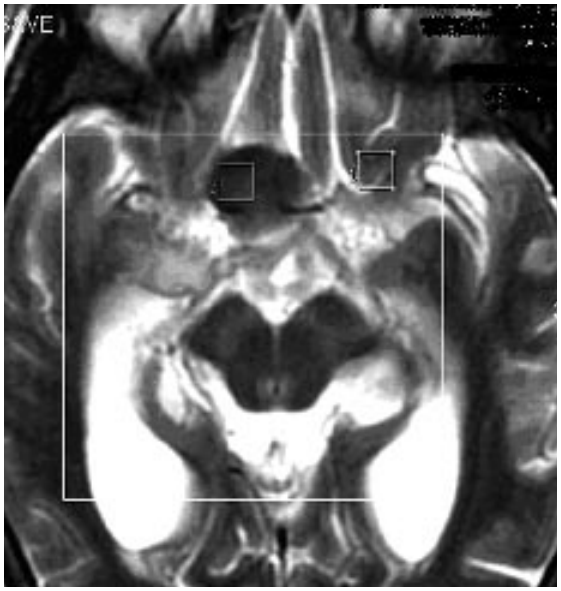


Fig. 3 Axial T2-W MR image shows the areas of interest, or voxels 1 (normal) and 2 (suprasellar mass) selected for spectroscopy.

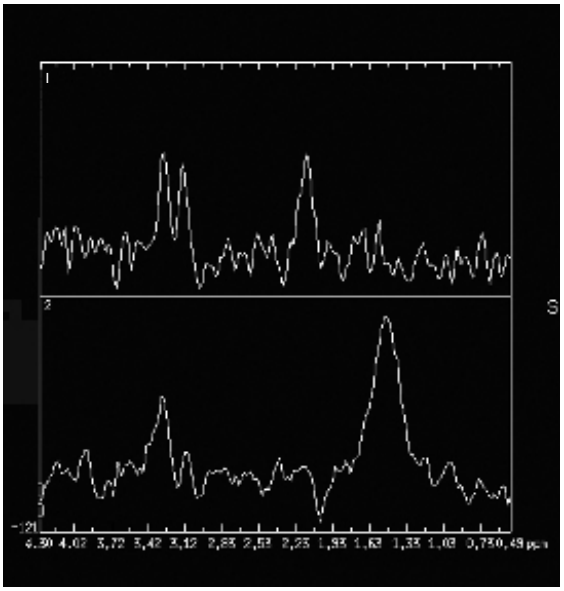


Fig. 4 MRS of voxels 1 and 2 (see Fig. 3). The upper panel shows the spectroscopic appearance of normal brain tissue. The lower panel shows a prominent lipid peak at 1.3 ppm, characteristic of a tuberculoma.

Investigations showed anaemia and raised erythrocyte sedimentation rate. The haemogram, biochemistry, electrocardiogram and chest radiograph were normal. MR imaging of the brain revealed an infarct of the right middle cerebral artery, thick enhancing exudates along the cortical sulci and basal cisterns, and features suggestive of tuberculous meningitis (Fig. 1). MR imaging also showed a 2 cm × 1.8 cm × 1.8 cm mass in the suprasellar cistern, which was isointense on T1-weighted images and hypointense on T2-weighted and fluid attenuated inversion recovery images (Fig. 2).

Cerebrospinal fluid (CSF) analysis showed five cells (100% lymphocytes) per high power field, 20 mg/dL of protein, and 45 mg/dL of glucose. CSF adenosine deaminase (ADA) level was 22 U/L (normal < 10 U/L). Serum immunoglobulin M for the anti-A60 tubercular antigen was positive. Cortisol, adrenocorticotropic hormone, follicle stimulating hormone, luteinising hormone, prolactin, T3, T4, and thyroid stimulating hormone levels were within normal limits.

The above clinical, laboratory, and MR imaging findings were suggestive of a tuberculoma; however,

an adenoma could not be reliably ruled out by MR imaging alone. In view of her pregnant state, MRS was performed in lieu of a transnasal suprasellar biopsy. MRS revealed a prominent lipid peak at 1.3 ppm, which was typical of a tuberculoma (Figs. 3 & 4). Anti-tuberculous drugs and intravenous steroids were prescribed. There was marked clinical improvement over the following weeks. She was discharged and was advised regular physiotherapy and follow-up.

DISCUSSION

The differential diagnoses of a pituitary mass include tumours (adenomas), developmental defects (Rathke's Pouch), haematomas (Sheehans' syndrome), granulomatous diseases (sarcoid, histiocytosis X) and infections (tuberculosis, toxoplasmosis, neurocysticercosis). MR imaging of the brain is the usual initial investigation employed when a suprasellar mass is suspected.[1] However, the diagnosis may be complicated by ambiguous neuroradiological findings; thus histopathological examination remains the gold standard. Newer, noninvasive tests are currently under development.

Tuberculosis has always been a significant health problem in Asia. It has shown a resurgence in the recent past due to the HIV/AIDS epidemic, increased travel and migration. Approximately 5%–10% of cases of tuberculosis have central nervous system (CNS) involvement. Tuberculosis of the CNS may present either with meningitis or a parenchymal space-occupying lesion.[2] A focal parenchymal tubercular lesion in the brain usually follows haematogenous dissemination of primary lung infection. More than half of the patients with a CNS tuberculoma have concomitant meningeal involvement.

Tuberculous granulomas of the CNS have fairly specific image morphology on MR imaging. These include enhancement of basal cisterns, granulomas, calcification, hydrocephalus, meningeal enhancement and basal ganglia infarction.[3] Iso- or hypointensity of intracranial lesions on both T1- and T2-weighted MR imaging are characteristic of the caseous necrotic content that is usually found in tuberculomas. Non-caseous tuberculomas typically display a hypointense signal on T1- and a hyperintense signal on T2-weighted imaging, with homogeneous enhancement after gadolinium administration.[4]

Pituitary tuberculomas are exceptionally rare.[5,6] These may be difficult to diagnose by MR imaging alone. Additional tests like CSF ADA level,[7] and the presence of antibodies to the tubercular antigen A60 in CSF, favour a tuberculoma.[8] MRS is an exciting new development in the field of radiodiagnosis, and is rapidly evolving as a reliable diagnostic tool.

MRS involves the study of the interaction of electromagnetic radiation with matter. It uses magnetic fields with radiofrequency energy to detect specific chemicals in tissues of interest. These chemicals include choline, creatine, lactate, phosphoserine, lipid, myoinositol, N-acetylaspartate, and glutamate. The signal obtained from water is first suppressed, as it overwhelms the signals emanating from these less abundant metabolites. The resonance signal from these molecules, which is proportional to the amount of these chemicals present within the tissue, is then displayed in a graphic format as a spectrum.[9]

The spectrum obtained depends on the chemical constitution of the tissue being studied; thus a particular pathology is reliably and reproducibly associated with a specific spectral pattern. MRS of a tuberculoma shows prominent lipid peaks at 0.9, 1.3, 2.0 and 2.8 ppm; and for phosposerine at 3.7 ppm. Pretell et al studied the MRS patterns of ten patients with either neurocysticercosis (n = 6) or intracranial tuberculoma (n = 4). They found that tuberculomas had higher lipid peaks, more choline, less N-acetylaspartate, and less creatine than neurocysticercosis.[10] The choline/creatine ratio was greater than 1 in all tuberculomas but in none of the cysticerci. Batra and Tripathi evaluated spectroscopy findings in 16 patients with focal cerebral tuberculosis. MRS revealed a lipid peak at 0.9–1.3 ppm in all of the 14 lesions evaluated.[11] An increase in the normalised choline:creatine ratio was found in all the lesions in which the spectra were obtained with the tissue of interest (or voxel), including a variable portion of the lesion wall.

MRS of a tuberculoma demonstrates lipid peaks at 0.9, 1.3, 2.0, and 2.8 ppm; and a phosphoserine peak at 3.7 ppm.[12] The lipid resonances, at 0.9 and 1.3 ppm, occur due to the presence of methylene and terminal methyl groups on fatty acids found in the caseous material in the centre of tuberculomas. A similar pattern may, however, also be observed in patients with toxoplasmosis and primary CNS lymphoma.[13]

MRS is rapidly evolving from a mere investigative tool to a clinically-useful diagnostic modality. When faced with specific diagnostic problems, MRS might help increase diagnostic accuracy by adding another piece of vital information.[9] Although currently limited by a lack of standardisation, MRS may soon become a useful and safe technique for investigating intracranial lesions in patients who cannot undergo more invasive procedures.

REFERENCES

1. Connor SE, Penney CC. MRI in the differential diagnosis of a sellar mass. Clin Radiol 2003; 58:20-31.
2. da Rocha AJ, Maia AC Jr, Ferreira NP, do Amaral LL. Granulomatous diseases of the central nervous system. Top Magn Reson Imaging 2005; 16:155-87.
3. Wasay M, Kheleani BA, Moolani MK, et al. Brain CT and MRI findings in 100 consecutive patients with intracranial tuberculoma. J Neuroimaging 2003; 13:240-7.
4. Chang KH, Han MH, Roh JK, et al. Gd-DTPA enhanced MR imaging in intracranial tuberculosis. Neuroradiology 1990; 32:19-25.
5. Desai KI, Nadkarni TD, Goel A. Tuberculomas of the hypophysis cerebri: report of five cases. J Clin Neurosci 2003; 10:562-6.
6. Sharma MC, Arora R, Mahapatra AK, et al. Intrasellar tuberculoma--an enigmatic pituitary infection: a series of 18 cases. Clin Neurol Neurosurg 2000; 102:72-7.
7. Mishra OP, Loiwal V, Ali Z, Nath G, Chandra L. Cerebrospinal fluid adenosine deaminase activity for the diagnosis of tuberculous meningitis in children. J Trop Pediatr 1996; 42:129-32.
8. López-Cortés LF, Nogales-Pérez MC, Gómez-Mateos J, et al. Antibodies to antigen A60 in cerebrospinal fluid from patients with tuberculous meningitis. Eur J Clin Microbiol Infect Dis 1994; 13:490-5.
9. Castillo M, Kwock L, Mukherji SK. Clinical applications of proton MR spectroscopy. AJNR Am J Neuroradiol 1996; 17:1-15.
10. Pretell EJ, Martinot C, Garcia HH, et al. Differential diagnosis between cerebral tuberculosis and neurocysticercosis by magnetic resonance spectroscopy. J Comput Assist Tomogr 2005; 29:112-4.
11. Batra A, Tripathi RP. Diffusion-weighted magnetic resonance imaging and magnetic resonance spectroscopy in the evaluation of focal cerebral tubercular lesions. Acta Radiol 2004; 45:679-88.
12. Gupta RK, Pandey R, Khan EM, et al. Intracranial tuberculomas: MRI signal intensity correlation with histopathology and localised proton spectroscopy. Magn Reson Imaging 1993; 11:443-9.
13. Jayasundar R, Raghunathan P, Banerji AK. Proton MRS similarity between central nervous system non-Hodgkin lymphoma and intracranial tuberculoma. Magn Reson Imaging 1995; 13:489-93.