DISCUSSION

Despite improving resuscitation practices, the outcome of most patients after a cardiac arrest remains poor. Overall, out-of-hospital cardiopulmonary resuscitation (CPR) for cardiac arrest has a success rate of 10%.[83] Even when patients are resuscitated in the hospital, fewer than one in five patients survive to discharge.[84]

 when severe, a post-resuscitation anoxic ischemic encephalopathy leaves patients comatose, awakening generally takes place within 3 days after CPR, and neurologic impairment is expected if a patient fails to do so.[85] These patients are often left in a severely cognitively disabled and fully dependent state; some remain in a minimally conscious or vegetative state, and very few awaken neurologically intact. [86]

In the last few decades, several clinical and electrophysiological variables have been reported to be strongly associated with a poor outcome in comatose survivors of cardiac arrest; these include absence of pupillary and corneal reflexes, absent motor response to pain [87] myoclonus or epilepticus status [88] , an increase of neuron specific enolase (NSE) in serum [89], and a burst-suppression , isoelectric electroencephalography (EEG) pattern.[88] However, in most cases, evidence predictive of poor outcome remains to be determined.[89]

Although the absence of various cranial nerve reflexes beyond day 1 as found in a study by young is strongly supportive of lack of neurological recovery, such reflexes are most often regained, even in those who fare badly. [90]

As regards demographic data in our study, there was no significant difference between bad and good outcome groups as regards age and sex. This was the same results in other studies about this subject.[91-93]

Circumstances of arrest as place, mode of arrest, and resuscitation time showed no significant difference between bad and good outcome groups. In large prospective study involving 774 patients, who had a 6-month rate of death of more than 80%, variables that were not strongly correlated with a poor outcome included age, sex, cause of arrest, type of arrhythmia (e.g., ventricular fibrillation or asystole), total arrest time, and duration of CPR.[92]

Regarding our study, corneal reflex was lost in 8 patients on day 1 but on day 3, it was lost in 6 patients, both groups had poor prognosis as GOS had mean (1.17 ± 0.41) and (1.25 ± 0.46) respectively. While pupillary reflex was lost in 18 patients on day 1 but on day 3 it was lost in 6 patients, both groups had poor prognosis as GOS had mean (1.17 ± 0.41) and (1.44 ± 0.62) respectively.

The absence of a pupillary reaction to light suggests a poor prognosis but has unclear specificity when assessed early after a cardiac arrest. In prospective studies involving 491 patients, 152 of Who had an absent pupillary light reflex on hospital admission. However, all

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108 patients whose pupillary reactions were absent at day 3 after cardiac arrest had poor outcomes.[94]

In two prospective studies, the absence of a corneal reflex at 72 hours was associated with no false positives for a poor outcome.[94]

In our study we found that time of resuscitation showed no significant difference between good and bad outcome however in other studies which included out hospital arrest cases showed that duration of resuscitation are related to poor outcome after CPR.[95]

In this result Glasgow Coma Scale (GCS) showed lower scores in bad outcome group than good outcome group, also there was positive significant correlation between GCS with glascow outcome score(GOS).

In other study it was reported that Glasgow Coma Scale motor score of 2 at 72 hours had an FPR of 0% (95% CI 0% to 3%) for predicting poor outcome.[100] Also the Glasgow Coma Scale score especially a low motor component score is associated with poor outcome in study by Sacco et al.[101] Also in study by chan there was significant coorelation between GCS after arrest and GOS.[95]

Regarding brain edema there was no significant difference between bad and good outcome groups however Torbey et al. showed a correlation between GM/WM ratios measured at the level of the basal ganglia and neurological outcomes in patients after ROSC[98]

Regarding myoclonus in our patients, myoclonus is present in 4 patients (19%) in bad group with no statistically significant correlation with GOS. Compared to our results, in a group of 67 patients with epileptic myoclonus 22% had a good outcome, myoclonic phenomena in the 1st 3 days were predictors of poor outcome.[99] Nevertheless, Young et al concluded that the presence of purposeful movements and EEG reactivity within the first 3 days after cardiac arrest suggest a more favourable prognosis.[100]

Our results regarding EEG findings revealed that 21 patients had bad outcome (vegetative or dead). Burst suppression was found in 6 patients on first day and 3 patients on seventh day. Unreactive background was found in 2 patients on first day and in 5 patients on seventh day. Low voltage background < 20 microvolts was found in 13 patients on first and seventh day. EEG on first day, burst suppression have PPV of 88.24 % topredict bad outcome, while unreactive background and low voltage background had PPV of 100 % to predict bad outcome.

As regards EEG on seventh day, burst suppression have PPV of 94.74 % topredict bad outcome, while unreactive background had PPV of 100 % and low voltage background had PPV of 88.89 % to predict bad outcome.

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In a study by Young et al., patients with burst suppression or who had low voltage less than 20 μV did not survive.[101] While in a study by Rossetti et al., patients with burst suppression, unreactive background or low voltage < 20μv had PPV OF 100% to predict bad outcome.[102]

In the American Academy of Neurology (AAN) recommendations, so-called "malignant" EEG patterns (such as generalized background suppression< 20μv; burst-suppression with generalized epileptiform activity, or unreactive background) during the three first days after cardiac arrest were associated with poor outcome with a FPR of 3% (95% CI: 0.9-11%).[103]

Regarding nonconvulsive seizures (NCSz) and status (NCSE) our study showed that 7 patients (33%) and 5 patients (23.8%) in bad outcome patients experienced NCSZ and NCSE respectively. There was statistically significant difference between incidence of non-convulsive seizures between bad and good outcome groupes. Seizure activity especially nonconvulsive seizures (NCSz) or nonconvulsive status epilepticus) is common in patients with anoxic–ischemic encephalopathy and may contribute to brain damage and prolonged coma.[104]

In a series of comatose patients with NCSE, 42% of the patients had hypoxic-anoxic injury after cardiac arrest.[105]

Regarding neuron specific enolase (NSE) sampling, NSE on first day had mean of 36.48 ± 19.61 µg/l in patients of bad outcome (vegetative or dead), while had mean of 14.15 ± 24.23 µg/l in patients of good outcome with statistically significant difference between two groups (p = 0.001).

While on third day NSE had mean of 24.33 ± 13.68 µg/l in bad outcome group, while had mean of 11.31 ± 20.74 µg/l in patients of good outcome with statistically significant difference between two groups (p< 0.001). NSE in predicting bad outcome with cut-off point > 35 µg/L, it came clear that NSE level had positive predictive value of 92.31 % to predict bad outcome in first day and 75 % in third day after arrest.

In one prospective, multicenter study involving 231 patients, NSE level of more than 33 µg per liter, sampled between 1 and 3 days after cardiac arrest, was strongly predictive of a poor outcome.[106] Other study showed that an NSE cutoff of 71.0 g/L drawn between 24 and 48 hours after ROSC was required to achieve an FPR of 0% (95% CI (0% to 43%) for predicting poor outcome with a sensitivity of 14%.[111] Numerous other studies show various thresholds from 30 to 65 g/L for poor outcome and mortality.[106-110]

As regards correlation between EEG coma scale and NSE levels, there was statistically significant positive correlation between EEG scale on day 1 with neuron specific enolase on day 1 and 3 as p value < 0.002 and < 0.001 respectively. There was statistically

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significant positive correlation between EEG scale on day 7 with   neuron specific enolase on day 1 and 3 as (p value < 0.001).

Rossetti et al, found also strong correlation was found between NSE and EEG findings in post cardiac arrest patients ($p= 0.001$).[111] Moreover, elevated NSE levels have been found in conditions related to neuronal death in humans, such as brain trauma and stroke.[112-113] Thus, the present findings suggest that EEG abnormalities are not simply a sign of transient post-CA global dysfunction, but rather represent apathophysiologic correlate of postanoxic neuronal injury.

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Limitations:

1. the sample size is limited; thus our results are to be considered preliminary and will need further confirmation by other groups and larger studies.
2. Some subjectivity may also be related to the scoring of EEG reactivity; however, we used the same method described in our study.
3. Serum NSE was not collected continuously, but the relatively long half-life of 24 hours justifies this practice.

SUMMARY

Anoxic–ischemic encephalopathy resulting from cardiac arrest is the third leading cause of coma requiring intensive care of comatose survivors. Certain malignant electroencephalographic (EEG) patterns have been shown to correlate with poor prognosis. Neurone specific enolase (NSE) released after cardiac arrest is regarded as a severity indicator of postanoxic neuronal injury

The aim of the study to investigate the EEG findings in post-cardiac arrest patients and to correlate these findings with clinical data, serum neuron-specific enolase levels and define their prognostic value.

This study included 34 Egyptian patients after successful resuscitation from cardiopulmonary arrest and were subjected to coma scoring scales, brainstem reflexes EEG, NSE measurement, computed tomography (CT) and Glasgow outcome scale (GOS).

According to results of our study there was statstically significant negative correlation between EEG coma scale on day 1 and 7 with GOS. There was statistically significant negative correlation between NSE on day 1 and 3 with GOS.There was statistically significant positive correlation between EEG scale on day 1and 7 with neuron specific enolase on day 1 and 3.

So we can conclude in post cardiopulmonary arrest patients, certain malignant EEG patterns are correlated with high serum levels of NSE and they are good predictors of poor outcome of these patients.