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Quantitative Analysis of Troponin I Serum Values in Patients with Acute Cholecystitis

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A B S T R A C T

The diagnosis and staging of acute cholecystitis, upon a lot of diagnostic methods and some scoring systems, is still a great clinical problem. The aim of the study was to investigate if serum Troponin I is elevated in patients with acute cholecystitis. Following informed consent, 65 patients with clinical and laboratory signs of acute cholecystitis were enrolled. All patients had measured serum Troponin I level and an abdominal ultrasound was done before definitive treatment was performed. Increased serum Troponin I level was found in most patients with severe form of acute cholecystitis ($p<0.00001$). It reached sensitivity of 94.5% and specificity of 57.1% of this test. In multiple regression analysis Troponin I significantly correlated ($p<0.05$) with the serum aspartate aminotransferase ($r=0.27$), gamma-glutamyl transferase ($r=0.25$) and gallbladder wall (>6 mm) thickness ($r=0.58$). Our study confirms that in most patients with severe and acute cholecystitis, serum Troponin I is increased. Troponin I level is in a lower range than it would be in patients with cardiac muscle damage or necrosis. Measuring serum Troponin I is a fast, reliable and widely performed test that could, with other routinely measured parameters, help in early diagnosis of the severe form of acute cholecystitis.

Key words: *acute cholecystitis, Troponin Ic, staging*

Introduction

The diagnosis of acute cholecystitis is, in most cases, a straight forward task. On the contrary, the predictability of its complications and severity of disease for everyday practice is still a challenge. A new and improved algorithm is necessary and important, because the introduction of timely and appropriate therapy is essential[1,2]. Other authors have attempted to resolve this problem by utilizing various other parameters, such as: leukocytosis, right upper quadrant (RUQ) tender mass, elevated body temperature, elevated serum level of liver function enzymes, the presence of pericholecystitic fluid, fluid in the abdominal cavity, presence of acute pancreatitis, and higher blood flow velocity in the gallbladder wall on Doppler imaging, among other[3–8]. Introduction of the APPACHE II score for intensive care patients with acute cholecystitis improved outcome prediction and disease severity determination[6,7]. Finally, the Tokyo guidelines included parameters that represented the local status of inflammation and systemic response to acute cholecystitis, depending on disease severity like hypoxia and circulation[6,9]. We studied Troponin I as one potentially easily measurable and widely performed test that can help in the early diagnose of the severe form of acute cholecystitis which can then be promptly and more aggressively treated. Significantly elevated Troponin I level (>0.5 ng/mL) represents a myocardial hypoxia and infarction but non-

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-specific elevation of Troponin I (<0.5 ng/mL) is a sign of myocardial hypoxia but not infarction[3,10,11–15]. By measuring Troponin I in combinations with gallbladder wall thickness, we attempted to make the detection of disease severity more precise and widely applicable. Measurement of Troponin I is simple, precise and available in most laboratories which make it a practical parameter to be incorporated into everyday practice.

Materials (Patients) and Methods

Materials (patients)

All investigations were done and all specimens were collected from the 65 subjects upon their written informed consents, according to the Helsinki Declaration and the approval of Hospital Ethics Committee.

In 65 patients with acute cholecystitis we measured the Troponin I (cTnI) level. Acute cholecystitis was diagnosed by standard clinical, laboratory and ultrasound based signs and symptoms[1,3,9]. The severity assessment of acute cholecystitis was done according to Tokyo guidelines[1,9]. The criteria for moderate cholecystitis was local and systemic signs of inflammation, leucocytes >18.00/mm^3, palpable tender mass in the right upper abdominal quadrant, duration of complaints more than 72 hours, marked local inflammation (biliary peritonitis, pericholecystitis, abscess, hepatic abscess, gangrenous cholecystitis, emphysematous cholecystitis)[1]. The criteria for severe cholecystitis was that the severe acute cholecystitis was accompanied by dysfunction in any one of the following organs or systems: cardiovascular dysfunction (hypotension requiring treatment with dopamine >5 μg/kg per minute, or any dose of doputamine), neurological dysfunction (decreased level of of consciousness), respiratory dysfunction (PaO_2 /FiO_2 ratio <300), renal dysfunction (olyguria, elevated creatinine), hepatic dysfunction (Protrombine time-INR>1.5), hematological dysfunction[1].

Methods

Inclusion criteria

Clinical symptoms of acute cholecystitis in all patients were upper right quadrant pain 1–2 hours after a meal, which propagates towards the scapula and back in association with elevated body temperature[1,3,4,9]. Laboratory signs were high leukocyte count and elevated serum C-reactive protein value (CRP). Beside a significantly elevated leukocyte count and the duration of symptoms for more than 24 hours, patients with severe form of acute cholecystitis had a palpable abdominal mass in right upper quadrant. Abdominal ultrasound findings of acute cholecystitis, according to standard criteria, were thickness of the gallbladder wall more than 4 mm with signs of dissection[4]. The gallbladder wall measurements were made on its front wall and by two experienced ultrasonographers in random fashion. We also checked for presence of pericholecystic fluid[1,3,9].

Exclusion criteria

Patients with an acute coronary incident were excluded from the study on clinical ground, non revealing ECG, serum values of creatine kinase and its MB fraction[3,9]. Furthermore, all studied patients with mild and severe acute cholecystitis were tested for coronary artery disease by treadmill stress test two months after acute cholecystitis was healed[3]. Only patients that were negative for both, acute coronary syndrome and cardiac stress test were included in the study. Renal function was measured by serum creatinine and urea values[1,3,9]. Only patients with normal findings were included while patients with renal failure were excluded from the study. Excluded were also patients with biliary obstruction[1,3,9]. An obstruction was detected by abdominal ultrasound with common biliary duct measuring more than 7 mm, elevated serum values of bilirubin, gamma-glutamyl transpeptidase and alkaline phosphatase[3,9]. Excluded from the study were also patients with complications such as acute pancreatitis or ileus. Upper gastrointestinal endoscopy was done to exclude all other differential diagnoses[3]. Up to two months after an episode of severe acute cholecystitis that was conservatively treated and healed, all patients underwent trans-abdominal or laparoscopic surgical cholecystectomy[1,3,9]. Gallbladder tissue sample analysis was done according to standard procedure. All patients with pathological findings that fulfilled inclusion criteria were included in the study while other causes of gallbladder diseases were excluded (e.g. tumor, etc.).

The Troponin I measurements

The Troponin I serum values were measured by immunoflorometric method (Dimension, Dade Boehringer). We considered Troponin I values up to 0.5 ng/mL while those values between 0.5–1.5 ng/mL were excluded as a possible acute myocardial infarction[3].

Other measurements

In our studies we used ultrasound system with convex 3.5 MHz probe. All laboratory parameters (leukocyte, CRP, amylase, bilirubin, aspartate aminotransferase, alanin aminotransferase, gamma-glutamyl transpeptidase, alkaline phosphatase, urea and creatinine) were measured according to standard procedures.

Statistics

We used software STATISTICA 2.1 for statistical analyses that included mean, standard deviation, ANOVA, test of homogeneity of variance and multivariate multiple regression test, and logistic regression. We calculated accuracy etc. by using ROC analysis.

Results

Enrolled patients were divided into two groups, moderate (N=18) or severe (N=47). Most patients with severe acute cholecystitis have increased serum level of Troponin I. The patients with severe acute cholecystitis had significantly elevated leucocyte count, CRP, Tropinin, serum amylase level and had presence of pericholecystitis ($p<0.05$; Levene test of Homogeneity) (Table1). We

TABLE 1
THE MEANS AND RANGES OF PARAMETERS ACCORDING TO TOKYO CLASSIFICATION OF DUSEASE SEVERITY IN PATIENTS WITH ACUTE CHOLECYSTITIS

	Moderate	(score=2)	Severe	(score=3)	
	Median	Range	Median	Range	p
Age (yrs.)	64.6	37	60.7	47.0	0.539
Sex (m/f)	1.77	1.0	1.7	1.0	0.199
Leucocytes ($\times 10^9$/L)	12.54	12.7	14.6	13.0	0.0284
CRP (mg/L)	19.8	37.7	49.5*	233	0.0002
Troponin I (μg/L)	0.004	0.05	0.22*	0.48	0.0001
US gallbladder wall thickness (mm)	5.5	5.0	6.36	4.0	0.011
US/pericholecystitis (y/n)	0.16	1.0	0.27*	1.0	0.046
CK (U/L)	65.8	82.0	81.1	135.0	0.478
Bilirubin (μmol/l)	14.4	28.0	16.6	31.3	0.131
AST (U/L)	21.1	31.0	26.4	37.0	0.451
ALT (U/L)	21.5	34.0	23.1	34.0	0.767
GGT (U/L)	33.3	58.0	45.1	112.0	0.171
ALP (U/L)	71.0	80.0	67.0	85.0	0.066
Amylase – s (U/L)	69.10	88.0	63.6*	122.0	0.028
Amylase – u (U/L)	131.1	200.0	143.0	264.0	0.518

* Statistical significance for Troponin I was at *p<0.05 level (Levene test of Homogeneity). Legend: CRP: C-reactive protein, AST: aspartate aminotransferase, ALT: alanine aminotransferase, GGT: gamma-glutamyl transpeptidase, ALP: alkaline phosphatase, Amylase-s: amylase serum level, amylase-u: amylase in urine, CK: creatine kinase.

have found no statistical difference between other parameters according to the Tokyo assessment of disease severity.

We had 35 out of 47 patients with Troponin I values in the range of 0.05–0.5 ng/mL (Table 2). No elevation of Troponin I above 0.5 ng/mL was measured. In the group of patients with the moderate form of disease, Troponin I was at level 0.00 ng/mL in 16 patients and two were above 0.00 ng/mL (Table 1). The Troponin I serum level reached sensitivity level of 94%, specificity of 57%, false positive value of 5.4%, and accuracy of 78.4%.

Troponin I, calculated by multiple regression analysis, significantly correlated with increase of gallbladder wall thickness (p<0.001; multiple regression). Not surprisingly, a significant correlation was also seen with Troponin I and aspartate aminotransferase (p<0.03; multiple regression) and also with Troponin I and gam-

TABLE 2
RESULTS OF SERUM TROPONIN I VALUES IN PATIENTS WITH EITHER MODERATE OR SEVERE FORM OF ACUTE CHOLECYSTITIS

	Moderate (N)	Severe (N)	Total (N)
Troponin = 0 ng/mL	16	12	28
Troponin > 0 ng/mL	2	35	37
	18	47	65

Result of χ^2-test presents statisticaly significant diference (p< 0.005; with χ^2=21.26 and df=1) between the group with troponin level 0.00 ng/mL vrs. the group with troponin level higher than 0.00 ng/mL.

TABLE 3
TROPONIN I IN MULTIPLE REGRESSION ANALYSIS WITH OTHER PARAMETERS

	US wall	gallbladder thickness	Troponin	
	r	p	r	p
Age	0.04	0.77	–0.07	0.60
Sex	0.16	0.21	–0.10	0.44
Leucocytes	0.14	0.26	–0.03	0.79
CRP	0.42*	0.001	0.10	0.42
TROPONIN	0.58*	0.001	/	/
US/Gallbladder thickness (>6 mm)	/	/	0.58*	0.001
US/pericholecystitis	0.16	0.213	0.05	0.68
CPK	–0.12	0.33	–0.03	0.81
Bilirubin	0.13	0.31	0.08	0.52
AST	0.09	0.48	0.27*	0.03
ALT	0.04	0.76	0.17	0.18
GGT	0.22	0.079	0.26*	0.048
ALP	–0.01	0.96	–0.15	0.23
Amylase (serm.)	0.05	0.68	0.10	0.42
Amylase (urine)	0.08	0.53	0.10	0.41

Statistical significance was at *p<0.05 leve (Multiple regresion analysis). Legend: CRP: C-reactive protein, AST: aspartate aminotransferase, ALT: alanine aminotransferase, GGT: gamma-glutamyl transpeptidase, ALP: alkaline phosphatase, Amylase-s: amylase serum level, amylase-u: amylase in urine, CK: creatine kinase.

TABLE 4
THE DIFFERENCES BETWEEN THE PARAMETERS ACORDONG TO ULTRASOUND MEASURED GALLBLADDER WALL THICKNESS IN PATIENTS WITH ACUTE CHOLECYSTITIS

	Gallbladder thickness wall (mm)		
	<6 X±SD	>6 X±SD	p
Age (yrs.)	58±9	63±13	0.067
Sex	1.66±0.4	1.74±0.4	0.257
Leucocytes (x10^9/L)	14±4.45	14±3.1	0.115
CRP (mg/L)	22±11	48±55*	0.00028
Troponin I (ug/L)	0.033±0.09	0.21±0.19*	0.00001
US/pericholecystitis (y/n)	0.17±0.38	0.27±0.45	0.046
CK (U/L)	80±38	76±28	0.078
Bilirubin (μmol/l)	17±7	16±7	0.531
AST (U/L)	22±11	26±9	0.445
ALT (U/L)	20±10	23±9	0.657
GGT (U/L)	35±18	44±22	0.368
ALP (U/L)	65±22	69±22	0.678
Amylase-s (U/L)	58±21	72±38	0.006
Amylase-u (U/L)	134±60	142±63	0.483

Statistical significance for Troponin I was at *p<0.0001 level (Analysis of variance). Legend: CRP: C-reactive protein, AST: aspartate aminotransferase, ALT: alanine aminotransferase, GGT: gamma--glutamyl transpeptidase, ALP: alkaline phosphatase, Amylase-s: amylase serum level, amylase-u: amylase in urine, CK: creatine kinase.

ma-glutamyl transferase (p<0.048; multiple regression) (Table 3). Gallbladder wall thickness significantly correlated with CRP (p<0.001; multiple regression).

Logistic regression analysis for severity of cholecystitis, according to the all parameters, found proportion of variance accounted for =.8463 and R=0.919 with variance explained of 84.634%. Testing observed versus predicted values, observed values were 0.099+0.9001* predicted values, with correlation r=0.9282 at regression of 95% confidence.

Test of variance of homogeneity detected highly statistically significant difference when comparing the patients with US measures gallbladder wall thickness <6 mm with the patients with >6 mm, in the Troponin I level (p<0.00001), in CRP level (p<0.00028), serum amylase level (p<0.006), presence of pericholecystitic fluid (pericholecystitis) (p<0.046) and patients age (p<0.06). We found no statistical significant difference by this test between other parameters (Table 4).

Discussion

The Tokyo guidelines recognize three degrees of acute cholecystitis. In the first, the disease is only localized to the gallbladder. The second degree is described with illness in the surrounding tissues but the patient is without systemic complications and operative therapy has higher risk. The third degree represents systemic complications such as high leukocyte count, abdominal palpable mass, and illness duration for more than 24 hours[6]. Similar criteria that include C-reactive protein, pericholecystitic fluid or gallbladder thickness detectable by ultrasound, were historically used for better assessment[1–4,6,9].

Our results in the majority of patients with a severe form of acute cholecystitis showed elevated serum levels of Troponin I. On the contrary, patients with the mild form of the disease had no detectible level of Troponin I. Troponin I has high sensitivity (94%) and much lower specificity (57 %) for severe cholecystitis with only 5.4% of false positive results (Table 2). All measured serum Troponin I levels in patients with the severe form of cholecystitis were in a lower range than for patients with acute myocardial infarction or acute coronary syndrome. A literature search found other medical conditions that can lead to a false positive rise in Troponin I levels and possible to a misdiagnosis. Congestive heart failure, sepsis, muscular activity, pulmonary embolism, supraventricular tachycardia, arterial hypertension all can lead to a rise in serum Troponin I levels also in the intervals that are non characteristic for acute myocardial infarction[11–18]. Authors explain that in this conditions elevation of Troponin I (and Troponin T) is due to cholecystitis itself or the systemic inflammation, which may have been associated with transient bacteraemia despite the negative blood cultures, and led to release of troponin from the myocardium[16–18]. Meta analysis presents that the elevation of Troponin T is positively related to higher mortality, but the meta analysis results, for the elevation of Troponin I is unclear, largely because of the lack of standardization of assays[18–20].

Literature gives us some other theoretical explanations for noncardial elevation of Troponine I. Local and circulating inflammatory markers including tumor necrosis factor α, interleukin 6 and reactive oxygen species, as well as bacterial endotoxins, may lead to direct myocardial injury by cytotoxic effects[19]. Moreover, elevated cTn values provide prognostic information and the extent of cTn elevation seems to correlate with the severity of the disease process[19].

Other parameters can be used in the detection of disease severity such as body temperature, leukocytosis, C-reactive protein, transferrin, and alpha 1-antitripsin[6,21,22]. Reasons for elevation of these parameters in acute cholecystits is well known, as it is well known that this parameters are more elevated in severe than in the mild form of acute cholecystitis, and it is no need to explain it now[1,3,9]. But we have to explain that the elevation of these parameters is associated with the elevation of Troponin I in acute cholecystitis. The reasons of Troponin I elevation mentioned above, are similar to the reasons for elevation of this parameters as are leucocyte, CRP, etc. When Troponin I was compared in multiple regression analysis with other parameters, we found a significant correlation with the increased gallbladder thickness, the aspartate aminotransferase and the gamma-

-glutamyl transpeptidase, parameters that represent liver and biliary injury (Table 3). Increased gallbladder wall thickness of 6 mm or more has the best correlation, at very high and statistically significant level with Troponin I ($p<0.0001$) and CRP (Table 4). CRP has been already described in the literature and in the Tokyo guidelines as a borderline parameter of statistical significance to distinguish between mild and severe forms of the disease[1,6,22,23].

Taken together, the determination of Troponin I in conjunction with gallbladder wall thickness, other parameters (leukocyte count, CRP, aspartate aminotransferase gamma-glutamyl transpeptidase *etc*.) and clinical presentation could be a very sensitive, useful and simple tool, easily incorporated into daily practice. Clinically, it is very important to determine the degree of acute and non obstructive cholecystitis (mild or severe form, with or without complications). This can lead to a more precise determination of disease severity ensuring timely and appropriate therapy resulting in lower costs and fewer complications.

Conclusion

We can conclude that the measurement of Troponin I level in acute cholecystitis patients, can improve medical decision making in the field of diagnosis, severity disease assessment, and therapy.

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KVANTITATIVNA ANALIZA SERUMSKOG TROPONINA I U BOLESNIKA S AKUTNOM UPALOM ŽUČNJAKA

SAŽETAK

Dijagnozu i stupnjevanje akutne upale žučnjaka i dalje je, unatoč brojnim dijagnostičkim metodama, ostao težak zadatak. Cilj ovog rada bio je istražiti ulogu povišenih serumskih vrijednosti Troponina I u bolesnika s akutnim kolecistitisom. Nakon informiranog pristanka u studiju smo uključili 65 bolesnika s kliničkim i laboratorijskim znakovima akutne upale žučnjaka. Svim bolesnicima je mjerena razina serumskog Troponina I te im je učinjen i transabdominalni

ultrazvuk prije početka liječenja. U većine bolesnika s teškim oblikom akute upale žučnjaka nađene su povišene vrijednosti Troponina I ($p<0,00001$). Postigli smo osjetljivost testa od 94,5% i specifičnost testa od 57,1%. Testom multiple regresije statistički značajnija korelacija ($p<0,05$) postignuta je za Troponin I, za serumsku aspart aminotransferazu ($r=0,27$), gamaglutamil transferazu ($r=0,25$) i za debljinu stjenke žučnjaka (>6 mm) ($r=0,58$). Naša studija potvrđuje da je većina bolesnika s teškim oblikom akutne upale žučnjaka imala povišene vrijednosti Troponina, ali u vrijednostima koje nisu karakteristične za oštećenje ili nekrozu srčanog mišića. Mjerenje Troponina I brza je i široko primjenjiva metoda koja uz ostale rutinske parametre može pomoći u dijagnostici i stupnjevanju teških oblika akutne upale žučnjaka.