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Forensic and clinical significance of serum amylase, lipase and gamma glutamyl transferase as predictors of outcome in head injured patients

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ABSTRACT

Head trauma is one of the leading causes of death and disability worldwide. Combined head lesions consist of more than one form of lesions. Biochemical markers of brain injury are used in determining the extent of brain injury and predicting its outcomes. The aim of this study was to investigate the forensic and clinical significance of serum amylase, lipase and gamma glutamyl transferase (GGT) as predictors of the outcome in head injured patients.

Patients and methods: Sixty head injured patients were enrolled and subjected to personal history taking, general and local physical examination. Glasgow coma scale (GCS), head computed tomography scan and pelvi-abdominal ultrasound were performed. Two blood samples (each 3 mL) were drawn at the time of admission and after 24 h for measuring serum amylase, lipase and GGT levels using special kits.

Results: Most cases of head trauma occurred accidentally during daytime, in the street as a result of falls and road traffic accidents (RTA). Significant increase of serum amylase, lipase and GGT levels on re-evaluation after 24 h from admission were demonstrated in combined head lesions. There was a high significant negative correlation between GCS and these enzymes both on admission and 24 h after admission. Serum levels of measured enzymes were significantly higher in non-survivors as compared to survivors.

Conclusion: Serum amylase, lipase and GGT are good predictors of the outcome in head injured patients. This could be useful for forensic experts to deduce that the poor outcome of the victims was primarily related to the effects of head trauma and its sequences.

1. Introduction

Head is the most frequently damaged part of the body in multiple trauma patients. Head injuries are increasing due to wide use of vehicles in low and middle income countries. Head trauma is one of the leading causes of death and disability worldwide.[1]

Traumatic brain injury (TBI) is caused by sudden impact or acceleration deceleration injury. Approximately one quarter of individuals admitted to hospital with TBI die within six months of injury.[2] TBI results in diffuse and variable pathophysiological effects, causing heterogeneous and diffuse brain injury. Combined head lesions consist of more than one form of lesions that involve cranium, meninges and brain. Heterogeneous pathology and clinical presentation of TBI are the main reasons for inaccuracies in predicting mortality.[3]

Various clinical and radiological characteristics of TBI have already been used as independent predictors of mortality.[4] Biochemical markers of brain injury are suggested to have the potential of determining the extent of brain injury and to predict outcomes in moderate and severe TBI. This will minimize the rates of malpractice suits against neurosurgeons.[5–7] The most widely studied biomarkers for TBI are S100β, neuron-specific enolase, glial fibrillary acidic protein, and Tau. None of these supposed biomarkers have proceeded in the clinical setting.[8]

Serum amylase (AMY), lipase and gamma glutamyl transferase (GGT) are routine biochemical markers for investigating pancreatic and hepatobiliary disorders, respectively.[9,10] Patients with brain injuries and neurosurgical intervention are found to have higher pancreatic enzyme levels compared to those with non-brain insults.[11]

Hence, the aim of this work was to investigate the forensic and

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clinical significance of serum amylase, lipase and gamma glutamyl transferase as predictors of the outcome in head injured patients.

2. Materials and methods

2.1. Patients' selection

Sixty head injured patients who were admitted within 6 h s of sustaining head trauma to neurosurgery department in Emergency Hospital, Tanta University, Egypt, during the period from the start of April 2015 to the end of September 2015 were randomly selected by simple randomization method[12] to be included in this study. Patients who were excluded from the study involved those who returned for check-ups after trauma and those with abdominal trauma, history of liver, pancreatic diseases, or preexisting pathologies (inflammation, fatty infiltration or fibrosis/cirrhosis) that may have influenced serum AMY, lipase and GGT levels before the insult. Patients with neurological, psychiatric diseases and alcohol or drug abuse were also excluded.

In order to exclude the possibility of pharmacological interactions, all patients in our series were evaluated upon admission to the emergency room before starting any medical treatment,[13] and none who had cardiopulmonary arrest or shock (manifested by hypotension, tachycardia, rapid shallow breathing, hypothermia and cold clammy extremities) after head trauma were enrolled in this study as they may be associated with raised serum amylase and lipase which might affect the relation between the type of head injuries and the elevation in enzymes level.[14]

A signed written informed consent was obtained from all individual participants included in the study or their guardians or their relatives after complete extensive description of the study. The Ethics Committee of Tanta Faculty of Medicine, Egypt approved the design of the study. The confidentiality of records of the patients was maintained by giving a code number for everyone.

All enrolled patients were subjected to thorough personal history taking (patient code, age, gender, residence, chronic disease, types of drugs used before injury and history of previous admission for traumatic injury), and circumstances of trauma including time, place, cause, type and manner of injury and time elapsed between trauma and admission to the hospital (pre hospitalization period).

They underwent general and local physical examination (neurological, cardiovascular, respiratory and abdominal). Glasgow coma scale (GCS) assessed the initial severity of traumatic head injury at the time of admission to the hospital. Patients were categorized into two groups: patients with high GCS (GCS 13–15) and patients with low GCS (GCS 3–12) according to Lee et al.[15]Moreover, head computed tomography (CT) scan was performed as soon as the time of admission to determine the presence and types of intracranial injuries. Pelvi-abdominal ultrasound was done to exclude abdominal injuries. Treatment provided to each patient in addition to duration of hospital stay, ICU admission, any detected complication and mortality were recorded.

Two blood samples (each 3 mL) were drawn using sterile syringes at the time of admission before receiving any treatment and after 24 h for measuring serum amylase, lipase and gamma glutamyl transferase (GGT) levels. Blood was collected into a clean tube and left to clot for 20 min at 37 °C and then centrifuged at 3000 rpm for 15min, and the serum samples were separated and stored at −20 °C prior to analysis.

2.2. Analytical procedure

Amylase enzyme activity was measured using kits obtained from Biosystem S.A. Costa Brava, 30.0030 Barcelona (Spain) according to the method of Winn-Dess et al.[16] and Gella et al.[17] Reference range: 22–80U/L.

Lipase enzyme activity was measured by using kits obtained from Biosystem S.A. Costa Brava, 30.0030 Barcelona (Spain) according to the method of Neumann et al.[18] Reference range: ≤ 38U/L.

GGT enzyme activity was measured using the kinetic kit (Randox Laboratories, 55 Diamond Road, Crumlin, County Antrium, BT 294 QY, United Kingdom) according to the method of Teitz.[19] Reference range: Women 8–36U/L, Men 12–55U/L.

2.3. Statistical analysis

The collected data were organized and statistically analyzed using SPSS software statistical computer package for windows version 22. For quantitative data, the Shapiro-Wilk test for normality was performed. For data that were not normally distributed, median and interquartile range (expressed as 25^{th}-75th percentiles) were calculated and Mann-Whitney U was used for comparison between two groups and Kruskal Wallis test for comparison between more than two groups followed by Dunn-Bonferroni test. For comparison between two related groups, Wilcoxon signed ranks test was used. Spearmann's rank order correlations were calculated. For qualitative data, Chi square goodness of fit test, Pearson's Chi-square test or Fisher's exact test was calculated. Receiver–operating characteristic (ROC) curves for predicting the outcome in head injured patients from the data were used. Areas under ROC curve, sensitivity, specificity, positive and negative predictive values were calculated. Significance was adopted at $p < 0.05$ for interpretation of results of tests.[20]

3. Results

Most of enrolled patients in the present study were rural (70%) male (96.67%) workers (53.33%). They were of median age of 35.5 (4.5–66years) with no significant difference between the age groups (Table 1). Most cases of head trauma occurred accidentally (86.67%) during daytime (70%), and mainly in the street (55%) as a result of falls (46.67%) or RTA (36.67%) and half the cases were transferred to hospital within less than 1 h after trauma (Table 2).

Severity of head injury in the studied patients was assessed by GCS which revealed no significant difference between numbers of patients with low and high GCS scores ($P = 0.071$). Conservative treatment that includes stabilization of air way and circulation, prevention of secondary injury through maintaining mean arterial pressures above 90 mm Hg; arterial saturations greater than 90% and reducing intracranial pressure was the main line of treatment (56.67%) with no significant difference with operative interference (43.33%) ($P = 0.366$). Most of our patients (75.0%) stayed from one to 7days in hospital with significant difference from other periods of hospital stay. Mortality rate recorded in the current study was 20% (Table 3).

CT scan findings of head in the studied patients revealed a significant increase of combined head lesions (43.3%) compared to other

Table 1
Demographic data of all studied head injured patients (N = 60).

		N = 60	%	Chi square goodness of fit test	
				X^2	P value
Age groups	≤18years	17	28.33	0.900	0.638
	> 18–45 years	23	38.33		
	> 45years	20	33.33		
Gender	Male	58	96.67	52.267	< 0.0001*
	Female	2	3.33		
Residence	Urban	18	30	9.600	< 0.003*
	Rural	42	70		
Occupation	Student	4	6.67	46.833	< 0.0001*
	Unemployed	8	13.33		
	Worker	32	53.33		
	Employed	13	21.67		
	Retired	2	5		

N: number; *: significant at p < 0.05.