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Diagnostic Potential of Ratio Between Creatine Kinase and Amylase in Acute Pancreatitis

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ABSTRACT

Background: Acute pancreatitis (AP) is an acute inflammatory illness of the pancreas representing a true question in diagnostic process. Laboratory markers of the hepatobiliary tract such as liver transaminases with pancreatic enzymes give a true hint of a hidden diagnosis together with urea, creatinine and creatine kinase (CK). **Objective:** This clinical study aims to show whether there is any correlation between alpha-amylase and CK or their ratio examining hospitalized patients with AP diagnosis. **Methods**: From total number of 99 patients with a clinical picture of AP, 71 patients in this retrospective analysis (including both genders) were included according to the presence of two biochemical markers in collected laboratory analysis at admission and 72 hours later on a laboratory check-up: CK and alpha-amylase. **Results:** The median CK value of AP cases was 92 (41.75 – 207.25) in the acute period and 73 (37 – 159) after 72h staying in the hospital without statistical significant (p=0.521; p<0.05). However, there was a statistically significant correlation between the parameters of CK at admission and creatine kinase after 72h staying in the hospital. The median value of CK/Amylase ratio in the acute period was 0.168 (0.069 – 0.532) and 0.386 (0.12 – 1.12) after 72 hours of staying in the hospital. There was a statistically significant difference between values of CK/amylase ratio in these two groups (p=0.000; p<0.01). **Conclusion:** In conclusion, a connection between CK and alpha-amylase needs to be elucidated in further studies and its existence must be researched both in physiological and pathophysiological conditions, and it is two-way and very complex. This study helped us obtain significant information about the perspective of AP in the potential relation to other non-standard laboratory markers for some diseases

Keywords: creatine kinase, amylase, CK/amylase ratio, acute pancreatitis.

1. BACKGROUND

Acute pancreatitis (AP) is an acute illness of the pancreas followed by the inflammatory process organ itself. Either it is classified as a disease, its diagnostic algorithm represents a clinical entity challenging medical physicians and bringing to the state-of-the-art due to the fact that a diagnosing process includes a presence of at least two criteria of three criteria in total: the worsening acute abdominal pain with propagation towards the back, increased values of pancreatic enzymes in the blood (an increase in the alpha-amylase at least 3 times of upper limit) and urine and pathognomic radiological findings caused by the inflammation of pancreas (1). Pancreas plays a pleiotropic role inside human bodies where it excretes hormones and peptides in charge for endogenous and exogenous actions whose influence is widespread between the endocrine system and digestive system. That ability of producing hormones and peptides is represented through classical and non-classical peptides corresponding as follows: insulin, glucagon, somatostatin, ghrelin, pancreatic polypeptide, cholecystokinin, glucagon like-peptide 1 and 2, gastrin, vasopressin, peptide tyrosine tyrosine (2). Basic functional morphological cellular constitution of pancreas consists of alpha cells, beta cells, PP cells and epsilon cells (1). Ordinary mechanism is related to a stacked outflow of activated enzymes to intes-

tine and blood circulatory system (3). Etiology is the most commonly related to the gallstones, alcohol abuse, and hypertriglyceridemia where gallstones and alcohol abuse represent the highest ranking causes of AP and each account for the underlying etiology in 30–50% of cases (4). Furthermore there are pathophysiological steps towards a final result of AP – a peripancreatic edema mightily aggravated by necrotization (3). Inflammatory cytokines are potently activated and act as a mediator in aforesaid process displaying high levels of interleukin 1 beta, interleukin 6, interleukin 8, tumor necrosis factor alpha (5). Therefore due to strong inflammatory reactions, AP is classified according to a severity rate found on the radiological imaging results applying Balthasar score (1). Besides the Balthasar score, the Ranson and APACHE scores are applied in clinical practice for a prediction of severity and a potential organ failure (1).

In terms of clinical decisions upon the first contact with a certain patient in the emergency department due to abdominal pain, it represents a wide spectrum of potential differential diagnoses – from a mild severity such as viral gastroenteritis up to life-threatening clinical diagnosis such as aortic dissection (6). Therefore abdominal pain is very commonly related to acute cardiovascular developments such as bradycardia or stubborn pain in the epigastrium extending below the left rib cage itself being caused by acute myocardial infarction (7). In the context of proper diagnostic tools, laboratory results represent a true anchor on the wavy sea. Laboratory markers of the hepatobiliary tract together with pancreatic enzymes give a true hint of a hidden diagnosis. They include liver enzymes such as alanine transaminase (AST), alanine transaminase (ALT), gamma-glutamyl transferase (GGT), alkaline phosphatase (ALP) or pancreatic enzymes such as alpha-amylase and lipase (8). Although those previously written markers represent a core of laboratory diagnostic tools related to the upper abdomen organs, medical practitioners extend their lab work with markers of urea and creatinine or creatine kinase (CK) and troponin I for the purpose of differential diagnosis (8). Respecting the biochemical properties of aforesaid biochemical markers commonly involved in a laboratory part of the diagnostic process, their high values might correspond to the group of macro-enzymes (9). Macro-enzymes are divided into two groups; where macro-enzymes group 2 consists of alpha-amylase and creatine kinase (9). The significance of macro-enzymes and their groups is still about to be researched in detail but they are capable of making bonds with autoantibodies making complex structures (9,10). Certain patterns of surges might be appreciated in blood laboratory analysis but still without final relations to systemic diseases or pathophysiological processes, even before disease clinical presentation (10, 11). Some of enzyme such as CK is a very profound enzyme located in heart, skeletal muscles, brain, etc (11). There are three isoenzymes, CK–MM, CK–MB, and CK–BB whose names correspond to their location respectively muscle, muscle/brain, and brain (11). A significant concentration of CK–MB isoenzyme is found almost exclusively in the myocardium and the appearance of elevated CK–MB levels in serum is highly specific and sensitive for myocardial cell wall injury (12).

Due to an enormous potential overlapping between basic biochemical characteristics between certain biochemical markers, it would be highly appreciable for everyday clinical practice in terms of AP diagnosing to elucidate potential relations. There is no previous study that questioned the potential relation of alpha-amylase with CK. Therefore there is no previous study that examined the involvement of creatine kinase in AP.

2. OBJECTIVE

This clinical study aims to show whether there is any correlation between alpha-amylase and CK or their ratio examining hospitalized patients with AP diagnosis.

3. PATIENTS AND METHODS

Patients

For study data collection of patients hospitalized in the General Hospital Prim.dr. Abdulah Nakaš at the Department of Gastroenterohepatology, took a period between 2016 and 2021 for analysis. Patients were excluded if they had any history of active oncologic illness, inflammatory bowel diseases, or chronic liver diseases. A group of 99 patients in this retrospective analysis with a clinical picture of AP including both genders were stratified according to the presence of two biochemical markers in collected data at admission and 72 hours later on a laboratory check-up: CK and alpha-amylase. After reviewing data, in total, there were 71 patients according to the aforementioned needed blood results on the admission and in control work-up after 72 hours. This research was approved by the Ethics Committee of the General Hospital Prim.dr. Abdulah Nakaš Sarajevo, Bosnia and Herzegovina and all patients signed informed consent. Only patients with mild or moderate AP (edematous or interstitial) were included in the study. Standard AP treatment options cover an intravenous fluid without solid food intake (nasogastric tube), the use of analgesics, and further supportive care regarding minerals. Patients were treated with antibiotics to avoid a development of septic complications.

Biochemical parameters and radiological diagnostic tests

All patients had a laboratory analysis of the values of the pancreatic enzyme as amylase in serum and creatine kinase or CK determined by the application of Jaffe reaction (using alkaline picrate) determined by the spectrophotometric method (Architect ci8200). Blood limits were disclosed according to the international harmonization standards as CK in referent range between 40 and 177 IU/L while alpha-amylase referent range is between 25 and 110 IU/L. Imaging methods that were used in determining morphological changes in the pancreas and other organ systems are as follows: percutaneous ultrasound of the abdomen, and computed tomography (CT) of the abdomen used for determining the degree of morphological damage to the pancreas.

Statistical analysis

Statistical analysis was performed by two differ-

ent programs, MS Excel (Microsoft Office Excel 2010) and SPSS (SPSS-Statistical Package for 27 Social Sciences) version 22.0. The Shapiro-Wilk tests were used to assess the normality of variable distribution. The mean value (X) and standard deviation (SD) for continuous independent variables that followed the normal distribution were determined, and the median and interquartile range for independent continuous variables did not follow the normal distribution. The Student t-test tested the significance of the difference for the independent variables that followed the normal distribution. In contrast, the Mann–Whitney U test tested the significance of the difference for the independent variables that did not follow the normal distribution. Correlations between continuous variables were assessed using Spearman's correlation coefficient. A $p<0.05$ was considered statistically significant.

	Values at admission	Values after 72h	p
Male/Female	44/27	44/27	p>0.05
Age	56.3 ± 12.87	56.3 ± 12.87	p>0.05
Creatine Kinase (U/L)	92 (41.75 – 207.25)	73 (37 – 159)	p=0.521
Amylase (U/L)	410 (147 – 1489)	182 (93 – 380)	p=0.001**
Creatine Kinase/Amylase ratio	0.168 (0.069 – 0.532)	0.386 (0.12 – 1.12)	p=0.000**

Table 1. Demographic and creatine kinase/ amylase values at two time periods; ap at admission and ap after 72 hours

Parameters	coefficient correlation (rho)	p
CK admission/CK 72h	0.533	0.000**
Amylase admission/amylase 72h	0.027	0.825
CK admission/amylase admission	0.146	0.229
CK admission/amylase 72h	-0.076	0.532
Amylase admission/CK 72h	-0.102	0.398

Table 2. Correlation between values of creatine kinase and amylase at the admission or after 72 hours

4. RESULTS

Basic demographic data are shown in Table 1. The sex distribution of patients was 44 males (61.9 %) and 27 females (38.1 %). The mean age was 56.3 ± 12.87 years. There were 17 patients (16.3%) over the age of 70 years. The Median Creatine kinase value of AP cases was 92 (41.75 – 207.25) in the acute period and 73 (37 – 159) after 72h staying in the hospital (Table 1) and this difference was not statistically significant ($p=0.521$; $p<0.05$) (Figure 1). However, there was a statistically significant correlation between the parameters of Creatine kinase at admission and Creatine kinase after 72h staying in the hospital, also there was not found statistically significant analysis between other variables(Table 2). The mean level of amylase of AP at the admission was 410 (147 – 1489)U/L and 182 (93 – 380)U/L after 72h staying in the hospital (Table 1), and median CK/Amylase in the acute period was 0.168 (0.069 – 0.532) and 0.386 (0.12 – 1.12 after 72h of staying in hospital. There was a statistically significant difference between values of the amylase and CK/amylase ratio in these two groups ($p=0.001$; $p<0.01$; $p=0.000$; $p<0.01$) (Figure 2).

5. DISCUSSION

This study aimed to investigate whether there is any correlation between alpha-amylase and CK in AP-diagnosed patients. This is the first study that combined two parameters in a ratio. Laboratory results represent a true puzzle to solve and the only possible way is to combine with clinical skills. AP cases are much more common in Eastern European countries and North America compared to available statistics for other parts of the world (13). A median worldwide cost of AP treatment surges up to $13,187 for hospitalized patients with mild severity and being rapidly expanded in case of deterioration in clinical scenario (14). Identifying and diagnosing AP as the clinical diagnosis through AP criteria, which require two or more of three known criteria, is crucial (1). Alpha-amylase and CK are presumed to share some common biochemical properties as seen in pathological states, along with other liver enzymes such as AST, ALT and ALP (9). AP predisposition of development depends on several factors, such as hypertriglyceridemia, hypercalcemia, or some viral causes (15). AP is also associated with an increased mortality rate respectively to the severity of cases. Notably, the overall mortality rate related to all AP cases was 21.1%. Therefore after a stratification upon the severity of cases, it turned out that hardly 2.22% of the AP patients with the severity of disease classified in a group of mild severity had a lethal outcome. In comparison, 45.63% of the AP patients belonging to the group of severe form had lethal outcomes in perspective time after diagnosis (16). AP prevalence varies in different parts of the world counting between 1,452,132.4 and 2,059,695.3 in 1990 while in 2019 between 2,414,361.3 and 3,293,591.8 withal it represents a surge of 62.9% compared between years of 1990 and 2019 (13).

It is believed that AP may rely on the theory of the activated pathway of lysosome and autophagy (17). Naturally, the easiest pathophysiological mechanism represents ischemia of pancreatic tissue leading to dysregulated enzymatic activities (19). The concept is based on the principle that trypsin activation is increased. At the same time, trypsinogen is simultaneously decreased as a common stem for AP mechanisms R. Although, lysosome represents a depot for the lysosomal hydrolase cathepsin B (CTSB) being very well-known as an activator of trypsinogen. CTSB is directly regulated by the activity of cathepsin B inside pancreatic parenchyma and when CTSB lower activity results in lowering of pancreatic parenchymal damage during AP R. Cathepsin B is proved to be in a direct correlation with the activity of alpha-amylase R. Additional a high activity of cathepsin B is seen in viral diseases such as SARS CoV-2. Still, in that particular case of interest usually, it was followed by a simultaneous increase in CK with alpha-amylase which hides a potential biochemical link between those two biochemical markers (19-21).

In light of the role of insulin in the AP pathophysiology due to the hypertriglyceridemia state as the known

risk factor for AP development, defective pancreatic cells contribute to insulin deficiency (15). Insulin is proven to decrease the hypertriglyceridemia state and in that way acts as a preventive care measure in terms of pancreatic parenchymal preservation (1, 17).

Our study's main finding was that there was a statistically significant difference between the amylase values on the admission and in the control check-up laboratory after 72 hours of admission while it was not proved for the CK parameters. Therefore CK showed up to have a statistically significant coefficient correlation between its values on the admission and in the control check-up laboratory after 72 hours of admission in terms of the control laboratory check-up. Additionally, combining those two parameters in the ratio between amylase and CK, showed that there is a statistically significant difference in CK/amylase ratio on the admission and the control laboratory work-up after 72h.

Previously only several case reports considered the potential role of CK in AP development. In 2022, Sheibani et al described a case of a 39-year-old man with AP where the CK activity peaked the day after admission but returned to normal 5 days after the admission (22). Karachaliou et al described a case report of a 64-year-old woman whose increased CK levels normalized throughout hospitalization in total of 12 days (23). Mao-Sheng Su et al published a case report about an alcoholic-cause AP but whose highly increased CK were related to another comorbidity – rhabdomyolysis (24). Shabestari et al described in their case report about the AP patients whose condition was caused by the anabolic steroids resulting in the acute increase of CK followed by high amylase levels (25). Marques et al described a case of AP in already diagnosed autoimmune disease – systemic lupus erythematosus whose CK level was very low (26). Corticosteroids are a potent minimizer of CK and that can represent a pathway when sometimes, in case of overlapping AP in autoimmune disease patients being treated with corticosteroids, CK might be very low (27).

Concerning the ratio encountering amylase, there is the presence of a ratio involving urinary and sera fractions of creatinine and amylase–amylase-creatinine clearance ratio (ACCR) which was researched for a diagnostic process of AP (28). Besides the diagnostic process related to AP, ACCR was increased in postoperative days after cardiothoracic surgeries when all patients had not any symptoms of AP and started to decline after day 2 of the postoperative recovery (30). Potentially discussed mechanisms of the increased value ACCR might be related to medicaments and that represents a true challenge in a diagnostic process in ED (29).

Regardless of potential biochemical relation in those biochemical markers, pain represents a huge ground for making connections between CK and alpha-amylase in AP patients. Ordinary AP patients describe the pain as tremendous and hazardous requiring either the strongest opioids for pain mitigation during the hospitalization (30). In case of enormous pain presence caused by the pancreatic parenchymal process involving trypsin and trypsinogen, there is a cholinergic stimulation related to the muscarinic receptors which for a final result gets a release of alpha-amylase (31). From the other side, the cholinergic stimulation causes the release of creatine kinase, as it is the case with organophosphate poisoning – more commonly expressed in male gender (32). Therefore CK is known as a marker of muscle damage and it could be present in organophosphate poisoning withal one of the most commonly expressed symptoms is abdominal pain (33). In that direction, CK and alpha-amylase might be increased due to cholinergic stimulation.

Regarding the complex pathogenesis of AP, it is highly needed to direct further research toward test discoveries that may predict AP potential complications development. AP patients usually display issues with lipids – hyperlipidemia or hypertriglyceridemia which is related to the atherosclerotic changes in cardiovascular and cerebrovascular systems (34). This is the first study that mutually evaluated a potential association of CK and alpha-amylase in AP.

6. CONCLUSION

AP is an important medical challenge due to its high mortality and morbidity. According to our results, we could not make a statistically significant difference between the values of amylase and CK. In conclusion, a connection between CK and alpha-amylase needs to be elucidated in further studies and its existence must be researched both in physiological and pathophysiological conditions, and it is two-way and very complex. This study helped us obtain significant information about the perspective of AP in the potential relation to other non-standard laboratory markers for some diseases. This proposes the aforementioned processes as a potential mechanism for further studies and a view of the alternatives in fully understanding AP's pathophysiologic mechanism and clinical aspect.

Limitations of the study

In interpreting the study's results, several limitations should be acknowledged. Firstly, the sample size was relatively small–the AP individuals were limited by the exclusion criteria in case of incomplete laboratory work-ups. Secondly, by searching the literature, we could not any results to compare with our results – most case reports were published exclusively on a single patient without causative relation.

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