Al-Azhar Med. J. *Vol. 45(2), April 2016, 331-343*
DOI: 10.12816/0029133/ORG. No. 91226 ***Issues_title_English:*** 2016 Vol.45 Issue 2

ELEVATED AT ADMISSION SERUM NEURON SPECIFIC ENOLASE AND HYPERGLYCEMIA ARE PREDICTORS OF POOR OUTCOME OF POST-RESUSCITATION PATIENTS

By

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ABSTRACT

Background: Optimal survival following sudden cardiac arrest requires heart and brain resuscitation. In patients who achieve cardiac resuscitation, brain recovery from anoxic injury is variable. Neurological sequelae range from complete recovery to coma with brain death. Thus, ideally outcome assessment would incorporate functional and neurologic status.

Objective: Evaluation of the prognostic yield of estimation of at admission random blood glucose (RBG) and serum neuron specific enolase (NSE) levels, and determination of the value of NSE serum level re-estimation 48 hours after admission in resuscitated post-cardiac arrest patients.

Patients and Methods: The study included 90 cardiac arrest patients; 75 were out-of-hospital and 15 were in-hospital cardiac arrest. All patients received mild therapeutic hypothermia irrespective of the initial rhythm admission. Blood samples were obtained for estimation of RBG and serum SNE. At 48-hours after admission, serum NSE was re-estimated, and the percentage of change in relation to at-admission level was calculated. Clinical evaluation was conducted using the Acute Physiology and Chronic Health Evaluation (APACHE II). Mortality rate throughout duration of ICU stay was determined. Neurologic outcomes were evaluated using the Cerebral Performance Category (CPC) score collectively as favorable neurological outcome (CPC score of 1-2) or unfavorable outcome (CPC score of 3-5).

Results: Mean time elapsed till return of spontaneous circulation (ROSC) since arrival to emergency department was 14.3±3.6; range: 8-19 minutes. Mean ICU stay was 16.3±7.4; range: 3-30 days. Thirty-one patients died (34.4%), 18 patients (20%) had unfavorable neurological outcome (CPC-3), and 41 patients (45.6%) had favorable outcome. Mean at admission RBG levels were significantly higher in non-survivors compared to survivors, with significantly higher levels in survivors had unfavorable outcome compared to those had favorable outcome. Mean levels of serum NSE estimated at admission and 48-hours, and percentage of change were significantly higher in non-survivors compared to survivors, with significantly higher levels in survivors had unfavorable outcome compared to those had favorable outcome. There was a negative significant correlation between survival rate and favorable neurologic outcome and levels of RBG and NSE, patient's age, time till ROSC and APACHE II score. ROSC curve and regression analyses were defined at admission hyperglycemia as the highly significant specific predictor for mortality and high serum NSE kinetics for prediction of unfavorable neurologic outcome.

Conclusion: Hyperglycemia could specifically predict mortality and high serum NSE levels estimated at admission and 48-hours later, with elevated percentage of increase which could specifically predict poor neurologic outcome of resuscitated post-arrest patients.

Keywords: Post-cardiac arrest, Neuron specific enolase, Serial estimations, Mortality, Neurological outcome.

INTRODUCTION

Overall survival rate from out-of-hospital cardiac arrest has not increased in parallel with the improvements in cardiopulmonary resuscitation ((CPR)) (**Fairbanks et al., 2007**).

The hospital discharge rate is 15% in a meta-analysis that included a total population of over 26,000 patients (**Nolan et al., 2007**). Additionally, the majority of patients who achieve return of spontaneous circulation after successful CPR have a high risk to death in the post-arrest period (**Gaieski et al., 2012**). Functional and neurologic status following cardiac arrest is a more meaningful clinical outcome than simply hospital survival when trying to judge the effectiveness of resuscitation care (**Raina et al., 2008**). CPC score overcomes is advantageous in that assessment does not require direct subject contact, does not require assessment at specified time points and corresponds to quality of life and functional status derived (**Stiell et al., 2009 and Ajam et al., 2011**). Enolase, also known as phosphopyrovate hydratase, is ametalloenzyme responsible for the catalysis of the conversion of 2-phosphoglycerate (2-PG) to phosphoenolpyrovate (PEP),the ninth and penultimate step of glycolysis.Enolase belongs to the family of lyases, specifically the hydro-lyasis,which cleave carbon-oxygen bonds. the systematic name of this enzyme is 2-phospho-D-glyceratehydro-lyase (phosphoenolpyrovate-forming).

The reaction is reversible, depending on environmental concentrations of substrates. The optimum pH for the human enzyme is 6.5. Enolase is present in all tissues and organisms capable of glycolysis or fermentation, functionally active can easily diffuse to the extracellular medium and cerebrospinal fluid when neuronal membranes are injured (**Cooper, 1994, Chai et al., 2004, Klenchin et al., 2004, Sims et al., 2006 and Kang et al., 2008**).

The current study aimed to evaluate the prognostic yield of estimation of at admission random blood glucose and serum neuron specific enolase levels, and to determine the value of NSE serum level re-estimation at 48 hours after admission in resuscitated post-cardiac arrest patients.

PATIENTS AND METHODS

The study included 90 patients developed cardiac arrest; 75 patients had out-of-hospital arrest and 15 patients had in-hospital arrest. Mean age of enrolled patients was 62.6±4.8; range: 53-73 years. There were 61 males (67.8%) and 29 females (32.2%). Forty six patients (51.1%) had underlying cardiac disorder which may be the cause of cardiac arrest, 23 patients (25.6%) had hypovolemia, 16 patients (17.8%) had COPD/ emphysema, 19 patients (21.1%) were hypertensive and 9 patients (10%) had previous cardiac surgery. Seventy patients (77.8%) were obese, 13 patients (14.4%) were morbidly obese, 6 patients (6.7%) were overweighed, and only one patient (1.1%) was of average BMI. History taking defined 23 diabetic patients (25.6%) while 67 patients (74.4%) had no previous history of diabetes mellitus.

The current study was conducted at Departments of Neurology, Anesthesia & ICU, and Clinical Pathology at Al Dar

hospital, KSA through the period of March 2013 till Oct 2014, after approval of the study protocol by the Local Ethical Committee and obtaining written fully informed near patients' relative consent .

Plasma glucose level was measured using enzymatic reference method with hexokinase, on the cobas Integra-400 plus analyzer (Roche Diagnostics GmbH, Mannheim, Germany). Serum NSE level was measured using a solid-phase electrochemiluminescence immunoassay "ECLIA" with double monoclonal antibodies (Sandwich principle) directed against NSE (Roche Diagnostics GmbH, Mannheim, Germany) on an Elecsys - 2010 instrument.

Six milliliters blood were withdrawn from every patient and divided into two vacutainer tubes; For blood glucose estimation, two milliliters blood were collected in gray vacutainer with fluoride additive. The remaining four milliliters blood were transferred to a red vacutainer with no additive for NSE measurement which was allowed to clot for 20-30 minutes. Both tubes were centrifuged at 3000 rpm for 10-15 minutes to separate plasma and serum respectively. Blood glucose was measured immediately and serum was stored frozen at -20 °C till NSE assessment (**Muley et al., 2003**).

Another four milliliters blood were obtained from all patients 48 hours after admission for estimation of serum NSE, and the difference in serum NSE levels was calculated in relation to at admission level as percentage of change for determination of NSE kinetics.

All patients received mild therapeutic hypothermia irrespective of the initial rhythm. Therapeutic hypothermia was initiated after admission with an intravenous infusion of cold saline (4°C, 1000 to 1500 ml bolus) followed by surface cooling with commercially available non-invasive devices (Arctic Sun2000® Medivance, Louisville, Colorado, USA). The target temperature was maintained for 24 hours. All patients received intravenous sedation and analgesia using a combination of midazolam (0.125 mg/kg/h) and fentanyl (0.002 mg/kg/h) in addition to muscle relaxation using repetitive administration of pancuronium (0.1 mg/kg) in order to prevent shivering.

At base line, demographic and clinical information were obtained using the Acute Physiology and Chronic Health Evaluation (APACHE II). For APACH-II score, zero points were usually assigned for the neurologic evaluation, since the majority of patients were arrested (**Knaus et al., 1985**).

Mortality rate throughout duration of ICU stay was determined. Neurologic outcomes were evaluated using the Cerebral Performance Category (CPC) score collectively as CPC score of 1-2 indicted favorable neurological outcome**, (Jennett and Bond, 1975).**

Statistical analysis: Obtained data were presented as mean±SD, ranges, numbers and ratios. Results were analyzed using Wilcoxon; ranked test for unrelated data (Z-test). Possible relationships were investigated using Pearson linear regression. Sensitivity and specificity of estimated parameters as predictors for outcome were evaluated using the receiver operating characteristic (ROC) curve analysis judged by the area under the curve (AUC) compared versus the null

hypothesis that AUC=0.05. Regression analysis using Stepwise Method was used for identification of specific predictors for poor outcome. Statistical analysis was conducted using the SPSS (Version 15, 2006) for Windows statistical package. P value <0.05 was considered statistically significant.

RESULTS

At time of admission, the initial cardiac rhythm was ventricular fibrillation in 57 patients (63.3%), asystol in 19 patients (21.1%), and 14 patients (15.6%) had pulseless electrical activity. For resuscitation, all patients received hypothermia as a basic line of management in conjunction with defibrillatory shock wave in 73 patients (81.1%), and 52 patients (57.8%) required coronary reperfusion therapy via PCA for acute coronary syndrome. Mean time elapsed till ROSC since arrival to emergency department was 14.3±3.6; range: 8-19 minutes. Forty-seven patients (52.2%) required >15 minutes till ROSC, 22 patients (24.4%) achieved ROSC within 10-15 minutes, and 21 patients (23.4%) achieved ROSC within <10 minutes.

Throughout a mean ICU stay of 16.3±7.4; range: 3-30 days, 31 patients died for a mortality rate of 34.4%. Out of the 59 survivors (65.6%), 41 patients (45.6%) had favorable outcome (CPC 1-2); 17 patients were CPC-1, while 24 patients were CPC2 and 18 patients (20%) had unfavorable outcome (CPC-3 - Table 1).

Mean at admission RBG levels were significantly higher in non-survivors compared to survivors with significantly higher levels in survivors had unfavorable outcome compared to those had favorable outcome, (Table 2). As regards CPC grading of patients had favorable outcome, patients had CPC2 showed non-significantly higher at admission serum RBG levels compared to those had CPC1 (Table 2).

Mean levels of serum NSE estimated at admission and 48-hours were significantly higher in non-survivors compared to survivors, with significantly higher levels in survivors had unfavorable outcome compared to those had favorable outcome. As regards CPC grading of patients had favorable outcome, patients had CPC1 had significantly lower at admission and 48-hours serum NSE levels compared to those had CPC2. Mean percentage of change of serum NSE in non-survivors was significantly higher compared to survivors, with significantly higher percentage of change of serum NSE in survivors had unfavorable outcome compared to those had favorable outcome. As regards CPC grading of patients had favorable outcome, CPC1 patients had significantly lower percentage of change of serum NSE compared to those had CPC2 (Table 3).

Survival of post-resuscitation patients showed negative significant correlation with RBG level, serum NSE level, patient's age, time till ROSC, APACHE II score and patient's BMI in decreasing order of significance. Favorable neurologic outcome showed negative significant correlation with serum NSE level, APACHE II score, RBG level, patient's age and time till return of spontaneous circulation in decreasing order of significance (Table 4).

ROC curve analysis for predictivity of mortality was defined at admission hyperglycemia as the highly significant specific predictor for mortality, followed by high serum NSE kinetics, patients' age, APACHE II score and high body mass

index in descending significance for prediction of mortality. For prediction of unfavorable neurologic outcome, high serum NSE kinetics were the highly significant specific predictor, followed by time till ROSC, patient's age and hyperglycemia, in descending order of significance (Table 5).

Regression analysis for predictivity of mortality defined at admission hyperglycemia as the highly significant specific predictor for mortality in 4 models, followed by persistently high serum NSE at 48-hr after admission in 3 models, patient's age in 2 models and APACHE II score in one model, (Table 6). For prediction of unfavorable neurologic outcome, high percentage of elevation of serum NSE was the highly significant : specific predictor in 5 models, followed by patient's age in 4 models, time till ROSC in three models, high at admission serum NSE in 2 models and hyperglycemia in one model (Table 7).

Table (1): Patients' enrollment data.

Data				No, % and range
Age (years)	Strata	>50-<60		26 (28.9%)
		>60-<70		58 (64.4%)
		>70		6 (6.7%)
	Total (mean±SD)			62.6±4.8 (53-73)
Gender; M:F				61:29
BMI data	Weight (kg- mean±SD)			89±6.7 (68-103)
	Height (cm- mean±SD)			165±2.9 (160-175)
	BMI(kg/m^2)	Strata	Average (<25)	1(1.1%)
			Overweight (25-30)	6 (6.7%)
			Obese (>30-35)	70 (77.8%)
			Morbid obese (>35)	13 (14,4%)
		Total (mean±SD)		**32.7±2.3 (24.4-37.2)**
Associated co-morbidities		Diabetes		23 (25.6%)
		Hypertension		19 (21.1%)
		CAD		46(51.1%)
		CHF		28(31,1%)
		Previous cardiac surgery		9(10%)
		COPD/emphysema		16(17.8%)!
Place ot arrest		Out-of-hospital		75 (83.3%)
		In-hospital		15(16.7%)
Cause ot arrest		Underlying cardiac disorder		38 (42.2%)
		Respiratory failure		18 (20%)
		Metabolic disorders		9(10%)
		Hypovolemia		23 (25.6%)
Initial cardiac rhythm		Ventricular fibrillation		45 (50%)
		Asystol		26 (28.9%)
		Pulseless electrical activity		19(21.1%)

Table (2): Mean ±SD at admission levels of random blood glucose(RBG) estimated in studied patients categorized according to outcome at time of discharge.

Studied patients			RBG (mg/dl)
Survivors	Favorable	CPC1 (n=17)	265.9±13.8 (240-285)
		CPC2 (n=24)	275.2±14.8 (250-315)
		Total (n=41)	271.3± 14.9 (240-315)
	Unfavorable (CPC3; n=18)		291.1±16.7 (260-315)t
	Total survivors (CPC3; n=59)		277.4±17.9 (240-315)
Non-survivors (CPC4-5; n=31)			310.4±16.9(270-330)t

Table (3): Mean ±SD levels of serum NSE estimated at admission and 48-h after admission in studied patients categorized according to outcome at time of discharge**.**

Levels of serum NSE / Patients			At admission	At 48-hours	% ot change
Survivors	Favorable	CPC1 **(n =17)**	31.4±10.2 (20-62)	35±11.6 (24-69)	11.6±3.9 (3.7-20)
		CPC2 (n=24)	43.2±12.4* (25-63)	51±14.5* (31-75)	18.4±5.9* (9.5-29.8)
		Total (n=41)	38.3±12.9 (20-63)	44.4±15.4 (24-75)	15.6±6.1 (3.7-29.8)
	Unfavorable (CPC3; **n=18)**		58.3±13.7† (30-77)	71.3±16† (36-92)	22.8±7.8† (11.4-42.6)
	Total survivors (n=59)		44.4±16 (20-77)	52.6±19.9 (24-92)	17.8±7.4 (3.7-42.6)
Non-survivors (CPC4-5; n=31)			70.9±l5.l ‡ (35-93)	89±16.3‡ (45-121)	27.lii3.5‡ (8.4-55.4)

Ranges are in parenthesis; *: significant difference versus CPC1 patients; †: significant difference patients had favorable outcome; ‡: significant difference versus survivors; NSE: neuron specific enolase.

Table (4): Correlation coefficient between constitutional, clinical and laboratory data and outcome of post-resuscitation data.

Correlations / Data	Survival		favorable neurologic outcome	
	r	p	R	p
Age (years)	-0.521	<0.001	-0.499	<0.001
Body weight (kg)	-0.049	>0.05	-0.200	>0.05
BMl(kg/m2)	-0.281	=0.007	-0.099	>0.05
APACHE 11 score	-0.287	=0.006	-0.530	<0.001
Time till ROSC (min)	-0.418	<0.001	-0.263	=0.044
RBG(mg/dI)	-0.671	<0.001	-0.582	<0.001
Serum NSE (ng/ml)	-0.630	<0.001	-0.513	<0.001

APACHE II: Acute Physiology and Chronic Health Evaluation, ROSC: return of spontaneous circulation, RBG: random blood glucose; NSE: neuron specific enolase; r: Pearson's coefficient.

Table (5): ROC curve analysis for specific predictors outcome of post-resuscitation patients as judged by AUC.

AUC / Parameters		Mortality					Unfavorable neurologic outcome				
		AUC	SE	P	CI 95%		AUC	SE	TSTg	CI 95%	
					Lower	Upper				Lower	Upper
(Age /years)		0.816	0.045	0.001>	0.728	0.904	0.825	0.053	0.001>	0.720	0.929
(BMI kg/m^2)		0.349	0.065	0.019=	0.221	0.478	0.550	0.087	0.05<	0.380	0.721
APACHE U score		0.750	0.052	0.001>	0.649	0.851	0.657	*0.080*	0.05<	0.500	0.814
(Time to ROSC/min)		0.508	0.062	0.05<	0.387	0.629	0.837	0.051	0.001>	0.738	'0.957
(RBG mg/dl)		0.919	0.030	0.001>	0.860	0.967	0.772	0.064	0.001 =	0.647	0.898
Serum NSE ng/ml `	At admission	0.701	0.063	0.002=	0.579	0.824	0.807	0.065	0.001>	0.680	0.934
	After 48-hr	0.881	0.036	0.001>	0.837	0.967	0.845	0.058	0.001>	0.732	0.958
	% of change	0.902	0.033	0.001>	0.810	0.952	0.864	0.054	0.001>	0.759'	0.970

APACHE II: Acute Physiology and Chronic Health Evaluation, ROSC: return of spontaneous circulation, RBG: random blood glucose; NSE: neuron specific enolase; AUC: Area under curve; CI: confidence interval.

Table (6): Regression analysis for specific predictors mortality of post-resuscitation patients.

Studied patients			RBG (mg/dl)
Survivors	Favorable	CPC1 (n=17)	265.9±13.8 (240-285)
		CPC2 (n=24)	275.2±14.8 (250-315)
		Total (n=41)	271.3± 14.9 (240-315)
	Unfavorable (CPC3; n=18)		291.1±16.7 (260-315)t
	Total survivors (CPC3; n=59)		277.4±17.9 (240-315)
Non-survivors (CPC4-5; n=31)			310.4±16.9(270-330)t

APACHE II: Acute Physiology and Chronic Health Evaluation, RBG: random blood glucose: S. NSE: serum neuron specific enolase.

Table (7): Regression analysis for specific predictors poor neurologic outcome of post-resuscitation patients**.**

Models	Parameters	T	P
Model 1	RBG (mg/dl)	2.257	=0.028
	S.NSE (ng/ml) at admission	2.519	=0.015
	ROSC	2.888	=0.006
	% of change of S. NSE	3.201	=0.002
	Age (years)	4.255	<0.001
Model 2	S.NSE (ng/ml) at admission	2.447	=0.018
	ROSC	3.179	=0.002
	% of change of S.NSE	3.300	=0.002
	Age (years)	4.149	<0.001
Model 3	ROSC	2.842	=0.006
	Age	4.298	<0.001
	% of change of S. NSE	4.426	<0.001
Model 4	Age	4.254	<0.00
	% of" change of S. NSE	6.010	<0.001
Model 5	% of change of S.NSE	6.113	<0.001

ROSC: return of spontaneous circulation, RBG: random blood glucose; S. NSE: serum neuron specific enolase.

DISCUSSION

One of alarming and interesting observation of the current study was that all enrolled patients were hyperglycemic, irrespective of being previously diabetics or not. Moreover, mean at admission, RBG level was significantly higher in non-survivors compared to survivors and in survivors who had unfavorable outcome (CPC3) compared to those had favorable outcome (CPC1-2) with non-significant difference between RBG levels in patients had CPC1 and CPC2. Additionally, statistical analyses showed a negative significant correlation between at admission RBG levels and outcome and it was found to be the highly significant specific predictor for mortality, but not for the neurologic outcome.

These findings pointed to a fact that cardiac arrest and resuscitation were events accompanied by stress hyperglycemia which if exaggerated worsens the prognosis of such patient irrespective of underlying cause of arrest or being diabetic or not. **Nielsen et al. (2011)** reported that adverse events were common after out-of-hospital cardiac arrest and sustained hyperglycemia and seizures treated with anticonvulsants were associated with increased mortality. **He et al. (2011)** determined blood glucose levels at four time points, including pre-cardiopulmonary bypass (CPB), pre-deep hypothermic circulatory arrest (DHCA), post-DHCA, and at admission to ICU, and found that blood glucose level at pre-DHCA was significantly higher than that of pre-CPB and was further elevated at the time point of post-DHCA and in-ICU compared with that of pre-CPB.

Cueni -Villoz et al. (2011) found increased blood glucose variability during therapeutic hypothermia is a predictor of

in-hospital mortality after cardiac arrest independent of injury severity and mean blood glucose levels. **Nurmi et al. (2012)** found that, among cardiac arrest patients, non-survivors showed significant increase of blood glucose estimated at admission compared to pre-hospital blood glucose level and concluded that patients who are resuscitated from out-of-hospital ventricular fibrillation, and whose outcome is unfavorable are characterized by significant increase of blood glucose in the ultra-acute post-resuscitation phase.

In support of the predictability of the weak predictability of blood glucose level for neurologic outcome of post-arrest patients, **Niemann et al. (2011)** evaluated neurologic outcome and early post-arrest hyperglycemia in cardiac arrest model in animals had a peak plasma glucose value > 226 mg/dl during the initial 60 minutes after resuscitation and found early post-resuscitation stress hyperglycemia did not appear to affect neurologic outcome.

Serum NSE levels were significantly higher in non-survivors compared to survivors and in survivors who had unfavorable neurologic outcome compared to those had favorable outcome. However, serum NSE levels showed less prognostic yield for mortality compared to at admission hyperglycemia, but showed significantly high specificity for neurological outcome and both ROC curve and regression analyses defined high at admission serum NSE as significant predictor for unfavorable neurologic outcome.These data went in hand with previous studies evaluating serum NSE level in various forms of brain affection. **Meric et al. (2010)** found the initial serum NSE levels in moderate and severe head trauma patients correlate inversely with Glasgow Coma Scale at one-month and by ROC analysis, serum NSE was 87 %sensitive and 82.1 % specific in predicting poor neurologic outcome in the head trauma patients.

Another interesting finding of the current study is that the sequential estimation of serum NSE at 48 hours after admission showed progressive increase in all patients, irrespective of their outcome, but the percentage of increase was significantly higher in those had unfavorable or poor outcome. These findings indicated progressive neuronal affection even in those had favorable outcome and assuring the trend for repeated estimation without reliance on at admission levels. In support of these data, regression analysis defined increased percentage of serum NSE increase as a significant specific predictor for unfavorable neurologic outcome and as a second specific predictor for mortality after hyperglycemia.

Cronberg et al. (2011) reported that patients with NSE levels <33 ug/L at 48 hours regained the capacity to obey verbal commands, while patients with NSE levels >33 ug/L failed to recover consciousness and their MRI studies defined extensive brain injury, and non-survivors who underwent autopsy had extensive severe histologic damage. **Suzuki et al. (2012)** found that prolonged elevation of NSE suggests that total brain necrosis might not be present at the time of clinical diagnosis of brain death. **Storm et al. (2012)** reported that, in arrest patients treated with hypothermia, prognostication of unfavourable outcome by NSE kinetics between admission and

48 hours after resuscitation may be superior to prognostication by absolute NSE levels. **Topjian et al. (2012)** found serial NSE measurement aids prognostication of brain injury in asphyxial cardiac arrest due to drowning.

Michael Mlynash et al (2013) found Absolute serum NSE levels of comatose cardiac arrest patients differ between laboratories. Any specific absolute cut-off levels proposed to prognosticate poor outcome should not be used without detailed data on how neurologic outcomes correspond to a particular laboratory's method, and even then only in conjunction with other prognostic variables.

Sandroni et al. (2014) found early status myoclonus, elevated values of neuron-specific enolase at 48-72 h from arrest, unreactive malignant EEG patterns after rewarming, and presence of diffuse signs of postanoxic injury on either computed tomography or magnetic resonance imaging were identified as useful but less robust predictors. Prolonged observation and repeated assessments should be considered when results of initial assessment are inconclusive.

Pascal Stammet et al. (2015) found Serial, high NSE values have a high predictive value of poor outcome in comatose out-of-hospital cardiac arrest patients. This predictive value of NSE is not significantly affected by target temperature at either 33°C or 36°C.

Alexandra Reynolds et al. (2016) found Among survivors of cardiac arrest who undergo TH, NSE level on day 3 has value in predicting short-term, but not long-term, disability.

In support of the diagnostic and prognostic applicability of NSE serum level estimation for neurologic outcome, found that regional apparent diffusion coefficient-based prognostication was accurate in comatose out-of-hospital cardiac arrest patients who were treated with mild hypothermia, however, it only provided additional prognostic information when the 48-h NSE levels indicated a good prognosis.

The obtained results and review of literature allowed concluding that at admission, hyperglycemia could specifically predict mortality, and high serum NSE levels estimated at admission, and 48-hours later with elevated percentage of increase could specifically predict poor neurologic outcome of resuscitated post-arrest patients.

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ارتفاع نسبة انزيم نيورون سبيسيفك اينوليز فى الدم وارتفاع نسبة السكر فى الدم عند دخول المستشفى يعطى تنبؤات بنتائج سيئة للانعاش القلبى الرئوى فى مرضى ما بعد توقف القلب والتنفس

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خلفية البحث : الحياة الكاملة التى تتبع توقف القلب المفاجئ تتطلب انعاش قلب رئوى جيد، وفى المرضى الذين حدث لهم انعاش قلبى فان تأثر المخ بسبب نقص الاكسجين مختلف ، وتتراوح درجات تأثر المخ من العودة الكاملة لكل الوظائف المخية إلى الغيبوبة مع الموت المخى. لذلك، فإن التقييم المثالى للنتائج لا بد ان يكون مشتملا على الحالة العصبية والوظيفية.

الهدف من البحث: تقييم مستوى التحسن بعد دخول المستشفى وذلك بحساب نسبة السكر فى الدم وانزيم نيورون سبيسيفك اينولاز عند الدخول وبعد ثمان وأربعين ساعة فى مرضى ما بعد الانعاش القلبى.

المرضى وطرق البحث: أجريت الدراسة على تسعين من مرضى توقف القلب 75 منهم حدث التوقف للقلب خارج المستشفى و15 منهم حدث توقف القلب داخل المستشفى وكل المرضى خضعوا لخفض درجة الحرارة مفتعل بغض النظر عن إنتظامية ضربات القلب فى البداية ،وقد اخذت عينات من هؤلاء المرضى لتحليل السكر عشوائيا وتحليل إنزيم نيورون سبيسيفك انوليز عند دخول المريض المستشفى، وبعد 48 ساعة من دخول المستشفى أخذت عينة من الدم لتحليل نسبة إنزيم نيورون سبيسيفك انوليز ومقارنتها بعد ذلك بالعينة الاولى التى اخذت اثناء دخول المريض المستشفى، وتم حساب نسبة التغير فى العلاقة بين العينتين .

وقد اجرى التقييم الإكلينيكى باستخدام اباتشى 2 وتحديد معدل الوفيات خلال فترة العلاج فى العناية المركزة وتقييم الكفاءة العصبية باستخدام اختبار مدى كفاءة المخ والتى تحدد بأنها جيدة مابين 1-2 وغير جيدة مابين 3-5.

النتائج: الوقت المحتسب من وصول المريض الى الطوارئ الى رجوع الدورة الدموية كان أربعة عشر دقيقة، وثلاث من عشرة من الدقيقة بزيادة ونقص ثلاث دقائق وستة من عشرة دقيقة بمعدل ثمانية الى تسعة عشر دقيقة، وكان معدل التنويم فى العناية المركزة من ثلاثة ايام الى ثلاثين يوم.

وقد توفى واحد وثلاثون مريضا وثمانية عشر عاشوا مع الإعاقة، وواحد وأربعون مريضا عاشوا بدون اعاقة وجاءت نسبة السكر عالية فى المرضى الذين ماتوا ايضا فى المرضى الذين عاشوا مع الإعاقة أيضاً وكانت نتائج انزيم نيورون سبيسيفك إينولاز مرتفعة فى هؤلاء المرضى الذين ماتوا والذين خرجوا بالإعاقة.

الاستنتاج: إرتفاع نسبة السكر أثناء الدخول للمستشفى وارتفاع نسبة إنزيم نيورون سبيسيفك انوليز اثناء الدخول وبعد 48 ساعة يدل على نتائج عصبية سيئة وغير مرضية فى المرضى الذين خضعوا للإنعاش بعد توقف القلب.