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Unusual emergent presentations of abdominal aortic aneurysm: Can simple blood tests predict the state of emergency?

Abdominal aort anevrizmasında nadir görülen acil klinik durumlar: Basit kan testleri aciliyetin tanımlanmasında yol gösterici midir?

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BACKGROUND

This paper attempts to see if simple blood test results can predict the state of an emergency aneurysm as being non-ruptured, contained leak or free rupture.

METHODS

Ninety-three patients who presented to our emergency room and were operated for abdominal aortic aneurysm (AAA) between January 1999 and March 2009 were evaluated retrospectively. Cases were classified as: chronic contained rupture (Group I), impending rupture (Group II), dissecting rupture (Group III), and free rupture (Group IV).

RESULTS

Chronic contained rupture was determined in 15 (16.1%), impending rupture in 31 (33.3%), dissecting rupture in 14 (15.1%), and true (free) rupture in 27 (29%) cases. Aortocaval fistula was present in 3 (3.2%) patients, aortoenteric fistula in 2 (2.2%) and aorto biliary fistula in 1 (1.1%). Group IV was significantly different from Groups I, II and III with regard to hematocrit levels, white blood cell counts, neutrophils and lymphocyte rates, bicarbonate levels, and mortality rates.

CONCLUSION

To avoid a delay in diagnosis, it is important to know the different presentations of emergency AAA. In the emergency room, simple laboratory parameters may be highly directive in suspicion of ruptured AAA.

Key Words: Abdominal aortic aneurysm; emergency; clinical presentations.

AMAÇ

Bu çalışmada, basit kan testlerinin acil abdominal aort anevrizmasının (AAA) klinik sunumu ile ilişkisinin araştırılması amaçlandı.

GEREÇ VE YÖNTEM

Ocak 1999 - Mart 2009 tarihleri arasında kliniğimiz acil servisine müracaat etmiş ve hastaneye AAA tanısı ile yatırılmış olan toplam 93 hasta geriye dönük olarak incelendi. Kronik sınırlandırılmış rüptür olguları "Grup I", rüptür tehdidi olguları, "Grup II", disekan rüptür olguları "Grup III" ve gerçek (serbest) rüptür olguları ise "Grup IV" olarak sınıflandırıldı.

BULGULAR

Kronik sınırlandırılmış rüptür hastaların 15'inde (%16,1), rüptür tehdidi 31'inde (%33), disekan rüptür 14'ünde (%15,1) ve gerçek (serbest) rüptür 27'sinde (%29) saptandı. Hastaların üçünde (%3,2) aortokaval fistül, ikisinde (%2,2) aortoenterik fistül, birinde (%1,1) aortobiliyer fistül tanımlandı. Grup IV hemotokrit seviyesi, beyaz küre sayısı, nötrofil ve lenfosit oranları, bikarbonat seviyesi ve mortalite oranları açısından Grup I, II ve III'e göre istatiksel olarak anlamlı olan farklı parametrelere sahipti.

SONUÇ

Tanıda gecikmeyi önlemek için AAA'nın acil kliniğinin değişkenlik gösterdiğinin bilinmesi önemlidir. Acil odasında alınan basit kan testleri serbest rüptür açısından oldukça yönlendirici olabilmektedir.

Anahtar Sözcükler: Abdominal aort anevrizması; acil; klinik bulgular.

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Abdominal aortic aneurysm (AAA) is an important cause of morbidity and mortality especially in male patients over the age of 65, and its prevalence is 1.0-8.9% in the general population.[1] Its incidence is reported to be increasing in the last 20 years.[2,3] It is estimated that there are 8000 deaths in England and 15000 deaths in the United States annually related to rupture of AAA.[1,4]

The treatment of ruptured AAA is emergent surgery or endovascular repair. The mortality rate is dependent on the hemodynamic condition of the patient during the intervention. The reported operative mortality is 30-70% in ruptured AAA.[5,6] The mortality of ruptured AAA is unchanged despite the advances in surgical procedure, anesthetic management and medical care in conventional operations for ruptured AAA.[6] However, in recent years, there have been encouraging studies reporting good results with endovascular stent graft treatment in patients with ruptured aortic aneurysms.[7,8]

The classic triad of ruptured AAA is abdominal or back pain, hemodynamic shock and pulsatile abdominal mass. The clinical presentation depends on the localization and degree of the rupture. The rupture is from the anterolateral wall of the abdominal aorta to the peritoneal cavity in 12% of the cases.[9,10] Rupture from the posterolateral wall to the retroperitoneal area constitutes 88% of cases and has a less mortal course than anterolateral wall rupture.[9,10] These groups of patients can reach health centers and operating rooms. However, an initially limited rupture with a small blood loss may progress into a large rupture within hours. Rarely, the ruptures can fistulize into the inferior vena cava, duodenum or biliary tract.[11]

It is important to note that emergency AAA encompasses a spectrum of disease from symptomatic non-ruptured aneurysms to the classic free intraperitoneal rupture. In the emergency room, the diagnosis of the ruptured AAA is established clinically, but also by ultrasound or computed tomography (CT) scan. There may be difficulties in obtaining correct hemodynamic data and laboratory results in a patient presenting with shock. Shock before surgery has been shown by univariate and multivariate analyses to be associated with increased mortality.[6,7] A pragmatic approach would dictate that prompt and correct diagnosis of emergency AAA in hemodynamically stable patients is vital to reduce the incidence of the development of shock before surgical intervention.[6] The purpose of this study was to detect subtle clinical and laboratory values for the early detection of ruptured AAA. We evaluated clinical and laboratory parameters and investigated whether these variables affected mortality in patients who present to the emergency room with ruptured and unusual forms of non-ruptured AAA.

MATERIALS AND METHODS

The study was approved by the Local Ethics Committee. The data collection methodology and data analysis strategies for this study were applied according to our institutional retrospective research practice. Ninety-three patients who were admitted to our emergency room and underwent operation for AAA between January 1999 and March 2009 were evaluated retrospectively. Three of the cases had aortocaval fistula, two had aortoenteric fistula and one had aorta biliary fistula. These patients with fistulae were excluded from the statistical analysis due to the insufficient number of cases. Patients were evaluated for demographic data, clinical presentation, operative findings, and rupture morphology. The patients who were diagnosed as emergency AAA were basically classified according to CT, and the diagnosis was confirmed by operative findings. In the operative findings, if the aneurysm wall was ripped and the retroperitoneal and peritoneal cavity were filled with massive blood, it was recognized as "*free rupture*"; if the rupture was limited with organized hematoma outside the aorta contained in a pseudoaneurysmal wall of retroperitoneal connective tissue, we recognized it as "chronic contained rupture" (Fig. 1). When the blood drained into the inferior vena cava, it was designated as "*aortocaval fistula*" (Fig. 2), while blood draining into the intestinal system was designated as "*aortoenteric fistula*" and to the biliary tract as "*aorto biliary fistula*". Respectively, an "*impending rupture*" is a symptomatic aneurysm that is about to rupture (Fig. 3). When the wall of the AAA was dissected, it was recognized as a "dissected rupture of AAA".

Cases were classified into four groups. Patients fulfilling criteria set by Jones and colleagues in 1986[12] for contained rupture constituted Group I (n=15), impending rupture[13,14] (acutely symptomatic non-rup-

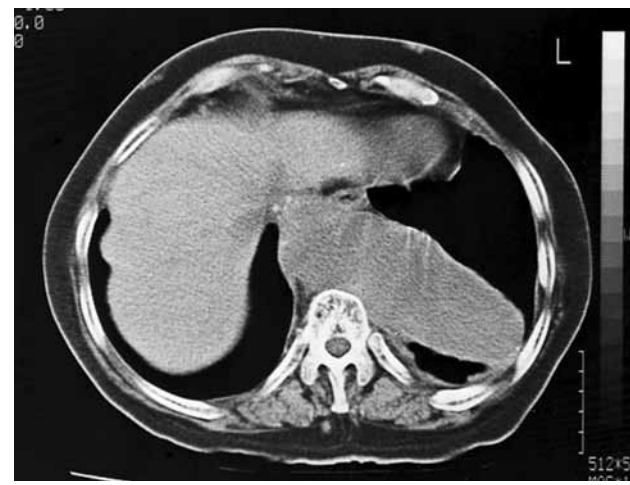

Fig. 1. Computed tomographic view of the chronic contained rupture of AAA.

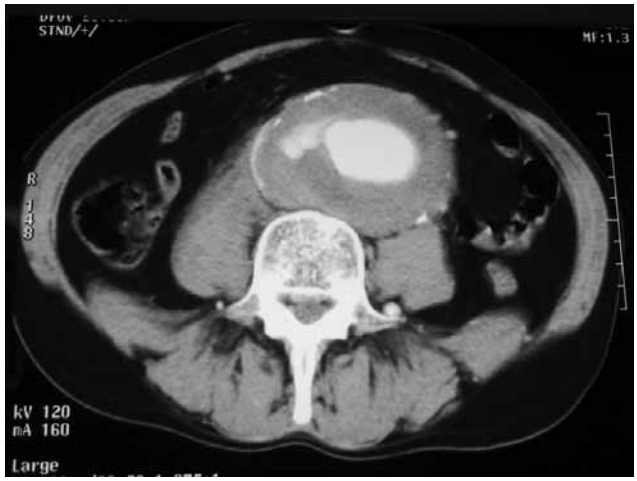

Fig. 2. Computed tomographic view of dilated iliac veins due to aortocaval fistula.

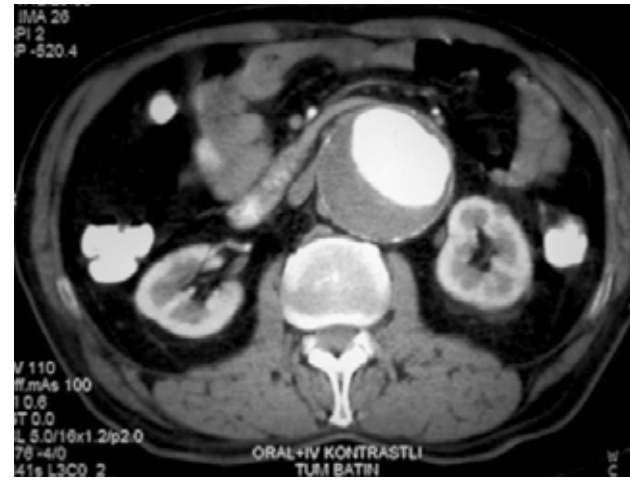

Fig. 3. Discontinuity of aortic wall calcifications and high-attenuating crescents in the wall of AAAs on unenhanced computed tomographic scans are signs of impending rupture of the AAA.[14] This patient admitted to the emergency room with abdominal pain and was taken to the operating room before rupture.

tured AAA) Group II (n=31), dissecting rupture Group III (n=14) (while forming this group, patients with intramural thrombus and/or hematoma were selected, with no distinction between chronic or acute dissection), and free rupture Group IV (n=27). Three of the cases had aortocaval fistula, two had aortoenteric fistula and one had aorta biliary fistula. These patients with fistulae were excluded from the statistical analysis due to the insufficient number of cases, and 87 patients were included in the final analysis. The clinical findings and laboratory parameters (hematocrit, white blood cell count, neutrophils, lymphocytes, blood pH, bicarbonate, base deficit, urea, and C-reactive protein [CRP] levels) that were measured during the admission to the emergency room and mortality rates were compared between the study groups. Freely ruptured AAAs were used as a "gold standard" for comparison with contained or impending ruptured cases.

Statistical Analysis

The Number Cruncher Statistical System (NCSS) 2007 and PASS 2008 statistical software (Utah, USA) were used for statistical analysis of data. For quantitative variables, in addition to descriptive statistical methods (mean and standard deviation), one way ANOVA test was used for the comparison of normally distributed parameters, and the Tukey HSD test was used to detect the group that caused difference. The Kruskal-Wallis test was used to compare non-normally distributed parameters, and the Mann-Whitney U test was used to detect the group that caused difference. A chi-square test was used for comparison of qualitative variables. P values <0.05 were considered statistically significant in the 95% confidence interval.

RESULTS

No significant difference was found between the groups in terms of age and gender (Table 1). The mean diameter of the aneurysm, median duration of symptoms, time interval before the operation, duration of hospital stay, mean arterial systolic and diastolic blood pressures, and known concomitant illnesses are summarized in Table 2. Hypertension was detected in 36 cases, chronic obstructive pulmonary disease (COPD)

Table 1. The distribution of AAA cases according to age, gender and deaths in groups

		Group I Mean±SD	Group II Mean±SD	Group III Mean±SD	Group IV Mean±SD	p
Age		64.40±9.33	67.67±6.98	63.71±8.01	69.70±8.86	0.083
		n (%)	n (%)	n (%)	n (%)	
Gender	Male	14 (93.3)	28 (90.3)	11 (78.6)	26 (96.3)	0.307
	Female	1 (6.7)	3 (9.7)	3 (21.4)	1 (3.7)	
Deaths (30-day mortality)	No	15 (100.0)	27 (90.0)	12 (92.3)	16 (59.3)	0.002**
	Yes	0 (0.0)	3 (10.0)	1 (7.7)	11 (40.7)	

+One way ANOVA test; ++Chi-square test; **p<0.01.

Table 2. Some clinical features of emergency AAA cases

	Range	Mean±SD
Diameter of aneurysm (mm)	40-130	73.11±21.11
Pulse (bpm)	11-130	81.38±25.38
The median duration of symptoms (day)	1-720	54.12±143.79
The time interval before the operation (hour)	0.40-16.0	4.32±4.08
Length of hospital stay (day)	1-60	10.08±9.80
Systolic blood pressure (mmHg)	20-216	121.90±41.44
Diastolic blood pressure (mmHg)	0-123	71.92±29.69
	Yes n (%)	No n (%)
Hypertension	36 (41.9)	50 (58.1)
Chronic obstructive pulmonary disease	34 (39.1)	53 (60.9)
Coronary artery disease	36 (41.4)	51 (58.6)

in 34 cases and coronary artery disease in 36 cases (Table 2).

The numbers of deaths in the study groups are presented in Table 1 (3 deaths in Group II, 1 in Group III and 11 in Group IV). There were no deaths in Group I. When groups were compared according to rate of mortality, a highly significant difference was detected between the groups (p=0.001). As an expected finding, Group IV, which was established from totally ruptured patients, had a significantly higher mortality rate than the other groups.

Seven patients had a delay in diagnosis and resultant free rupture. These patients were evaluated in Group IV. Five patients had clinical impeding rupture, 2 patients contained rupture and 1 patient dissected rupture. All of them progressed to free rupture in the emergency room or intensive care unit, and three patients died.

The laboratory parameters were compared with the reference values and classified as normal, high or low. The distributions of these values according to groups are presented in Table 3. All parameters in Group IV were abnormal (Fig. 4). Hematocrit levels lower than normal reference values were seen in 100% of patients in Group IV, 32% in Group III, 67% in Group II, and 53% in Group I. The white blood cell count was higher than normal reference values in 100% in Group IV, 42% in Group III, 58% in Group II, and 53% in Group I. All parameters, the number of patients and percentages are presented in Table 3.

The distributions of the levels of the laboratory parameters according to groups are presented in Table 4. The levels of hematocrit, white blood cell count, percentages of neutrophils and lymphocytes, and bicarbonate levels in blood were compared between the groups. Results of the Tukey HSD test showed that there was significant statistical difference between Group IV and the others (p: 0.001; p: 0.001; p: 0.01; p<0.01). The levels of hematocrit, number of white blood cells, percents of neutrophils and lymphocytes, and bicarbonate levels did not show differences between Groups I, II and III (p>0.05).

Table 3. The distribution of laboratory parameter levels in groups

		Group I n (%)	Group II n (%)	Group III n (%)	Group IV n (%)	Sum n (%)
Hematocrit (39-50%)*	Low	8 (53.3)	21 (67.7)	13 (92.9)	27 (100)	69 (79.3)
	Normal	7 (46.7)	10 (32.3)	1 (7.1)	0 (0)	18 (20.7)
White blood cell (4.3-10.3 x10^9/L)*	Low	0 (0)	1 (3.2)	0 (0)	0 (0)	1 (1.1)
	Normal	7 (46.7)	12 (38.7)	8 (57.1)	0 (0)	27 (31)
	High	8 (53.3)	18 (58.1)	6 (42.9)	27 (100)	59 (67.8)
Neutrophil (41-73%)*	Normal	4 (26.7)	10 (32.3)	3 (21.4)	0 (0)	17 (19.5)
	High	11 (73.3)	21 (67.7)	11 (78.6)	27 (100)	70 (80.5)
Lymphocyte (19-44.9%)*	Low	11 (73.3)	23 (74.2)	10 (71.4)	27 (100)	71 (81.6)
	Normal	4 (26.7)	8 (25.8)	4 (28.6)	0 (0)	16 (18.4)
Base deficit (0-2.5 mEq/L)*	Normal	7 (46.7)	14 (45.2)	5 (35.7)	2 (7.4)	28 (32.2)
	High	8 (53.3)	17 (54.8)	9 (64.3)	25 (92.6)	59 (67.8)
CRP (0-0.74 mEq/L)*	Normal	6 (40)	7 (22.6)	0 (0)	0 (0)	13 (14.9)
	High	9 (60)	24 (77.4)	14 (100)	27 (100)	74 (85.1)

*Normal reference values; CRP: C-reactive protein.

Table 4. The mean values of laboratory parameters in groups

	Group I Mean±SD	Group II Mean±SD	Group III Mean±SD	Group IV Mean±SD	p
Hematocrit (%)+	38.13±5.12	36.51±3.83	33.71±5.31	24.08±5.75	0.001**
White blood cell (x10^9/L)+	11.69±5.29	12.83±5.79	10.30±3.07	18.42±3.47	0.001**
Neutrophil (%)+	76.27±12.62	79.19±10.80	76.71±9.28	88.11±4.35	0.001**
Lymphocyte (%)++	16.29±9.79	13.94±10.71	14.39±6.15	8.43±2.67	0.006**
Blood pH	7.40±0.05	7.39±0.05	7.39±0.05	6.73±1.94	0.087
Bicarbonate (mEq/L)+	23.11±2.63	22.15±3.47	22.27±3.08	16.58±3.39	0.001**
Base deficit (mEq/L)++	2.48±1.46	3.15±1.80	3.54±2.28	9.98±4.45	0.001**
Urea (mg/dL)++	46.73±23.67	62.23±29.67	63.14±21.04	87.87±52.34	0.006**
CRP (mg/dL)++	0.89±0.44	1.31±0.65	1.52±0.71	4.22±1.36	0.001**

+One way ANOVA test; ++Kruskal-Wallis test; **p<0.01.

Blood pH levels were lower in Group IV than the others. However, there was no significant difference between the other three groups. Base deficit levels were found significantly different between the groups, and Mann-Whitney U test detected that this difference came from Group IV. The levels of base deficit did not differ between Groups I, II and III (p>0.05).

The levels of urea showed significant difference between the groups (p<0.01). The Mann-Whitney U test was performed to detect the group that caused difference. The levels of urea in Group IV were significantly higher than in Groups I and II (p: 0.001; p: 0.036, respectively). The urea levels of Group III were significantly higher than in Group I (p: 0.046; p<0.05). There were no differences between the levels of urea in the other groups.

The levels of CRP differed significantly between the groups (p<0.01). The Mann-Whitney U test was performed to detect the group that caused difference. The levels of CRP in Groups IV and III were significantly higher than in Group I (p: 0.001; p: 0.001; p<0.01). It was also found that the levels of CRP in Group IV were significantly higher than in Groups II and III (p: 0.001; p<0.01, respectively). There were no differences between the levels of CRP in the other groups (p>0.05).

DISCUSSION

Our data show that patients with emergency AAA presented unusual forms by a majority. Eighty-seven patients who were admitted as emergency AAA were basically classified according to operative findings. Contained rupture was determined in 15 (17.2%), impending rupture in 31 (35.6%), dissecting rupture in 14 (16.1%), and true (free) rupture in 27 (31.1%) cases. Clinical presentation of emergency AAA is not typical every time. Therefore, misdiagnoses and de-

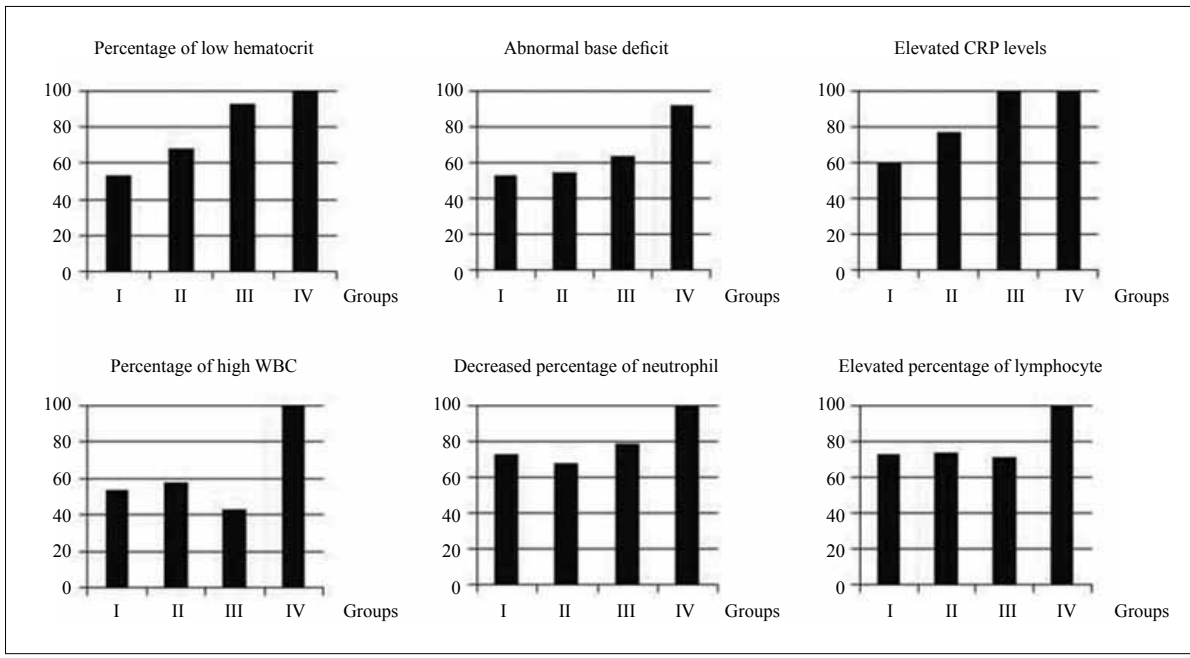

Fig. 4. The percentages of abnormal laboratory parameters in groups.

lays in the diagnosis are possible.[15,16] Davidovich et al.[17] presented a study of 41 cases with unusual forms in a series of 506 cases with ruptured AAA within a 14-year period. Eleven patients had chronic contained rupture, 5 aortoduodenal fistula, and 25 aortocaval fistula. The correct preoperative diagnosis was established in 6 (of 11) cases of chronic contained rupture, in 2 (of 5) cases of primary aortoduodenal fistula, and in 13 (of 25) cases of aortocaval fistula. They classified and investigated only ruptured aneurysms and unusual forms of ruptured AAA and did not focus on emergency presentations of AAA.

Campbell et al.[18] presented a 25-year study of emergency surgical admissions. This study documented the changes over 25 years in a district general hospital, and showed a progressive increase in ruptured aortic aneurysm. It is important to note that emergency AAA encompasses a spectrum of disease from symptomatic non-ruptured aneurysms to the classic free intraperitoneal rupture. There is a dilemma about the effect of misdiagnoses and delays in the diagnosis on mortality. Some studies have shown an increased mortality among patients with emergency AAA who were misdiagnosed. Several other studies have shown that misdiagnosis at presentation had no effect on mortality.[19-21] In a recent study, Gaughan et al.[6] reported that initially hemodynamically stable AAA patients who deteriorate after admission to the hospital had a poor prognosis. The 30-day mortality was 49% in that study, which emphasized the importance of starting prompt treatment with a correct diagnosis in reducing the mortality rates. One study investigated 98 patients; 56 of 98 patients were stable during the admission, and there were more misdiagnoses in this latter group compared to the group of patients presenting with shock.[6] Seven patients had delay in diagnosis and resultant free rupture in our series and three cases resulted in mortality.

Hans and Huang[22] retrospectively analyzed the records of 101 ruptured AAA patients. A better outcome was obtained in patients younger than 70 years, with a hematocrit of more than 35% at presentation and with emergency department to operating room time of less than 120 minutes. In the study of the Tochii et al.,[23] the rate of hospital deaths was 25.6% in 43 patients who underwent emergency operation for AAA. In our study, the mortality rate was low, at 6.25%, in the hemodynamically stable groups (Groups I, II, III). However, in patients with true rupture, the mortality rate was significantly higher than in these groups (40.7%). These findings suggest that the mortality rate can be reduced if hemodynamically stable patients who lack the classic signs of rupture are diagnosed and managed without delay before the aneurysms have ruptured.

Usually, ruptured or non-ruptured AAA has to be decided in the emergency room. The clinical examination plays an important part in the detection of AAAs and has moderate overall sensitivity; however, it cannot be relied upon to exclude them, especially if rupture is a possibility.[24] The accuracy of the physical examination in the diagnosis of AAA has a sensitivity of 68% and a specificity of 75%.[25] Ultrasound is substantially less accurate in the diagnosis of rupture, with a high false-positive rate of 33% and low specificity of 62%.[26] In cases of suspected rupture, CT has a sensitivity of 88% and a specificity of 88%.[27] However, CT is more time-consuming, and there is usually not enough time. The present study investigated whether any laboratory parameter could be used as a guide for differentiating a ruptured or non-ruptured emergency AAA.

The correct diagnosis of emergency AAA is difficult, and the reports from autopsy findings and retrospective studies reveal that there are misdiagnosed cases even in the most advanced medical centers.[28] The diagnosis can be missed especially when the patients present with atypical clinical symptoms such as hip pain,[29] groin pain,[24] hematuria, tenesmus, and hydronephrosis.[25] These atypical clinical symptoms can lead to diagnostic error, such as urethral colic, lumbar disc herniation, sciatica, acute myocardial infarction, perforated peptic ulcer, acute pancreatitis, acute cholecystitis, mesenteric vascular occlusion, and acute diverticulitis.[28] Most patients who survive to be admitted to the emergency department are hemodynamically stable. We evaluated a series of patients with and without AAA rupture to determine which, if any, laboratory parameters may be used as a guide for differentiating ruptured or non-ruptured emergency AAA. The laboratory parameters were compared with the reference values and were classified as normal, high or low. The distributions of these values according to groups are presented in Table 3. All parameters in the free rupture group were abnormal (Fig. 4). Hematocrit levels lower than normal reference values were seen in 100% of patients in the free rupture group, 32% in the dissecting rupture group, 67% in the impeding rupture group, and 53% in the chronic contained group. Our data show that the hematocrit levels were not definitive for the rupture, as nearly 50% of the other groups also had abnormal hematocrit levels. The white blood cell count was higher than normal reference values in 100% in the free ruptured group, 42% in the dissected group, 58% in the impeding rupture group, and 53% in the chronic contained rupture group. High base deficit was present in 92.6% of patients in the free rupture group, and low hematocrit, high white blood cell count, elevated rate of neutrophil and decreased lymphocytes rates, and elevated CRP levels were present in all of the patients. Therefore, we can claim that our study, as expected, showed that increased base deficit,

low hematocrit, high white blood cell count, elevated neutrophil and decreased lymphocytes rates, and elevated CRP levels are relevant to the severity of the patient's condition of ruptured AAA. Especially when classic signs of rupture are lacking, simple hematological and biochemical tests can be directive in determining the timing of life-saving surgery in emergent AAA patients.[30]

Vainas et al.[31] reported that high sensitive CRP levels are associated with AAA size (CRP: 3.23 (2.96) mg/dl), and they hypothesized that CRP may be released from the aneurysmatic area. In Parry's paper,[32] it was reported that the aneurysmatic region is a inflammatory site, and they had documented elevated CRP levels (2.07 vs 1.29 ng/dl, p=0.005). However, Domanovits[33] found acute phase reactants showing inflammatory increase only in symptomatic AAA patients (p=.002). In our study, we found high CRP levels in almost all groups (reference CRP levels 0-0.74 mEq/ml, p=0.001), but when the scenario worsened, we observed a greater increase in CRP levels (Tables 3, 4).

The results of our study revealed lower mortality rates in hemodynamically stable patients compared to patients presenting with free rupture. Free rupture represents a serious clinical scenario with deteriorated laboratory parameters prompting early surgery, which can provide a low mortality rate in this group. With this study, we also attempted to attract attention to an acute phase reactant (CRP) and its association with AAAs. Outcomes similar to those reported in other clinical trials were also observed in our study.

Our study has some limitations. First, it is a retrospective study. Second, we did not consider the possible differences in the skill levels of surgeons and anesthetists. Third, the study was not designed in a blinded fashion, so observer bias cannot be excluded. Fourth, the included patients are only those who were operated. It would seem that in this group the diagnosis is obvious. Freely ruptured AAAs could be used as a type of "gold standard" for comparison with contained or impending ruptured cases. However, a difference in lab parameters for a group with a clear clinical presentation is not particularly helpful for diagnosis.

In conclusion, emergency AAA represents a spectrum of disease from symptomatic non-ruptured aneurysm to free intraperitoneal rupture, which has significantly worse outcomes in patients with shock before surgery. Even before shock presents, laboratory parameters may indicate the extent of the threat in ruptured patients. Among groups, the worst laboratory parameters were determined in the free rupture group. Hematocrit level was lower, neutrophils dominated, white blood cell counts were higher, CRP levels were elevated, and blood pH deteriorated toward acidosis. We conclude that especially when classic signs of rupture are lacking, simple hematological and biochemical tests can be directive in determining the timing of life-saving surgery in emergent AAA patients.

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