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Role of MRI in Diagnosis of Doubtful Cases of Eclampsia

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Summary

Eclampsia is one of the leading causes of maternal mortality in our country. Though largely a preventable disease, it is still persisting in India due to ignorance and lack of antenatal care. It is associated with marked cerebrovascular and renal perfusion changes. MRI is a sensitive means of detecting these changes. The present study found significant hyperintensity in parietal lobe (66.67% cases) followed by occipital (53%) and frontal (50%) lobes. These changes were of both diagnostic and prognostic significance. On renal angiography there was narrowing of renal arteries with diminished perfusion in 6(40%) out of the 15 study group cases.

Introduction

Eclampsia is the leading cause of maternal mortality in our country. The present study was designed to identify the cerebrovascular changes associated with eclampsia and their significance in relation to diagnosis and prognosis of the disease

Materials and Method

The study was conducted on 25 patients admitted in Queen Mary's Hospital, KGMC, Lucknow. These were divided into 2 groups.

Study group A of 15 cases out of which 13 had eclampisa and 2 had preeclampsia.

Control group B comprised 10 normal pregnant and postnatal women.

A detailed history & examination including neurological examination was done in all patients. Investigations including renogram, urine examination, LFT, Blood sugar, electrolytes and renal function tests were also done. All patients were subjected to MRI of brain and MR angiography of brain and renal arteries during 3rd trimester or within 3 to 6 days after last convulsion. Repeat MRI was performed 15 days later in the study group. Control group was subjected to the imaging only once.

All studies were performed on 1.5 T MR system, using circulatory polarised head coil for studies of brain

and circulatory polarised body coil for renal perfusion studies.

The brain MR imaging protocol included axial spine echo proton density & T2 weighted images repetition time 2200/20-80 (in msec/echo time) with a 192*256 matrix. Section thickness was 0.5 cm, and 0.5 mm intersection space. MR cerebral angiograms were obtained with a three dimensional time of flight magnetisation transfer prepared sequencies. The volume slab was oriented in axial plane to cover the region of Circle of Willis. Imaging volume was 70-80 mm with 64 partitions with effective slice thickness 1.25 mm. Data sets were reconstructed on a micro-vax II computer by means of a ray tracing technique that incorporated maximum intensity projection. Thirty six views of imaging volumes were calculated at 10^0 increments from 0^0 to 360^0.

MR renogram and renal perfusion was performed in all these patients using the contrast gadolinium diethylene triamine penta acetic acid (GDL DTPA) 4 ml given intravenously as a bolus. MR imaging was performed using a circulating polarised body coil. A T1 weighted Turbo FLASH (fast low angle shot) sequence was used with TR/TE/Fan = 4.9/2/8/1 in the coronal plane. Slice thickness was 10 mm and matrix size was 128 x 128. To measure the renal perfusion, time versus signal intensity curves were plotted.

Results

Table – I shows the details of the patients in the study group. Seventy percent patients belonged to 20-30 years age group, and 60% had antepartum eclampsia, 20% had postpartum eclampsia and 13% had PET. There was only one patient of intrapartum eclampsia.

Table-II shows the results of MR imaging of the above patients within 3-5 days of last convulsion and a repeat imaging 15 days later. Almost all patients showed some hyperintensity in T2 weighted images but in different areas. Parietal lobe was involved in 66.67% (n=10), occipital in 53% and frontal in 50% cases.

Table – 1
Clinical profile of patients with pre-eclampsia and eclampsia

Sr. No.	Age (Years)	Parity	Gestational age (weeks)	Diagnosis	BP (mmHg) At the time of admission	BP (mmHg) Follow up	Outcome of Labour	Result
1.	26	P_{0+0}	40	APE	180/120	110/78	FTND	TR
2.	22	P_{0+0}	36	APE	178/128	126/76	Forceps	TR
3.	23	P_{1+0} PNC	1st day	PPE	180/110	116/76	Forceps	TR
4.	24	P_{1+0}	36	APE	160/96	130/90	FTND	TR
5.	23	P_{0+0}	38	APE	158/114	140/100	FTND	HT
6.	26	P1+0 PNC	1st day	PPE	168/112	126/78	FTND	TR
7.	30	P2+0	36	APE	182/124	126/70	FTND	TR
8.	30	P0+0	40	APE	210/140	108/80	LSCS	TR
9.	18	P0+0	36	APE	140/98	128/90	Forceps	TR
10.	20	P0+0	36	APE	140/90	120/80	FTND	TR
11.	17	P1+0	40	APE	150/90	90/60	FTND	TR
12.	18	P0+0	40	IPE	160/128	112/68	FTND	TR
13.	21	P0+0	34	PET	156/98	130/80	Outlet Forceps	TR
14.	20	P1+0	1st day PNC	PPE	158/96	128/84	FTND	TR
15.	25	P0+0	36	PET	140/96	128/94	Outlet Forceps	HT

APE – antepartum eclampsia
PPE – postpartum eclampsia
IPE – intrapartum eclampsia
HT – hypertensive
TR – total recovery

Table – II
MR imaging of the Brain (T2 Hyper-intensity) \
MR imaing finding

S.No.	Diagnosis	1st MR Imaging	2nd MR Imaging
1.	APE	Both BGS, RtCR, RtP. Lobe	Normal
2.	APE	Both side CR	Normal
3.	PPE	Both side EC, ICRt, O	Normal
4.	APE	LtEC, PL, CR, IC, O	N
5.	APE	F, P, BG	F.P.
6.	PPE	Bilateral F multiple O	Normal
7.	APE	LtF, P, subcortical	Normal
8.	APE	P small infarct	Normal
9.	APE	O, LtP subcortical	Normal
10.	APE	BG	Normal
11.	APE	O, P subcortical	Normal
12.	IPE	F, P, O, T	Small infarct P, O
13.	PET	F, P, O granuloma in Lt Parietal lobe-posteriorly	Normal
14.	PPE	F, P, O, T subcortical	Normal
15.	PET	Normal	Normal

F – Frontal
P – Parietal
O – Occipital
T – Temporal
CR – Corona Radiata
EC – External Capsule
IC – Internal Capsule
BG – Basal Ganglia

Changes were present in subcortical areas in 50% cases. Temporal lobe was involved in 13% basal ganglia and corona radiata in 20%, external capsule in 20% and internal capsule in 7% cases. One patient also showed healed granuloma in occipital lobe.

There was full recovery at the second scan in 86.67% cases. Only 13.33% (n=2) cases showed infarction at follow up – one of these two patients also had persistent hypertension. Repeat MRI findings were normal in patients having persistent hypertension and in the 2 patients with psychosis.

Table – III shows the results of MR angiography of brain at both occasions. Fourteen of the 15 patients showed significant changes in the calibre of the cerebral arteries. Anterior cerebral artery was most commonly involved i.e. in 66% of cases. Posterior cerebral artery was involved in 60%, middle cerebral artery in 40% and Circle of Wills in 20% cases.

On follow up, 15 days after the last convulsion, there was total recovery in 80% (i.e. 12) patients. Changes persisted in 20% (i.e. 3) patients. Two of these 3 patients also showed persistent MR changes.

Table – IV and Table – V show the results of renal antiography and renal perfusion studies. Five patients (i.e. 33.3%) showed narrowing of the renal arteries, out of which 4 (i.e. 80%) showed bilateral narrowing. On follow up, changes persisted in only one patient.

Renal perfusion was found to be diminished in 6 patients (i.e. 40%), out of which 5 were the ones having narrowing of renal arteries. It is not clear why perfusion was diminished in one patient with normal calibre of renal arteries. On follow up reduced perfusion persisted in 2 out of 6 patients. Four (i.e. 66.67%) out of these 6 patients with diminished renal perfusion had abnormal renal function tests. These tests were normal in all patients with normal renal perfusion.

None of the above changes were present in any of the control group patients.

Discussion

MR imaging showed brain changes in almost all patients of the study group, most commonly in parietal lobe (66.7%) followed by occipital lobe (53%).

Table-III
MR imaging angiography of brain

S. No.	Diagnosis	First study	Second Study
1.	APE	Lt ACA-N, Both PCA-N	Normal
2.	APE	Both MCA-N, ACA-N	Normal
3.	APE	ACA-N, ACA and MCA-N, Prox. PCA	Normal
4.	APE	Hypoplastic ACA, N-M_1M_2 junction N-PCA	N-M1 M2 junctin, ACA
5.	APE	CW-N	N-MCA
6.	PPE	CW-N	Normal
7.	APE	ACA-N, MCA-N, PCA-N	Normal
8.	APE	ACA-N, Proximal PCA-N	Normal
9.	APE	PCA-N, MCA	Normal
10.	APE	ACA-N, MCA-N, PCA-N	Normal
11.	APE	ACA-N, PCA-N, BA-N	Normal
12.	IPE	CW-N, ACA-N	Residual changes +nt in ACA
13.	PET	MCA-N, ACA-N	Normal
14.	PPE	Proximal-PCA, N-RICA bifurcation	Normal
15.	PET	Normal	Normal

ACA – Anterior cerebral artery
PCA – Posterior cerebral artery
MCA – Middle cerebral artery
CW – Circle of Willis
BA – Basilar artery
ICA – Internal carotid artery
N – Narrowing
M1 & M2 – Junction of horizontal & vertical portions of MCA

Table – IV
Renal Angiography

S. No.	Diagnosis	1st MR angiography	2nd MR angiography
1.	APE	Narrowing – Lt RA	N
2.	APE	N	N
3.	APE	Narrowing of both RA	N
4.	APE	Narrowing of both RA	Narrow RA
5.	APE	N	N
6.	PPE	N	N
7.	APE	N	N
8.	APE	N	N
9.	APE	Narrowing of both renal artery	N
10.	APE	N	N
11.	APE	N	N
12.	IPE	Narrowing of both RA	N
13.	PET	N	N
14.	PPE	N	N
15.	PET	N	N

RA – Renal artery
N – Normal

Table – V
Renal Perfusion Changes

S. No.	Diagnosis	First Study	Second Study
1.	APE	↓ Bilat	Normal
2.	APE	Normal	Normal
3.	APE	↓ Bilat	↓ Bilat
4.	APE	↓ Bilat	Normal
5.	APE	Normal	Normal
6.	PPE	Normal	Normal
7.	APE	Normal	Normal
8.	APE	Normal	Normal
9.	APE	↓ Bilat	Normal
10.	APE	↓ Bilat	↓ Bilat
11.	APE	Normal	Normal
12.	IPE	↓ Bilat	Normal
13.	PET	Normal	Normal
14.	PPE	Normal	Normal
15.	PET	Normal	Normal

Similar changes have been reported by Schwartz and Meldrum (1985) who demonstrated almost complete reversion to normal on follow-up, as seen in our study.

Our findings on cerebral angiography are similar to those of Tromer et al (1988). These findings appear to be unique to eclamptic women and are of diagnostic and prognostic value particularly in patients with an atypical presentation e.g. with focal neurological deficit during pregnancy.

Conclusion

There are significant changes on MR imaging, cerebral and renal angiography and renal perfusion studies in eclamptic women. Changes of T2 hyperintensity are characteristic of eclampsia and help to differentiate it from other causes of convulsions, where the diagnosis is in doubt. These changes are not evident on computed tomography.

Larger studies are required to further delineate the significance of changes on cerebral and renal angiography and perfusion studies.

As these are expensive investigations, they should be reserved for patients whose diagnosis is in doubt, where they can prove to be very useful.

References

1. Schwartz R, Meldrum B. Lancet 2, 1403, 1985.
2. Tromer B L, Homer D, Mikhael M A. Stroke 19, 326, 329, 1988.