AIM OF THE WORK

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

PATIENTS

This study will be conducted on 34 patients surviving after successful cardiopulmonary resuscitation who are admitted to the critical care medicine department in Alexandria Main University Hospital (AMUH).

The study was approved by the Medical Ethics Committee of Alexandria Faculty of Medicine and an informed consent was taken from the patients' next of kin before their enrolment in the study.

The selected patients will fulfill the following inclusion criteria:

1. Adults (16 years of age or older)
2. Critically ill normothermic patients after successful cardiopulmonary resuscitation.
3. Haemodynamically stable.

Exclusion criteria

1. Patients aged less than 16.
2. Medical history of recent cerebral insult before arrest.
3. Brain death (flat recording of the EEG).
4. Coincident head injury.
5. Patients on sedation or receiving neuromuscular blocking agents.
6. Patients on drugs that lower seizure threshold.

METHODS

The study was approved by the ethical committee of the Alexandria faculty of medicine and an informed consent signed by patients relatives.

All patients were subjected to the following:

1. **Complete history taking:** age, sex, place of arrest (inicu or outside icu), duration of resuscitation to restoration of circulation, type of arrhythmia , cause of arrest.

2. **Neurological examination :** pupillary and corneal reflexes Glascow Coma Scale (day 1 and 3 after arrest), presence or absence of myoclonus .

3. **Laboratory investigations :** BUN, serum creatinine, SGOT, SGPT, sodium, ionized calcium were withdrawn on day first, third and seventh day after arrest to exclude metabolic abnormalities.

4. **Digital EEG recording** was done using Nicolet (21 channels) machine, using both bipolar and referential montages with the electrodes (21 electrodes) placed according to the international 10-20 system using EEG free electrodes with filter setting lowcut filter of 1.6Hz and high cut filter 35 Hz:

- The EEG monitoring was done within the first day after arrest for about 30 minutes and repeated on the seventh days after cardiac arrest. Stimulation (nail bed pressure, passive eye opening, and shouting in the ear) was routinely performed to show the reactivity of background.

- EEG background on days 1 and 7 post-arrest was classified according to EEG coma scale by Young et al.[81]

I. Delta/theta > 50% of recording.

II. Triphasic waves.

III. Burst-suppression pattern: with or without epileptiform activity.

IV. Alpha/theta/spindle pattern coma (no reactivity).

V. Suppression (generalized) : <20 microvolts

- EEG patterns will be divided into benign and malignant according to Synek et al.[69] Benign consisted of patterns I&II,while malignant consisted of III, IV & V.

5. **Neuron specific enolase (NSE):**was sampledat both day 1 and 3 after cardiac arrest. Venous blood samples were taken from patients and collected in a plain tube. The blood samples were clotted 30±15 min. at room temperature. After clotting, the samples were

centrifuged at room temperature for 10 minutes. Subsequently, the serum samples were stored at - 70 °C until the NSE concentration was measured. Serum concentrations of NSE measured by using solid phase enzyme immunoassay ELISA kit. (Reference value <10 µg/l).

6. **Radiological examination:** Computed tomographic (CT) images are usually normal immediately after cardiac arrest, but by day 3 they often show signs of brain oedema in the form of inversion of the gray–white densities, obliteration of sulci and slit ventricles, hence we did it on day 3 to reduce risk of transport to the Radiology department.

7. **All patients will be followed for 3 months after cardiac arrest or until death:**

- Neurological status after 1 month will be assessed using modified Rankin Scale (mRS).
- Outcome after 3 months will be assessed, by telephone interview for patients discharged alive from the intensive care, using the 5-grade Glasgow outcome scale (GOS).

8. **The cost** of digital EEG recording will be compared to the cost of neuron specific enolase sampling as regard cost effectiveness. Cost effectiveness is ratio between total cost of the test and health effect (AUC: test performance detected by ROC curve) [82]

Statistical Analysis:

Qualitative data were described using number and percent.

Quantitative data were described using mean and standard deviation, median, minimum and maximum.

Agreement of the different predictives with the outcome was used and was expressed in sensitivity, specificity, positive predictive value, negative predictive value and accuracy.

Receiver operating characteristic curve (ROC) was plotted to analyze a recommended cut off, the area under the ROC curve denotes the diagnostic performance of the test. Area more than 50% gives acceptable performance and area about 100% is the best performance for the test.

RESULTS

The study was carried on 34 adult male and female patients after cardiopulmonary resuscitation who were admitted to the department of critical care medicine in Alexandria Main University Hospital.

The patients were grouped according to their Glascow outcome scale (GOS) into good outcome group (scale: 3, 4 &5) and bad outcome group (scale :1& 2)

Table (3): Comparison between the two studied groups according to demographic data (n=34)

	GOS				Test of sig.	p
	Good (I) (n = 13)		Bad (II) (n = 21)			
	No.	%	No.	%		
Sex						
Male	8	61.5	14	66.7	$\chi^2 = 0.092$	$^{FE}p = 1.000$
Female	5	38.5	7	33.3		
Age						
Min. – Max.	30.0 – 69.0		19.0 – 72.0			
Mean ± SD	56.38 ± 11.11		47.48 ± 16.27		t = 1.735	0.092
Median	59.0		53.0			

χ^2: value for Chi square
FE: Fisher Exact test
t: Student t-test

Table (3): showsdemographic distribution there was no difference in males and females distribution in the two groups where 8 males with good outcome (group I) with mean 61.5 % compared to 14 males with bad outcome (group II) with mean 66.7 % and 5 females in group I with mean 38.5 % and 7 females in group II with mean 33.3 % with no significance difference between the two groups (p = 1.000).

Regarding age where ranging between 30 – 69 year with a mean 56.38 ± 11.11 in group I compared to group II where age ranging between 19-72 year with a mean 47.48 ± 16.27 with no significance difference between the two groups (p = 0.092).

Table (4): Comparison between the two studied groups according to different parameters (n=34)

	GOS				Test of sig.	p
	Good (I) (n = 13)		Bad (II) (n = 21)			
	No.	%	No.	%		
Place of arrest						
Inside ICU	9	69.2	15	71.4	$\chi^2 = 0.019$	$^{FE}p = 1.000$
Outside ICU	4	30.8	6	28.6		
Mode of arrest						
VF	10	76.9	10	47.6		
Asystole	3	23.1	7	33.3	$\chi^2 = 3.521$	$^{MC}p = 0.160$
PEA	0	0.0	4	19.4		
Resuscitation time						
Min. – Max	4.0 – 15.0		4.0 – 20.0			
Mean ± SD.	8.31 ± 3.47		9.62 ± 4.08		Z = 0.826	0.409
Median	8.0		8.0			
Cause of arrest						
Cardiac	10	76.9	8	38.1	$\chi^2 = 4.859^*$	0.028^*
Resp	3	23.1	13	61.9		

χ^2: value for Chi square
MC: Monte Carlo test
FE: Fisher Exact test
Z: Z for Mann Whitney test
*: Statistically significant at $p \leq 0.05$

Table (4): shows regarding circumstances of arrest , place of arrest there were 9 inside ICU in group I with mean 69.2% while 15 inside ICU in group II with mean 71.4% and there were 4 outside ICU in group I with mean 30.8% while 6 outside ICU in group II with mean 28.6% with no significant difference between two groups (p = 1.000).

As regards mode of arrest there were 10 patients with ventricular fibrillation(VF) in group I with mean 76.9% while 10 in group II with mean 47.6% and 3 patients with asystole in group I with mean 23.1% while 7 in group II with mean 33.3% and no patients with pulseless electrical activity (PEA) in group I while 4 with (PEA) in group II with mean 19.4%. There was no significant difference between the two groups as regards mode of arrest as (P = 0.160).

As regards resuscitation time its range was 4-15 minutes in group I with mean 8.31 ± 3.47 while it ranges 4-20 minutes in group II with mean 9.62 ± 4.08. there was no significant difference between the two groups as regards resuscitation time.

As regards cause of arrest there were 10 patients with cardiac causes in group I with mean 76.9 % while 8 patients with mean 38.1% in group II and there were 3 patients with respiratory causes in group I with mean 23.1% while 13 patients in group II with mean 61.9%.there was statistically significant difference between the two groups as regards cause of arrest (p = 0.028).

Table (5): Comparison between the two studied groups according to corneal and pupillary reflexes (n=34)

	GOS				χ^2	p
	Good (I) (n = 13)		Bad (II) (n = 21)			
	No.	%	No.	%		
Corneal reflex 1st day						
Preserved	13	100.0	13	61.9	6.476*	$^{FE}p = 0.013^*$
Lost	0	0.0	8	38.1		
Corneal reflex 3rd day						
Preserved	13	100.0	15	71.4	4.510	$^{FE}p = 0.062$
Lost	0	0.0	6	28.6		
Pupillary reflex1st day						
Preserved	12	92.3	4	19.0	17.298*	<0.001*
Lost	1	7.7	17	81.0		
Pupillary reflex3rd day						
Preserved	13	100.0	15	71.4	4.510	0.062
Lost	0	0.0	6	28.6		

χ^2: Chi square test

FE: Fisher Exact test

*: Statistically significant at $p \leq 0.05$

Table (5): shows on first day after arrest the corneal reflex was preserved in 13 patients in group I with mean 100% while preserved in 13 patients in group II with mean 61.9% and corneal reflex was not lost in first day in any patient in group I while was lost in 8 patients in group II with mean 38.1%. There is statistically significant difference between the two groups as regards corneal reflex in first day after arrest as ($p = 0.013$).

On third day after arrest the corneal reflex was preserved in 13 patients in group I with mean 100% while preserved in 15 patients in group II with mean 71.4% and corneal reflex was not lost in third day in any patient in group I while was lost in 6 patients in group II with mean 28.6%. There is no statistically significant difference between the two groups as regards corneal reflex in third day after arrest as ($p = 0.062$).

On first day after arrest the pupillary reflex was preserved in 12 patients in group I with mean 92.3% while preserved in 4 patients in group II with mean 19% and it was not lost in one patient in group I with mean 7.7% while was lost in 17 patients in group II with mean 81%. There is statistically significant difference between the two groups as regards pupillary reflex in first day after arrest as(p value <0.001).

On third day after arrest the pupillary reflex was preserved in 13 patients in group I with mean 100% while preserved in 15 patients in group II with mean 71.4% and corneal reflex was not lost in third day in any patient in group I while was lost in 6 patients in group II with mean 28.6%.There is no statistically significant difference between the two groups as regards pupillary reflex in third day after arrest as (p value = 0.062).

Table (6): Comparison between the two studied groups according to Glascow coma scale (GCS). (n=34)

GCS	GOS		t	p
	Good (n = 13)	Bad (n = 21)		
1st day				
Min. – Max.	6.0 – 12.0	3.0 – 7.0		
Mean ± SD.	8.38 ± 2.06	5.0 ± 1.26	5.952*	<0.001*
Median	8.0	5.0		
3rd day				
Min. – Max.	5.0 – 15.0	3.0 – 6.0		
Mean ± SD.	12.08 ± 3.28	4.71 ± 1.06	7.851*	<0.001*
Median	13.0	5.0		

t: Student t-test

*: Statistically significant at p ≤ 0.05

Table (6):shows on first day after arrest the Glascow Coma Scale (GCS) ranges from 6-12 in group I with mean 8.38 ± 2.06 while ranges from 3-7 in group II with mean 5.0 ± 1.26. There is statistically significant difference between the two groups as regards Glascow coma scale in first day after arrest as (p <0.001).

On third day after arrest the Glascow Coma Scale (GCS) ranges from 5-15 in group I with mean 12.08 ± 3.28 while ranges from 3-6 in group II with mean 4.71 ± 1.06. There is statistically significant difference between the two groups as regards Glascow coma scale in third day after arrest as (p <0.001).

Table (7): Comparison between the two studied groups according to Myoclonus (n=34)

	GOS				χ^2	p
	Good (I) (n = 13)		Bad (II) (n = 21)			
	No.	%	No.	%		
Myoclonus						
No	0	0.0	4	19.0	2.806	0.144
Yes	13	100.0	17	81.0		

χ^2: Chi square test

Table (7) shows myoclonus is absent in group I while present in 4 patients(19%) in group II. There was no statistically significant difference between the two groups as regards incidence of myoclonus.

Table (8): Comparison between the two studied groups according to brain edema (n=34)

	GOS				χ^2	^{FE}p
	Good (I) (n = 13)		Bad (II) (n = 21)			
	No.	%	No.	%		
Brain odema						
No	1	7.7	3	14.3	0.336	1.000
Yes	12	92.3	18	85.7		

χ^2: value for Chi square
FE: Fisher Exact test

Table (8): shows CT brain finding , brain edema was found in 3 patients in bad outcome group (II) while present in 1 patient in good group with no statistically significant difference between the two groups (p = 1.000).

Table (9): Comparison between the two studied groups according to laboratory investigations (n=34)

	GOS		t	p
	Good (n = 13)	Bad (n = 21)		
BUN				
DAY1	21.92 ± 4.41	23.10 ± 4.46	0.748	0.460
DAY3	21.92 ± 6.96	22.24 ± 5.87	0.142	0.888
DAY7	21.85 ± 4.71	23.33 ± 4.72	0.894	0.378
Cr				
DAY1	0.83 ± 0.20	0.88 ± 0.28	0.512	0.612
DAY3	1.07 ± 0.50	1.07 ± 0.43	0.016	0.987
DAY7	0.88 ± 0.29	0.91 ± 0.21	0.438	0.664
SGOT				
DAY1	37.27 ± 4.18	34.17 ± 6.08	1.615	0.116
DAY3	30.77 ± 4.57	35.71 ± 17.97	1.200	0.242
DAY7	30.08 ± 3.07	29.52 ± 4.23	0.409	0.685
SGPT				
DAY1	39.47 ± 3.74	35.33 ± 5.97	2.235*	0.032*
DAY3	38.25 ± 3.98	34.48 ± 5.14	2.258*	0.031*
DAY7	34.15 ± 6.28	29.38 ± 4.68	2.532*	0.016*
Na				
DAY1	139.15 ± 3.41	140.43 ± 2.27	1.311	0.199
DAY3	138.54 ± 7.69	137.62 ± 5.99	0.390	0.699
DAY7	138.54 ± 5.92	137.95 ± 4.80	0.316	0.754
Ion.Calcium				
DAY1	1.22 ± 0.12	1.17 ± 0.11	1.200	0.239
DAY3	1.18 ± 0.11	1.15 ± 0.12	0.883	0.384
DAY7	1.15 ± 0.11	1.24 ± 0.11	2.294*	0.028*

t: Student t-test

*: Statistically significant at $p \leq 0.05$

Table (9): As regards laboratory investigations (BUN, Cr, SGOT, SGPT, ioniz. calcium, Na) on day 1, 3, and 7, results were in normal range values of every investigation. This excluded any associated metabolic derangments in the study cases.

Table (10): Comparison between the two studied groups according to Nonconvulsive seizures and status (n=34)

	GOS				χ^2	^{FE}p
	Good (n = 13)		Bad (n = 21)			
	No.	%	No.	%		
Nonconvulsive seizures						
No	13	100.0	14	66.7	5.457*	0.029*
Yes	0	0.0	7	33.3		
Nonconvulsive status						
No	12	92.3	16	76.2	1.435	0.370
Yes	1	7.7	5	23.8		

χ^2: value for Chi square
FE: Fisher Exact test
*: Statistically significant at $p \leq 0.05$

Table (10) Nonconvulsive seizures were not found in group I while were found in 7 Patients (33.3%) in group II. There was stastically significant difference between the two groups as regards incidence of nonconvulsive seizures (p = 0.029). Nonconvulsive status was present in 1 patient in group I while was present in 5 patients(23.8%) in group II. There was no stastically significant difference between the two groups as regards incidence of nonconvulsive status (p = 0.37).

- **Correlation results between clinical and radiological parameters with outcome scales**

Table (11): Correlation between GCS with mRS and GOS in total sample (n=34)

GCS	Day 1		Day 3	
	r	p	r	p
MRS	-0.698*	<0.001	-0.890*	<0.001
GOS	0.724*	<0.001	0.914*	<0.001

- r: Pearson coefficient
- *: Statistically significant at $p \leq 0.05$

Table (11) shows that there was statistically significant negative correlation between Glascow coma scale on day 1, 3 and modified Rankin scale (p <0.001). There was statistically significant positive correlation between Glascow coma scale on day1 , 3 and glascow outcome score (p < 0.001).

Table (12): Correlation between myoclonus with mRS and GOS in total sample

	MRS		GOS	
	r_s	p	r	p
Mycolonus	-0.275	0.115	0.318	0.067

r: Pearson coefficient

r_s: Spearman coefficient

Table (12) shows there was no statistically significant correlation between myoclonus with modified Rankin scale (P <0.115). There was no statistically significant correlation between myoclonus with Glascow outcome scale (P <0.067).

Table (13): Correlation between mRS and GOS with brain edema

	Brain edema	
	r	p
MRS	-0.283	0.105
GOS	0.143	0.418

r: Pearson coefficient

Table (13) shows there was no statistically significant correlation between brain edema with modified Rankin scale (P <0.105). There was no statistically significant correlation between brain edema with Glascow outcome scale (P <0.418).

- **correlation between EEG patterns with outcome scales**

Table (14): Correlation between Nonconvulsive seizures and status with mRS and GOS

	Nonconvulsive seizures		Nonconvulsive status	
	r_s	p	r_s	p
mRS	0.281	0.107	0.200	0.256
GOS	-0.216	0.221	-0.343*	0.047

r_s: Spearman coefficient
*: Statistically significant at $p \leq 0.05$

Table (14) shows there was no statistically significant correlation between nonconvulsive seizures with modified Rankin scale (P value 0.107) or Glascow outcome scale ($p < 0.221$).

There was statistically significant correlation between nonconvulsive status epilepticus withGlascow outcome scale ($p < 0.047$).

Table (15): Correlation between EEG coma scale with mRS and GOS

	EEG			
	1st day		7th day	
	r_s	p	r_s	p
mRS	0.696*	<0.001	0.663*	<0.001
GOS	-0.670*	<0.001	-0.722*	<0.001

r_s: Spearman coefficient
*: Statistically significant at $p \leq 0.05$

Table (15) shows there was statstically significant positive correlation between EEG coma scale (on day 1 and 7) with modified Rankin scale as ($p < 0.001$).

There was statstically significant negative correlation between EEG coma scale (on day 1 and 7) with Glascow outcome score as ($p < 0.001$).

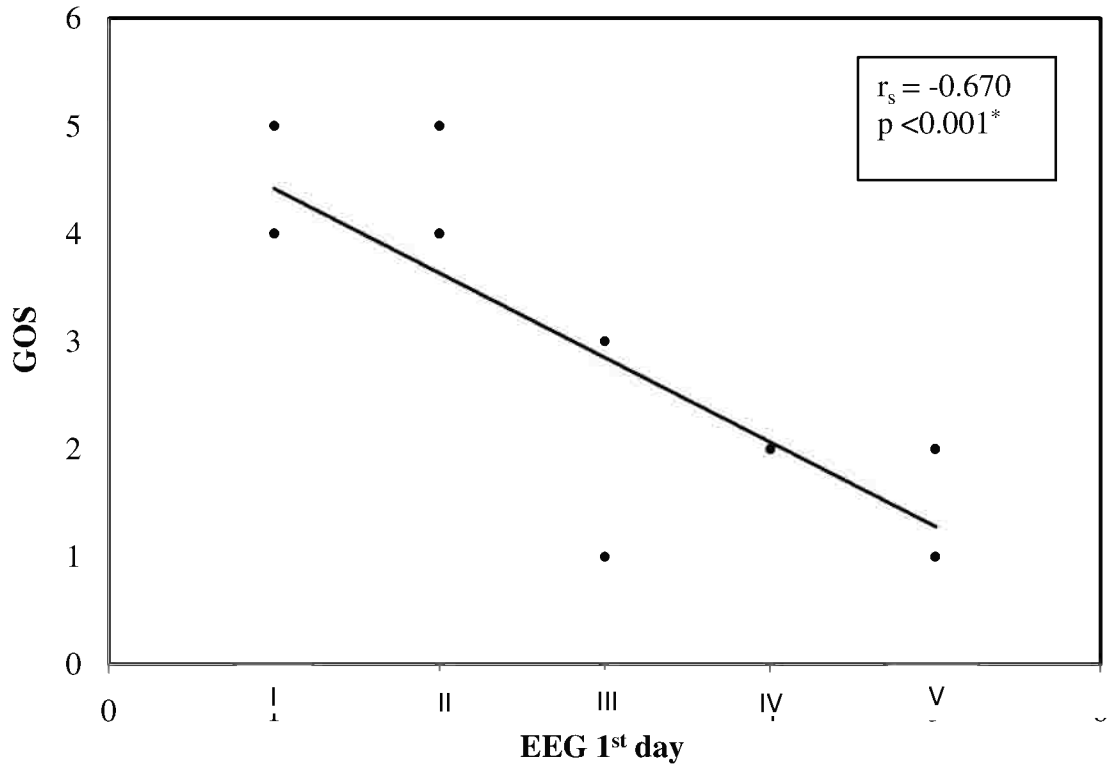

Figure 4: Correlation between EEG scale 1st day and GOS

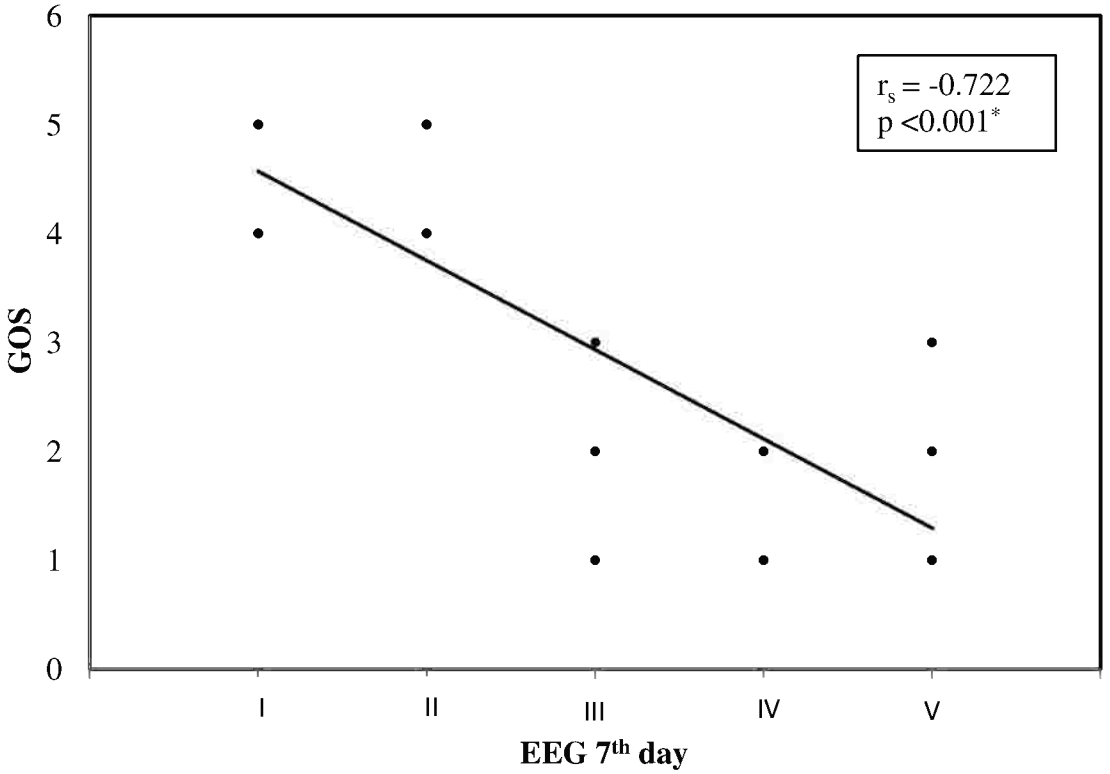

Figure 5: Correlation between EEG scale 7th day and GOS

Table (16): Impact of EEG patterns on outcome (n=34)

EEG	GOS			
	Good (n = 13)		Bad(vegetative or dead) (n = 21)	
	No.	%	No.	%
1st day				
I (delta/theta > 50%)	7	53.8	0	0.0
II (triphasic waves)	4	30.8	0	0.0
III (burst suppression)	2	15.4	6	28.6
IV (unreactive background)	0	0.0	2	9.5
V (low voltage < 20 μV)	0	0.0	13	61.9
Mean ± SD.	1.62 ± 0.77		4.33 ± 0.91	
Z (p)	4.818* (<0.001*)			
7th day				
I (delta/theta > 50%)	9	69.2	0	0.0
II (triphasic waves)	2	15.4	0	0.0
III (burst suppression)	1	7.7	3	14.3
IV (unreactive background)	0	0.0	5	23.8
V (low voltage < 20 μV)	1	7.7	13	61.9
Mean ± SD.	1.62 ± 1.19		4.48 ± 0.75	
Z (p)	4.483* (<0.001*)			

Z: Z for Mann Whitney test

*: Statistically significant at $p \leq 0.05$

Table (16) shows outcome after 3 months, 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.

Table (17): Prognostic predictive value of malignant EEG patterns in bad outcome

	1st day		7th day	
	PPV	**NPV**	**PPV**	**NPV**
EEG				
I	77.78	100.0	84.0	100.0
II	70.0	100.0	65.63	100.0
III (burst suppression)	88.24	64.71	94.74	80.0
IV (unreactive background)	100.0	86.67	100.0	72.22
V (low voltage < 20μv)	100.0	50.0	88.89	48.0

Table (17) shows 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.

Table (18): Correlation between neuron specific enolase with mRS and GOS

	NSE			
	1st day		3rd day	
	r_s	P	r_s	p
mRS	0.695*	<0.001	0.710*	<0.001
GOS	-0.624*	<0.001	-0.673*	<0.001

Table (18) shows correlation between NSE with outcome scales there was statistically significant positive correlation between modified Rankin scale with NSE on day 1 and 3 and there was statistically significant negative correlation between Glascow outcome scale with NSE on day 1 and 3 ($p< 0.001$).

Table (19): Impact oF NSE level on outcome

NSE	GOS		Z	p
	Good (n = 13)	Bad (n = 21)		
1st day				
Min. – Max.	4.0 – 90.0	4.0 – 88.0	3.301*	0.001*
Mean ± SD.	14.15 ± 24.23	36.48 ± 19.61		
3rd day				
Min. – Max.	4.0 – 80.0	5.0 – 56.0		
Mean ± SD.	11.31 ± 20.74	24.33 ± 13.68	3.815*	<0.001*
Median	5.0	22.0		

Z: Z for Mann Whitney test

*: Statistically significant at $p \leq 0.05$

Table (19) shows NSE on first day had mean of 36.48 ± 19.61 in patients of bad outcome (vegetative or dead), while had mean of 14.15 ± 24.23 in patients of good outcome with statistically significant difference between two groups ($p = 0.001$).

While on seventh day NSE had mean of 24.33 ± 13.68 in bad outcome group, while had mean of 11.31 ± 20.74 in patients of good outcome with statistically significant difference between two groups ($p< 0.001$).

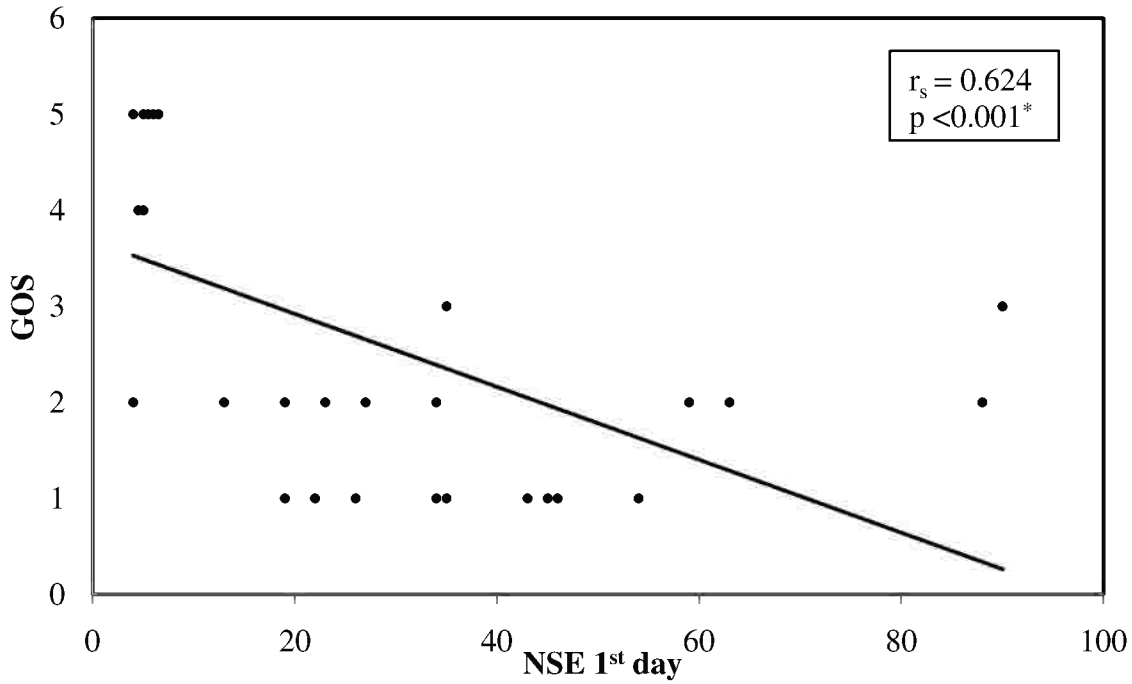

Figure 6: Correlation between NSE 1st day with GOS

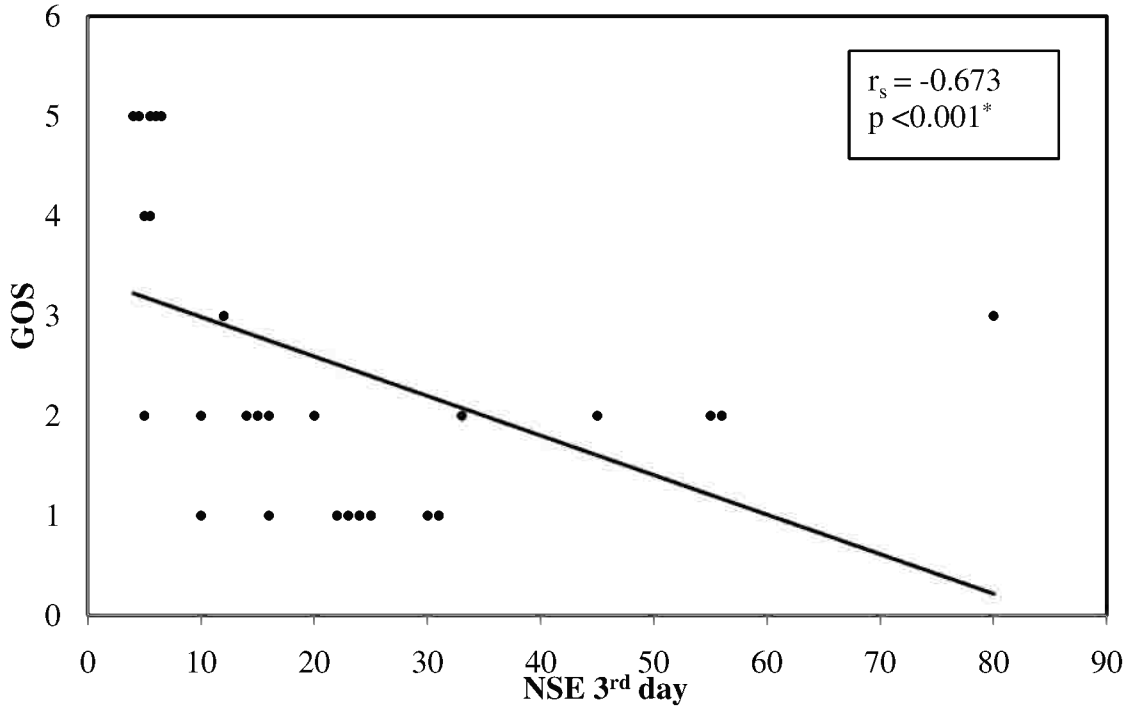

Figure 7: Correlation between NSE 3rd day with GOS

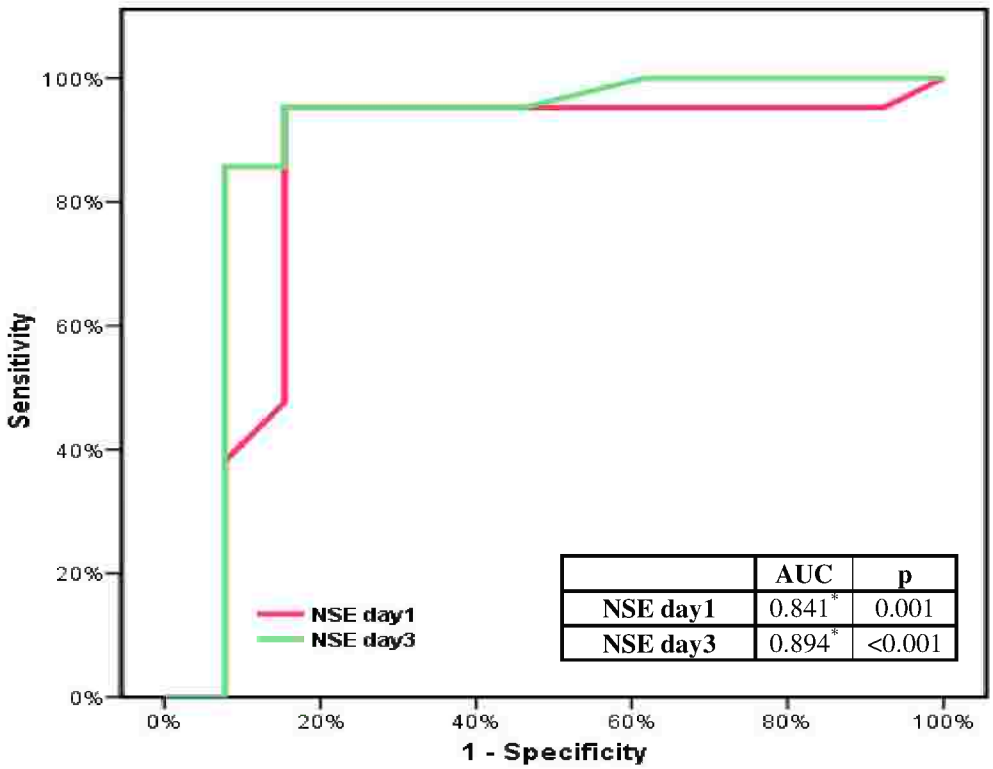

Figure (8): ROC curve for NSE in 1st and 3rd days to predict bad GOS

Table (20): Agreement (sensitivity, specificity and accuracy) for NSE in 1st and 3rd days with bad GOS

		Good GOS	Bad GOS	Sensitivity	Specificity	PPV	NPV	Accuracy
NSE day1	**≤35**	12	13	38.10	92.31	88.89	48.0	58.82
	>35	1	8					
NSE day3	**≤35**	12	18	14.29	92.31	75.0	40.0	44.12
	>35	1	3					

Figure (8) and **table (20)** showed the agreement (sensitivity,specificity,and accuracy) for neuron specific enolase (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 .

Table (21): Correlation between EEG coma scale with NSE

	EEG			
	1st day		7th day	
	r_s	p	r_s	p
NSE day 1	0.508*	0.002	0.652*	<0.001
NSE day3	0.612*	<0.001	0.738*	<0.001

r_s: Spearman coefficient
*: Statistically significant at $p \leq 0.05$

Table (21) shows there was stastically significant positive correlation between EEG scale on day 1 with neuron specific enolase on day 1 and 3 as ($p < 0.002$) and ($p< 0.001$) respectively.There was statstically significant positive correlation between EEG scale on day 7 with neuron specific enolase on day 1 and 3 as ($p < 0.001$).

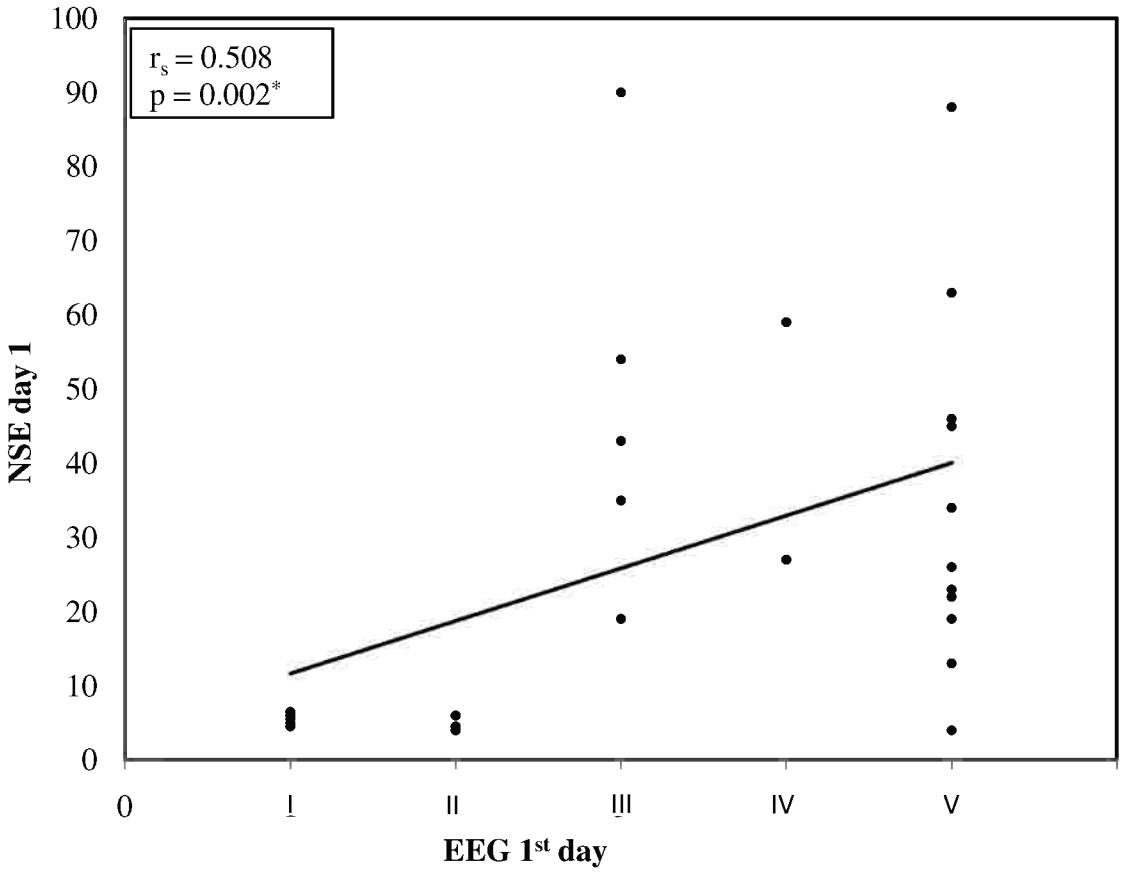

Figure 9: Correlation between EEG scale 1st day with NSE 1st day

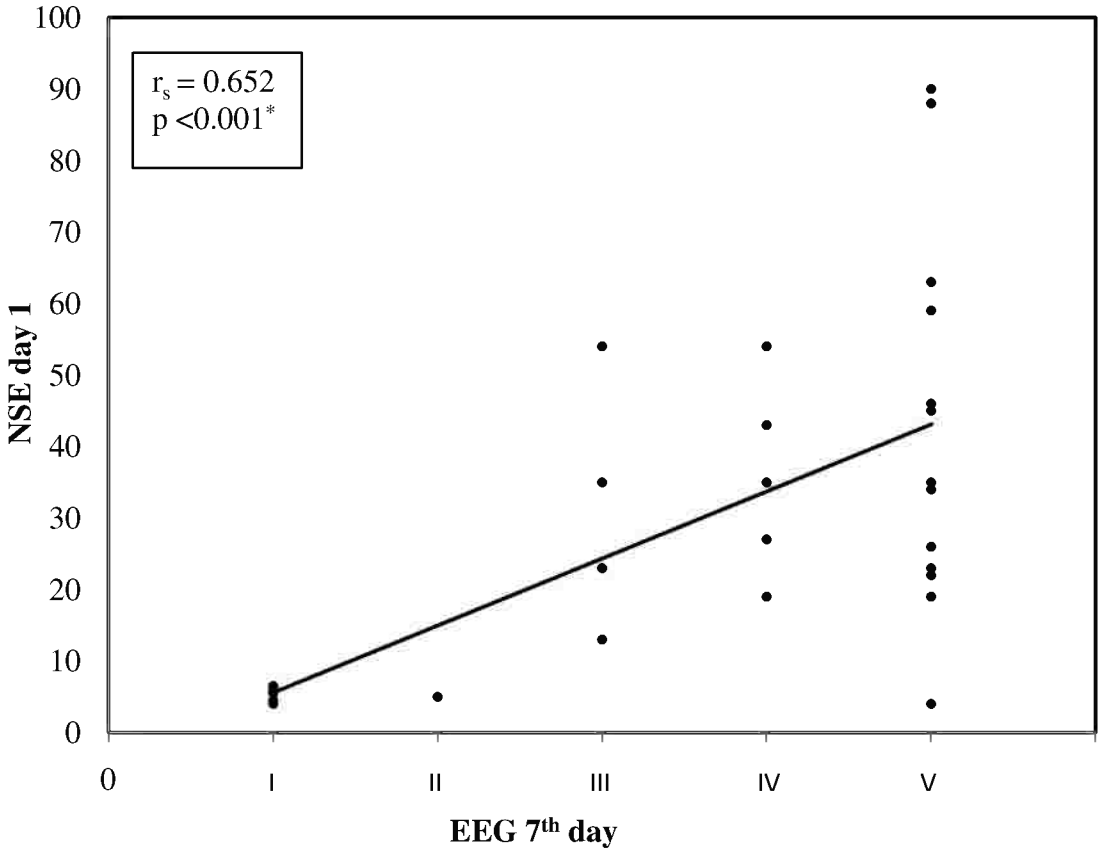

Figure 10: Correlation between EEG scale 7th day with NSE 1st day

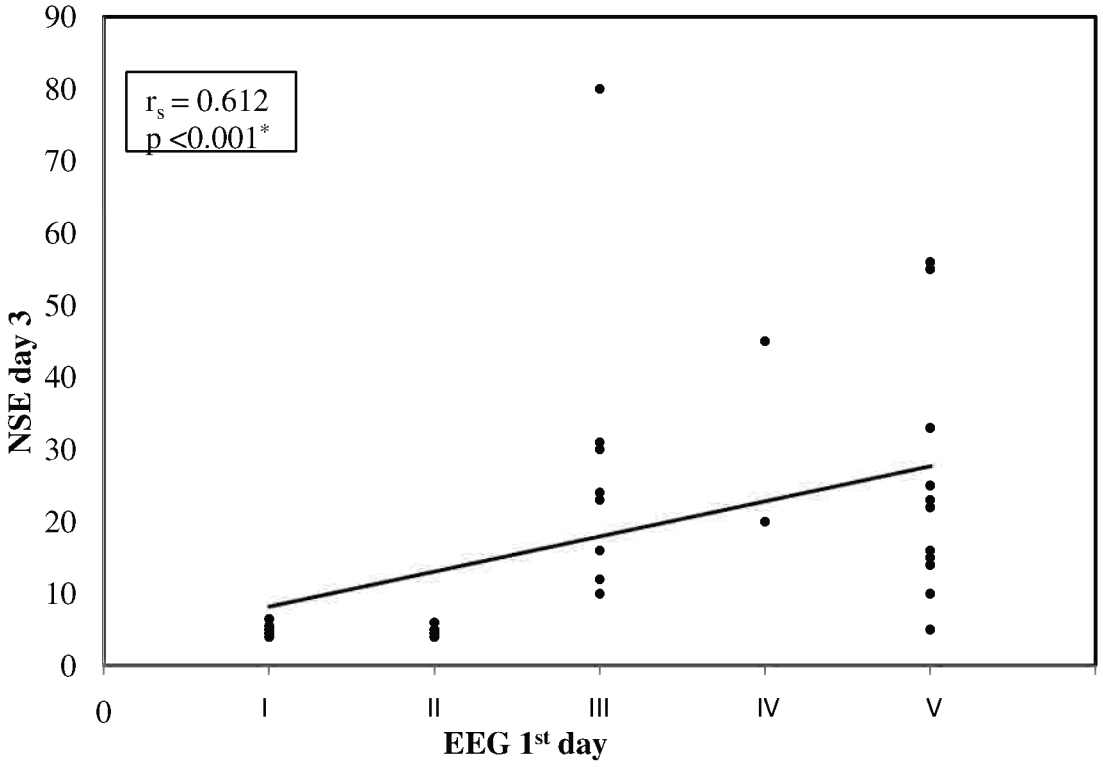

Figure 11: Correlation between EEG scale 1st day with NSE 3rd day

- **Comparison between EEG and NSE regarding direct cost**

The estimated cost of EEG for every patient is 50 L.E. so the total cost of all patients was 3400 L.E. while the cost of NSE for every patient was 100 L.E. so the total cost of all patients was 6800 L.E.

Table (22): Cost effectiveness ratio between EEG recording and NSE sampling in total sample.

	Cost (L.E.)	Health effect (AUC: test performance)	Cost effectiveness ratio
EEG			
1ST DAY	1700	0.857	1983.6
7TH DAY	1700	0.890	1910.1
NSE			
1ST DAY	3400	0.841	4042.8
3RD DAY	3400	0.894	3803.1

It came clear that EEG done on seventh day had better cost effectiveness ratio than that done on first day. And so EEG had better cost effectiveness than NSE sampling.