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Study of MRI Brain in children suffering from cerebral malaria in tertiary care hospital of Bathinda Punjab

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Abstract

Background: Cerebral malaria is the leading cause of seizures and encephalopathy and a major cause of death in the pediatric age group. Our objective was to look for magnetic resonance imaging (MRI) changes in CM and to correlate the findings with clinical parameters and outcome.

Materials and Methods: This cross-sectional study was conducted from January 2022 to January 2024 in the pediatrics department of a tertiary care teaching hospital, Adesh Institute of Medical Sciences, Bathinda, Punjab. Children suffering from CM during the study period were subjected to MRI study of brain. All the data were analyzed with appropriate statistical methods.

Results: Twenty cases were included. The mean age was 8.5 (±3.52) years, with males being 75%. Headache and vomiting were the presenting symptoms (30% each), and the most common signs were pallor and seizure. The mean duration of altered sensorium was 18.55 (±11.19) h, and the mean coma recovery time was 14.65 (±6.8) h. Retinal hemorrhages were present in 40%. The study did not find any statistical significance between MRI finding and Glasgow Coma Scale level. None of the MRI findings revealed any significant association with parasitemia. No significant association was found between focal, diffuse swelling of brain and restricted diffusion in the MRI brain with the outcome.

Conclusions: In children with CM, MRI findings did not correlate with clinical parameters and outcomes. All the MRI abnormalities disappeared within a short period. Further studies with larger sample size are needed to support or refute the present study findings.

Keywords: Cerebral edema, magnetic resonance imaging, malaria, mortality, pediatric, Plasmodium falciparum

INTRODUCTION

Malaria is one of the most important parasitic diseases of humans. Malaria is a mosquito-borne infectious disease of humans and other animals caused by parasitic protozoans belonging to the Plasmodium species. It affects around 5% of the world's population at any time and causes somewhere between 0.5 and 2.5 million deaths each year.[1] Malaria is a major public health problem in the state of Punjab especially Malwa region of Punjab. Even though it comprises only 4% of the total population of India, it contributes the highest number (22%) of malaria cases, 43% of the total *Plasmodium falciparum* cases, and about 50% of all reported deaths due to malaria in the country.[2] The World Health Organization (WHO) defines cerebral malaria (CM) as severe *P. falciparum* malaria with cerebral manifestations, usually having alteration in sensorium (Glasgow Coma Scale [GCS] <11 and Blantyre Coma Scale <3).[3] Malaria with altered sensorium persisting for >30 min after a seizure episode is considered to be CM.[3] CM is the most severe neurological complication of P. falciparum infection and is a major cause of acute nontraumatic encephalopathy in tropical countries.[4] CM is the most common complication of *P. falciparum* infection, which may result in severe complications, including long-term neurologic impairment, multi-organ failures, and high mortality in the absence of prompt diagnosis and appropriate treatment.[5] CM is the leading cause of seizures and encephalopathy and a major cause of death in the pediatric age group.[6] The pathogenesis is heterogeneous, and the neurological complications are often part of a multisystem dysfunction.[6] The clinical presentation and pathophysiology differ between adults and children.[6] The incidence of neurological sequelae in malaria occurs due to severe complications of malaria such as CM, acidosis, acute renal failure, and severe anemia. This occurs even if there is parasite clearance

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by antimalarial drugs, suggesting that the cause of mortality is not only directly by invasion of tissues by the parasites but also due to various other factors and may include structural and/or functional changes in brain parenchyma. Fatality rates of 10%–30% have been reported among children referred to hospital with severe malaria.[7] These rates are even higher in rural and remote areas where patients have restricted access to adequate treatment.[7] Worldwide, the case fatality rate in CM ranges from 8% to 33%.[8] Studies in India report a case fatality rate varying from 12% in Cuttack (Odisha) to 21% in central India.[9] The incidence of neurological sequelae in survivors of CM among children has been estimated to be up to 26.3%.[10] Magnetic resonance imaging (MRI) may reveal direct evidence of structural as well as functional changes in brain parenchyma which might be contributing to death. In this study, we tried to find reliable imaging correlate of cerebral malaria that could be helpful in understanding the pathophysiology of cerebral edema leading to death or sequalae.

MATERIALS AND METHODS

This cross-sectional study was conducted on the inpatients of pediatric intensive care unit (PICU), department of pediatrics of a tertiary care teaching hospital of Adesh Institute of Medical Sciences, Bathinda, Punjab. The study was conducted over 2-year period from January 2022 to 2024. The inclusion criteria included children aged 5–15 years being admitted to pediatric ward or PICU with CM as defined by the WHO (severe falciparum malaria with coma [GCS <11 and Blantyre Coma Scale <3], malaria with coma persisting for >30 min after a seizure is considered to be CM, severe anemia with hemoglobin <5 g/dL, or packed cell volume <15%).[3] CM is usually associated with repeated generalized seizures, which are described as more than 2 seizures (≥3) observed within 24 h.[9]

Exclusion criteria: Exclusion criteria included seizures and loss of consciousness due to other central nervous system infections, as per a clinical and cerebrospinal fluid study, altered sensorium due to electrolytes imbalance, head trauma, or metabolic causes, preexisting neurological disorder, and hemodynamically instability precluding MRI study. Children whose parents denied consent were also excluded. Prior to enrollment in the study, patients with malaria (smear positive and/or rapid test positive) with loss of consciousness and satisfying criteria for CM as defined by the WHO were screened. During this initial process, informed consent was obtained from the parents/guardians. The consent form was read out and explained verbally in Punjabi to those unable to read. After that, they were asked to sign it (for those who could not sign, a thumb impression was taken on the form).

Detection of cases and methodology: Patients, who met clinical case definition of CM as defined by the WHO, were enrolled in the study. After taking proper history and clinical examination, the following laboratory evaluations were performed: immunochromatography test, complete blood count and differential count (AcT 5 Differential Coulter), hematocrit (AcT 5 Differential Coulter), malaria parasite for thick and thin film (peripheral blood smear (PBS)/microscopy), blood glucose (by GOD-POD method), blood culture (BACTEC method), liver function test (IFCC method), renal function test (urea by GLDH method; creatinine by Jaffe's method), and electrolytes (by indirect ISC method). All biochemical parameters are done in AU5800 Beckman Coulter Machine. MRI was performed within 12 h of admission and repeated after 48 h of the first scan, on day 2, if any abnormality was found in the first MRI. If the first MRI was normal, then the second MRI was not done. All patients were screened for MRI compatibility prior to undergoing their subsequent examination. After initial stabilization upon admission, the patient was scanned using research MRI protocol as soon as possible. The contrast agent and the normal saline were administered. MRI scanning was done in 1.5 Tesla MRI symphony (Siemens Health Care). For MRI, the patient was placed in a transmit/receive head coil and a compatible pulse oximeter monitor was applied to monitor oxygen saturation and heart rate. Most of the patients were unconscious and sedation was not required. If sedation was required, the patient was administered sedation or anesthesia if required. MRI protocol will include the following sequence: axial T1-Turbo spin echo (TSE) and TSE-fluid attenuation inversion recovery (FLAIR) for lesion identification, axial T2-TSE and TSE-FLAIR for lesion identification, and axial trace – diffusion-weighted imaging (DWI) (b-values 0, 500, and 1000 s/mm2) + creation of apparent diffusion coefficient maps. MRI images were evaluated to see the following parameters: changes of brain swellings, changes of all parts of gray and white matter, changes of cortex, brain stem, extrapyramidal system abnormalities, vascular abnormalities, other changes such as involvement of basal ganglia thalamus, corpus

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callosum, and cerebellum, and any other associated abnormalities. The cost of MRI procedure was borne out of the MRI project in our hospital. All the data were recorded in the case record forms prior to analysis.

Sample size and statistical tools: The sample size has been determined on the basis of the data on CM from our hospital. On an average, 21 cases are available in the year, and the 90th centile (between the first standard deviation [SD] [68.3%] and the second SD [95.4%]) is 20. Therefore, the sample size was fixed at 20. Data were collected on different parameters as per the protocol of the study. These were codified and entered into SPSS-16 software for statistical analysis (IBM SPSS Statistics for Windows, Version 24.0. IBM Corp., Armonk, NY). Categorical data were expressed as numbers and percentages and analyzed using Chi-squared test. Continuous data were expressed as mean (SD) in case normal distribution and median (interquartile range) in case they are skewed. Analysis was done by either t-test or Mann–Whitney U-test, depending on the distribution. $P < 0.05$ was considered statistically significant.

RESULTS

The mean age of the cases was 8.5 ± 3.52 years, with the range lying between 5 and 15 years. The cases were evenly distributed among the age group of 5–8 and ≥9 years ($P = 0.824$). Among the cases, males have preponderance with a high proportion of 75%. A proportion of males and females were significantly different ($P = 0.041$). The most common presenting complaints were headache and vomiting, found in 30% of cases each. Physical examination of the study subjects revealed that all the 20 cases presented with pallor, 11 (55%) cases presented with icterus, and only 2 (10%) had edema. All the subjects had seizure, of which 16 cases were generalized tonic colonic seizure (GTCS) and 4 cases focal. There were no meningeal signs. Eye signs were normal for all. Plantar reflex was up (positive Babinski sign) going in 13 (65%) cases. Altered sensorium was present in all the cases. The mean hours of altered sensorium was 18.55 (±11.19) h, with a median value of 15.5 and a range of 8.0–48.0 h. About 50% of cases have altered sensorium more than 15.5 h and 25% more than 23.0 h. GCS 6 or below was found among 40% of cases and above 6 was found among 60% of cases.

For studying the association of MRI finding and GCS, the patients were divided into two groups: one with GCS 6 or less and the other group with GCS >6. The mean coma recovery time was 14.65 (±6.8) h, with a median value of 14.0 h and ranging between 4.0 and 30.0 h. About half of the subjects have a coma recovery time of more than 14.0 h and 25% more than 19.5 h. Laboratory parameters are described in Table 1. The mean hemoglobin level was found to be 4.4 (±0.5) g/dL. The platelet count was low, with a mean level of 62,429 (±24,909)/mm3. The mean level of total leukocyte count was 13.0 (±5.9) 1000/mm3. The mean random blood sugar (RBS) was 124 (±34) mg/dL. The mean bilirubin level was 1.2 (±0.7) mg/dL. The mean alanine transaminase (ALT) level was 56.4 (±40.2) IU/L, with >50% of the subjects having ALT level above the normal value. The mean urea level was 56.8 (±22.6) mg/dL. The mean creatinine level was 0.8 (±0.5) mg/dL. Almost all the cases had raised serum urea level with a creatinine level. The mean serum sodium was 134.5 (±3.49) mmol/L. The mean serum potassium was 3.94 (±0.54) mmol/L. The mean serum calcium was 7.8 (±0.37) mg/dL.

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Table 1: Blood investigation of the study subjects

Parameters	Mean	SD	Median	Maximum	Minimum
Hemoglobin	4.4	0.5	4.6	4.9	3.2
Platelet count	62,429	24,909	60,000	102,000	20,000
Total leukocyte count	13	5.9	12.1	27.2	4.5
Blood glucose	124	34	119	196	27
Total bilirubin	1.2	0.7	0.9	3.6	0.4
Alanine transaminase	56	40	42	164	23
Urea	57	23	50	135	36
Creatinine	0.8	0.5	0.7	2.7	0.5
Sodium	134.5	3.49	134	140	127
Potassium	3.94	0.54	3.8	5.2	3.2
Calcium	7.77	0.37	7.8	8.4	7.1

SD: Standard deviation

Retinal hemorrhage was present in 8 (40%) cases. No papilledema, macular/peripheral whitening, or vessel changes were evident in fundoscopy. MRI findings were as follows: brain swelling focal was found among 2 out of 20 patients (10%) in the first MRI, but in the second MRI, it was found to have recovered and brain swelling diffuse was found among 10 (50%) cases, but by the time the second MRI was done, it reduced to 4 (20%) cases. Restricted diffusion was found among 7 (35%) cases in the first MRI, which reduced to 1 case only. They are described in Table 2. When brain swelling focal was present, the mean GCS was 7.5 (±2.12), and for no focal brain swelling, the mean GCS was 7 (±1.49) with P = 0.74. Thus, the mean GCS was not associated with the presence or absence of brain swelling. Similarly, for patients with diffuse brain swelling, the mean GCS was 7.11 (±1.36), and for patients without diffuse brain swelling, it was 7 (±1.67). The respective median was 7 in both the cases. The mean was not statistically different (P = 0.84).

Table 2: Magnetic resonance imaging findings of the study subjects

MRI parameters	First MRI, *n* (%)	Second MRI, *n* (%)
Brain swelling (focal)		
Present	2 (10)	0
Absent	18 (90)	10 (100)
Brain swelling (diffuse)		
Present	9 (45)	4 (40)
Absent	11 (55)	6 (60)
Other findings (DW1 and ADC)		
Restricted diffusion	7 (35)	1 (10)
No	13 (65)	9 (90)

DW: Diffusion weighted, ADC: Apparent diffusion coefficient, MRI: Magnetic resonance imaging

For cases with or without restricted diffusion, the respective mean of GCS was 6.86 (±1.34) and 7.15 (±1.62) and the median was same, i.e., 7 for both the groups of cases. The difference was not significant (P = 0.65). The association of GCS with MRI findings was studied and is presented in Table 3. The presence or absence of focal brain swelling did not have a significant association with GCS level (P = 0.76). Similarly, cases with or without brain swelling diffuse have no association with GCS level (P = 0.58). Further cases with or without restricted diffusion did not present any significant association (P = 0.84). None of the MRI findings revealed any significant association with parasitemia. Table 4 presents the association of MRI findings by outcome. Out of 20 cases, 18 recovered and 2 succumbed to death. Chi-squared test of association did not find any association between brain swelling focal, brain swelling diffuse, and restricted diffusion in the MRI findings with outcome. The respective “P” values were 0.62, 0.18, and 0.27, respectively.

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Table 3: Association of Glasgow Coma Scale with magnetic resonance imaging findings

	GCS			χ^2, *P-value*
	6 or less, *n* (%)	**Above 6, *n* (%)**	**Total, *n* (%)**	
Brain swelling (focal) first MRI				
Present	1 (50.0)	1 (50.0)	2 (100)	0.093, 0.761
Absent	7 (38.9)	11 (61.1)	18 (100)	
Total	8 (40.0)	12 (60.0)	20 (100)	
Brain swelling (diffuse) first MRI				
Present	3 (33.3)	6 (66.7)	9 (100)	0.303, 0.582
Absent	5 (45.5)	6 (54.5)	11 (100)	
Total	8 (40.0)	12 (60.0)	20 (100)	
Other first MRI findings (DW1 and ADC)				
Restricted diffusion	3 (42.9)	4 (57.1)	7 (100)	0.037, 0.848
No	5 (38.5)	8 (61.5)	13 (100)	
Total	8 (40.0)	12 (60.0)	20 (100)	

DW: Diffusion weighted, ADC: Apparent diffusion coefficient, MRI: Magnetic resonance imaging, GCS: Glasgow Coma Scale

DISCUSSION

In the present study, the mean age of the cases was 8.5 ± 3.52 years with male preponderance (75%). The mean duration of altered sensorium was 18.55 (±11.19) h, and the mean coma recovery time was 14.65 (±6.8) h. The study did not find any statistical significance between MRI finding and GCS level. None of the MRI findings revealed any significant association with parasitemia. A significant association between retinal hemorrhage and diffuse brain swelling and also restricted diffusion was noted. The mortality rate was 10% among the study group. No significant association was found between brain swelling focal, brain swelling diffuse, and restricted diffusion in the MRI brain with the outcome. In a prospective study of brain MRI in children with acute CM, basal ganglia were the most common area of involvement.[11] Other MRI findings included severely increased cerebral volume with herniation, focal cortical abnormalities, periventricular white matter changes, involvement of the corpus callosum, and abnormalities in the deep gray matter. Millan et al. described MRI findings in CM using conventional T1-weighted and T2-weighted imaging to demonstrate hemorrhage and infarction in a single case.[12] Mohanty et al. reported a series of 12 patients with CM who received MRI, only 3 of whom demonstrated any findings suggestive of a pathologic process on conventional T1 and T2 sequences (cortical infarcts [one case] or hyperintense areas of white matter [two cases] on T2-weighted and fluid-attenuated inversion-recovery sequences). The diffuse hyperintensity was probably due to edema, whereas focal lesions were probably associated with gliosis.[13].

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Table 4: Association of outcome and magnetic resonance imaging findings

	Outcome		χ^2, *P-value*
	Recovered *n* (%)	**Death *n* (%)**	
Brain swelling (focal) first MRI			
Present	2 (11.1)	0	0.247
Absent	16 (88.9)	2 (100)	0.619
Total	18 (100)	2 (100)	
Brain swelling (diffuse) first MRI			
Present	9 (50)	0	0.818
Absent	9 (50)	2 (100)	0.178
Total	18 (100)	2 (100)	
Other first MRI findings (DW1 and ADC)			
Restricted diffusion	7 (38.9)	0	1.197
No	11 (61.1)	2 (100)	0.274
Total	18 (100)	2 (100)	

DW: Diffusion weighted, ADC: Apparent diffusion coefficient, MRI: Magnetic resonance imaging

The present study found that the GCS was not associated with the presence or absence of brain swelling, diffuse brain swelling, and restricted diffusion. In a study, GCS has been correlated with computed tomography (CT) brain. In this study, cerebral edema with thalamic and cerebellar white matter hypoattenuation was seen in five patients. All had a GCS score of 6 or less, a median APACHE II score of 26, and multi-organ failure; none survived.[14] In the present study, none of the MRI findings revealed any significant association with parasitemia (P > 0.05). In a study, the parasite density of bisexual forms of P. falciparum ranged from 50 to 300,000 parasites/μL of blood with a mean of 16,058 parasites/μL of blood.[15] There was no statistical correlation between hyperparasitemia and mortality in CM in the study. Another study reported that parasite density did not influence the outcome in CM. [16] Data on association between parasitemia and MRI findings in children are lacking. In pediatric patients, retinal micro vessels have been shown to sustain damage comparable to the ones occurring in the brain, making them an easily observable surrogate marker to assess the severity of cerebral pathology during CM. [17,18] Recent research indicates that a unique malaria retinopathy can be identified acutely to confirm the diagnosis of CM with 95% sensitivity and 100% specificity in fatal cases. [17,19,20].

In a study, none of the children with retinal hemorrhages due to CM had normal brain MRI. The MRI abnormalities in these groups of patients included a markedly increased brain volume, abnormal T2 signal intensity, and DWI abnormalities in the cortical, deep gray, and white matter structures.[11] The mean level of the parameters was not significantly different between survived and non-survived cases. One study reported that depth of coma is an important prognostic factor, especially in the development of neurological sequelae in survivors of CM. [10] In a study, impaired consciousness and neurological involvement were not found to be associated with hyponatremia. In the same study, hyponatremia was not significantly associated with increased mortality.[21] In the present study, 18 recovered and 2 succumbed, with a mortality rate being 10%. There was no significant association between brain swelling focal, brain swelling diffuse, and restricted diffusion with the MRI findings. Even with appropriate antimalarial therapy and intensive care, 15%–25% of patients die, and mortality may reach 50% if more than 10% of erythrocytes are parasitized.[22] There are few reports of neuroimaging findings in CM in children. Mohanty et al. reported the absence of conspicuous lesions on CT scans.[23].

CONCLUSION

In the present study, the MRI findings did not correlate with clinical parameters such as coma recovery time and mortality in children with CM. All these changes actually disappear within a short period. We did not aim at follow-up of cases with subsequent MRI due to various technical reasons such as cost, paucity of time, and patient compliance. Further study with larger sample size is suggested to support or refute the present study findings.

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