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Study of the Prognostic criteria in Post Cardiac arrest Patients Treated with Therapeutic Hypothermia

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Abstract

Background: Cardiac arrest (CA) cause over 500,000 deaths / year in North America. Neurologic injury is a main cause of death in (OHCA) and contributes in the mortality of (IHCA) .Lowering body and brain temperature to 32 to 34°C during the early hours after CA decrease the risk of neurologic deterioration. Prognosis assessment in patients post-CA is challenging clinically. As it's important to judge medical decision making as regard withdrawal of care, no single test accurately predicts poor clinical outcomes. This pilot study assesses the value of different prognostic parameters in post-CA patients. **Objective:** To formulate a prognostic criteria for post-CA arrest patients treated with therapeutic hypothermia. **Patients and Methods:** The current study was carried on 53 patients admitted to the Alexandria Main University Hospital over a period of six months after cardiac arrest resuscitations and ROSC. According to our study group, 9 patients died before the end of the first post-CA month. Regarding the other 44 patients, CPC scale was performed 1month after cardiac arrest. Patients with favorable outcome (group A) includes patient with CPC scale from 1 to3.It includes 17 patients with a percentage of 38.6% and Patients with bad outcome(group B) includes patients with CPC scale 4 and 5. It includes 27 patients with a percentage of 61.4%. CEEG monitoring was connected to all patients after cardiac arrest and for 24h after. Routine EEG was done for all patients 72h after CA and TCD was performed 2,6, 24,48 and 72 hours after ROSC. Clinical examination including GCS, pupillary and corneal reflexes were performed 72h after CA. **Results:** After 72 h of ROSC, a lower voltage EEG(using 10Mv as a cut off value) is considered as a positive predictor for bad outcome as about 6% of patients in group A had a low voltage compared with 89% of patients in group B with a sensitivity of 88.9%, specificity 94% and accuracy 90.9%.Also, a non reactive EEG was considered as a positive predictor for bad outcome as 96.3% of patients in group B had a non reactive background compared with 17.6% in group A with a 96.3% sensitivity, 82.3% specificity and accuracy of 90.9% .Regarding EEG pattern and background, about 91% of patients in group A had a background which was either normal(defined as a reactive alpha or beta rhythm with voltage more than or equal 10Mv) or showed interictal discharges or status epilepticus compared with only 26% of patients in group B showed this pattern and were considered as a positive predictor for good outcome. However, 74% of patients in group B showed a slow background or intermittent burst-suppression pattern compared with only 5.9% in group A with positive prediction of bad outcome with sensitivity 74%, specificity 95% and accuracy 81.8%.Regarding absent pupillary reflex, it has a sensitivity of 70.34% and specificity 64.7% with accuracy 68.18% for detecting patients with bad clinical outcome with about 70.4% of patients in group B compared with 35% of patients in group A had absent bilateral pupillary reflex 72h after ROSC. However, bilateral absent corneal reflex has a low sensitivity and specificity (59.2%, 58.8% respectively) for detecting those with bad clinical outcome as about41.2% of patients in group A and 59.3% of patients in group B had absent corneal reflex after 72h of ROSC. Regarding TCD mean values, a ROC curve analysis for the cut off value of MFV and PI reveals that a cut off MFV equal or less than 25cm/sec associated with 55.6% sensitivity and 100% specificity for prediction of bad clinical outcome with area under the curve equal 0.742, while a pulsatility index cut off value of more than 1 is associated with 55.6% sensitivity and 94% specificity for bad clinical outcome with 0.702 area under the curve. **Conclusion:** A combination of TCD data with clinical examination through GCS assessed 24 hours after rewarming will increase GCS specificity (using a cut off value of less than 8 a s a predictor for bad outcome) for prediction of bad clinical outcome from 29.4% to 70.59% with 0.865 area under the curve.

Thus, adding both EEG and TCD parameters to clinical neurological examination will have positive impact and will improve the specificity of bad outcome detection which of great help in prognostication after CA.

Keywords: TTM, therapeutic hypothermia, post-CA care, TCD.

Introduction

Cardiac arrest (CA)cause over 500,000 deaths / year in North America[1]. However, advances in CPR and post-arrest care have improved the outcomes in selected group of patients[2-3]. The care of post CA patients is complicated and includes sedation, mechanical ventilation, coronary interventions, seizure management, circulatory support, and more[4,5]. The HIBI affects different organ systems, mainly the brain[4].Brain Specific areas as the cerebral cortex, thalamus, cerebellum, hippocampus, and striatum are more affected by ischemia-reperfusion damage[6].So, clinical convulsions are common after CA and found in approximately 1/3 of the patients[7]. About 80% of admissions to the ICU after resuscitation from OHCA are comatose [8] and 2/3 of them will die as a result of HIBI[9,10].Neuronal death and diffuse brain eodema can result from HIBI[11–12,13, 14]. However, only a small percentage of these deaths are a direct consequence of severe neuronal injury (i.e. brain death)[15]. In fact, deaths mainly resulted from HIBI caused by (WLST) after prognostication of a poor outcome[16-17-18]

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The main steps involved in management of these patients include:[19,20,21]

- Assessment and stabilization of pulmonary and cardiac status.
- Searching for the cause of arrest.
- Neuroprotection.
- Avoiding recurrence of arrest.

Target temperature Management in post CA patients (TTM)

Neurologic injury is a main cause of death in (OHCA) and contributes in the mortality of (IHCA)[9]. Lowering body and brain temperature to 32 to 34°C during the early hours after CA decrease the risk of neurologic deterioration[19]. An observational study of 151post CA patients, deaths increases for every one degree over 37°C during the early 48 hours after CA. (OR 2.26; 95% CI 1.24-4.12)[19].

The ideal duration and best speed for reaching the target temperature are unknown[20]. We suggest achieving a core temperature between 32 to 34°C in a six hours and keep it for twelve to twenty four hours[20].Patients should be cooled using surface or intravascular cooling methods that are familiar and available[2]. Many patients after (ROSC) are hypothermic (about 35 to 35.5°C) from the mixing of core blood and cooler peripheral blood[2,3,21]. So, minimally invasive methods can often achieve the target temperatures rapidly[21]. Shivering raises core body temperature and must be controlled in patients treated with TTM[22,23,24]. Inability to control shivering is a common cause of delay in reaching target temperatures when instituting TTM[24]. So, we suggest adding increasing doses of sedation to achieve shivering suppression, rather than using a well known standard sedation scales[24]. High sedatives doses are frequently needed to achieve target temperature without shivering[24].

Neuromuscular blocking drugs are highly effective at shivering control, but can mask convulsions, which develop in about three to forty four percent of post-CA patients[2,25,26,27]. CEEG monitoring is highly needed for the safe use of NMB agents[27].

Core temperature should be continuously monitored during TTM[28]. The gold standard method is central venous temperature, but several similar methods are available. In order of preference include lower esophageal temperature, bladder or rectal probes[28].The most accurate of them in following core body temperature is esophageal temperature measurement[28,29].

Rewarming should be achieved gradually, with a rate of temperature increase not exceeding 0.5°C/hour and its recommended that a rate of 0.2 to 0.25°C/hour is satisfactory[24,29,30]. Rapid rewarming results incerebral edema, electrolyte abnormalities as hyperkalemia, seizures, and many other problems[31].

Prognostication after cardiac arrest

Prognosis assessment in patients post-CA is challenging clinically[32].As it's important to judge medical decision making as regard withdrawal of care, no single test accurately predicts poor clinical outcomes, especially during the first twenty-four hours after CA[32] .And so, more than one

prognosticators should be used to evaluate the intensity of neurological damage[33]. Before doing neuroprognostication, any confounding factors such as seizures, hypotension, neuromuscular blockers or toxins must be excluded[34]. Neuroprognostication exact timing in patients who undergo TTM remains unknown[34]. The 2010 AHA guidelines recommends that all neuroprognostication tools should be delayed after the first 72-hours post-CA in patients undergo TTM[32]. In patients not subjected to TTM, neuroprognostication can be done in the first 24-hours post-CA[32].

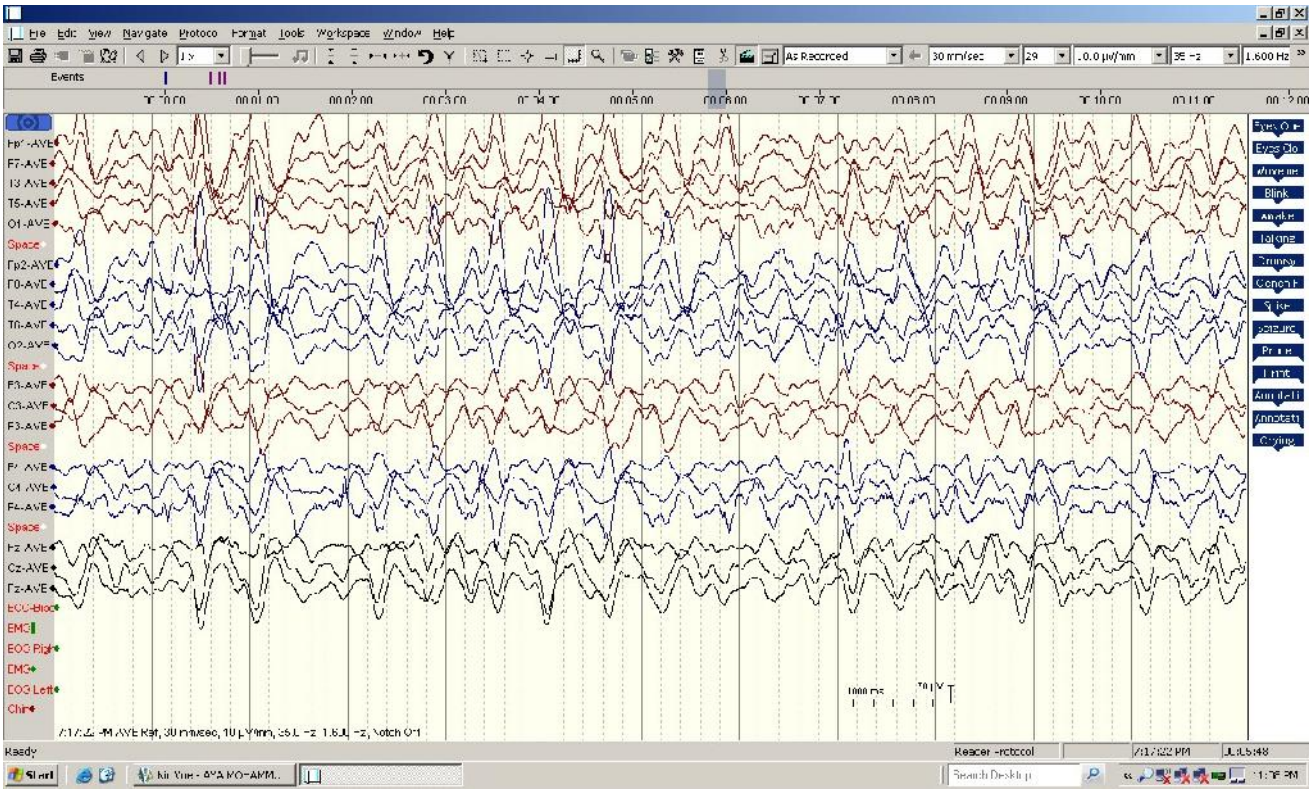

Figure (1) Post-CA patient with Non convulsive seizures.

Electroencephalography (EEG) has been extensively studied in comatose post-CA patients before the use of TTM[35]. In general, EEG reactivity and early improvement of brain activity were associated with good outcome; however, clinical and EEG evidence of seizures correlates with bad outcome[36]. Prior the era of TTM, many EEG patterns have been associated strongly with bad outcome, most notably burst-suppression background with generalized epileptiform discharges, a low voltage background (< 20 µV) and periodic generalized complexes on a suppressed or flat background; however, none of those patterns predicts bad outcome accurately[37].

Trans cranial Doppler (TCD) is now considered as one of the most common neurocritical care tool used in various aspects and it was first used by Aaslidin 1982[38].It is easy, inexpensive and a non-invasive tool that can be helpful for rapid and bedside repeatable measurement of CBF velocity (FV)[39]. Waveform analysis can help indirectly in providing information about CBF, ICP and cerebro-vascular resistance in neurocritically ill patients[39]. During cerebral heamodynamics monitoring, TCD can assess patency of cerebral vessels, changes in basal arteries diameter of the Willis circle through alteration in FV, autoregulation (a stable FV within a MBP range of 50-150 mmHg) and reactivity of brain vessels to carbon dioxide[39,40].

Brain death confirmation ancillary testing remains controversial [41]. Guidelines from the AAN and AAP report an insufficient evidence for brain death determinination with ancillary tests[40,41]. However, various ancillary tests still an essential tool in confirmation of brain death when apnea test cannot be used safely or hypothermia or barbiturate treatmentprevent proper brain death confirmation[42,43,44]. In such situations, ancillary testing may help in confirmation of brain death[43]. TCDis useful ancillary test for confirmation of brain death as it is

noninvasive and safe(45). TCD can confirms brain death by demonstrating cerebral circulatory arrest (CCA), which has a specific flow patterns: oscillatory (reverberating) flow which represents reversal of flow during diastole and systolic peak representing lack of any forward flow(45).

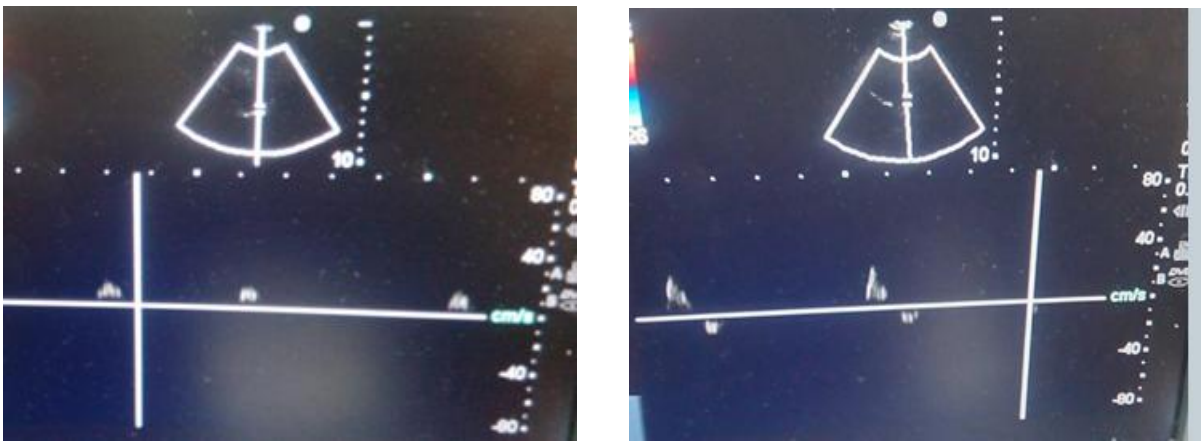

Figure (2): Some TCD patterns in post-CA Patients.

Patients and Methods

The current study was carried on 53 patients admitted to the Alexandria Main University Hospital over a period of six months after cardiac arrest resuscitations and ROSC. According to our study group, 9 patients died before the end of the first post-CA month. Regarding the other 44 patients, CPC scale was performed 1month after cardiac arrest. Patients with favorable outcome(group A) includes patient with CPC scale from 1 to3.It includes 17 patients with a percentage of 38.6% and Patients with bad outcome(group B) includes patients with CPC scale 4 and 5. It includes 27 patients with a percentage of 61.4%. CEEG monitoring was connected to all patients after cardiac arrest and for 24h after. Routine EEG was done for all patients 72h after CA and TCD was performed 2, 6, 24,48 and 72 hours after ROSC. Clinical examination including GCS, pupillary and corneal reflexes were performed 72h after CA.

Results

According to the demographic data, about 52% of the study group was males and 47.7% was females. Regarding age, the percentage of patients with age more than or equal 60 years old was higher and was around 56.8% with mean age 60 years old and median 62 years old.

According to the cause of admission, the most common cause in our study was septic shock(15.9%) followed by renal failure (11.4%) , multiple trauma patients excluding those with TBI(11.4%), CAP(9.1%), acute exacerbation of COPD (9.1%), decompensated HF(9.1%) , acute myocardial infarction (9.1%), ARDS (6.8%), malignancy(excluding CNS tumours) (6.8%), DKA(4.5%), pulmonary embolism (4.5%), and 1 patient with intraoperative cardiac arrest. Regarding the cause of cardiac arrest, the most common cause of cardiac arrest was hypoxia and accounts for (33.9%) of all causes of CA followed by metabolic acidosis which accounts for (22.6%),myocardial infarction (16.9%), hyperkalemia(9.4%), hypoventilation (7.5%), hypovolemia (3.77%), pulmonary embolism (3.77%) and hypokalemia(1.88%).

According to CPC scale, there was a significant positive correlation between CPC score and duration of CPR, age in years and TCD pulsatility index.Also, there was a significant negative correlation between GCS assessed 2 hours after ROSC,24h after rewarming(72h after ROSC) ,mean BP and outcome measured by CPC scale.

So, group A in our study has a mean duration of CPR of 3cycles compared to 6,5 cycles in group B.Also, the mean age in group A was around 57 years compared to 67 years in group B.A higher TCD MFV and a lower pulsatility index was noticed in group A(55.9cm/sec and 0.71 ,respectively) compared to group B who had TCD MFV around 40.5cm/sec and PI around 1.1. Regarding GCS assessed 2 hours and 24 hours after ROSC, group A had a GCS around 6 two hours after ROSC compared to GCS around 5 2 hours after ROSC in group B, and a mean GCS of 12 in group A and of 5 in group B examined 24 hours after rewarming which was approximately 72 hours after ROSC.

Table (1): correlation between different parameters and CPC scale.

	CPC Scale 1 month after ROSC	
	r^S	p
No of CPR Cycles	0.597*	<0.001*
Age (Years)	0.301*	0.047*
TCD		
MFV	-0.490*	0.001*
PI	0.432*	0.003*
GCS 2 h after Cardiac arrest	-0.633*	<0.001*
GCS 2 h after rewarming	-0.731*	<0.001*
Mean Blood Pressure	-0.422*	0.004*

r^S : Spearman Coefficient

*: Statistically significant at $p \leq 0.05$

After 72 h of ROSC, a lower voltage EEG(using 10Mv as a cut off value) is considered as a positive predictor for bad outcome as about 6% of patients in group A had a low voltage compared with 89% of patients in group B with a sensitivity of 88.9%, specificity 94% and accuracy 90.9%.Also, a non reactive EEG was considered as a positive predictor for bad outcome as 96.3% of patients in group B had a non reactive background compared with 17.6% in group A with a 96.3% sensitivity, 82.3% specificity and accuracy of 90.9% .Regarding EEG pattern and background, about 91% of patients in group A had a background which was either normal(defined as a reactive alpha or beta rhythm with voltage more than or equal 10Mv) or showed interictal discharges or status epilepticus compared with only 26% of patients in group B showed this pattern and were considered as a positive predictor for good outcome. However, 74% of patients in group B showed a slow background or intermittent burst-suppression pattern compared with only 5.9% in group A with positive prediction of bad outcome with sensitivity 74%, specificity 95% and accuracy 81.8%.Regarding absent pupillary reflex, it has a sensitivity of 70.34% and specificity 64.7% with accuracy 68.18% for detecting patients with bad clinical outcome with about 70.4% of patients in group B compared with 35% of patients in group A had absent bilateral pupillary reflex 72h after ROSC. However, bilateral absent corneal reflex has a low sensitivity and specificity (59.2%, 58.8% respectively) for detecting those with bad clinical outcome as about41.2% of patients in group A and 59.3% of patients in group B had absent corneal reflex after 72h of ROSC. Regarding TCD mean values, a ROC curve analysis for the cut off value of MFV and PI reveals that a cut off MFV equal or less than 25cm/sec associated with 55.6% sensitivity and 100% specificity for prediction of bad clinical outcome with area under the curve equal 0.742, while a pulsatility index cut off value of more than 1 is associated with 55.6% sensitivity and 94% specificity for bad clinical outcome with 0.702 area under the curve.

Table (2): Agreement (Sensitivity, specificity) for MFV, PI, GCS, and their combination to predit bad outcome cases.

	AUC	p	95% C.I	Cut off	Sensitivity	Specificity	PPV	NPV
MFV	0.742*	0.007*	0.577-0.906	≤25	55.56	100.0	100.0	58.62
PI	0.702*	0.026*	0.527-0.876	>1	55.56	94.12	93.75	57.14
GCS 24h after rewarming	0.846*	<0.001*	0.720-0.973	≤8	100.0	29.41	69.23	100.0
Combination (MFV+PI+GCS)	0.865*	<0.001*	0.750-0.980		85.19	70.59	82.14	75.0
AUC: Area under a curve								

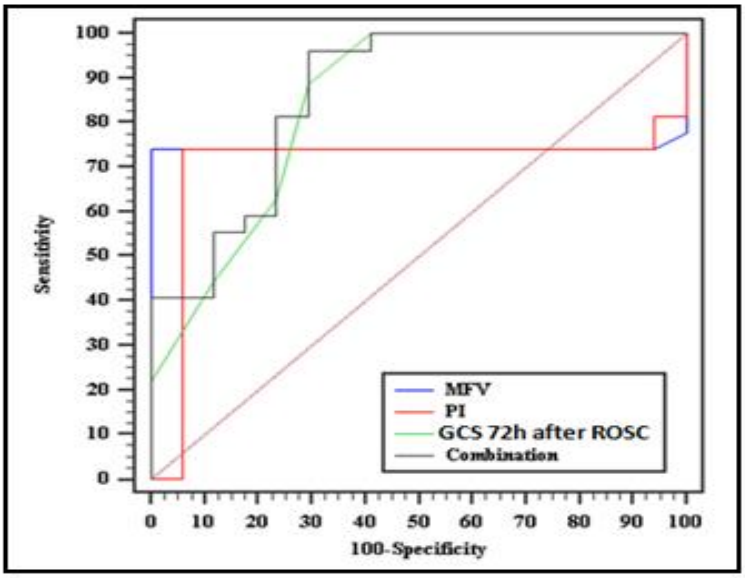

Figure (3): ROC curve for MFV, PI, GCS 72h after ROSC and their combination to predict bad outcomes cases.

Discussion

Many studies were carried out during the last 10 years for prognostication after CA. Different parameters were used as a prognostic indicators after CA including brain biochemical markers mainly NSE, SSEP and EEG. However, limited studies used different parameters to produce a full prognostic criteria. Also, limited studies addressed TCD parameters and values for prognostication after CA. Regarding TTM, also not all studies done to evaluate the accuracy of different prognostic parameters introduced in the era of TTM and hypothermia induction which clinically changes the overall outcome.

In our study, CPC scale was used as a predictor for good and bad outcome. 17 patients were considered to have a favorable final outcome however, 27 patients were considered to have a bad clinical outcome. In the reverse to the most studies using CPC scale for outcome detection, we used grades 1, 2 and 3 as a predictor for good outcome and grades 4 and 5 as a predictor for bad outcome. In most of the other studies, grade 3 CPC scale was considered as a predictor for poor outcome. In our study, we considered any patients with preserved awareness to have a good outcome (i.e CPC scale 1 to 3). This because most patients who were aware but with severe disability (CPC grade 3) at one month post-CA improved clinically at 3 month in the reverse of patients with a persistent vegetative state who will not likely to improve as shown in most of the studies.

Many neuroprotection clinical trials on in the modern resuscitation era failed to show significant outcome benefit[46,47]. Because recent interventions targeting the preservation of brain function, the (CPC) was developed as the CNS outcome measure[48]. In Utstein-style reporting, the CPC prognostic scale became the most commonly applied standard for post-CA outcome measurement[49]. The CPC was adapted from the GOS for TBI. The strengths of the CPC are simplicity, extensive use and stratify patients in to good and bad outcomes. Despite the widespread use of the CPC scale and historical importance, however, no reliability or validity studies have ever been done for any post-CA time points for which it has been used. In our study, we assess CPC scale at 1 month post-CA. The CPC purports to assess aspects of functioning after ROSC, with scores from 1 (good cerebral performance/ normal life) to 5 (brain death)[50]. Each score, however, includes multiple aspects of function. For example, a CPC grade of two represents three domains of function: impairment (eg, the presence of seizures, hemiplegia, dysarthria, or permanent mental or memory changes), level of performed activities (eg, ability to dress independently, to travel by common transportation, to prepare food), and participation level (sufficient brain function to work as part-timer in a sheltered environment). Unfortunately, it has not been proved that the CPC scale has sufficient sensitivity to assess all domains. Moreover, rater bias as to the domain that is the primary focus may differ if the CPC score is derived early in the emergency department (consciousness), one month later in a rehabilitation facility (cognition), or one year after discharge to home (activities of daily living). The CPC has been applied as an measure of outcome in multiple follow-up studies of CA, For example, studies have examined the relationship of the CPC and neurological[,(48,50,51)]cognitive[52],functional[50], or quality-of-life4[50,53,54)] final outcomes at six months after CA. A numerous studies with enhanced methodology, prolonged periods of observation, and more detailed measurements show that outcomes remain unclear. These studies, for example, those by Hsu et al[50] and Raina et al [55] which used discharge CPC scores to detect outcomes at one month after CA, raise concerns about overestimation of positive longer-term outcomes. Likewise, Tiainen et al[52] showed that among 93% of patients who were classified as having a favorable outcome (CPC score 1 or 2) three months after CA, 34% had moderate or severe deficits in standardized neuropsychological measures. However in our study, only about 39% of the study group showed a favorable outcome(CPC 1,2,3) and 61% showed a poor outcome (with mean age 60±15 years), however, CPC scale was performed after one month in our study. Comparable to us, in a study carried out by Gamil NM et al.[56], 28.9% of patients developed a favorable outcome(CPC 1-2) and about 71% of the patients had a bad final outcome in a study of 45 adult patients (mean age 51 ±12 yr) successfully resuscitated from CA with grade 3 CPC scale includes only 3 patients. A systematic review of twenty-eight studies examining cognitive impairment three months after OHCA found impairment (mainly attention, memory and executive function) in 6% to 100% of patients[57]. In the same report, the three largest prospective studies showed significant rates of impairment, ranging from 42%[58] to 50%[59] to 60%[60] at 3 months. In another study, up to 74% of survivors have low societal participation at 3 years[61]. This diversity of results depends on the exact timing of performing CPC scale, use of TTM after ROSC, number of patients recruited and the domain of CPC that accurately tested. A direct comparison of the CPC and the Health Utilities Index showed that the CPC is an important tool indicating broad functional outcome categories that are useful for a number of key clinical and research applications but should not be considered a substitute for the Health Utilities Index[62,63]. Collectively, these studies suggest that although the patient may survive, some neurological dysfunction is perhaps more common than realized.

Clinical examination

A-Brainstem Reflexes

Absence of one or more of brainstem reflexes during the first hours after ROSC cannot be considered as being specific for bad outcome, as some patients who did not have these reflexes early may in fact regain their consciousness in the following days[64]. Conversely, patients who didn't regain their brainstem reflexes, without presence of sedation 72 hours after ROSC are unlikely to regain their consciousness; however, the sensitivity of absent brainstem reflexes for outcome detection at this point of time is low[64].

The Brain Resuscitation Clinical Trials found a 19% sensitivity and a 0% false-positive rate for lack of pupillary reflex at 72h in a 262 patients [65]. A study done by Zandbergen et al. which did not use TTM after ROSC and involved 407 patients found a 22% sensitivity for prediction of bad outcome of lack of pupillary reflexes and 28% for absent corneal reflex at 72h , with a 0% false-positive rate [66].In our study, As regards pupillary reflex , there was a significant correlation with CPC scale as patients with preserved pupillary reflex 72 hours after ROSC were associated

with better outcome with mean CPC of 3 compared with 4 in those patients with absent pupillary reflex. However, there was no significant correlation between outcome and presence of corneal reflex. Regarding absent pupillary reflex, it has a sensitivity of 70.34% and specificity 64.7% for detecting patients with bad clinical outcome. In another study carried out by Gamil NM et al. [56], the overall clinical examination including GCS, pupillary and corneal reflexes showed a sensitivity of 87.1%, specificity of 84.6% for bad outcome detection.

B- Motor Response

Reports on the accuracy of prognosis on the basis of best motor response to mechanical stimuli in post-CA patients shown less consistent results [67]. In general, its accuracy is higher when it is assessed after 72h than after 24 or 48h post-CA [66,67]. In a study conducted by Levy et al.'s, none of the patients who had extensor posturing, flexor posturing, or absent motor response (M-score ≤ 3) to painful stimuli 72h after the CA recovered with a satisfactory neurological outcome[68].

A meta-analysis of 25 (prospective and retrospective) studies showed that at day two and three after CA the motor score (M-score ≤ 3) is a predictor for bad outcome with accuracy similar to SSEPs, however, these findings cannot be necessarily applied to patients undergo TTM [69].

A study of 37 patients subjected to TTM showed that 2 of 14 patients without motor response after 3 days post-CA regained their awareness [70]. This indicates that early post arrest absent or abnormal motor activity do not always predict bad outcome after TTM [70]. It was found that an M-score more than 3 on the day one after sedation stoppage predicts good outcome (CPC 1 and 2), with a specificity 100% and 43% sensitivity [71].However, this study also reveals that an M-score of one up to four days after stoppage of sedation not always predict bad outcome in each patient [71]. A GCS of four at day four after sedation stoppage predicted bad outcome, with a 95% specificity and a 47% sensitivity [72].In our study, GCS of eight assessed 72 hours after ROSC (24 hours after rewarming) showed a 100% sensitivity and 29% specificity for bad outcome detection .However, adding different TCD parameters to clinical examination (GCS score) will increase the specificity of GCS for bad outcome prediction to 70% . Comparable to us, a study of Gamil NM et al. [56] revealed that combination of clinical examination(including GCS and brain stem reflexes) and TCD measurement after 72 hr of CPR raised the percentage of positive prediction of poor outcome to 100%, with sensitivity of 90.6%, specificity of 100% and accuracy of 93.3% than the use of single modality alone.

C- Myoclonic Status Epilepticus

Myoclonic SE is defined as repetitive, spontaneous, unrelenting and generalized multifocal jerks affecting the extremities, face and trunk in comatosed patients[64]. Myoclonic SE presents typically in the first day ROSC[48]. Many patients have evidence of SE by EEG, but it is not a requirement for diagnosis confirmation[48]. Prior to TTM era, myoclonic SE was usually associated with a bad outcome in patients after ROSC [48]. In rare situations good outcomes was shown in patients with myoclonic SE after a CA secondary to respiratory failure [48].

The prevalence of myoclonic SE after CA was different between studies[66]. In a study carried out by Zandbergen et al. of 407 patients, Myoclonic SE was observed in four percent of patients at twenty four hours after CA compared to 6% in our study showed myoclonis SE early after CA and all of them had a bad outcome[66].

Electroencephalography

EEG has been extensively studied in comatose post-CA patients before the use of TTM[35]. In general, most studies agree that EEG reactivity and early improvement of brain activity were associated with good outcome; however in some studies and mainly before the era of TTM, clinical and EEG evidence of seizures correlates with bad outcome[36]. Also, prior the era of TTM, many EEG patterns have been associated strongly with bad outcome, most notably burst-suppression background with generalized epileptic form discharges, a low voltage background (< 20 μV) and periodic generalized complexes on a suppressed or flat background; however, none of those patterns predicts bad outcome accurately[64].A meta-analysis of 408 post-CA comatose patients conducted by Bassetti and Scollo-Lavizzari through EEG recorded six hours or more after ROSC showed good CNS recovery in 79% of patients with a normal dominant alpha activity and 0% in those showing low-voltage background dominated by delta activity , alpha coma, , periodic-generalized complexes with flat or low-voltage background and isoelectric EEG (< 10 μV)[39]. A 43% of patients with predominant mixed delta-theta activity had a favourable outcome[39].

In our study, we also noticed a significant correlation between EEG voltage, reactivity and pattern and CPC scale. Regarding cEEG, patient with normal pattern was having a mean CPC scale of 2, however, those with low voltage EEG was having a mean CPC scale of 4. Regarding the routine EEG performed 24 hours after rewarming, patients with normal voltage and reactivity was associated with a mean CPC scale of 2, however, those with a low voltage non reactive EEG was associated with CPC scale of 4 or 5. For EEG pattern, a normal pattern (defined as a background alpha or beta with voltage equals or more than 10Mv) was associated with CPC scale ranging from 1 to 3, a generalized background slowness (defined by background non reactive delta or theta activity) or intermittent burst suppression pattern were associated with mean CPC of 4 and an epileptic form EEG with diagnosis of interictalepilepti form discharges or SE was associated with mean CPC scale of 3. A 3 patients in our study diagnosed clinically as a brain dead with their EEG showed an isoelectric non reactive pattern. And it was significantly noticed that patients with initial diagnosis of seizures and SE have a better outcome as compared to those with a low voltage, slow background or non-reactive EEG. In a study carried out by Andrea O Rossetti.et.al[73], Continuous EEG recording was started 12 ± 6 hours after CA and lasted 30 ± 11 hours and found that a nonreactive cEEG background (12 of 15 (75%) among nonsurvivors versus none of 19 (0) survivors; $P < 0.001$) and prolonged intermittent "burst-suppression" activity (11 of 15 (73%) versus none of 19; $P < 0.001$) were significantly associated with mortality. Similarly In our study, regarding EEG voltage, a lower voltage EEG is considered as a positive predictor for bad outcome as about 6% of patients in group A had a low voltage compared with 89% of patients in group B with a sensitivity of 88.9%, specificity 94% and accuracy 90.9%.

Also, a non reactive EEG was considered as a positive predictor for bad outcome as 96.3% (compared to 75% in Andrea O Rossetti.et.al study with a positive predictive value of 100% after TTM) of patients in group B had a non reactive background compared with 17.6% in group A with a 96.3% sensitivity, 82.3% specificity and accuracy of 90.9% .Regarding EEG pattern and background, about 91% of patients in group A had a background which was either normal or showed interictal discharges or status epilepticus compared with only 26% of patients in group B showed those patterns and were considered as a positive predictor for good outcome. However, 74% of patients in group B showed a slow background or intermittent burst-suppression pattern compared with only 5.9% in group A with positive prediction of bad outcome with sensitivity 74%, specificity 95% and accuracy 81.8%. However, Rossetti.et.al found that patients with nonreactive background or seizures/epileptic form discharges on cEEG showed no improvement after TTM, but in our study, we consider seizures as a favourable outcome if detected early and adequately treated .In a recent study carried out by Erik Westhall and his colleagues[74], they found that whether patients with electrographic status epilepticus may benefit from antiepileptic medication is unclear as continuous EEG monitoring was not available in most centers of this study and some patients with intermittent electrographic seizures were missed in reverse to our study as all patients monitored continuously by EEG for the first 48h ,so, seizures were detected early. However, those patients have a CPC scale of 3 which was considered as a bad outcome grade in most of the other studies.

Transcranial Doppler

Few studies used TCD as a prognostic indicator after CA. It is easy, inexpensive and a non-invasive tool that can be helpful for rapid and bedside repeatable measurement of CBF velocity (FV) [75].However, TCD is considered as important ancillary testing for confirmation of brain death if apnea test cannot be performed. A recent review[76] revealed that in patients who still comatose 2 hr after ROSC, the main TCD patterns includes decreased MFV and high PI. Normal levels should be reached after 72 hr as there were no complications. If this hypodynamic pattern persists, this is an indicator of bad outcome. The etiology of CBF abnormalities shown after ROSC is not fully understood. The suggested possibilities are cerebral oedema, vasospasm and blood cell aggregation and this is responsible for the suggested TCD changes seen after CA[77,78]. A study carried out by Gamil NM et al. [56], the mean values of MFV were low immediately after ROSC in both groups, which significantly increased in group I (good outcome) in comparison to group II(bad outcome). In the same study, PI was high in both groups after ROSC but significantly decreased in group I with a TCD (MFV and PI) measured 72 post-ROSC shown to be a positive predictor of bad outcome with sensitivity 84.4% and specificity of 92.3%, and accuracy of 86.7%.In our study, a ROC curve analysis for the cut off value of mean MFV and PI reveals that a cut off MFV equal or less than 25cm/sec associated with 55.6% sensitivity and 100%

specificity for prediction of bad clinical outcome while a pulsatility index cut off value of more than 1 is associated with 55.6% sensitivity and 94% specificity for bad clinical outcome detection. The mean value of MFV in the study carried out by Gamil NM et al. [56] was 47±11 compared to 40.5±30 in our study, so we used a lower cut of value for MFV explaining the lower sensitivity and higher specificity seen in our study. The wide range of MFV in our study can be explained by that we used the mean values of MFV measured 2,6, 24,48 and 72 hours after ROSC, while in Gamil NM et al. , the author depends only on the value of MFV measured 72h after ROSC. However, we suggested that the mean changes of TCD parameters in the same patient over the first 3 days after ROSC will predict CBF changes in the same patient over time which will be more accurate for outcome detection compared to a single reading. However, our results were in agreement with that of Wessels et al.[78] who carry a study on 39 patients after ROSC and observed that the mean value of peak SFV in MCA were higher significantly in group 1 (survivors) done at 1.5, 4, 8, 16, 24 and 72 h after ROSC. However, both the studies done by Gamil NM et al. [56] and Wessels et al.[78]Did not used TTM after ROSC. Another study carried out by Álvarez-Fernández etal.[79] revealed that persistence of a diffuse hypodynamic TCD changes (low MFV and high PI) predicts bad neurologic recovery. Also, Early or late presence of a diffuse hyperdynamic TCD changes (high MFV and low PI) is also associated with bad prognosis using serial TCD measurements because the progression to intracranial hypertension and brain death. Limited studies using TCD for prognostic purposes after CA used TTM and hypothermia.

Conclusion

A combination of TCD data with clinical examination through GCS assessed 24 hours after rewarming will increase GCS specificity (using a cut off value of less than 8 a s a predictor for bad outcome) for prediction of bad clinical outcome from 29.4% to 70.59% with 0.865 area under the curve. Thus, adding both EEG and TCD parameters to clinical neurological examination will have positive impact and will improve the specificity of bad outcome detection which of great help in prognostication after CA.

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