Predictors of Early-Onset Seizures after Recent Intracerebral Hemorrhage among a Sample of Egyptian Patients

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Abstract: Background: Post-stroke seizures were reported in about one fifth of patients. Prediction is very important for early initiation of anti-epileptic treatment. **Aim of the work:** To evaluate the possible predictors of early post intracerebral hemorrhage seizures by clinical, laboratory, radiological or electrophysiological studies. **Patient and Methods:** The present study included 100 patients who presented with first time intracerebral hemorrhage within the first 24 hours. Patients were followed up for 7 days for the occurrence of seizures; and patients were classified into two groups; seizure group and non-seizure group. All were submitted for full history taking, clinical examination, laboratory investigations, computed tomography and electroencephalogram at admission and computed tomography were re-done after 48 hours. **Results:** There was statistically significant increase of stroke severity, blood pressure, serum glucose, and significant decrease of serum sodium in group with seizures when compared to group without seizures. CT examination revealed significant increase of lobar cortical site, increased hematoma volume and perihematomal edema and midline shift in group with seizures. Serum sodium, hematoma volume and site detected by CT at admission, hematoma expansion and intraventricular extension at 48 hours by CT and focal epileptiform discharge were the predictors of development of seizures in our patients. **Conclusion:** Results of the present study revealed that, many clinical parameters as low GCS, NIHSS, high blood pressure and high temperature, laboratory findings as decreased serum sodium level, radiological findings as Volume and site of hemorrhage detected by CT at admission, hematoma expansion and intraventricular extension at 48 hours by CT were associated with the onset of seizures, and focal epileptiform discharge detected by EEG were predictors for development of early seizures.

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1. Introduction

Stroke is the leading cause of mortality above 60 years of age and the fifth leading cause in people aged 15-59 year **(Reddy *et al.*, 2016).** Seizures are common complications after intracerebral hemorrhage (ICH), the reported incidence being approximately 10% **(Passero *et al.*, 2002).** Seizures by the time of onset after stroke usually classified as early or late, but the definitions for the time frame differ widely, although the cut-off point has generally been 1-2 weeks after the onset of the stroke **(Gilmore *et al.*, 2010)**. Only a small minority of patients with ICH later develop epilepsy **(Bladin *et al.*, 2000),** although prolonged seizures and/or status epilepticus could damage the brain and cause secondary brain injury such as herniation or re-bleeding **(Gilmore *et al.*, 2010).**

Electrographic seizures occur in 28% of patients with ICH during the initial 72 h after admission **(Vespa *et al.*, 2003),** and may be associated with expanding hemorrhages and periodic electrical discharges during cortical ICH **(Claassen *et al.*, 2007).**

Early Seizures after spontaneous intracerebral hemorrhage (ICH) can be explained by the sudden development of a space-occupying lesion with mass effect, focal ischemia, and blood products. Several studies report that the factors provoking seizures following ICH are related to hemorrhage volume, hemorrhage location within the cerebrum, cortical involvement, and the severity of neurological deficits **(De Herdt *et al.*, 2011; Rossi, *et al.*, 2013).**

Understanding of the risk factors of seizures following ICH is needed to predict which patient will require treatment. Literatures concerning seizures in patients with spontaneous intracerebral hemorrhage are few and data concerning identifying predictive factors of such seizures are lacking **(Berges *et al.*, 2000).**

The aim of the present study was to evaluate the possible predictors of early Seizures (within 1 week after the attack of hemorrhage) by (clinical, laboratory, EEG) and neuro-radiological parameters (CT brain) in patients with spontaneous intracerebral hemorrhage.

2. Methodology

The present study was designed as a prospective study conducted during the period from January 2015 to December 2016. It included 100 patients presented with first time intracerebral hemorrhage within the first 24 hours. All were selected from Al-Hussein University Hospital.

Inclusion criteria:

Patients with first time spontaneous ICH presented within the first 24 hours of their symptoms.

Exclusion criteria:

Patient with one or more of the following was excluded from the study: 1) traumatic ICH; 2) subarachnoid hemorrhage, arteriovenous malformations, subdural hematoma, hemorrhagic infarct, or inflammatory vascular disease; 3) hemorrhage caused by a primary or metastatic brain tumor; 4) preexisting neurological deficits such as stroke, head trauma and hypoxic ischemic encephalopathy; 5) epilepsy prior to ICH and positive family history; 6) occurrence of seizures after 7 days from the start of symptoms.

Patients were followed up for 7 days for the occurrence of seizures; and patients were classified into two groups; seizure group and non-seizure group.

On admission for their stroke, detailed medical characteristics included were as follows: sex, age, National Institute of Health Stroke Scale (NIHSS) score on admission (1-4 stands for mild stroke; 5-15 stands for moderate stroke; 16-20 moderate to severe stroke; and 21-42 for severe stroke) **(Glymour *et al.*, 2007),** lesion and etiology of ICH, treatment, presence of the first seizure and modified Rankin Scale (mRS) score at discharge (0 = no disability, 1= No significant disability, able to carry out all usual activities, despite some symptoms; 2= Slight disability, able to look after own affairs without assistance, but unable to carry out all previous activities; 3= Moderate disability, requires some help, but able to walk unassisted; 4= moderately severe disability, unable to attend to own bodily needs without assistance, and unable to walk unassisted; 5= Severe disability, requires constant nursing care and attention, bedridden, incontinent; and 6= dead).

Laboratory investigations included the following: 1) Random blood sugar (RBS) level; 2) serum creatinine; 3) sodium; 4) potassium and calcium.

Radiological investigation in the form of computed tomography (CT) brain was done for all patients at admission and after 48 hours of presentation. At admission the scan was done to detect and confirm presence of ICH. The site of hematoma classified as lobar cortical, lobar subcortical, thalamic, basal ganglionic, brain stem and cerebellar). The size of hematoma was categorized into two groups: 1) small (<30 cc) and 2) large (≥ 30 cc). The size was calculated using ABC/2 method. The CT slice with the largest area of hemorrhage had to be identified. (A) equal the largest cross-sectional diameter, while B equals the largest diameter perpendicular to A on the same slice, and C equals the approximate number of 10 mm slices on which the ICH is seen. A×B×C/2 equals approximately the volume of an ellipsoid **(Kothari *et al.*, 1996).** Other signs on CT brain such as intraventricular extension, perihematomal edema and midline shift also were documented. The second CT scan was done for detection of hematoma expansion, midline shift, intra ventricular extension, perifocal edema and hydrocephalic changes. Electroencephalography (EEG) within the first 48 hours of symptoms was done and the following abnormalities were assessed: 1) focal epileptiform discharges. 2) diffuse slowing. The patients received standard medical treatment according to the department protocol, No surgery was done to any patient. The study protocol was approved by the local ethics committee in Al-Azhar University after obtaining informed consent.

Statistical analysis:

Data are expressed as mean ± standard deviation (SD) for continuous variables and as relative frequencies and percentages for discrete variables. Comparison between two groups was done by unpaired student (t) test for continuous variables and Chi square (X^2) for categorical variables. *P* value < 0.05 was considered significant. All statistics were done by statistical package for social science (SPSS) computer package version 16 (SPSS Inc., Chicago, USA) running on IBM-compatible computer.

3. Results

As regarding to patient characteristics, there was no significant difference between group with seizures and those without seizures as regard to age or sex distribution. However, the NIHSS score at admission revealed high score (more severe stroke) in group with seizures; and GCS was significantly lower in group with seizures. Finally, there was no significant difference between both groups as regard modified Rankin scale (mRS) at discharge (Table 1).

As regard clinical data, there was statistically significant increase of systolic BP, diastolic BP and temperature in group with seizures when compared to group without seizures (166.8±12.8, 118.0±10.7 and 38.0±0.3 vs 159.9±9.6, 106.4±10.2 and 37.9±0.1 respectively). Laboratory data also revealed significant increase of blood glucose and significant decrease of serum sodium in group who developed seizures (128.5±26.9 and 136.9±3.85 vs 112.9±23.3 and 138.6±2.9 respectively) (Table 2).

Ass regard CT findings, there was statistically significant increase of lobar cortical site in group with seizures when compared to group without seizures

(85% vs 5.0% respectively). In addition, there was significant increase of large volume (>= 30 cc) in group with seizures (90.0%) when compared to group without seizures (13.8%). Also, edema around hematoma, hematoma expansion, midline shift was significantly increased in group with seizures. At 48 hours after admission, there was significant increase of hematoma expansion, midline shift, and intraventricular extension in group with seizures. The EEG changes showed significant increase of focal epileptiform discharge in group with seizures when compared to no seizures (55% vs 0.0% respectively (Table 3).

Table (1): Patient characteristics, stroke severity and disability at discharge of studied populations

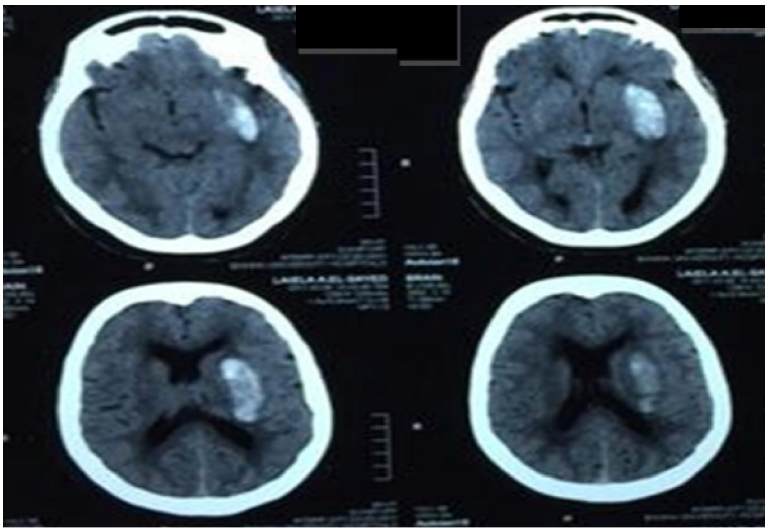
Variable		Seizures (n=20)	No seizures (n=80)	test	*P*
Age		56.0±8.9	55.1±6.8	0.48	0.62(ns)
Sex	Male	13(65.0%)	49(61.3%)	0.10	0.75(ns)
	Female	7(35.0%)	31(38.8%)		
NIHSS Score	Moderate stroke	10(50.0%)	65(82.3%)	19.18	<0.001*
	Moderate to sever	6(30.0%)	14(17.7%)		
	Severe stroke	4(20.0%)	0(0.0%)		
	Mean±SD	18.3±6.0	11.4±3.3	6.85	<0.001*
GCS		10.6±1.2	11.8±1.0	4.61	<0.001*
mRS at discharge	2	2(10.0%)	7(8.8%)	5.77	0.12(ns)
	3	7(35.0%)	43(53.8%)		
	4	10(50.0%)	30(37.5%)		
	5	1(5.0%)	0(0.0%)		
	Mean ±SD	3.5±0.8	3.3±0.6	1.30	0.19(ns)

Table (2): clinical and laboratory data in studied populations

Variable	Seizures (n=20)	No seizures (n=80)	test	*P*
Age	56.0±8.9	55.1±6.8	0.48	0.62(ns)
Systolic BP	166.8±12.8	159.9±9.6	2.64	0.010*
Diastolic BP	118.0±10.7	106.4±10.2	4.53	<0.001*
Temperature	38.0±0.3	37.9±0.1	2.63	0.010*
Blood glucose	128.5±26.9	112.9±23.3	2.58	0.011*
Creatinine	0.72±0.2	0.73±0.20	0.37	0.70(ns)
Sodium	136.9±3.85	138.6±2.9	2.23	0.028*
Potassium	3.91±0.24	3.90±0.23	0.1	0.98(ns)
Calcium	8.9±0.6	9.0±0.3	0.37	0.71(ns)

Table (3): CT and EEG findings in studied populations

	Variable		Seizures (n=20)	No seizures (n=80)	test	*P*
CT At admission	Site	Lobar cortical	17(85.0%)	4(5.0%)	62.45	<0.001*
		Lobar subcortical	0(0.0%)	7(8.8%)		
		Thalamic	0(0.0%)	33(41.3%)		
		Basal ganglia	3(15.0%)	36(45.0%)		
	Volume	Small (<30cc)	2(10.0%)	69(86.3%)	45.18	<0.001*
		Large (>=30cc)	18(90.0%)	11(13.8%)		
	Edema around hematoma		13(65.0%)	12(15.0%)	21.33	<0.001*
	Midline shift		14(70.0%)	6(7.5%)	39.6	<0.001*
	Side	Right	14(70.0%)	51(63.8%)	0.27	0.60
		Left	6(30.0%)	29(36.3%)		
CT At 48 hours	Hematoma expansion		6(30.0%)	5(6.3%)	9.21	0.002*
	Midline shift		14(70.0%)	7(8.8%)	36.18	<0.001*
	Intraventricular expansion		5(25.0%)	4(5.0%)	7.81	0.015*
	Edema around hematoma		1(5.0%)	0(0.0%)	4.04	0.20(ns)
	Hydrocephalic changes		1(5.0%)	1(1.3%)	1.14	0.28(ns)
EEG changes	Diffuse slowing		8(40.0%)	31(38.8%)	0.02	0.91(ns)
	focal epileptiform discharge		11(55.0%)	0(0.0%)	49.43	<0.001*



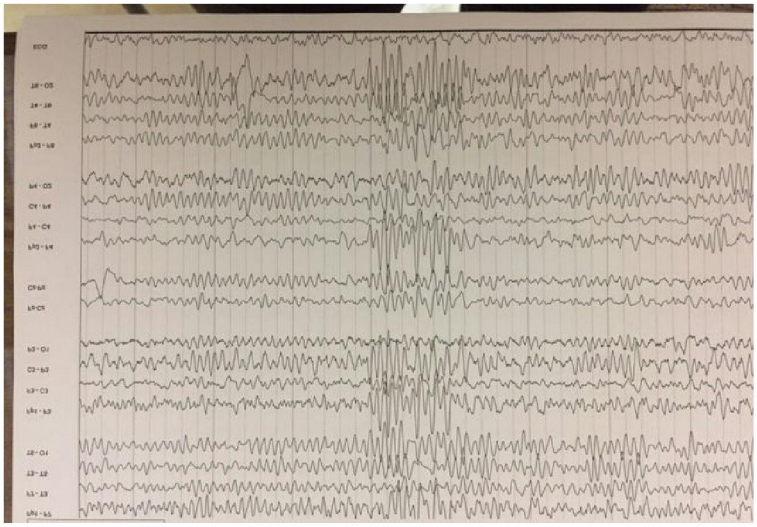


Figure (2): EEG shows ikes with contralateral propagation of the epil

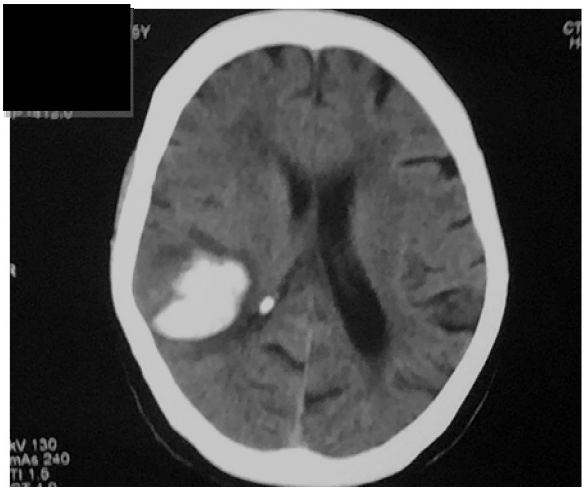


Figure (3) Right parital hemorrhage with surrounding edema

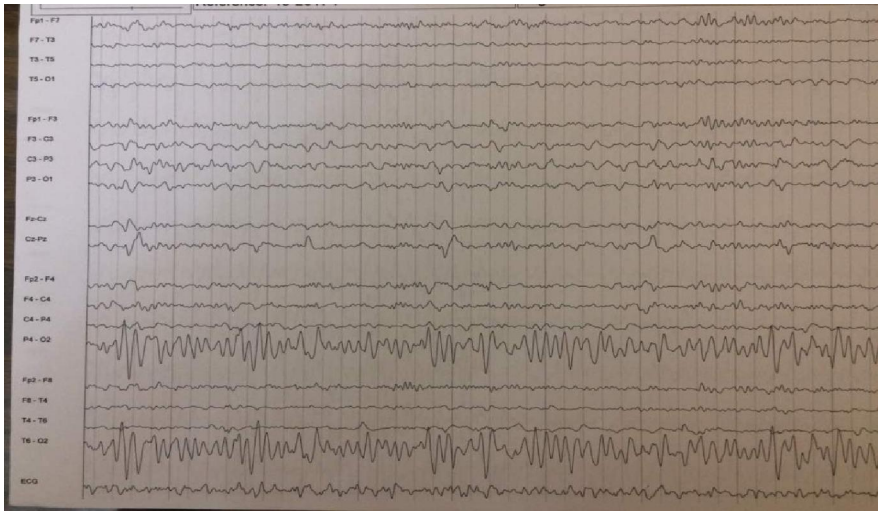


Figure (4) Right parito temporal focal spikes and slowness

Running regression analysis, we found that, serum sodium, Volume and site detected by CT at admission, hematoma expansion and intraventricular extension at 48 hours by CT and focal epileptiform discharge were the predictors of development of seizures in our patients; using these variables in multivariate analysis, all stay as predictors.

4. Discussion

According to the ILAE, seizures after stroke are classified as early seizures (ES) when they occur within 7 days of a stroke, and late seizures (LS) when they take place over 7 days after a stroke **(Guidelines for Epidemiologic Studies on Epilepsy, 1993).** In the current study, post-stroke epilepsy was defined according to the most recent report by the ILAE **(Fisher *et al.*, 2014),** which indicated that patients with a single seizure episode associated with an enduring condition that could cause epilepsy (such as stroke) met the criteria for epilepsy.

In the present work, the incidence of seizures after intracerebral hemorrhage was 20%. This percentage lies close to that reported in the previous literature s of **Bladin *et al.* (2002) Passero *et al.* (2002) and Vespa *et al.* (2003),** which reported that, early seizures occur in 2.7–17%, after ICH and the majority of seizures occurring at, or near the onset of ICH. In addition, **Osama *et al.* (2014)** reported that, of the seventy patients with spontaneous ICH included in the study, 18.6% developed seizures within the first 7 days of presentation. Also, **Beghi *et al.* (2011)** reported incidence rate of 16.2%. On the other hand, **Neshige *et al.* (2015)** reported that the incidence of post-stroke seizures (PSS) in 1920 patients with ICH is 6.6%, and that seizures occur frequently following a cortical hemorrhage. The incidence rate of late seizures after ICH is reported to be 2.0–26.1% and the wider variance is attributed to the divergent definitions of late seizures between studies. Some reports define late seizures as seizure occurring 24 h after ICH (**De Reuck *et al.*, 2007; Andaluz and Zuccarello, 2009; Rossi *et al.*, 2013).**

Sung *et al.* (1999) reported a low prevalence of 12%, while **Bladin *et al.* (2000)** reported 10.6%. On the other hand, **Lancman *et al.* (1993)** reported prevalence of 25%, while **Claassen *et al.* (2007)** reported 31%, and **De Reuck *et al.* (2007)** reported 42.9%. These variations could be attributed to the heterogeneity in methodology, time windows to define early seizures, study designs and differences in use of neuroimaging techniques **(Osama *et al.*, 2014).**

In the early stage after ICH, the mechanisms resulting in early seizures have been implicated in a combination of the sudden development of a space-occupying lesion with mass effect, focal ischemia, and blood products **(Bladin *et al.*, 2000).** During the late stage after ICH, the mechanisms contributing to seizure include abnormal electrical discharge due to gliosis or irritability after brain injury. However, the exact mechanism producing seizure in ICH remains unclear **(Neshige *et al.*, 2015).**

In the present study, there was statistically high significant increase in the mean systolic and diastolic blood pressure at time of admission in patients of seizure group than in patients of non-seizure group. These results agree with those reported by **Krakow *et al.* (2010)** who considered arterial hypertension as one of the parameters that associated with early post-stroke seizures.

In the present study, we found higher statistical significant increase of temperature in group that developed seizures when compared to those not develop seizures. These results are in agreement with **Osama *et al.* (2014)** who found similar situation **and**

Kammersgaard *et al.* (2005) who considered fever as the most apparent mechanism promoting the seizures. Other factors like administration of antibiotic drugs or metabolic disturbances related to infection might also affect the seizure threshold. These were confirmed in the present work by the significant increase of glucose levels and significant decrease of sodium in patients developed seizures. **De Herdt *et al.* (2011)** considered hyperglycemia and accumulation of metabolites decrease the threshold of epileptogenesis.

In the present work, we found that, subcortical location, large size of hematoma, hematoma expansion, intraventricular extension, focal epileptiform discharge and serum sodium were predictors of early seizures occurrence. These results are comparable to those reported by **Qian *et al.* (2014)** who reported that, a subcortical location of the hematoma, a low GCS score on admission and a large hematoma volume to significantly predict immediate seizures after ICH, in the present work GCS score was significantly low in group developed seizures when compared to those did not develop seizures.

Subcortical location is a well-known risk factor for seizures after ICH **(Passero *et al.*, 2002; Rossi *et al.*, 2013).** In addition, **Arboix *et al.* (1997)** reported that cortical location founded to be associated with early seizures (75.9%). **Giroud *et al.* (1994)** reported that lobar location is a potent predictor of early post stroke seizures. Meta-analysis by **Zhang *et al.* (2014)** found that, subcortical location to be the powerful predictor of early onset post-stroke seizures. These results confirmed by the present study.

Conflicting observations have been reported previously concerning the role of hematoma size, however, as both small **(Passero *et al.*, 2002; Weisberg *et al.*,1991)** and large hematomas **(Yang *et al.*, 2009)** have been found to increase the risk of seizures, and there have even been reports of no significant effect of hematoma volume on the seizure risk **(Woo *et al.*, 2012; De Herdt *et al.*, 2011).** Our result supports the findings of those studies as immediate seizures were more common among patients with large hematomas.

Midline shift on CT scan at the time of admission was statistically significant more frequent in the seizure group than in non-seizure group, but multivariate analysis could not found it as a predictor of early seizure occurrence. These results are in agreement with **Bladin *et al.* (2000) and Berges *et al.* (2000)** who reported that intracranial shift did not influence the occurrence of early seizures. On the other hand, **Vespa *et al.* (2003)** reported that increase in midline shift was associated with occurrence of seizures.

Computed tomography 48 hours after admission demonstrated that hematoma expansion and midline shift was statistically highly significantly more frequent in the seizure group. Results also confirmed that hematoma expansion and intraventricular extension were predictors for early seizures. **Yang *et al.* (2009)** reported that any increase in ICH volume increases the seizure rate by 2.7%. Intraventricular extension as a predictor of early seizures was explained by association of intraventricular hemorrhage with marked midline shift. On the other hand, **Bladin *et al.* (2000)** reported that IVH did not influence the occurrence of early post stroke seizures. **Vespa *et al.* (2003)** reported that there is a trend for higher ICH volumes on follow-up CT in patients with electrographic seizures. They also reported greater midline shift on CTs obtained 48 to 72 hours after hemorrhage for patients with seizures.

In the present study, focal epileptiform discharge detected by EEG was statistically significant increase in seizure group and were a predictor for early development of seizures. These results are in agreement with **Arboix *et al.* (1997) and Claassen *et al.* (2007).).** However, EEG shows variety in the literature due to the difference in timing, duration and type of recording. Thus, its role in prediction of early post-stroke seizures is questioned **(Claassen *et al.*, 2007).**

Stroke severity measured by NIHSS and GCS scores revealed increased severity in those developed seizures when compared to those without seizures. But, multivariate analysis did not found stroke severity to be a predictor of early onset seizures. Previous studies reported the association between stroke severity and post-stroke seizures. However, different assessment methods, such as the Scandinavian Stroke Scale (SSS), Glasgow Coma Scale, Barthel Index, and NIHSS, were used to assess stroke severity. **Jungehulsing *et al.* (2013) and Lossius *et al.* (2002)** have found that stroke severity could predict seizure onset using the Barthel Index and SSS, respectively. However, **Kammersgaard and Olsen (2005)** and **Graham *et al.* (2013)** showed opposite results.

Conclusion

Results of the present study revealed that, many clinical parameters as low GCS, NIHSS, high blood pressure and high temperature, laboratory findings as decreased serum sodium level, radiological findings as Volume and site of hemorrhage detected by CT at admission, hematoma expansion and intraventricular extension at 48 hours by CT were associated with the onset of seizures, and focal epileptiform discharge detected by EEG were predictors for development of early seizures.

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