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FULL PAPER

Evaluation of intracranial tuberculomas using diffusion-weighted imaging (DWI), magnetic resonance spectroscopy (MRS) and susceptibility weighted imaging (SWI)

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Objective: The present study was aimed to evaluate patients of suspected intracranial tuberculomas with diffusion-weighted imaging (DWI), magnetic resonance spectroscopy (MRS) and susceptibility-weighted imaging (SWI).

Methods: The present study evaluated 116 patients known or suspected of having central nervous system tuberculosis with advanced MRI techniques comprising of DWI, MRS and SWI in addition to the conventional MRI.

Results: Apparent diffusion coefficient value of tuberculomas was not significantly different ($p > 0.05$) from apparent diffusion coefficient value of metastatic lesions and high-grade gliomas. MRS revealed that NAA/Cr and NAA/Cho ratios of tuberculomas were not significantly different ($p > 0.05$) from that of malignant brain lesions. However, Cho/Cr ratio of tuberculomas (1.36 ± 0.41) was

significantly lower from that of malignant brain lesions (2.63 ± 0.99). SWI revealed a complete and regular hypointense peripheral ring in 42 cases of tuberculomas (58%) and in none of the malignant brain lesions.

Conclusion: DWI offers no clear advantage in differentiating tuberculomas from metastasis and gliomas. Tuberculomas may be differentiated from metastases and gliomas by their unique metabolite pattern on MRS. Presence of a complete and regular peripheral hypointense ring in SWI favors the diagnosis of tuberculomas.

Advances in knowledge: The results from the present study suggest promising role of SWI in the discrimination of tuberculomas from metastatic brain lesions and gliomas with the presence of a complete and regular peripheral hypointense ring favoring the diagnosis of tuberculomas.

INTRODUCTION

Tuberculosis caused by *Mycobacterium tuberculosis* causes a wide spectrum of human diseases, with lungs being the most common affected organ.¹ Central nervous system (CNS) tuberculosis occurs from hematogenous seeding of brain parenchyma. CNS tuberculosis may occur either as diffuse disease (leptomenigitis) or in a localised form as tuberculoma, abscess or cerebritis.² Tuberculoma is a mass of granulomatous tissue which develops from Rich's focus.^{3,4}

Tuberculoma commonly occurs as a nodular or discrete lesion, however, uncommonly in the form of multilocular or en plaque forms. Tuberculoma can be found anywhere in the brain parenchyma and also extra-axially in cisterns and fissures.⁵ Though tuberculomas are rare in developed countries, in developing countries they constitute 5–30% of

all intracranial mass lesions.⁶ The pandemic of HIV has led to the resurgence of CNS tuberculosis worldwide.

Intracranial tuberculoma being potentially curable disease must be differentiated from other space-occupying lesions of the brain for prompt institution of antituberculous therapy. The diagnosis of brain tuberculomas in the absence of menigitis is challenging with a broad repertoire of differential diagnosis on conventional imaging like CT and MRI.

MRI cannot confidently diagnose tuberculomas in many cases as substantial overlap is known to occur with other focal brain lesions. However, non-invasive diagnosis of tuberculomas would obviate the need for biopsy, an invasive procedure fraught with risks.⁷ Thus, there is a need for additional imaging modalities, such as MR spectroscopy

(MRS), perfusion MRI, diffusion-weighted imaging (DWI) and susceptibility-weighted imaging (SWI) which may aid in improving the diagnosis of these brain lesions.

With application of new MRI techniques such as Proton MRS, DWI and SWI the MR specificity can be significantly increased in the diagnosis of intracranial tuberculomas.

Proton MRS provides information on the chemical composition of brain which can prove vital in the diagnosis of tuberculomas. DWI provides quantitative information about the microscopic motion of water molecules in a tissue by calculating apparent diffusion coefficient (ADC).⁸

SWI, originally called BOLD venographic imaging, using the susceptibility differences helps in detection of paramagnetic substances like venous blood, hemorrhage and iron storage.⁹ It is a well-known fact that most of the tubercular lesions have a capsule rich in paramagnetic ions with consequent hypointense signal on T_2W images and hypodense rim on non-contrast CT scan. As such, it is expected that the capsule will show a more profound hypointense signal on SWIs, which is exquisitely sensitive to the presence of paramagnetic ions. The susceptibility weighted sequence, as such, is expected to add to the diagnostic confidence in the differentiation of granulomatous lesions from neoplastic lesions. This will complement the information obtained from other modalities (like contrast enhanced MRI, Spectroscopy etc.).

METHODS AND MATERIALS

116 patients, comprising 70 males and 46 females ranging from 1 to 76 years of age (mean age, 45 years), suspected of having CNS tuberculosis were prospectively evaluated with MRI, DWI, MRS and SWI after taking due clearance from the institutional ethical committee. In all cases, informed consent was taken from the patient or his/her attendant. The following patients were included in the study:

- (1) Known cases of CNS tuberculosis or patients suspected of having CNS tuberculosis.
- (2) Ring or disc enhancing lesions on CT other than metastasis (*i.e.* patients with no known malignancy elsewhere in the body).

Patients in whom MRI, as such, is contraindicated (metallic implants, pacemaker etc.) were excluded from the study.

MR imaging protocol and image analysis

All participants were examined by 1.5 T superconducting magnetic resonance imager (Magnetom Avanto, Siemens Medical System,) with a standard head coil. After the preliminary localizing sequence, the imaging protocol included:

- (1) Axial T_1 weighted (T_1W) spin echo sequence [repetition time/echo time (TR/TE) 500 ms/11 ms; slice thickness 5 mm; field of view (FOV) 230 mm].
- (2) Axial T_2 weighted (T_2W) turbo spin echo sequence (TR/TE 3500 ms/110 ms; slice thickness 5 mm; FOV 230 mm).

- (3) Axial fluid attenuated inversion recovery sequence (TR/TE/inversion time 8000 ms/108 ms/2500 ms; slice thickness 5 mm; FOV 230 mm).
- (4) Sagittal T_1W spin echo sequence (TR/TE 450 ms/10 ms; slice thickness 5 mm; FOV 230 mm).
- (5) DWIs were obtained by using an axial echoplanar SE sequence (TR/TE 3000 ms/87 ms), 2 averages, 5 mm section thickness, 230×230 FOV. DW images and ADC maps were acquired by using b-values of 0, 500, 1000 $S\text{ mm}^{-2}$. ADC values were calculated on a picture archiving and communication system workstation by placing the region of interest on the lesion and the values obtained were expressed in $10^{-3}\text{ mm}^2\text{ s}^{-1}$. In lesions with cystic as well as solid component ADC was calculated from the solid component.
- (6) SWI images were obtained in axial plane by using following parameters: TR = 49 ms; TE = 40 ms; section thickness = 2.5 mm and FOV = 230 mm. SW images were analyzed for the presence or absence of a hypointense ring along the periphery of the lesions. In cases showing hypointense ring the characteristics of the ring (complete or incomplete and regular or irregular) were noted.
- (7) Multivoxel three-dimensional chemical shift imaging was performed by using point resolved spectroscopy sequence with water signal suppression and avoiding contamination from scalp fat. MRS acquisition was performed after injecting 0.1 mmol/kg of Gadodiamide (Omniscan; GE Healthcare). Spectroscopic data was obtained by using point resolved spectroscopy sequence with following parameters: TR = 1690 ms; TE = 135 ms; flip angle = 90° ; section thickness = 15 mm; bandwidth = 1000 Hz. Metabolic peaks studied were NAA at 2.02 ppm, choline at 3.22 ppm, creatine at 3 ppm, lipids at 0.9–1.3 ppm and lactate at 1.3 ppm. The lactate assignment was made on the basis of an inverted doublet due to J coupling of lactate bound protons at a TE of 135 ms. Metabolic values were calculated automatically from the area under each metabolic peak and metabolic ratios NAA/Cr, NAA/Cho and Cho/Cr were obtained. Spectrum was also analyzed for the presence or absence of lipid and lactate peaks.

The decision regarding institution of antitubercular therapy (ATT) was based on the clinical profile of the patients as well as the MRI features. Patients with focal brain lesion(s) with associated clinical symptoms and signs of meningitis and supportive laboratory findings like positive cerebrospinal fluid examination were started on ATT. Patients with a focal brain lesion with absence of meningitis were empirically started on ATT based on the conventional MRI features. Patients put on ATT were subjected to follow-up MRI examinations at close intervals of 3 and 6 months to evaluate the initial response to drug therapy.

Patients in whom MRI suggested a diagnosis of primary brain tumor underwent surgery or stereotactic biopsy (in lesions involving vital or eloquent parts of brain). Patients in whom MRI suggested a diagnosis of metastasis were extensively evaluated to locate the primary site of tumor and cases in whom the primary site of tumor could not be established were subjected to stereotactic biopsy with histopathology establishing the metastatic nature of these lesions.

Table 1. T_1 W and T_2 W morphology of various lesions

S.no.	Pathology	T_1 morphology		T_2 morphology	
		Isointense	Hypointense	Hypointense	Hyperintense
01.	Tuberculoma	44 (61%)	28 (39%)	42 (58.3%)	30 (41.7%)
02.	Metastasis	14 (53.8%)	12 (46.2%)	10 (38.4%)	16 (61.6%)
03.	Neurocysticercosis	–	6 (100%)	–	6 (100%)
04.	High-grade glioma	4 (40%)	6 (60%)	–	10 (100%)
05.	Abscess	–	2	–	2

T_1 W, T_1 weighted; T_2 W, T_2 weighted.

RESULTS

116 patients known or suspected of having CNS tuberculosis or presenting with ring/disc enhancing lesions on CT scan were evaluated with advanced MRI techniques comprising of DWI, MRS and SWI in addition to the conventional MRI. 72 patients out of total 116 had tuberculomas on final diagnosis, whereas 26 were found to be metastatic. 10 came out to be gliomas, whereas 6 proved to be neurocysticercosis (NCC) and 2 pyogenic abscesses. The final diagnosis was confirmed by radiological response to ATT in 68 patients of tuberculomas and to albendazole in 6 patients of NCC ($n = 74$, i.e. 63%), whereas in 4 patients of tuberculomas, 16 patients of metastasis, 10 patients of gliomas and 2 patients of pyogenic abscesses, the final diagnosis was confirmed by histopathology ($n = 32$, i.e. 27.5%). 74 patients, in whom the final diagnosis was reached upon by clinical and radiological follow-up, the mean duration of follow up was 11.5 months (range: 4–19 months).

Patients in this study group had age ranging from 1 to 76 years (mean age 45 years). Patients of tuberculomas had age ranging from 1 to 76 years (mean age 41 years). With regards to sex of the study group, 70 (60%) patients were males and 46 (40%) were females.

The T_1 W and T_2 W characteristics of various focal brain lesions are described in Table 1.

Ring enhancement was the most common pattern of contrast enhancement shown by all categories of brain lesions. On DWI, tuberculomas revealed a mean ADC value of $1.027 \times 10^{-3} \text{ mm}^2 \text{ s}^{-1}$ with T_2 W hypointense tuberculomas showing a significantly higher ($p \leq 0.0001$) ADC values than T_2 W hyperintense tuberculomas (Tables 2 and 3).

Table 2. Mean ADC value of various brain lesions

S.no	Pathology	n	Mean ADC value ($\times 10^{-3} \text{ mm}^2 \text{ s}^{-1}$)	Std. deviation
1.	Tuberculoma	72	1.027	0.26
2.	Metastasis	26	0.978	0.124838
3.	Neurocysticercosis	6	1.64	0.13
4.	High-grade glioma	10	0.892	0.143
5.	Abscess	2	0.396	–

ADC, apparent diffusion coefficient.

In MRS, we evaluated lesions for various metabolites (Table 4). Though NAA/Cr ratio of metastatic lesions is less than that of tuberculomas, it did not reach to the level of significance ($p = 0.138$) on statistical analysis. Given the small number of patients of high-grade glioma, NCC and pyogenic abscess, they were not subjected to statistical comparison. Cho/Cr ratio of metastatic lesions (2.63) is significantly higher ($p < 0.05$) than that of tuberculomas (1.36). Given the small number of patients of high-grade glioma, NCC and pyogenic abscess, they were not subjected to statistical comparison. Though considered to be the biochemical fingerprint of the tuberculomas, lipids were also seen in a significant number (66%) of malignant brain lesions.

42 (58.3%) patients of tuberculomas showed a complete peripheral hypointense ring on SWI (Table 5). Whereas 41.7% ($n = 30$) patients did not reveal peripheral hypointense ring on SWI. Using Fischer's exact test, the difference was statistically significant ($p \leq 0.0001$). None of the metastatic lesions or high-grade gliomas revealed a complete peripheral hypointense ring. On statistical analysis, we found a significant difference ($p \leq 0.0001$) between tuberculomas and metastatic lesions with regard to the presence or absence of complete peripheral hypointense ring.

DISCUSSION

On T_1 W images, tuberculomas showed iso- or hypointense (Table 1 and Figure 1a) signal intensity, whereas on T_2 W images, tuberculomas displayed hypointense (Table 1 and Figure 1b) or hyperintense (Table 1) signal intensity. Tuberculomas with solid caseating centre show hypointense signal intensity on T_2 W images owing to granulation tissue in the core of the lesions.^{5,10} Tuberculomas with central liquefactive caseation and non-caseating tuberculomas exhibit hyperintense signal on T_2 W images.^{5,10} On post-contrast study, 64 (88.8%) patients revealed

Table 3. ADC values of tuberculomas

Pathology	T ₂ W morphology	n	Range of ADC values (×10 ⁻³ mm ² s ⁻¹)	Mean ADC value (×10 ⁻³ mm ² s ⁻¹)	Std. deviation	p-value
Tuberculoma	Hypointense	42	0.404–1.414	1.1769	0.213125	≤0.0001
	Hyperintense	30	0.411–1.040	0.817	0.17	

ADC, apparent diffusion coefficient; T₂W, T₂ weighted.

ring enhancement of the lesions (Figure 1c) and 8 (11.2%) patients showed disc or homogenous enhancement of the lesions. Non-caseating granulomas exhibit homogenous contrast enhancement while as tuberculomas with solid or liquid central caseation exhibit ring enhancement.^{5,10}

Tuberculomas were divided into two groups on the basis of their T₂W morphology. T₂W hypointense tuberculomas showed mean ADC value of $(1.176 \pm 0.21) \times 10^{-3} \text{ mm}^2 \text{ s}^{-1}$. ADC value ranged from 0.404×10^{-3} to $1.414 \times 10^{-3} \text{ mm}^2 \text{ s}^{-1}$. T₂W hyperintense tuberculomas had a mean ADC value of $(0.817 \pm 0.17) \times 10^{-3} \text{ mm}^2 \text{ s}^{-1}$ (range 0.411×10^{-3} to $1.040 \times 10^{-3} \text{ mm}^2 \text{ s}^{-1}$) (Figure 1d and e). On statistical analysis, we found a significant difference (p -value ≤ 0.001) between the ADC of T₂W hypointense and T₂W hyperintense tuberculomas. Combined ADC of all tuberculomas including both T₂W hypointense and T₂W hyperintense lesions in our study was $(1.027 \pm 0.26) \times 10^{-3} \text{ mm}^2 \text{ s}^{-1}$. Our results are in concordance with the results of Gupta et al¹¹ who found an ADC value of $(1.24 \pm 0.32) \times 10^{-3} \text{ mm}^2 \text{ s}^{-1}$ in T₂W hypointense tuberculomas and $(0.80 \pm 0.08) \times 10^{-3} \text{ mm}^2 \text{ s}^{-1}$ in T₂W hyperintense tuberculomas. Our results are also in concurrence with the studies done by Vasudev et al,¹² Chatterjee et al,¹³ and Batra and Tripathi.¹⁴

The ADC value of metastatic lesions varied from 0.738×10^{-3} to $1.197 \times 10^{-3} \text{ mm}^2 \text{ s}^{-1}$ with a mean ADC value of $(0.978 \pm 0.12) \times 10^{-3} \text{ mm}^2 \text{ s}^{-1}$ (Figure 2). Our results are comparable to the results of Chatterjee et al,¹³ Berg Hoff et al.¹⁵ ADC values of metastatic lesions varied from 0.35 to $1.37 \times 10^{-3} \text{ mm}^2 \text{ s}^{-1}$ with a mean of $0.796 \times 10^{-3} \text{ mm}^2 \text{ s}^{-1}$ in a study performed by Kinu Ko et al.¹⁶ The variation in ADC values can be attributed to the site of primary cancer with a higher prevalence of lung and breast cancer as the primary carcinoma in our study. The ADC value of metastatic tumors depends on the cellularity of the tumor and hence ADC value is dictated by the site of primary cancer. Statistical analysis did not reveal any significant difference ($p > 0.523$) between the ADC values of tuberculomas and metastatic brain lesions. This finding is in concordance with the findings of

Chatterjee et al,¹³ who also did not find any significant difference between the ADC values of tuberculomas and metastatic brain lesions.

High-grade gliomas revealed a mean ADC value of $(0.892 \pm 0.14) \times 10^{-3} \text{ mm}^2 \text{ s}^{-1}$ (Figure 3). These results are in concordance with the results of Kinu Ko et al.¹⁶

Among NCC (Figure 4) patients, all the six cases showed free diffusion on DWI with a mean ADC value of $(1.64 \pm 0.13) \times 10^{-3} \text{ mm}^2 \text{ s}^{-1}$ which is similar to ADC value of $(1.66 \pm 0.29) \times 10^{-3} \text{ mm}^2 \text{ s}^{-1}$ in a study conducted by Gupta et al.¹¹ Pyogenic abscesses revealed diffusion restriction with an ADC value of $0.396 \times 10^{-3} \text{ mm}^2 \text{ s}^{-1}$.

Spectroscopic analysis of tuberculomas revealed reduction in N-acetylaspartate and creatine with relative increase in choline integral value. Consequently, NAA/Cr and NAA/Cho ratios were reduced with a value of (0.83 ± 0.30) [mean $\pm$ standard deviation (SD)] and (0.64 ± 0.23) (mean $\pm$ S.D) respectively. Peng Juan et al reported NAA/Cho ratio of 0.55, 0.58 and 0.74 ± 0.35 in three consecutive studies.^{17–19} An increase in the Cho/Cr ratio in tuberculomas (1.36 ± 0.41) was found in our study. Elevated choline peak can be seen in tuberculoma due to increased cellularity by inflammatory cellular infiltrates.²⁰ These findings are comparable to the findings of Kumar et al²¹ and Pretell et al.²² However, in two patients we found a markedly raised Cho/Cr ratio of 2.59 and 2.52. Atypical MRS finding in the form of markedly elevated Cho/Cr ratio and marked reduction in NAA/Cho ratio has been reported by R. P. Tripathi et al.²³ Elevated Cho/Cr ratio in tuberculomas was also reported by Batra and Tripathi.¹⁴

Spectroscopic analysis of tuberculomas revealed lipid peak at 0.9 and 1.33 ppm in 54 patients (75%) (Figure 1g). Among them, only lipid was seen in 32 patients while 22 patients showed combined lipid-lactate peaks. Eight patients (11%) showed only lactate peak. The lipid peak in tuberculoma is due to the presence of lipids within the cell wall of tubercle bacillus.²⁴

Table 4. MRS: list of various metabolite peaks and ratios noted in various pathologies

S.no	Pathology	n	NAA/Cr	NAA/Cho	Cho/Cr	LAC (no. of patients)	LIPID (no. of patients)
1.	Tuberculoma	72	0.83	0.64	1.36	30	54
2.	Metastasis	26	0.698	0.564	2.63	18	18
3.	Neurocysticercosis	6	1.09	1.03	0.95	2	–
4.	High-grade glioma	10	0.646	0.40	3.34	8	6
5.	Abscess	2	0.72	0.63	0.51	–	–

MRS, magnetic resonance spectroscopy.

Table 5. SWI features of various lesions

S.no	Pathology	SWI		
		Complete hypointense ring	No Hypointense ring	Incomplete hypointense ring
1.	Tuberculoma	42 (58.3%)	30 (41.7%)	–
2.	Metastasis	–	26 (100%)	–
3.	Neurocysticercosis	2 (33%)	4 (67%)	–
4.	High-grade glioma	–	8 (80%)	2 (20%)
5.	Abscess	2	–	–

SWI, susceptibility-weighted imaging.

Lactate is the result of anaerobic glycolysis within the tuberculoma.²⁴ A number of studies have reported elevated lipids in tuberculomas.^{13,22,23,25,26}

Metastatic lesions revealed a markedly increased choline with reduced N-acetylaspartate and creatine. NAA/Cr and NAA/Cho ratios were (0.69 ± 0.15) and (0.56 ± 0.22) (mean $\pm$ SD) respectively. Cho/Cr ratio of metastatic lesions was (2.63 ± 0.99) (mean $\pm$ SD).

In high-grade gliomas, we found a substantial increase in the choline integral value with simultaneous reduction in NAA and Cr levels contributing to decreased NAA/Cr and NAA/Cho ratios with concurrent increase in Cho/Cr ratio. High-grade gliomas revealed NAA/Cr ratio of 0.64, NAA/Cho ratio of 0.40 and Cho/Cr ratio of 3.34. Our findings are consistent with the results of Kumar et al,²¹ Alam MS et al²⁵ and Peng Juan et al.¹⁷ Lactate peak was seen in 6 metastatic tumors whereas lipid-lactate peak was seen in 12 metastatic tumors

(Figure 2). Six patients of high grade gliomas showed both lipid-lactate peak whereas two patients showed isolated lactate peak (Figure 3).

Statistical analysis performed between the two largest groups of patients of tuberculomas and metastatic brain tumors revealed a significant difference ($p < 0.05$) in the Cho/Cr ratio of tuberculomas (mean = 1.36) and metastatic brain tumors (mean = 2.63). Though the NAA/Cr and NAA/Cho ratios of metastatic brain tumors were less than that of tuberculomas, however, it did not reach to the level of statistical significance (p values of 0.138 and 0.277 respectively). Our results are consistent with the results of other studies.^{17,19}

Presence of lipid-lactate peaks was not helpful in differentiating between tuberculomas and metastatic lesions. This finding is consistent with the findings of Peng Juan et al¹⁷ and Alam et al²⁵ who also drew a similar conclusion. Kumar et al²¹ and Kim et al²⁷ have also reported similar results.

Figure 1. Tuberculoma T_1W image (a) reveals a hypointense lesion in midbrain with associated hydrocephalus. The lesion is hyperintense with a peripheral hypointense rim on T_2W image (b). Post-contrast image (c) reveals ring enhancement of the lesion. DWI (d) and corresponding ADC map (e) reveals restricted diffusion in the lesion. A complete, regular peripheral hypointense ring is seen on SWI (f). MRS (g) reveals reduced levels of NAA, Cr and Cho with a lipid-lactate peak at 1.3 ppm. ADC, apparent diffusion coefficient; DWI, diffusion-weighted imaging; MRS, magnetic resonance spectroscopy; SWI, susceptibility-weighted imaging; T_1W , T_1 weighted; T_2W , T_2 weighted.

Figure 2. Metastasis T_1W image (a) reveals an isointense lesion with surrounding vasogenic edema in left posterior parietal region. T_2W image (b) shows the lesion to be slightly hypointense. On post-contrast image (c) the lesion shows thick ring of enhancement. DWI (d) and corresponding ADC map (e) shows restricted diffusion in the lesion. No peripheral hypointense ring is seen on SWI (f). MRS (g) shows marked elevation of choline peak with reduced NAA and creatine peaks. ADC, apparent diffusion coefficient; DWI, diffusion-weighted imaging; MRS, magnetic resonance spectroscopy; SWI, susceptibility-weighted imaging; T_1W , T_1 weighted; T_2W , T_2 weighted.

Neurocysticercosis patients revealed NAA/Cr, NAA/Cho and Cho/Cr ratios of 1.09, 1.03 and 0.95 respectively. Cho/Cr ratio was <1 in 4 patients whereas it was slightly >1 in 2 patient. None

of the patients revealed lipid peak while two patients showed lactate peak (Figure 4). Two cases of pyogenic abscess revealed presence of cytosolic amino acids on spectral analysis.

Figure 3. High-grade glioma T_1W image (a) showing an isointense lesion in left parietal region. The lesion is hyperintense on T_2W image (b). On post-contrast image (c) the lesion shows ring enhancement. DWI (d) and corresponding ADC map (e) reveals areas of both restricted diffusion (centre) and free diffusion (periphery) in the lesion. SWI (f) reveals an irregular and incomplete peripheral hypointense ring. MRS (g) shows markedly elevated choline levels with reduced NAA and creatine peaks. ADC, apparent diffusion coefficient; DWI, diffusion-weighted imaging; MRS, magnetic resonance spectroscopy; SWI, susceptibility-weighted imaging; T_1W , T_1 weighted; T_2W , T_2 weighted.

Figure 4. Neurocysticercosis T_1W (a) hypointense and T_2W (b) hyperintense lesion showing ring enhancement on post-contrast sequence (c) is seen in left posterior parietal region. The lesion shows free diffusion on DWI (d) and corresponding ADC map (e). On SWI (f), the lesion shows a peripheral hypointense ring. MRS (g) reveals slightly reduced NAA and Cr with Cho/Cr ratio slightly >1 with a lactate peak at 1.3 ppm. ADC, apparent diffusion coefficient; DWI, diffusion-weighted imaging; MRS, magnetic resonance spectroscopy; SWI, susceptibility-weighted imaging; T_1W , T_1 weighted; T_2W , T_2 weighted.

High grade gliomas, NCC and pyogenic abscess were excluded from statistical comparison because of their small sample size.

To our knowledge, the present study is the first attempt to study the role of SWI in the evaluation of brain tuberculomas. Previously, SWI has been studied by Lai et al²⁸ in the evaluation of pyogenic brain abscesses and by Toh et al²⁹ who attempted to explore the role of SWI in the differentiation of pyogenic brain abscesses and rim enhancing glioblastomas. We observed a complete and regular hypointense peripheral ring in 42 cases of tuberculomas (Figure 1g), 2 cases of NCC (Figure 4g) and 2 cases of pyogenic brain abscess. The signal intensity of the ring varied from slightly hypointense to profoundly hypointense ring. None of the metastatic brain tumors or high-grade gliomas revealed a complete hypointense peripheral ring. Hence, there was significant difference in the presence of hypointense peripheral ring between tuberculomas and metastatic brain lesions ($p \leq 0.0001$). Two cases of high-grade gliomas showed an irregular and incomplete peripheral hypointense ring (Figure 3g). The sensitivity, specificity, positive likelihood ratio, negative likelihood ratio, positive-predictive value and negative-predictive value of complete peripheral hypointense ring on SWI in diagnosis of tuberculomas were 58.33%, 90.91%, 6.42, 0.46, 91.30% and 57.14%, respectively. In case of tuberculomas and inflammatory granulomas, peripheral hypointense ring on SWI is seen because of the paramagnetic free radicals in the periphery of the lesion.²⁸ In the follow-up MR examinations, we observed disappearance of complete peripheral hypointense ring on SWI in tuberculomas in 30 patients at 3 months and rest 12 patients at 6 months post-treatment. In high-grade gliomas, an incomplete, broken and irregular ring is likely due to deposition of hemorrhagic

products at the edges of the lesion.²⁸ It is thus expected that SWI will help in the differentiation of tuberculoma (and possibly other inflammatory focal brain lesions) from neoplastic lesions. At this stage, however, it is admittedly too early to extrapolate the potential of SWI based on the limited data presented in our study. Nevertheless, we believe that the analysis, as shown in our study, could serve the purpose of idea stimulation for investigators who are interested in proceeding with further exploration in this direction. Further studies with a large number of brain tuberculomas should be performed to validate the findings of our study.

CONCLUSION

DWI offers no clear advantage in differentiating tuberculomas from metastasis and gliomas. Tuberculomas may be differentiated from metastases and gliomas by their unique metabolite pattern comprising of reduced NAA/Cr and NAA/Cho ratios, elevated Cho/Cr ratio with presence of lipids whereas, markedly elevated Cho/Cr ratio, reduced NAA/Cr and NAA/Cho ratios with/without lipid or lactate peak favors a diagnosis of neoplastic brain lesion. The results from the present study suggest promising role of SWI in the discrimination of tuberculomas from metastatic brain lesions and gliomas with the presence of a complete and regular peripheral hypointense ring favoring the diagnosis of tuberculomas. Nevertheless, a larger prospective study is needed to ascertain the usefulness of SWI in the discrimination of tuberculomas and neoplastic brain lesions.

ETHICAL CLEARANCE:

The study was duly cleared by the institutional ethical committee (IEC) under the No. SIMS 1 31/IEC-SKIMS/2014-75

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