

A CROSS SECTIONAL STUDY ON EEG PROFILE IN ACUTE ISCHEMIC STROKE IN TERTIARY CARE

Medicine

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KEYWORDS

INTRODUCTION

Stroke is responsible for a high disability, mortality and economic burden worldwide. Emergency treatments like intravenous thrombolysis and mechanical thrombectomy can improve outcomes. Surface electroencephalography (EEG) shows promise for stroke identification and outcome prediction[1]. The use of EEG in the acute ischemic stroke (AIS) in the clinical routine is rather limited nowadays, as neuroimaging plays the most important role in diagnosis and evaluation. EEG use in Stroke Centers is usually restricted to post-stroke epilepsy or to differential diagnosis of possible seizures as stroke mimics[2].

AIMS AND OBJECTIVES

The EEG changes in ischemia was described long ago: a) decreased beta-range fast activities; b) increased slowing in theta and delta ranges; c) loss of normal background rhythms such as the alpha rhythm; and d) decreased overall amplitude[3]. Electroencephalography detects changes in brain function immediately following onset of brain ischemia, before cell death. It can be used as an advantage for early pre hospital stroke diagnosis in mobile units. Electroencephalography (EEG) provides a direct measure of the functional neuroelectric activity in the brain, which may be used to assess neural function and predict post-stroke recovery.[4]

Computerized tomographic (CT) scans done within 24 hours after an ischemic stroke may be appear normal initially, though CT abnormalities eventually. It is useful to have a test verifying the clinical impression of stroke and its location immediately. Magnetic resonance imaging (MRI) are more sensitive, but MRI is often impractical on an emergency basis. EEG sensitivity (76%) for cortical ischemic stroke is good. Although focal EEG slowing is not specific for ischemic stroke, it does document the presence and lateralization of a lesion affecting cortical function, which can confirm the clinical impression of stroke. [5]

METHODOLOGY

We aimed to evaluate the value of routine EEG in a group of AIS (Acute Ischemic stroke) patients. Stroke patients who were admitted in Stroke Unit, Neurology Department(MIN), Madras Medical college in February 2023 2nd week were included for study. The diagnosis of AIS was made after history taking, neurological examination and MRI. Demographic details including name, age, risk factors, neurological deficits due to stroke, NIHSS score, MRI imaging are entered. After formal consent EEG was done by portable apparatus with standard protocol.

All patients underwent EEG with 21 electrodes placed according to the 10–20 system (19 scalp electrodes plus 2 ear reference electrodes) using a digital EEG record. Bipolar longitudinal (double banana) and average referential montages were used for evaluation. The EEG filter configuration was as follows: 50 Hz filter; low pass filter: 0.5 Hz; high

pass filter: 70 Hz. EEG was evaluated visually with all available channels, as it is for a normal EEG interpretation. EEG was considered abnormal if any of the following abnormality found. 1. Delta/theta Focal slowing 2. Diffuse slowing 3. Periodic discharges 4. Alpha wave amplitude and frequency reduction 5. asymmetry[6].

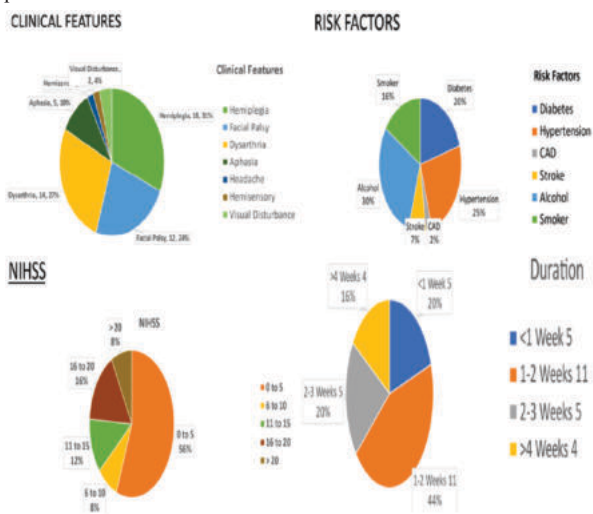
RESULTS

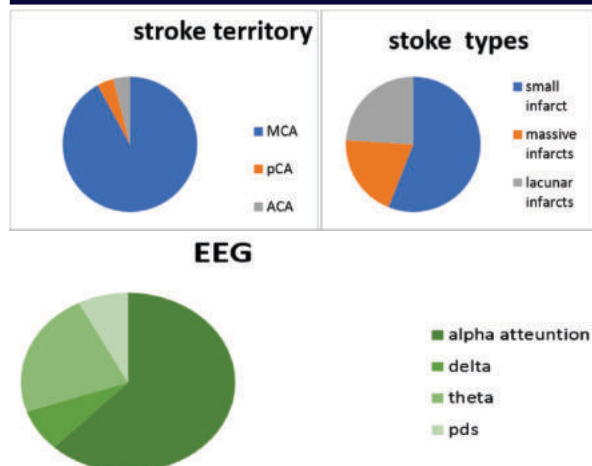
EEG was done for total of 25 ischemic stroke (MCA-23, ACA-1, PCA-1) patients. Mean age was 56 years (range 32–80). Patients were mostly men (19; 76%). Median NIHSS score on admission was 7.24 (range: 0–26). The most common stroke pattern were small infarcts (14; 56%) (massive MCA 5, 20%), and lacunar (6; 24%).

Most strokes were located in the anterior circulation (23; 92%), while strokes in the posterior and anterior circulations each one (8%). As about 56% of our patients had a rather low initial NIHSS score (≤ 5 points).

Among 25 patients EEG were abnormal in 13 patients. All massive MCA infarcts had abnormal EEG. Out of 6 lacunar infarct one was abnormal. In remaining 14 small infarcts 8 were abnormal. EEG was normal in 12 patients (48%). Among normal EEG 5 were lacunar infarcts and 7 were small infarcts.

Within the abnormal EEG in 13 patients (52%), asymmetry noted in all 13 patients (52%), alpha amplitude reduction 10 patients (76%), theta slowing noted in 3 patients (23%), delta slowing 1 (8%), periodic sharp wave discharges 1 patient (8%). Asymmetry noted in electrode channels over ischemic regions. Abnormal EEG was seen in patients with large infarcts, high NIHSS score and with less than 1 week period of stroke onset.





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DISCUSSION

Common visual EEG abnormalities in stroke include, ipsilateral attenuation or loss of faster alpha activity (8–13 Hz). slow activity like delta (1–4 Hz), theta (5–7 Hz) waves. About 52 % of our patients had an abnormal EEG. Similar figures can be found in older studies, between 48% and 86%, depending on stroke population and stroke size [2,12]. One patient with lacunar stroke had pathological EEG (periodic sharp waves). It is known that lacunar strokes are much less probable to cause EEG changes, in up to 9% in the literature [7]. None of the patients had a history of seizures, post-stroke seizures during the hospital stay (9 ± 6 d).

Alpha amplitude reduction was the most common manifestation of EEG changes (76 % of EEG abnormalities). Patients with abnormal EEG had significantly higher NIHSS scores on admission. As the EEG reflects the brain dysfunction occurring due to the neuronal damage alpha amplitude reduction, theta slowing, delta slowing occurred in the same lateralization as the ischemic lesion. As the NIHSS score corresponds to the severity and in most cases size of the infarct, this finding seems to be in agreement with previous observations of EEG changes being more common in larger ischemic lesions [7].

EEG features predicting poor prognosis, among others, were unilateral presence of delta and theta activity in previous studies. Another study also included visual EEG analysis and reported that the degree of background abnormality, i.e. slowing of the background resting state activity analogous to generalized slowing, was most important in the prediction of outcome [8].

Limitations

This study has several limitations. The sample size is rather small and the follow-up was limited only to hospitalization time. This paper does not intend to ignore sophisticated EEG evaluation techniques like quantitative EEG analysis. We kept the evaluation quite simplistic, as we aimed to assess the value of the normal, easily accessible, routine EEG in a setting with a typical Stroke Unit. This brings the study closer to the reality of clinical routine and may facilitate practical implementation of the results, even in the absence of available quantitative EEG analysis.

CONCLUSIONS

We conclude that EEG abnormalities described for stroke can be used in prehospital and transport settings to detect ischemic strokes early, differentiate from stroke mimics and for prognosis to plan treatment. Abnormal EEG and generalized slowing in particular are associated with clinical deterioration after AIS. Of course, larger, more detailed studies are needed to confirm our results.

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