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Peri-ictal magnetic resonance imaging in status epilepticus: Temporal relationship and prognostic value in 60 patients

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ABSTRACT

Purpose. Magnetic resonance imaging (MRI) changes associated with status epilepticus (SE) have been described in recent studies. Our aim was to evaluate the diagnosis and prognosis of the peri-ictal MRI changes detected in SE patients.

Method. All adults diagnosed with SE and examined by MRI within 240 h after SE onset were enrolled (2011–2017). Demographic, clinical and electroencephalography data, and functional status at admission and discharge were collected. MRI findings were recorded and relationships between clinical and MRI data, and between these data and functional outcome were analyzed.

Results. Sixty patients included, 50% women, mean age 57.5 years. Median duration of SE was 51.46 h and median time from SE onset to MRI was 86.5 h. Of the total, 41.7% had a restricted diffusion pattern on diffusion-weighted imaging (DWI) and 63.3% had hyperintensities suggestive of edema on T2-weighted (T2WI)/FLAIR sequences. The factors independently associated with T2WI hyperintensities were the presence of acute cerebral lesions ($p = 0.023$), baseline STESS ($p = 0.007$), and MRI performed within 84 h ($p = 0.007$). Variables independently associated with diffusion restriction were a potentially fatal cause ($p = 0.020$), SE duration > 24 h ($p = 0.022$), and MRI performed within the first 84 h ($p = 0.045$). In patients undergoing MRI within 84 h, the DWI and T2WI abnormalities were both highly associated with an unfavorable outcome.

Conclusions. Characteristic signal changes on DWI and T2WI sequences were seen in approximately half our SE patients undergoing early (< 84 h) brain MRI studies, and were independently related to the patients' functional status at discharge.

1. Introduction

When the mechanisms responsible for terminating epileptic seizures fail, status epilepticus (SE) may occur, resulting in prolonged seizures that can have long-term consequences [1]. In clinical practice, magnetic resonance imaging (MRI) is commonly used in SE patients as an aid to establish the etiological diagnosis. However, recent studies have described acute MRI changes associated with SE [2–4], which are mainly restriction in diffusion-weighted imaging (DWI) in different brain areas. Based on the findings in animal models, these features are usually interpreted as stemming from changes in cellular metabolism occurring in the presence of edema secondary to epileptic activity [5]; both vasogenic and cytotoxic edema have been described. The incidence of these imaging changes is reported at 0.07% [6] to 29.4% [7], a considerable range that is likely due to the heterogeneity of the related studies, which are mostly retrospective and contain limited samples. The DWI abnormalities have been topographically related to the epileptogenic focus [8] as well as to motor seizures, altered level of consciousness [9], and lengthy duration of the episode [10]. Abnormalities associated with the epileptogenic focus have also been detected in T2WI and T2-fluid-attenuated inversion recovery (T2-FLAIR) sequences in varying percentages, from 36.8% [11] to 71% [12]. The most common location of these findings (70%–80%) is the hippocampal region [13,14].

Despite this accumulated evidence, there are no definitive clinical data regarding the evolution of these MRI abnormalities over time [6] or their clinical significance. Hence, the aim of this study was to

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evaluate the diagnosis and prognosis of peri-ictal MRI changes detected in patients with SE.

2. Material and methods

This is a retrospective, cross-sectional study including MRI data from patients older than 16 years diagnosed with SE in a tertiary care center and prospectively recorded in a dedicated database between 2011 and 2017. By protocol, all patients in our center with new focal-onset SE undergo MRI study when clinical conditions allow it, regardless of the SE type, underlying etiology, or the patient's prognosis, except in end-of-life situations. To achieve our objective of delineating the evolution of MRI findings over time, we included patients with MRI acquired within the first 240 from the onset of the SE in the present study. We collected information on their demographic characteristics, clinical findings and electroencephalography (EEG) data, and established their functional status before the SE episode, and at discharge using the modified Rankin scale (mRS) [15].

The final diagnosis of SE was established in accordance with the clinical semiology of the event, including the level of consciousness (dichotomized in the analysis into alert-lethargic or stuporous-comatose), and the patient's EEG findings (using Salzburg criteria for non-convulsive SE) [16]. SE was classified according to the latest proposed ILAE classification [1]. SE cases diagnosed before 2015 were reassessed to retrospectively adapt them to the new subtypes. SE severity was determined using the Status Epilepticus Severity Score (STESS), based on the patients' level of consciousness, age, worst type of seizure, and previous history of epilepsy [17].

The etiology of SE was classified as acute symptomatic, chronic, progressive, or cryptogenic, according to the latest ILAE classification. As has been done in other studies [18–20], the cause was considered potentially fatal if, in itself, it could lead to the patient's death regardless of the SE episode. These included stroke with great vessel occlusion occurring less than 7 days previously, acute intracranial hemorrhage, central nervous system infection, severe infection, malignant brain tumor, AIDS with central nervous system complications, renal failure on dialysis, systemic vasculitis, metabolic abnormality sufficing to provoke coma in the absence of SE, eclampsia, and brain tumor surgery.

The duration of SE (in hours) was estimated according to the time of clinical onset of the SE as reported in the medical history. The time of SE resolution was defined as time when the clinical symptoms recovered or an EEG recording showed an absence of ictal epileptogenic activity indicated that the episode had terminated. The SE type was classified according the more prominent clinical semiology described in the clinical history and the EEG ictal findings. The cessation of the non-convulsive SE was overall confirmed with EEG. As to the EEG characteristics analyzed, apart from those related to SE, we also included the presence of lateralized periodic discharges (LPDs) and their specific relationship with the MRI changes.

MRI was performed on a 1.5-T system, using a 12-channel phased array coil, according to a standardized protocol for SE patients that included: 1. transverse T2WI (TE/TR voxel/FA^{o}/slices/GAP: 91 ms/4000 ms/0.8 × 0.6 × 3mm/150/46/0 mm); 2. transverse 2D-FLAIR (TE/TR/TI voxel/FA^{o}/slices/GAP: 92 ms/8500 ms/2439 ms/1 × 0.9 × 1mm/36/0.4 mm) and 3. DWI sequences (TE/TR voxel/slices/GAP: 102 ms/8200 ms/1.2 × 1.2 × 3 mm/0 mm, b Value: 0/500/1000). The images were reviewed by an experienced neuroradiologist (S.S.) blinded to the patients' data with the exception of the SE diagnosis. The MRI studies were specifically reassessed to search for the the presence of areas of restricted diffusion on isotropic DWI (hyperintensity) and T2WI hyperintensities suggestive of edema. In the image review, special focus was placed on brain regions known to be associated with SE-related abnormalities, which encompassed the mesial temporal lobe structures (hippocampus, amygdala, and entorhinal cortex), insula, pulvinar nucleus of thalamus, and lateral temporal lobe. In addition, the neuroradiologist included lesions in other cortical areas, which in her judgement, were considered unrelated to damage induced by the underlying cause. In parallel and when they were available, previous and later MRI studies were assessed to further define the features associated with SE, of special interest in cases related to tumors.

At hospital discharge, we recorded all deaths and whether patients had the same functional status as that at baseline or a functional decline relative to baseline. Reduced functional status and death were considered unfavorable outcomes.

Lastly, we performed an analysis to determine which clinical characteristics were independently associated with the MRI findings, and to seek relationships between the MRI characteristics and the outcome at discharge.

2.1. Statistical analysis

Descriptive and frequency statistics were obtained and comparisons were made using the IBM SPSS Statistics software, version 22.0. Categorical variables are reported as frequencies (percentages) and continuous variables as the mean ± standard deviation (SD) or the median and interquartile range (IQR), as appropriate. Statistical significance for intergroup differences was assessed by the Pearson chi-square or Fisher exact text for categorical variables, and the Student *t* or Mann-Whitney *U* test for quantitative variables. Receiver operator characteristics (ROC) curves were configured to calculate cut-off points for the time from SE onset to MRI examination and the SE duration with the best sensitivity and specificity to predict the response variables. Variables found to be associated with MRI changes and a poor outcome in the bivariate analysis were entered into forward stepwise multiple logistic regression models to identify factors independently associated with restricted diffusion, presence of T2 hyperintensities, and unfavorable outcome at discharge. A p-value < 0.05 was considered statistically significant.

3. Results

From February 2011 and May 2017 we attended 285 SE with a mean age of 63.1 (SD 18.1) years. Of them, 141 (49.47%) had MRI but only 60 (21.05%) were performed in the acute phase. The patients included have a mean age of 57.5 years (SD 18.5) and 30 (50%) were women. The median duration of the SE episode was 51.5 h (IQR 22.2–93), and the median gap time from SE onset to the MRI examination was 86.5 h (IQR 42.3–145.3). MRI was performed during the SE in 23 (38.3%) patients. In the remaining 37 (61.7%) MRIs performed after SE resolution the median time from the end of SE to MRI acquisition was 61.2 h (IQR 26.5–121.6). The patients' clinical and demographic data according to the MRI features detected are summarized in Table 1.

3.1. MRI findings

The overall analysis of the MRI findings showed that 25 patients (41.7%) had a restricted diffusion pattern on DWI and the corresponding ADC maps, and 38 (63.3%) had hyperintensities suggestive of edema on T2WI/FLAIR sequences. Twenty-four of these patients showed abnormalities in all sequences. The distribution of the most commonly found abnormalities is shown in Table 2.

3.2. * T2 hyperintensities

The brain regions where a hyperintensity was most often detected were the frontal (21, 55.3%) and temporal lobes (10, 26.3%) (Fig. 1). Analysis of the factors related to these features showed that they were more common in patients with a more markedly decreased level of consciousness (39.5% vs 9.1%, p = 0.01), those with acute or

Table 1
Relationship between the patients' clinical characteristics and MRI findings. n, number of patients; DWI, diffusion-weighted imaging; T2WI, T2-weighted image; SD, standard deviation; A-L/S-C, alert-lethargic/stuporous-comatose; SE, status epilepticus; NCSE, non-convulsive status epilepticus; STESS, Status Epilepticus Severity Score; LPDs, lateralized periodic discharges; mRS, modified Rankin Scale.

	General	DWI			T2WI		
		Restriction	No restriction	p	Signal abnormalities	No signal abnormalities	p
N	60	25 (41.7%)	35 (58.3%)	—	38 (63.3%)	22 (36.7%)	—
Sex, female, n (%)	30 (50%)	17 (68%)	13 (37.1%)	0.02	22 (57.9%)	8 (36.4%)	0.18
Age, years, mean (SD)	57.5 (18.5)	55.4 (14.8)	59.0 (17.6)	0.78	59.0 (16.9)	54.8 (21.1)	0.40
Previous epilepsy, n (%)	23 (38.3%)	9 (36.0%)	14 (40.0%)	0.18	13 (34.2%)	10 (45.5%)	0.16
Potentially fatal cause, n (%)	21 (35%)	13 (52%)	8 (22.9%)	0.02	18 (47.4%)	3 (13.6%)	< 0.01
Status epilepticus type, n (%):							
Convulsive	6 (10%)	4 (11.4%)	2 (8.0%)	0.18	2 (9.1%)	4 (10.5%)	0.32
Myoclonic	2 (3.3%)	0 (0%)	2 (8.0%)		0 (0%)	2 (5.3%)	
Focal motor	14 (23.3%)	9 (25.7%)	5 (20.0%)		5 (22.7%)	9 (23.7%)	
NCSE with coma	5 (8.3%)	1 (2.9%)	4 (16.0%)		0 (0%)	5 (13.2%)	
NCSE without coma	33 (55%)	21 (60.0%)	12 (48.0%)		15 (68.2%)	18 (47.4%)	
Level of consciousness, A-L/S-C, n (%)	43/17 (61.7/28.3%)	15/10 (60/40%)	28/7 (80/20%)	0.09	23/15 (60.5/39.5%)	20/2 (91.9/9.1%)	0.01
SE duration, hours, median (IQR)	51.46 (22.2-93)	67.5 (33-113)	40 (12.5-84)	0.12	53.62 (30.1-86)	51.2 (27.8-92)	0.65
Duration > 24 hours, n (%)	41 (68.3%)	21 (84%)	20 (57.1%)	0.04	28 (73.7%)	10 (52.6%)	0.242
STESS, median, (IQR)	2 (1-3)	3 (2-3)	2 (1-3)	0.04	3 (2-4)	2 (1-3)	< 0.01
LPDs, n (%)	17 (28.3%)	9 (36.0%)	8 (22.9%)	0.27	14 (36.8%)	3 (13.6%)	0.05
mRS deterioration at discharge, n (%)	27 (45%)	17 (68%)	10 (28.6%)	< 0.01	21 (55.3%)	6 (27.3%)	0.07

Table 2
Anatomic distribution of MRI abnormalities observed in the patients.

	T2 (n = 38) (63.3%)	DWI (n = 25) (41.7%)
Thalamus (Pulvinar nucleus)	4 (10.5%)	6 (24%)
Hippocampus-amygdala	11 (28.9%)	11 (44%)
Lateral temporal lobe	10 (26.3%)	8 (32%)
Insula	6 (15.8%)	8 (32%)
Extratemporal cortex	21 (55.3%)	10 (40%)

progressive SE vs remote SE (p = 0.04), and those in whom SE had a potentially fatal cause (47.4% vs 13.6%, p < 0.01) and was more severe (STESS 3 [IQR 2–4] vs 2 [IQR] 1–3), p < 0.01) (Table 2). Another related factor was the presence of LPDs in the EEG tracing (36.8% vs 13.6%, p = 0.05). Specific analysis of the time at which MRI was carried out showed that changes were detected more often in patients who had undergone the study more promptly after symptoms onset (73.3 h vs 128.4 h, p < 0.01) (Fig. 2). The best cut-off point for detecting MRI abnormalities in these patients was within the first 84 h after onset of the episode (86.2% vs 41.9%, p < 0.01).

In the analysis of associations between the imaging findings and underlying causes classified into groups, only the presence of acute cerebral lesions was independently associated with the presence of high signal on T2WI (p = 0.002). Logistic regression models adjusted by etiology yielded the following variables independently associated with the presence of edema on T2WI: baseline STESS (OR = 2.347; 95% CI 1.264–4.357; p = 0.007) and MRI performed within the first 84 h (OR = 11.540; 95% CI 2.247–59.269; p = 0.007).

3.3. * Restriction on DWI

The temporal (17, 68.0%) and frontal lobes (4, 16.0%) were the regions most commonly showing a restricted diffusion pattern (Fig. 1). Patients showing restriction on DWI were most often women (68% vs 37.1%, p = 0.02), with no differences related to age. With regard to the clinical characteristics of SE, restricted diffusion was seen more often in patients with SE having a potentially fatal cause (52% vs 22.9%, p = 0.02), in episodes lasting longer than 24 h (84% vs 60%, p = 0.04), and in more severe episodes according to the STESS (3 [IQR 2–3] vs 2 [IQR 1–3], p = 0.04) (Table 1). Once again, we found that DWI abnormalities were seen more often in patients undergoing an earlier examination, although the differences were not significant (74.1 h vs

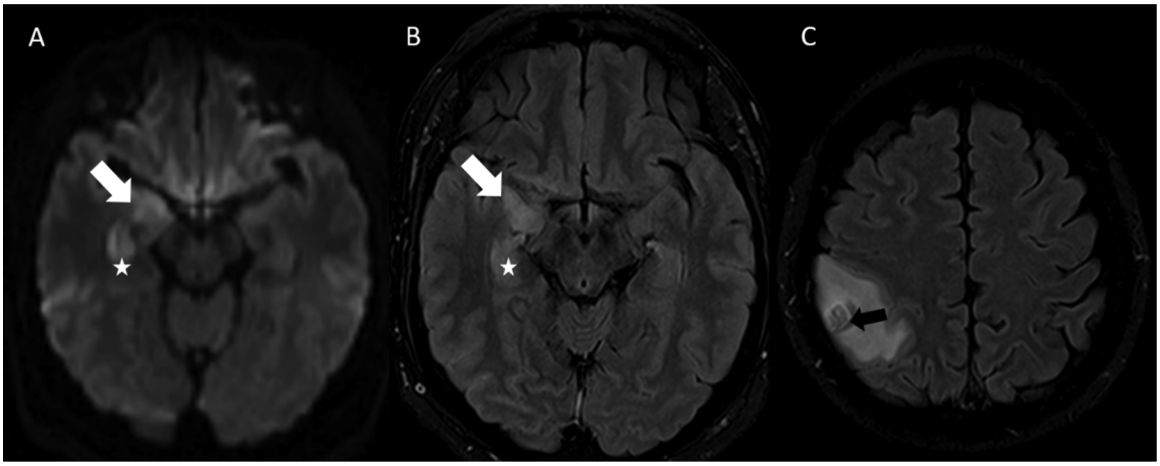

Fig. 1. Brain MRI in a patient with status epilepticus. A hyperintense area is seen on diffusion-weighted images (A and B), affecting the amygdala (arrow) and hippocampus (star) on the right side, related to cytotoxic edema. A right parietal metastatic lesion (arrow in C) surrounded by vasogenic edema is seen on an axial T2-FLAIR image.

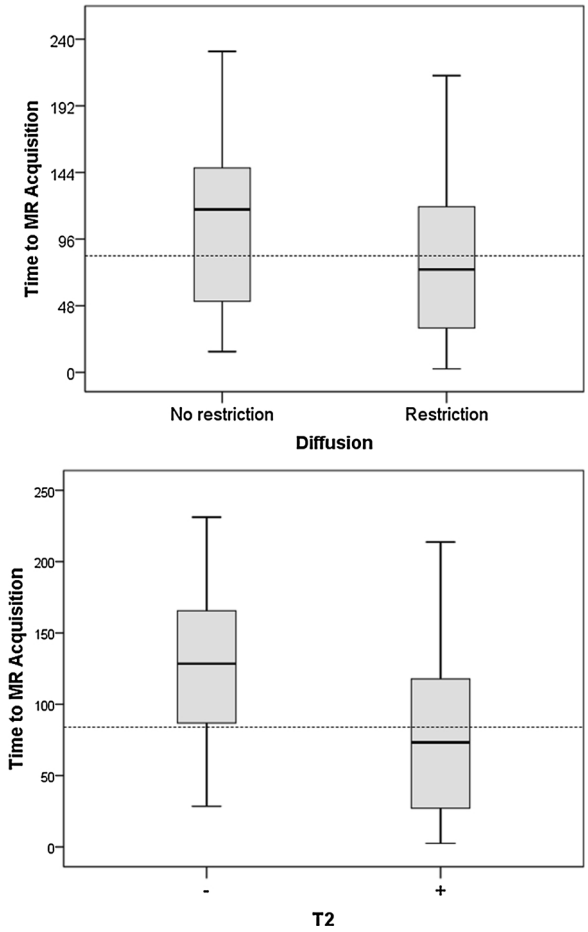

Fig. 2. Relationship between the time interval from status epilepticus onset to MRI examination and presence of MRI changes. Time elapsed was shorter in patients with T2WI ($p < 0.01$) and diffusion-weighted ($p = 0.22$) imaging abnormalities.

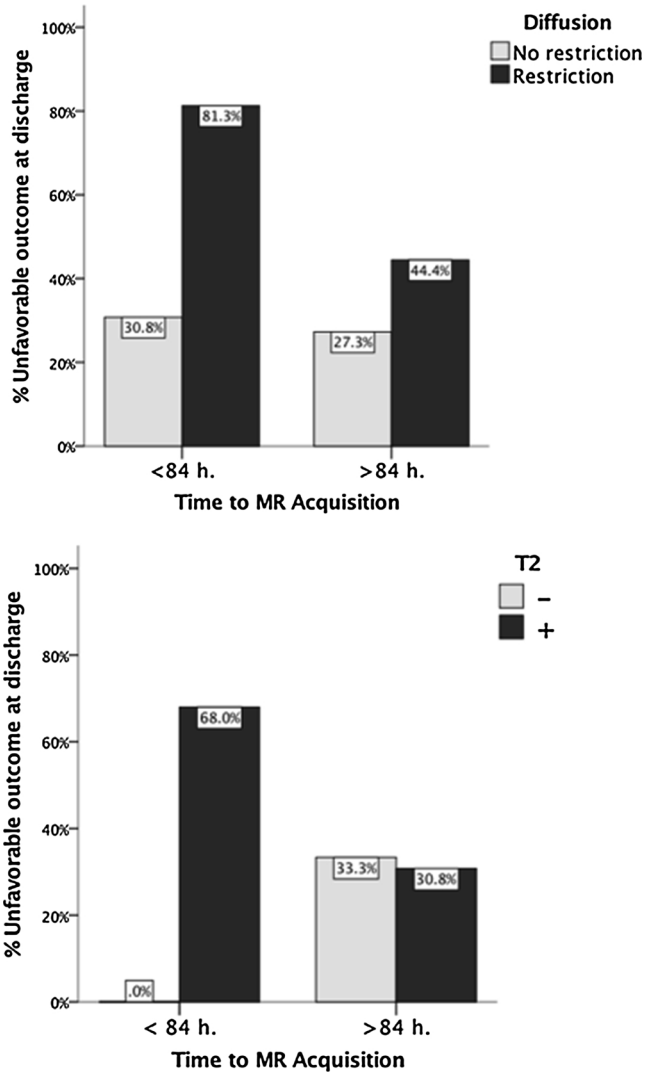

Fig. 3. Relationship between findings on T2WI and diffusion-weighted images and discharge outcome according to the time elapsed from status epilepticus onset to the MRI examination.

117.3 h, $p = 0.22$) (Fig. 1). Nonetheless, DWI abnormalities were found in a higher percentage of patients when MRI was carried out within the first 84 h than when it was performed after this time (55.2% vs 29.0%, $p = 0.04$).

On logistic regression analysis adjusted by etiology, the variables independently associated with the presence of DWI lesions were a potentially fatal underlying cause (OR = 7.698; 95%CI 1.378–42.997; $p = 0.020$), MRI performed within the first 84 h (OR = 4.321; 95%CI 1.035–18.033; $p = 0.045$), and SE duration > 24 h (OR = 6.192; 95%CI 1.304–29.416; $p = 0.022$).

3.4. Outcome at discharge and relationship with MRI findings

At discharge, 27 (45%) patients had unfavorable outcome. These included 19 patients with poorer functional status than before the SE episode and 8 patients who died. The clinical factors associated with an unfavorable outcome were a potentially fatal cause (51.9% vs 21.2%, $p = 0.01$), lower consciousness level (44.4% vs 15.2%, $p = 0.02$), SE duration longer than 65 h (63% vs 30.3%, $p = 0.01$), and higher STESS value (3 [IQR 2–3] vs 2 [IQR 1–3], $p = 0.03$) (Supplementary material). As to the MRI features, an unfavorable outcome was associated with detection of T2WI hyperintensities (77.8% vs 51.5%, $p = 0.04$) and areas of restricted diffusion (63.0% vs 24.2%, $p < 0.01$).

The variables independently associated with a poor outcome after adjusting by etiology were the presence of DWI restriction (OR = 4.511; 95%CI 1.242–16.338; $p = 0.022$), consciousness impairment (OR = 5.689; 95%CI 1.321–24.494; $p = 0.020$), and SE duration > 65 h (OR = 4.769; 95%CI 1.276–17.822; $p = 0.020$).

This association between the MRI findings and the patients' prognosis was more evident in the group that had undergone an earlier examination (< 84 h). Within this time window, DWI changes (81.3% vs. 30.8%, $p < 0.01$) and T2WI abnormalities (68% vs 0%, $p = 0.02$) were both significantly associated with an unfavorable outcome (Fig. 3).

4. Discussion

This study investigated relationships between MRI peri-ictal changes in SE patients and the patients' prognosis. The prevalence of DWI and T2WI abnormalities was 41.7% and 63.3%, respectively, values similar to those described in previous studies [2–9]. Our data show that these lesions were detected more often when MRI studies were performed within the first few days after the onset of the episode. Furthermore, they were related to the cause of SE and clinical factors associated with greater severity of the episode, and their presence on early studies was associated with a poorer functional outcome after SE had resolved. That is, restricted diffusion and T2WI signal abnormalities seen on MRI within the first 84 h were associated with an unfavorable outcome at hospital discharge.

The studies in this line published to date are mainly retrospective,

with the exception of one by Jabeen et al. [10], and overall, they do not establish a conclusive relationship between the MRI changes associated with SE and the patients' functional outcome. In the present study, a DWI restriction pattern and hyperintensity on T2WI sequences were related with clinical data indicating an unfavorable prognosis, in accordance with the most important studies published in this regard [4,8–10]. In the prospective study by Jabeen et al. [10], MRI abnormalities were more common in patients with a more prolonged SE episode, as was also seen in our series.

In at least some of cases and particularly in those related with structural lesions, these findings could be secondary to the lesion itself. On the other hand, a DWI restrictive pattern was usually seen at a distance from the lesions, often involving the limbic system or thalamus, and was equally represented in all the etiologic groups. Therefore, this finding was likely a direct consequence of SE [3,8,14].

In the largest study performed to date, which included 69 SE patients [9], MRI abnormalities were found to be related to a low level of consciousness, concurring with our results. Other reported factors related to peri-ictal MRI features in the remaining studies [4,8] such as the absence of previous epilepsy, higher score on the STESS scale, and LPDs on EEG study were also observed in our series [14]. However, beyond the results from these studies, we found that an independent association between the MRI findings and a poor functional outcome was primarily seen when the imaging examination was carried out promptly.

Detection of T2WI and DWI changes was significantly higher in SE patients undergoing MRI in the first 84 h after onset of the episode, and the findings obtained within this window of time had greater prognostic value. These data suggest that although the all MRI changes found cannot be conclusively defined as being secondary to SE [21], early examination with MRI may increase the value of this technique in SE patients.

With regard to the long-term prognosis, peri-ictal changes are potentially reversible, but they have been associated with neurological deficits in more than half the patients in some studies. Speech impairment, motor deficits, and the development of epilepsy at long term have been described in relation to SE-associated MRI changes, and these sequelae are consistent with the topography of the lesions [6,11]. Moreover, the presence of T2WI high signal or DWI restriction predicted poorer outcome independently of the etiologic subgroup, suggesting that SE-related edema may have prognostic value beyond the mortality risk associated with the underlying cause.

Some longitudinal studies have associated SE-related MRI abnormalities with laminar necrosis and atrophy on follow-up imaging [22–26]. Others, however, have reported that there are no long-term radiologic sequelae [8].

The retrospective design and the lack of protocolled follow-up studies to evaluate whether the imaging abnormalities found had reversed, are some of the limitations of our study. All the patients were not included during the study period due to the lack of conditions to acquire the MRI. Nonetheless, MRIs were performed following the same protocol in all patients during the acute phase and reassessed to find SE-related findings. Despite the limitations, our results suggest that SE-related findings in the acute MRI can be a prognostic marker. These results, added to the previously published, could be useful in clinical practice to take treatment decision.

5. Conclusion

Signal changes on DWI and T2WI sequences are seen in up to half of SE patients, and are more likely to be detected on examinations performed within the first 84 h after the onset of the episode. Regardless of the underlying cause of SE, the presence of edema predicted a poorer functional outcome in these patients.

Approval statement

This study was approved by the local ethics committee (PR(AG)33/2010) (Comité Ético de Investigación Clínica).

Consent statement

Due to the retrospective character of the study, the need for written informed consent was waived.

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Ethical publication statement

We confirm that we have read the Journal's position regarding ethical publication and affirm that this report is consistent with those guidelines.

Appendix A. Supplementary data

Supplementary data associated with this article can be found, in the online version, at https://doi.org/10.1016/j.seizure.2019.08.013.

References

[1] Trinka E, Cock H, Hesdorffer D, Rossetti AO, Scheffer IE, Shinnar S, et al. A definition and classification of status epilepticus – report of the ILAE task force on classification of status epilepticus. Epilepsia 2015;56(October (10)):1515–23. PubMed PMID: 26336950.
[2] Mendes A, Sampaio L. Brain magnetic resonance in status epilepticus: a focused review. Seizure 2016;38(May):63–7. PubMed PMID: 27156207.
[3] Cole AJ. Status epilepticus and periictal imaging. Epilepsia 2004;45(Suppl 4):72–7. PubMed PMID: 15281962.
[4] Goyal MK, Sinha S, Ravishankar S, Shivshankar JJ. Peri- ictal signal changes in seven patients with status epilepticus: interesting MRI observations. Neuroradiology 2009;51(March (3)):151–61. PubMed PMID: 19057899.
[5] Hong KS, Cho YJ, Lee SK, Jeong SW, Kim WK, Oh EJ. Diffusion changes suggesting predominant vasogenic oedema during partial status epilepticus. Seizure 2004;13(July (5)):317–21. PubMed PMID: 15158702.
[6] Raghavendra S, Ashalatha R, Krishnamoorthy T, Kesavadas C, Thomas SV, Radhakrishnan K. Reversible periictal MRI abnormalities: clinical correlates and long-term outcome in 12 patients. Epilepsy Res 2007;73(January (1)):129–36. PubMed PMID: 17125968.
[7] Huang YC, Weng HH, Tsai YT, Huang YC, Hsiao MC, Wu CY, et al. Periictal magnetic resonance imaging in status epilepticus. Epilepsy Res 2009;86(September (1)):72–81. PubMed PMID: 19541453.
[8] Di Bonaventura C, Bonini F, Fattouch J, Mari F, Petrucci S, Carni M, et al. Diffusion-weighted magnetic resonance imaging in patients with partial status epilepticus. Epilepsia 2009;50 Suppl 1(January):45–52. PubMed PMID: 19125848.
[9] Rennebaum F, Kassubek J, Pinkhardt E, Hubers A, Ludolph AC, Schocke M, et al. Status epilepticus: clinical characteristics and EEG patterns associated with and without MRI diffusion restriction in 69 patients. Epilepsy Res 2016;120(February):55–64. PubMed PMID: 26719998.
[10] Jabeen SA, Cherukuri P, Mridula R, Harshavardhana KR, Gaddamanugu P, Sarva S, et al. A prospective study of diffusion weighted magnetic resonance imaging abnormalities in patients with cluster of seizures and status epilepticus. Clin Neurol Neurosurg 2017;155(April):70–4. PubMed PMID: 28267656.
[11] Canas N, Breia P, Soares P, Saraiva P, Calado S, Jordao C, et al. The electroclinical-imagiological spectrum and long-term outcome of transient periictal MRI abnormalities. Epilepsy Res 2010;91(October (2-3)):240–52. PubMed PMID: 20728314.
[12] Xiang T, Li G, Liang Y, Zhou J. A wide spectrum of variably periictal MRI abnormalities induced by a single or a cluster of seizures. J Neurol Sci 2014;343(August (1-2)):167–72. PubMed PMID: 24950900.
[13] Chatzikonstantinou A, Gass A, Forster A, Hennerici MG, Szabo K. Features of acute DWI abnormalities related to status epilepticus. Epilepsy Res 2011;97(November (1-2)):45–51. PubMed PMID: 21802259.
[14] Giovannini G, Kuchukhidze G, McCoy MR, Meletti S, Trinka E. Neuroimaging alterations related to status epilepticus in an adult population: definition of MRI findings and clinical-EEG correlation. Epilepsia 2018(August). PubMed PMID: 30129213.
[15] Wilson JT, Hareendran A, Grant M, Baird T, Schulz UG, Muir KW, et al. Improving the assessment of outcomes in stroke: use of a structured interview to assign grades on the modified Rankin Scale. Stroke 2002;33(September (9)):2243–6. PubMed

PMID: 12215594.
[16] Leitinger M, Beniczky S, Rohracher A, Gardella E, Kalss G, Qerama E, et al. Salzburg consensus criteria for non-convulsive status epilepticus–approach to clinical application. Epilepsy Behav 2015;49(August):158–63. PubMed PMID: 26092326.
[17] Rossetti AO, Logroscino G, Milligan TA, Michaelides C, Ruffieux C, Bromfield EB. Status Epilepticus Severity Score (STESS): a tool to orient early treatment strategy. J Neurol 2008;255(October (10)):1561–6. PubMed PMID: 18769858.
[18] Rossetti AO, Hurwitz S, Logroscino G, Bromfield EB. Prognosis of status epilepticus: role of aetiology, age, and consciousness impairment at presentation. J Neurol Neurosurg Psychiatr 2006;77(May (5)):611–5. PubMed PMID: 16614020. Pubmed Central PMCID: 2117456.
[19] Novy J, Logroscino G, Rossetti AO. Refractory status epilepticus: a prospective observational study. Epilepsia 2010;51(February (2)):251–6. PubMed PMID: 19817823.
[20] Rossetti AO, Lowenstein DH. Management of refractory status epilepticus in adults: still more questions than answers. Lancet Neurol 2011;10(October (10)):922–30. PubMed PMID: 21939901. Pubmed Central PMCID: 3202016.
[21] Toledo M, Munuera J, Sueiras M, Rovira R, Alvarez-Sabin J, Rovira A. MRI findings in aphasic status epilepticus. Epilepsia 2008;49(August (8)):1465–9. PubMed PMID: 18522643.
[22] Bauer G, Gotwald T, Dobesberger J, Embacher N, Felber S, Bauer R, et al. Transient and permanent magnetic resonance imaging abnormalities after complex partial status epilepticus. Epilepsy Behav. 2006;8(May (3)):666–71. PubMed PMID: 16503204.
[23] Donaire A, Carreno M, Gomez B, Fossas P, Bargallo N, Agudo R, et al. Cortical laminar necrosis related to prolonged focal status epilepticus. J Neurol Neurosurg Psychiatr 2006;77(January (1)):104–6. PubMed PMID: 16361606. Pubmed Central PMCID: 2117425.
[24] Didelot A, Kremer S, Schmitt E, Maillard L, Gibot S, Cravoisi A, et al. MRI findings in a case of prolonged status epilepticus. J Neuroradiol 2006;33(April (2)):121–5. PubMed PMID: 16733426. Suivi iconographique d'un etat de mal epileptique prolonge.
[25] Wieshmann UC, Woermann FG, Lemieux L, Free SL, Bartlett PA, Smith SJ, et al. Development of hippocampal atrophy: a serial magnetic resonance imaging study in a patient who developed epilepsy after generalized status epilepticus. Epilepsia 1997;38(November (11)):1238–41. PubMed PMID: 9579926.
[26] Gong G, Shi F, Concha L, Beaulieu C, Gross DW. Insights into the sequence of structural consequences of convulsive status epilepticus: a longitudinal MRI study. Epilepsia 2008;49(November (11)):1941–5. PubMed PMID: 18494785.