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ORIGINAL ARTICLE

Role of Acute Symptomatic Seizures and Hippocampal Sclerosis in Development of Post-Stroke Epilepsy

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ABSTRACT

Aim: To analyze the risk and role of acute symptomatic seizure and hippocampal sclerosis in the development of post-stroke epilepsy.
Study Design: Retrospective study.
Place and duration of study: Department of Neurology, Gujranwala Medical College, Gujranwala from 1st July 2021 to 31st December 2021.
Methodology: One hundred patients were enrolled on the basis of acute ischemia or hemorrhagic stroke occurrence. These two conditions were confirmed through MRI imaging. Atrophy formation, alterations in signals visibility in T1 as well as T2 weighted-sequences in addition to internal architecture loss with atrophy of hippocampal was the MRI criteria for diagnosis of hippocampal sclerosis. A control group was also generated of fifty patients to assess the clinical diagnostics of hippocampal sclerosis patients against normal controls.
Results: There were 57% males and 43% females with a mean age of 74.2±13.2 years. Ischemia was seen in 40% of the cases while hemorrhagic stroke was present in 62% of all the patients enrolled. Acute symptomatic seizures were present in 30 cases while it was not observed in 70 other cases. The EEG of hippocampal sclerosis presented fronto-parietal stroke and left parieto-temporal strokes while in controls with majority as cerebral venous sinus thrombosis, in addition to basal ganglia hemorrhage.
Conclusion: Acute hemorrhage from stroke in combination with the acute and symptomatic seizure results into epileptic episodes and in many cases to secondary formation of hippocampal sclerosis.
Keywords: Acute symptomatic seizures (ASS), Hippocampal sclerosis, Post-stroke epilepsy

INTRODUCTION

Neuropathological lesions such as hippocampal sclerosis are the most commonly presenting with a high frequency of childhood occurrence. Epilepsy of temporal lobe which is resistant to drugs is an underlying associated condition. This type of epilepsy is observed in almost 70% of cases to be presented since their childhood as mentioned earlier.[1] The acquiring of hippocampal sclerosis in either youth or adulthood is a rare event. Within these ages it could be described as an inflammatory condition or a neurocysticercosis disease[2] or human-herpes[3] as well as autoimmune-limbic encephalitis[4]. Aging in addition to neurodegenerative ailment can also be the underlying cause.[5,6] However, acute cerebrovascular illness has not been reported as a reason for hippocampal sclerosis in youth or adults.

Epileptic event or complete epilepsy can be a stroke complication but not frequently. In a cohort study it was recorded that 9% of the patients with stroke had also suffered from acute symptomatic seizures (ASS). These types of seizures are termed as provoked seizures with a double hemorrhagic risk than seen in ischemic stroke patients[7]. Late onset of epilepsy was documented in only 2.5 percent of the stroke cases[8].

The previous term provoked seizures have now been replaced by acute seizure with both referring to the same condition with seizure being formed related to situation.[9] Within recent years it has been observed that MRI lesions in acute symptomatic seizures are not always benign and have been later observed for high relapse risk which is similar to the risk seen in genetic-epilepsy cases[10,11]. The present study was designed for analyzing this risk and contribution of acute symptomatic seizure and hippocampal sclerosis in formation of post-stroke epilepsy. The results of this study will assist in providing reliable and authentic data for future prevention of avoidable events and healthier outcomes.

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MATERIALS AND METHODS

This retrospective study was performed at Department of Neurology, Gujranwala Medical College, Gujranwala from 1st July 2021 to 31st December 2021. A total of 100 patients were included on the basis of acute ischemia or hemorrhagic stroke occurrence. These two conditions were confirmed through MRI imaging. Patients who were included in this study had no previous cerebrovascular disease history or episodes of epileptic seizures. Moreover, the inclusion criteria also relied on registering only those patients whose second MRI was conducted post two year of last stroke. Identification of neurological deficits as well as existing deficits aggravation was the main aim for opting this inclusion criteria and ruling out novel lesions in the patients. Ethical approval as well as patient consent was obtained before study beginning. Those patients whose age were below 18 years and had pre-existed lesions/atrophy of hippocampus or of brain injury were excluded. A complete analysis of patient's medical files was done for obtaining relevant medical information including demographic details and clinical data. Symptomatic seizure was termed as focal /generalized seizure temporary occurrence in clearly known settings. Atrophy formation, alterations in signals visibility in T1 as well as T2 weighted-sequences in addition to internal architecture loss with atrophy of hippocampal was the MRI criteria for diagnosis of hippocampal sclerosis. A control group was also generated of fifty patients to assess the clinical diagnostics of hippocampal sclerosis patients against normal controls. Acute symptomatic seizures were reserved in cases where post seven days of stroke still a seizure appeared. The sample size of this study was 30 which was based on sample size calculations using 80% power of test and 95% confidence interval. Data analysis was based on chi square and Mann Whitney test of SPSS version 25.0 where a comparative analysis of acute symptomatic seizures with EEG was done as spikes against non-spikes as well as types of strokes. A p value < 0.001 was taken significant.

RESULTS

There were 57% males and 43% females with a mean age of 74.2±13.2 years. Ischemia was seen in 40% of the cases while hemorrhagic stroke was presented in 62% of all the patients enrolled. Out of these 62% patients 42 patients had primary aetiology while twenty had vascular malformations. Ischemia was presented in 40% of the cases (Table 1).

Acute symptomatic seizures were presented in 30 cases while it was not observed in 70 other cases. In the cases where acute symptomatic seizures was present 83.3% of the cases were having epilepsy while on the contrast in patients with no acute symptomatic seizure only 5 cases reported with epilepsy. The year of epilepsy formation in ASS present and absent groups were ranging between 2-5 years with 3.8±2.1 and 4.6±2.5 years respectively and statistically the significant ($P<0.01$) difference between years of epilepsy formation and epilepsy (Fig. 1).

Mann Whitney results showed that there was no significant difference seen in probability of gender and age. The medical history presented with Cholangiocarcinoma, basilar migraine in Hippocampal sclerosis while in control group relevant medical history was seen. The EEG of hippocampal sclerosis presented fronto-parietal strokeand left parieto-temporal strokes while in controls with majority as cerebral venous sinus thrombosis, in addition to basal ganglia hemorrhage. Follow up MRI showed no hippocampal sclerosis in control group (Table 2).

Cerebral MRI of patients was performed at the initial and post stages. The results presented no hippocampal sclerosis in the initial MRI while it could be observed at later stages. In few patients (4 cases) Acute hemorrhagic lesion formation with symmetrical hippocampi presence at the site of the stroke and hippocampal sclerosis were main findings at two year's post stroke (Fig. 2).

Table 1: Frequency of age, gender and clinical features (n=100)

Variable	No.	%
Gender		
Male	43	43.0
Female	57	57.0
Mean age (years)	74.2±13.2	
Hemorrhagic stroke	62	62.0
a. Primary aetiology	42	67.7
b. Vascular malformation	20	32.2
Ischemia	40	40.0

Table 2: Comparison between Hippocampal sclerosis and normal control patients

Variable	Hippocampal sclerosis	*Control*	P value
Male/Female	43/57	88/71	1.00
Age	74.2±13.2	78.3 ± 14.6	0.12
Medical history	Cholangio-carcinoma, 50% L carotid stenosis, Basilar migraine	Relevant	0.22
ASS	30 (30)	14 (28%)	0.068
MRI	L fronto-parietal stroke L parieto-temporal stroke	Majority of Stroke with few of cerebral venous sinus thrombosis, and basal ganglia hemorrhage	Stroke localization: $p = 0.13$ Lateralization of stroke (L VS R): $p = 0.62$
EEG	L parieto-temporal spikes, L fronto-temporal slowing, L parietal slowing	Routine EEG; presented IEDs	0.06
Type of stroke	Hemorrhagic	Ischemic, hemorrhagic	1.00
Follow-up MRI	L HS	No HS	NA

L: left, HS: hippocampal sclerosis

Fig. 1: Comparison of epilepsy within acute symptomatic seizure and without acute symptomatic seizure cases

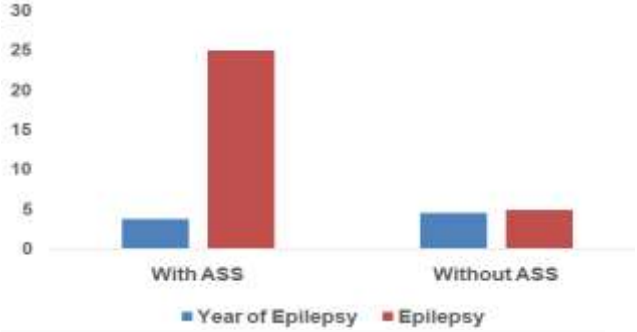
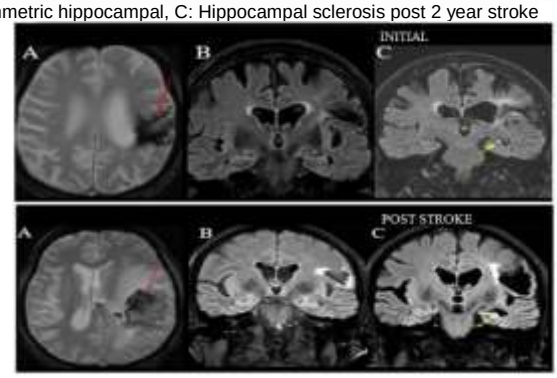


Fig 2: Initial and post-stroke MRI images; A: acute hemorrhagic lesion, B: symmetric hippocampal, C: Hippocampal sclerosis post 2 year stroke



DISCUSSION

Epilepsy association has strongly been found with the primary as well as secondary stroke and a 2.5x greater chance of epilepsy formation in primary/secondary stroke[12]. This can be attributed to presence of acute symptomatic seizures. Previously intracranial hemorrhagic patients' reports have shown a presence of 10 to 16% of acute symptomatic seizure prevalence[13,14]. The current study results present a high incidence of epilepsy formation in cases with acute symptomatic seizure post stroke[15] with emphasis on focal epilepsy as predicted through the epilogentic-discharge observed in the EEG. Similar results have been elaborated in various other researches[16,17].

There were a few patients who developed hemorrhagic and stroke a hippocampal sclerosis was seen to an extent. The precise mechanism involved in development of such a severity and hippocampal sclerosis is not properly understood due to limited data availability in the related context.[18,19] In hippocampal sclerosis patients with renal inadequacy there is a high risk of secondary vasogenic-edema formation.[20,21] However, the present study found no relevant evidence for the aforementioned state.

It is significant to observe that all the hippocampal alterations were observed on the left side. The number of cases reported in the current study was not that sufficient to confirm this condition and might be related to chance. Still literature supports that left side is more vulnerable to hippocampal sclerosis than the right side. Child hood associated temporal-lobe epilepsy has also been reported for its strong association with epilepsy formation than right side[22-24].

CONCLUSION

Acute hemorrhage from stroke in combination with the acute and symptomatic seizure results into epileptic episodes and in many cases to secondary formation of hippocampal sclerosis.

Conflict of interest: Nil

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