Original Research Article

Indian Journal of Emergency Medicine
Volume 4 Number 3, July - September 2018
DOI: http://dx.doi.org/10.21088/ijem.2395.311X.4318.3

Retrospective Analysis of Continuous EEG in Spontaneous Intracerebral Hemorrhage: A Study from Central India

Sunil Athale[1], Dinesh Chouksey[2], Rahul Jain[3], Akshay N Lakhotia[4]

Author's Affiliation:
1Professor and HOD [2,3]Assistant Professor [4]Ex Resident, Neurology Department, Sri Aurobindo Medical College and Post Graduate Institute, Indore, Madhya Pradesh 453555, India.

Corresponding Author:
Dinesh Chouksey, Assistant Professor, Neurology Department, Sri Aurobindo Medical College and Post Graduate Institute, Indore, Madhya Pradesh 453555; India.

E-mail: dineshmd09@yahoo.com

Received on 13.06.2018,
Accepted on 11.07.2018

Abstract

Background: Intracerebral hemorrhage (ICH) is a neurological emergency and it is the most severe form of acute stroke, accounting for 15–25% of all strokes, and 50% of stroke mortality [1,2]. The intracerebral hemorrhage complicated by seizures is often a life threatening condition. The subclinical seizures (Electrographic seizure) also gained attention even if they are infrequent, to prevent further ongoing neuronal damage [3]. EEG provides insight into the thalamocortical functions in patients with impaired consciousness and will help to detect the subclinical seizures before clinical manifestation and at the time of reversible stage. Role of prophylactic antiepileptic in ICH is still a matter of discussion. Subclinical seizures are identified with the help of continuous EEG monitoring. The goal of our study is to look for the EEG changes in different ICH locations and identification of electrographic seizures. ***Aim:*** To look for EEG changes in different locations of ICH and to identify the electrographic seizures. ***Method:*** A Retrospective observational study of 46 patients of ICH admitted in Department of Neurology, SAMC and PGI, Indore from January 2012 to March 2017. ***Result:*** Total 16 patients (35%) had EEG abnormalities, 8 of them having abnormal epileptiform activities and rest 8 were having generalized low amplitude fast Beta activity. The electrographic seizures were seen in 4 patients (9%) and all of them having subcortical bleed. Two patient of cortical bleed had clinical seizures and were showing abnormal epileptiform activities in EEG record. Commonest site of ICH in our study was Basal ganglia and commonest cause is hypertension. ***Conclusion:*** The continuous EEG monitoring in ICH patients is cost effective to identify subclinical seizures because they are detrimental to these patients. Our observation suggests it is better to use prophylactic antiepileptic drugs in lobar bleeds.

Keywords: EEG in ICH; Nonconvulsive Seizures in ICH; Antiepileptic Drugs Inich; Seizures in ICH; Subclinical Seizure

Introduction

Intracerebral hemorrhage (ICH) is a neurological emergency and it is the most severe form of acute stroke, accounting for 15–25% of all strokes, and 50% of stroke mortality [1,2]. The acute phase of an ICH is frquently complicated by seizures and possibly reflecting the disruptive effect of the hematoma and edema. The clinical seizure are known to cause neuronal death, primarily from activation of NMDA-mediated glutamatergic excitotoxic pathways. The subclinical seizures (Electrographic seizure) also gained attention evevn if they are infrequent to prevent further ongoing neuronal damage [3]. EEG provides insight into the thalamocortical functions in patients with impaired consciousness and will help to detect the subclinical seizures before clinical manifestation and at the time of reversible stage. Vespa et al. [5] reported electrographic seizures in 28% ICH patients and found their association with increasing midline shift. ICH was the reported cause of status epilepticus in 19% patients [2]. Ongoing nonconvulsive seizure activity is detected in 14% of patients after convulsive status epilepticus [4]. There were no such study which demonstrate different type of EEG changes in different locations of spontaneous ICH. We just want to describe our findings of EEG in different locations that will may give insight in

future for larger scale study. There is still dilema to give prophylactic antiepileptic drugs to patients of all types of ICH, we also observe this burning issue in our small study. The goal of our study is to look for the EEG changes in different ICH locations and identification of electrographic seizures.

Material and Methods

This study was conducted in rural based neurology intensive care unit of Sri Aurobindo Institute of Medical Science and PG Institute, Indore located in central India. We retrospectively studied all patients with spontaneous ICH who underwent continuous EEG monitoring at the time of admission from January 2012-March 2017. All the included patients were managed on the basis of published guidelines for treatment of ICH [8,9]. Prophylactic antiepileptic medications were not given routinely; only those who developed clinical seizures were treated (Table 3).

Continuous EEG recording was done on RMS (21 channel Maximus version 4.2.54) with electrodes placed according to international 10-20 placement system by trained EEG technician and reported by experienced faculty of Department of Neurology. The continuous EEG monitoring was started on the day of admission and was continued for duration upto 48 hours.

All acute spontaneous intracerebral hemorrhage patients of any age group presenting with baseline metabolic profile, Brain imaging and continuous EEG record were included in the study.

Those with traumatic ICH or subarachnoid hemorrhage and incomplete data were excluded.

After the evaluation of EEG record the predesigned proforma consisting of demographic details (including name, age, gender, residential address, contact details, date of admission, length of hospital stay) was filled and clinical, radiological diagnosis was noted from clinical records.

Outcome of patient were subdivided into expired, left against medical advice, discharge after physician advice (from ICU/Ward) and entered in to proforma (Table 1).

Table 1: Demographic table

Variables	Number of patients	Percentage (%)
a) Gender		
Male	29	63
Female	17	37
b) Age		
≥ 50 years	29	63
< 50 years	17	37
c) Stage of presentation		
Hyperacute	26	56.5
Acute	11	24
Early subacute	6	13
Late subacute	3	6.5
d) Clinical seizures		
GTCS	9	19.6
Focal	3	6.5
No	34	73.9
c) Site		
Basal ganglia	24	52.2
Thalamic	14	30.5
Brainstem	3	6.5
Cerebellar	2	4.3
Temporal lobe	1	2.2
Frontal lobe	1	2.2
Intraventricular	1	2.2
d) Sensorium		
Drowsy	19	41.3
Comatose	9	19.6
Conscious	18	39.1
e) Outcome		
Discharge	22	47.8
Expired	3	6.5
Leave against medical advice	21	45.6
f) Etiology		
Hypertensive	44	95.6
Coagulopathy	1	2.2
Vascular malformation	1	2.2

Table 2: Site of bleed and associated electrographic abnormalities.

Site of bleed	EEG abnormalities	Number of patients	Percentage (%)
1. Basal ganglia without intraventricular extension	Right mid temporal continuous spike discharges	1	2.2
	Left centrotemporal sharp waves with left side theta activity	1	2.2
	Low amplitude Beta activity	3	6.5
	Normal	12	26
2. Basal ganglia with intraventricular extension	Low amplitude beta activity	2	4.4
	Normal	5	10.8
3. Thalamus without intraventricular extension	Intermittent non rhythmic theta activity	1	2.2
	Low amplitude beta activity	1	2.2
	Normal	7	15.2
4. Thalamus with intraventricular extension	Left anterior temporal polyphasic periodic discharges	1	2.2
	Left anterior frontal phase reversal with left hemispheric sharp waves	1	2.2
	Normal	3	6.5
5. Brainstem (Pons) without intraventricular extension	Low amplitude beta activity	2	4.4
	Normal	1	2.2
6. Cerebellum without intraventricular extension	Normal	1	2.2
7. Cerebellum with intraventricular extension	Normal	1	2.2
8. Temporal lobe with intraventricular extension	Right hemispheric spike and wave discharges	1	2.2
9. Frontal lobe	Generalized slowing	1	2.2
10. Intraventricular	Left focal PLED's	1	2.2

Table 3: showing seizures in ICH patients.

Variables	Number	Cortical location	Subcortical location
Clinical seizure with EEG abnormality	4	2	2
Clinical seizure without EEG abnormality	8	0	8
Electrographic seizure	4	0	4

Metabolic parameters were also entered in this proforma which consisted of random blood sugar (mg/dl), serum sodium, potassium, blood urea (mg/dl), serum creatinine (mg/dl).

Electrographic seizures was defined as rhythmic discharges or a spike-and-wave pattern with definite evolution in frequency, location, or morphologic features lasting at least 10 seconds; evolution in amplitude alone did not qualify [10].

Stages of ICH [11] were divided into hyperacute with less than 24 hours duration, acute with 1-3 days duration, early subacute with more than 3 days duration and late subacute of more than 7 days duration.

Results

This retrospective study included 46 patients of intracerebral hemorrhage. 29 patients (63%) were male and remaining were of female gender (37%). Sixty three percent of patients belong to more than 50 years of age. Out of total 46 patients 26 were (56.5%) presented in hyper acute stage, 24 % (n=11) of patients reported in acute stage whereas 13% (n=6) and 6.5% (n= 3) reported in early and late subacute stage respectively.

Clinical seizures were present in 12 patients (26%) of ICH. Electrographic seizures were recorded in 4 patients (9%) and no seizures were recorded in 30 (65%) patients.

Commonest cause of ICH was hypertension (95.6%) whereas coagulopathy and vascular malformation was the etiology in remaining 2 patients.

The EEG abnormalities were seen in total 16 patients and commonest finding in 8 patients

(17.4%) was low amplitude beta activity.

Basal ganglia was the most common site of bleed in 24 patients (52.2%) and associated intraventricular extension was in 7 patients. Youngest patient seen was 30 years old while eldest was 80 years old. Duration of hospital stay ranged from minimum 1 day to maximum 64 days. Five patients had generalized tonic clonic seizures and 1 patient had focal seizure.

Thalamus as the second most common site of hemorrhage was found in 14 patients (30.5%). There was associated intraventricular extension in 5 patients.

There were 6 patients of basal ganglia bleed who had clinical seizures and 2 of them having classical epileptiform discharges and 4 of them were not showing any interictal EEG abnormalities. Five patients of Basal ganglia bleed without clinical seizure were having generalized low amplitude beta waves in their continuous EEG record. Three patients with thalamic bleed had electrographic seizures with classical epileptiform discharges on continuous EEG record. Thalamic bleed patients did not have any clinical seizures.

Brainstem hemorrhage was found in 3 (6.5%) patients. All were male patients with age in the range from 49-60 years. There were no clinical and electrographical seizures seen in all three patients.

There were 2 patients (4.4%) who had cerebellar bleed. One patient had associated intraventricular extension. Age of patient was 50 and 70 years respectively. Metabolic derangement in the form of hypernatremia was evident in one of the patient. Both presented in hyperacute stage of bleed and their EEG record was normal.

One patient had pure intraventricular hemorrhage with generalized tonic clonic seizure and his EEG record was showing left focal periodic lateralized epileptiform discharges.

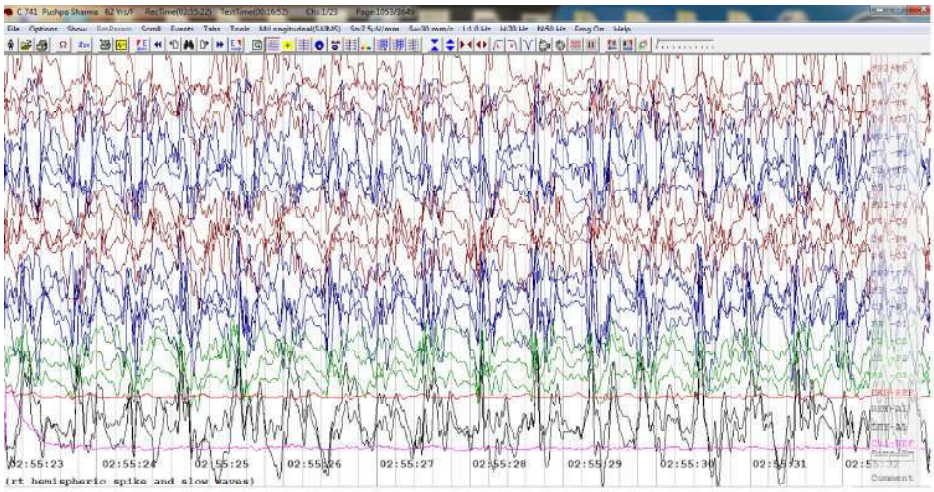
Temporal lobe as site of bleed was present in one patient (2.2%) presented in hyperacute stage with clinical seizure and his EEG showing interictal right hemispheric spike and wave epileptiform discharges (Figure 1).

There was one patient of right frontal lobar bleed (2.2%) possibly due to vascular malformation who had clinical seizures with EEG record showing generalized high amplitude frontal predominant slow waves.

Discussion

This study reports the profile of ICH patients and EEG findings with its role in identification of subclinical seizure. In our study deeply located subcortical bleeds were the commonest whereas intraventricular was rarest one. Hypertension was the commonest cause of intracerebral hemorrhage.

The clinical seizure occurred in 12 (26.1%) patients and electrographic seizures or subclinical seizure occurred in 04 patients (09%).The incidence of seizures after spontaneous hemorrhage have been reported ranging from 2.8 to 18.7% from various studies [4]. The debate of prophylactic antiepileptic drugs (AED) in ICH is still going on and several studies reported that prophylactic AEDs has to given in frontal and cortical location ICH. In previously published studies [5,13] it was established that lobar hemorrhage likely to produce more seizures than deep hemorrhage, we found



EEG 1 of patient of right temporal bleed with intraventricular extension showing right hemispheric spike and slow waves.

similar results as two of our patients with cortical bleed had clinical seizures.

In a study done by Vespa et al., [5] seizures occurred in 21% of subcortical bleed patients and he also reported worsened outcome after intracerebral hemorrhage complicated by seizures. In our study total 13 (28.2%) patients of sub cortical bleed were having seizures and 9 of them had clinical seizures and 4 of them had electrographical seizures.

The interictal EEG abnormality were seen in only 4 of the total 12 patients with clinical seizure of intracerebral hemorrhage. This will support the already established fact of approximate 50% sensitivity of EEG in detecting interictal epileptiform abnormality. The total patients with abnormal epileptiform discharges were 8 (17.4%) including 4 patients with subclinical seizures. This finding give us insight in significant role of continuous EEG in detecting seizure because it is detrimental to the patients, contributing to his sensorium status and lead to neuronal death if present for long duration. Identification and treatment of such seizure will improve the prognosis of ICH patient. The fact of continuous EEG monitoring burden have to be taken in to consideration because it will requires thorough time taken review by a qualified person and management of technical factors and cost effectiveness of the procedure. Future studies in large scale will required to answer the questions of need of continuous EEG in ICH patients and prophylactic use of antiepileptics in deep seated ICH too.

Electrographic epileptiform abnormalities of patients ranged from sharp waves, spike and slow wave to periodic discharges and low amplitude fast waves to generalised slow waves. Genjiro Hirose et al. [12] in his study of intracerebral hemorrhage also found similar EEG findings (Table 2).

Electrographic abnormalities as per location by Neidermyer [14] which included ipsilateral delta activity, lack of sleep spindles, reduction in alpha rhythm (in anteroventral thalamic bleed), alpha enhancement in posterior thalamic lesion. Diffuse theta waves from midbrain and preserved alpha rhythm that cannot be blocked by various modality of stimuli, diffuse low voltage tracings from lower brainstem lesions. Cerebellar lesions had high voltage delta activity in contralateral cerebellar hemisphere.

Generalised low amplitude fast Beta activity (13-30 Hz) as an ictal onset pattern is [14] already known however this finding not much described in previous studies. We had found this activity in 8 patients although no patient with this finding had clinical seizure. Low amplitude fast Beta waves are described in conditions like sedative drug effect and coma with other causes [15].

Thalamus with its extensive cortical and limbic networks has been established as a generator of spike wave discharges and also as a subcortical pacemaker of epilepsy. Gloor et al. [16], investigated the location of structural pathology that produced localized, lateralized or generalized EEG slow activity. Thalamic lesions generally produces focal or unilateral delta activity however the slow delta activity varied in time of onset, amplitude and degree of focality.

Electrographic findings were described in 32 patients with circumscribed thalamic lesions (68%) by Elene Tsoures et al. [17] They were heterogeneous and comprised regional and generalized slowing, triphasic waves, generalized periodic and regional epileptiform discharges. Regional slowing was associated with ipsilateral thalamic lesions, epileptiform discharges were related to lesions in the ipsilateral medial thalamus and periodic generalized discharges/triphasic waves with lesions in the anterior-ventromedial thalamus. Epileptic seizures were also more common in patients with medial thalamic lesions. Patients with regional epileptiform discharges responded to antiepileptic treatment whereas patients with triphasic waves and generalized periodic patterns did not. In our study electrographic abnormalities in thalamic bleed included low amplitude beta activity, polyphasic periodic discharges and sharp waves. Electrographic abnormalities in basal ganglionic bleed patients in our study ranged from low amplitude beta activity to sharp, spikes waves with theta activity.

Classen et al. [13] in his study found periodic discharges, frontal intermittent delta activity, stimulus induced rhythmic period discharges in EEG of hemorrhage patients.

The study gives an insight into electrographic abnormalities in intracerebral hemorrhage patients specially deep seated ICH and significant presence of sub clinical seizures in these patients. There is dearth of studies reported from developing countries.

Study was single center based with limited number of patients and retrospective nature of the study are limiting factors of study.

Conclusion

The current study brings into attention that seizures are frequent complication of intracerebral

hemorrhage and may be a target for improving ultimate outcome. Seizures can occur in both lobar and deep hemorrhage and with or without associated intra ventricular extension patients. The study calls for need of prospective study to correlate electrographic abnormalities with location of bleed and outcome of patients those having seizures with ICH; to further refine management of intracerebral hemorrhage patients in future.

Source(s) of support: No

Acknowledgement: Nil

Conflict of interest: Nil

References

1. Badjatia N Rosand J. Intracerebral hemorrhage. Neurologist 2005;11:311-324.
2. Poon MT Fonvite AF Al-Shahi Salman R. Long term prognosis after ICH: systemic review & meta analysis. J. Neurol Neurosurgery psychiatry 2014;85:660-7.
3. Nathan B Fountain. Is it time for routine EEG monitoring after ICH? Neurology 2007;69:1312-13.
4. Kwang meo woo, Segung yeob yang,Keun tee cho. Seizure after spontaneous intracerebral hemorrhage. J. Korean Neurosurgery Soc. 2012 Oct;52(4):312-319.
5. Vespa PM, O'phelan K, Shah M, Mirabelli J, Starkman S, Kidwell C, Saver J, Nuwer MR, Frazee JG, McArthur DA, Martin NA. Acute seizures after intracerebral hemorrhage A factor in progressive midline shift and outcome. Neurology. 2003 May 13;60(9):1441-6
6. Young BG, Jordan KG, Doig GS. An assessment of nonconvulsive seizures in the intensive care unit using continuous EEG monitoring an investigation of variables associated with mortality. Neurology. 1996 Jul 1;47(1):83-9.
7. Alroughani R, Javidan M, Qasem A, Alotaibi N. Non-convulsive status epilepticus; the rate of occurrence in a general hospital. Seizure. 2009 Jan 31;18(1):38-42.
8. Morgenstern LB, Hemphill JC, Anderson C, Becker K, Broderick JP, Connolly ES, Greenberg SM, Huang JN, Macdonald RL, Messé SR, Mitchell PH. Guidelines for the management of spontaneous intracerebral hemorrhage. A guideline for healthcare professionals from the American Heart Association/American Stroke Association. Stroke. 2010 Sep;41(9):2108-29.
9. Hemphill JC, Greenberg SM, Anderson CS, Becker K, Bendok BR, Cushman M, Fung GL, Goldstein JN, Macdonald RL, Mitchell PH, Scott PA. Guidelines for the management of spontaneous intracerebral hemorrhage. Stroke. 2015 Jul 1;46(7):2032-60.
10. Chong DJ, Hirsch LJ. Which EEG patterns warrant treatment in the critically ill? Reviewing the evidence for treatment of periodic epileptiform discharges and related patterns. Journal of Clinical Neurophysiology. 2005 Apr 1;22(2):79-91.
11. Atlas SW, editor. Magnetic resonance imaging of the brain and spine. Lippincott Williams & Wilkins; 2009.
12. Hirose G, Saeki M, Kosoegawa H, Takado M, Yamamoto T, Tada A. Delta waves in the EEGs of patients with intracerebral hemorrhage. Archives of neurology. 1981 Mar 1;38(3):170-5.
13. Classen J, Jette N, Chum F, Green R, Schmidt M, Choi H, Jirsch J, Frontera JA, Connolly ES, Emerson RG, Mayer SA. Electrographic seizures and periodic discharges after intracerebral hemorrhage. Neurology. 2007 Sep 25;69(13):1356-65.
14. Niedermeyer E, da Silva FL, editors. Electroencephalography: basic principles, clinical applications, and related fields. Cerebrovascular disease and EEG. Lippincott Williams & Wilkins; sixth edition; page367.
15. Stern JM. Atlas of EEG patterns. Lippincott Williams & Wilkins; 2005.
16. Gloor P, Ball G, Schaul N. Brain lesions that produce delta waves in the EEG. Neurology. 1977 Apr 1;27(4):326.
17. Tsoures E, Lewerenz J, Pinkhardt E, Ludolph AC, Fauser S. Electroencephalographic findings in patients with circumscribed thalamic lesions. Send to Epilepsy Res. 2017 Sep;135:115-122.