EJCM
ejcvsmed.org
E Journal of Cardiovascular Medicine
Volume 5 | Issue 4
October-December 2017
www.ejcm.org & www.ejcvsmed.org

E Journal of Cardiovascular Medicine

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EJCM
E Journal of Cardiovascular Medicine
ejcvsmed.org
E Journal of
Cardiovascular Medicine
www.ejcvsmed.org
www.ejcm.org
medikal Akademi
Volume 2 Issue 1
January-June 2014

www.ejcm.org
www.ejcvsmed.org

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Volume 5 | Number 4 | October-December 2017

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www.ejcm.org & www.ejcvsmed.org

EJCM 2017; 05 (4): 67-74 **Doi:** 10.15511/ejcm.17.00467

Correlations between hematological indicators and other known markers in acute coronary syndromes

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Abstract

Introduction: Recently, many hematologic markers have identified as prognostic and diagnostic indicators in different acute coronary syndrome (ACS) patients. In particular, neutrophil / lymphocyte ratio (NLR) and platelet / lymphocyte ratio (PLR) are recognized as markers in the diagnosis and prognosis of ACS. In this study, our aim was to investigate the correlation between the diagnostic yield of PLR and NLR values and other markers such as troponin in all ACS patients.

Material and methods: 319 patients admitted to our hospital with ACS and 283 control patients were included in the study. Leukocyte, neutrophil, platelet, lymphocyte counts, PLR, NLR and high sensitive troponin I (HsTnI) measurements were taken.

Results: Leukocyte, neutrophil and platelet counts were significantly higher in the ACS group than the controls ($p < 0.001$). Lymphocyte count was significantly lower in the ACS group than the controls ($p < 0.001$). NLR and PLR were found to be significantly higher in the ACS group than the controls (4.0 ± 3.3 vs 2.1 ± 1.4 and 126.3 ± 68.9 vs 106.9 ± 49.4, $p < 0.001$, respectively). NLR was showed significant correlation with HsTnI, PLR, angina time, presence of ST elevation and T wave negativity ($p < 0.05$), and PLR revealed significant correlation with NLR, HsTnI, ST elevation and T wave negativity ($p < 0.05$).

Conclusion: Hematologic markers were found to be significantly higher in the ACS group than the controls. The difference also continued in subgroup analyzes. NLR and PLR correlated with many other ACS indicators.

Key words: Platelet lymphocyte ratio, neutrophil lymphocyte ratio, acute coronary syndrome, troponin.

Ertürk M., Turhan Caglar FN., Bıyık İ., et al. Correlations between hematological indicators and other known markers in acute coronary syndromes. EJCM 2017; 05 (4): 67-74. Doi: 10.15511/ejcm.17.00467.

Introduction

Coronary artery disease (CAD) is a major cause of morbidity and mortality worldwide.[1] One of the most important causes of CAD is atherosclerosis and atherosclerosis is considered to be an inflammatory disease.[2] Previous studies have shown serious evidence that atherosclerosis is systemic rather than focal disease.[2] Recently, many studies have been published, which demonstrate that hematological markers measured in peripheral blood count such as platelet, neutrophil, lymphocyte are predictors in the diagnosis and the determination of the prognosis of acute coronary syndromes (ACS).[3, 4] Increased platelet activation has been reported to play a role in the onset and the progression of atherosclerosis.[5] It has been reported that increased platelet count is associated with platelet activation and may be associated with thrombotic tendency and major adverse cardiac events.[3, 5]

On the other hand, there are reports that low lymphocyte counts are associated with lymphocyte apoptosis in prolonged inflammation, decreased immune-reactivity, and may also cause major adverse cardiac events.[5-7] As a result, proliferation and relative thrombocytosis are seen in the megakaryocytic series with inflammation.[7] Thus, in some studies, high platelet / lymphocyte ratio (PLR) and neutrophil / lymphocyte ratio (NLR) are predicted to better reflect inflammation and pro-thrombotic states and may be predictors of prognosis in acute ACS patients.[7, 8] In our study, we aimed to investigate the diagnostic efficiency of PLR and NLR values and their correlation with other predictors such as high sensitivity troponin I in ACS patients admitted to our hospital.

Patients and Method

This single-center comparative study was conducted in a tertiary heart center. The study was approved by the local Ethics committee and informed consent of the patients and controls were obtained. 319 patients admitted to our hospital with the diagnosis ACS between 2015 and 2017 were included in the study. In the same time period, 283 healthy individuals who applied to our outpatient clinics were included in the study as a control group. Subsequently, the ACS group was divided into subgroups as unstable angina pectoris (USAP), non-ST elevation myocardial infarction (NSTEMI), and ST elevation myocardial infarction (STEMI). Patients with severe valve disease, decompensated heart failure, malignancy, hematological disease, systemic inflammatory disease, active infection, autoimmune disease, severe renal or hepatic insufficiency and those using steroids were excluded from the study.

The demographic and biochemical parameters of the patients and controls were obtained from the files. NLR and PLR were calculated from the neutrophil, platelet and lymphocyte counts obtained from the hemogram. PLR and NLR were calculated by dividing platelet count by lymphocyte count, respectively, by dividing the number of neutrophils by the number of lymphocytes. For the measurement of high sensitive troponin I (HsTnI) EDTA whole blood sample was used, HsTnI was measured by the method of the AQT90 FLEX cardiac Troponin I immunoassay (Radiometer Medical ApS, Copenhagen, Denmark), and the method is a one-step sandwich immunofluorometric assay based on the use of three monoclonal antibodies, two for the capture and one for the detection.[9]

Complete blood count (CBC) was measured from EDTA whole blood samples via BC 6800 auto analyzer (Mindray Medical International Limited, Shenzhen, China). Other biochemical measurements such as glucose, HDL, LDL, total cholesterol and triglycerides were made from serum samples via Cobas systems (Roche Diagnostic Basel, Switzerland) by using commercial kits (Roche Diagnostic Basel, Switzerland).[9] Two levels of internal quality controls were made for all devices.

Statistical analysis

Statistical analysis was performed using SPSS version 15.0 (SPSS Inc., Chicago, Illinois, USA). Continuous variables were described by mean and standard deviation. Categorical variables were described in percent. Mann-Whitney U-test and student t-test were used for continuous variables, while chi-square test was used for categorical variables. Kruskal-Wallis test and Mann-Whitney U-test were used for subgroup analysis. Spearman test was used for correlation analysis. The diagnostic accuracy of high sensitive troponin I, NLR and PLR for ACS was calculated by receiver operating characteristic (ROC) analysis.

Results

319 ACS patients (219, 68.7%, male) and 283 controls (181, 64%, male) were included in the study. There were 114 (35.7%) patients with USAP, 101 (31.7%) patients with NSTEMI and 104 (32.6%) patients with STEMI in the ACS subgroups. The demographic, clinical, electrocardiographic and biochemical parameters of the patients are shown in **Table 1**. Neutrophil and platelet counts were significantly higher in the ACS groups than in the control group (p <0.001 respectively). Lymphocyte count was lower in the ACS group than in the control group (p <0.001). NLR and PLR were significantly higher in the ACS group than in the control group (p <0.001).

In the ACS subgroup analysis, NLR and PLR values were significantly higher in STEMI patients than in USAP and NSTEMI patients (p <0.001) (**Table 2**). Correlation analyzes revealed that NLR correlated with HsTnI, PLR, ST elevation, ST depression, T wave negativity and angina duration (**Table 3**). PLR was correlated with NLR, HsTnI, ST elevation and T wave negativity (**Table 3**). Receiver operating characteristic (ROC) analysis results are given in **Table 4**. When the cut-off value for NLR was taken as 2.5, the sensitivity of NLR for the diagnosis of ACS was found 63.6%, specificity 80.2%, negative predictive value 66.1% and positive predictive value 78.6% (95% confidence interval 0.66-0.75). When the cut off value for PLR was taken as 101.2, the sensitivity of PLR for the diagnosis of ACS was determined as 54.9%, specificity, 54.4%, negative predictive value 51.7% and positive predictive value 57.6% (95% confidence interval 0.54-0.63).

Discussion

In recent years, NLR and PLR have become important markers easily measured from peripheral blood and have proven diagnostic and prognostic information in patients with ACS.[10] In our study, NLR and PLR values were found to be higher in all ACS patients than the control group in accordance with the literature. In subgroup analyzes, NLR and PLR values were significantly higher in STEMI patients than NSTEMI and USAP patients. NLR and PLR were correlated with each other and with other parameters which have proven prognostic information in ACS patients such as high sensitive troponin I (**Table 3**). Sezer et al. showed that the number of neutrophils and the mean platelet volume in patients with acute myocardial infarction were associated with reperfusion injury after infarct-related artery occlusion opened.[10] In many inflammatory conditions neutrophils are stimulated to release many cytokines and cytotoxic or proteolytic enzymes.[4]

These enzymes have adverse effects on the ischemic heart by damaging endothelial cells, stimulating the coagulation system, blocking the microvascular circulation, and increasing infarct expansion.[4] Lymphocytes, as parts of the adaptive immunity system, fight against inflammation and suppress inflammation.[2] Thus, low

Table 2. Subgroups comparisons of hematologic indicators and cardiac markers

	USAP (n=114, 35.7%)	NSTEMI (n=101, 31.7%)	STEMI (n=104, 32.6%)	
	Mean ± Standard Deviation	Mean ± Standard Deviation	Mean ± Standard Deviation	P value
HsTnI (µg/L)	0.0108 ± 0.0084	1.2659 ± 3.6638	5.5973 ± 7.8381	<0.001
NLR	2.61 ± 1.59	3.30 ± 2.36	6.13 ± 4.34	<0.001
PLR	99.27 ± 48.54	125.90 ± 65.59	156.37 ± 78.56	<0.001

HsTnI: *High sensitivity troponin I,* ***NLR:*** *neutrophil / Lymphocyte ratio,* ***PLR:*** *platelet / lymphocyte ratio*

Table 1. Demographic characteristics of patients and controls

	Patients (n=319)	Controls (n=283)	P value
Age (years)	56.6 ± 11.4	47.3 ± 13.6	<0.001
Male, n (%)	219 (68.7)	181 (64)	0.223
BMI, kg/m²	27.8 ± 4.2	27.5±4.3	0.697
History			
Known CAD, n (%)	109 (34.2)	54 (19.1)	<0.001
Previous PCI, n (%)	80 (25.1)	44 (15.5)	0.004
Previous AMI, n (%)	59 (18.5)	32 (11.3)	0.014
Previous CABG, n (%)	51 (16)	13 (4.6)	<0.001
Previous stroke / TIA, n (%)	17 (5.3)	7 (2.5)	0.074
Cardiovascular risk factors, n (%)			
Hypertension, n (%)	145 (45.5)	102 (36)	0.019
Diabetes mellitus, n (%)	57 (17.9)	39 (13.8)	0.172
Family history of CAD, n (%)	80 (25.1)	30 (10.6)	<0.001
Smoking, n (%)	156 (48.9)	127 (44.9)	0.323
Electrocardiographic findings in admission			
Normal, n (%)	87 (27.3)	247 (87.3)	<0.001
ST segment elevation, n (%)	100 (31.3)	0 (0)	
ST segment depression, n (%)	61 (19.1)	6 (2.1)	
T-wave inversion, n (%)	42 (13.2)	24 (8.5)	
Bundle branch block, n (%)	29 (9.1)	6 (2.1)	
Duration of angina, hours	7.9 ± 1.7	8.0 ± 1.8	0.886
Biochemical parameters			
Total cholesterol, mg/dL	202.2 ± 44.3	188.6 ± 42.7	<0.001
LDL cholesterol, mg/dL	140.0 ± 41.6	123.9 ± 37.8	<0.001
HDL cholesterol, mg/dL	44.2 ± 10.5	44.8 ± 14.1	0.321
Triglycerides, mg/dL	167.0 ± 103.1	188.8 ± 125.0	0.046
Glucose, mg/dL	137.4 ± 63.1	115.4 ± 47.1	<0.001
Creatinine, mg/dL	0.96 ± 0.5	0.88 ± 0.47	0.003
High sensitive Troponin I µg/L	2.2295 ± 5.4671	0.0102 ± 0.0054	<0.001
Hemogram parameters			
Hematocrit, %	41.4 ± 5.9	40.6 ± 5.1	0.422
WBC	10.4 ± 3.5	8.5 ± 2.3	<0.001
Neutrophil count 103 / µL	7.2 ± 3.3	5.0 ± 1.9	<0.001
Lymphocyte count 103 / µL	2.3 ± 1.1	2.7 ± 0.9	<0.001
Platelet count 103 / µL	244.5 ± 60.0	260.6 ± 64.8	<0.001
NLR	4.0 ± 3.3	2.1 ± 1.4	<0.001
PLR	126.3 ± 68.9	106.9 ± 49.4	<0.001

BMI: *body mass index,* ***CAD:*** *coronary artery disease,* ***AMI:*** *acute myocardial infarction,* ***CABG:*** *coronary artery bypass grafting,* ***TIA:*** *transient ischemic attack,* ***LDL:*** *low-density lipoprotein cholesterol,* ***HDL:*** *high-density lipoprotein cholesterol,* ***Htc:*** *hematocrit,* ***WBC:*** *white blood cell count,* ***NLR:*** *neutrophil / lymphocyte ratio,* ***PLR:*** *platelet / lymphocyte ratio.*

lymphocyte counts have been associated with athero-sclerosis progression and poor clinical outcomes.[2] Zouridakis et al found that low lymphocyte counts in unstable angina pectoris patients are strongly associated with future cardiac events.[11]

Although the pathophysiological mechanism of this condition is not fully understood, lymphocyte count is thought to reflect the early response of myocardial ischemia to physiological stress and systemic immunodeficiency.[4] Conversely, high lymphocyte counts during

Table 3. Correlation analyzes for NLR and PLR in ACS patients

Spearman's rho	Correlation Coefficient Sig. (2-tailed)	NLR	PLR
NLR	r	1.000	0.733**
	p	.	0.000
	n	319	319
HsTnI	r	0.466**	0.475**
	P	0.000	0.000
	n	319	319
PLR	r	0.733**	1.000
	p	0.000	.
	N	319	319
ST depression	r	-0.183**	0.051
	p	0.001	0.361
	n	319	319
Bundle branch block	r	-0.017	-0.045
	p	0.762	0.422
	n	319	319
Duration of angina	r	0.145**	0.085
	p	0.010	0.129
	n	319	319
Location of infarction	r	-0.023	0.068
	p	0.677	0.227
	n	319	319
T inversion	r	-0.285**	-0.221**
	p	0.000	0.000
	n	319	319
ST segment elevation	r	0.466**	0.365**
	p	0.000	0.000
	n	319	319
Type of angina	r	-0.075	-0.023
	p	0.181	0.676
	n	319	319

__HsTnI;__ high sensitivity troponin I, __NLR;__ neutrophil / Lymphocyte ratio, __PLR;__ platelet / lymphocyte ratio, __ACS;__ acute coronary syndrome

ACS reflect a strong immune response and the prognosis of these patients is better.[4] Instead of examining neutrophils and lymphocytes separately, NLR has been shown to be more prognostic.[2] Wang et al. have shown that NLR is predictive of all-cause mortality and cardiovascular events in patients undergoing coronary angiography.[12] Likewise, Tamhane et al. found that NLR value in patients with percutaneous coronary intervention is related to in-hospital and 6-month mortality.[13] Ayca et al. have shown that NLR is associated with stent thrombosis.[14]

NLR has been studied by different investigators in relation to different scoring systems showing CAD severity and it has been shown to be related to NLR by SYNTAX, GRACE and TIMI scores.[15-17] In our study, similarly, the NLR values were higher in the ACS group than in the control group. Correlation analyzes showed that NLR correlated with high sensitive troponin I, PLR, ST elevation, ST depression, T wave negativity and angina duration. NLR was found to have a sensitivity of 63.6% and a specificity of 80.2% for ACS diagnosis. The role of platelets in ACS is versatile.[4] The platelet count is associated with the underlying inflammation because inflammatory mediators stimulate megakaryocytic proliferation and result in a prothrombotic state with relative thrombocytosis.[4, 5]

As platelet levels increase, more platelet rich thrombosis is observed in atherosclerotic plaques, and antiplatelet therapy response is reduced.[4] It is known that platelets are associated with endothelial activation as well as inflammatory functions.[2] Active platelets release pro-angiogenic mediators and regulate the microvasculature of blood vessels.[18] As essential components of coronary thrombus formation, platelets play an important role in ACS.[2] In the CADILLAC study, increased platelet counts were also associated with restenosis and stent thrombosis.[19] PLR is thought to better reflect both inflammation and coagulation pathways.[4] In their study using optical coherence tomography, Wang et al showed that high PLR is associated with sensitive plaque properties such as thinner fibrous cap, wider lipid load in non-target vessels in ACS patients.[20]

The authors suggested that the relation between PLR and sensitive plaques is associated with immunologic and inflammatory pathways.[20] The relationship between PLR and CAD severity is also shown in other publications.[21] For example, Kurtul et al. have shown that high PLR is associated with moderate to high SYNTAX score in patients with ACS.[21] In another study, high platelet counts have been shown to be associated with microvascular plug formation, thrombus formation, and vasoconstriction and no-reflow development.[8] PLR has been shown to be associated with mortality in STEMI and NSTMI patients.[22] Oylumlu et al noted that high PLR values in ACS patients were associated with in-hospital mortality.[4]

In their meta-analysis, Li et al. showed that increased PLR is associated with in hospital and long-term mortality and cardiovascular events in ACS patients.[5] Yıldız et al. have also shown that high PLR values are associated with no-reflow development after percutaneous interventions.[23] Prajapati et al. showed a relationship between

Table 4. Receiver operating characteristic (ROC) analyzes of HsTnI, NLR and PLR for diagnosis of ACS

	AUC	SE	P-value	CI%95		Speci-ficity	Sensi-tivity	NPV	PPV	Cut off value
				Lower limit	Upper limit					
HsTnI	0.800	0.018	0.000	0.764	0.836	99.6	54.2	65.9	99.4	>0.0023
NLR	0.709	0.022	0.000	0.667	0.751	80.2	63.6	66.1	78.6	>2.5
PLR	0.586	0.023	0.000	0.541	0.632	54.4	54.9	51.7	57.6	>101.2

***HsTnI;** high sensitivity troponin I, **NLR;** neutrophil / Lymphocyte ratio, **PLR;** platelet / lymphocyte ratio, **ACS;** acute coronary syndrome, **NPV;** negative predictive value, **PPV;** positive predictive value* increased PLR and NLR and decreased HDL values in CAD patients.[18] The combined use of NLR and PLR suggests that more prognostic information will be obtained. In this context, Choi et al. found that combined NLR and PLR elevations were associated with long-term cardiovascular events (22). Hematological markers have also been shown in other publications as indicators of inflammation. Similar to other studies, in our study, NLR and PLR in patients with ACS were higher than in the control group. The difference of present study from the other studies is that all the ACS subgroups were included and examined in the same study and that the NLR and PLR were high in all the subgroups.

Conclusion

Inflammatory and thrombotic processes play a crucial role in the development of atherosclerosis, atherosclerotic plaque destabilization and clot formation after plaque rupture. In the diagnosis and prognosis of ACS, there are still needs for reliable, easy, inexpensive and fast detectable markers. PLR and NLR are cheap and easy to use markers in the diagnosis and the prognosis of ACS patients but definite cut off values are needed to use these markers. Large scale and comprehensive studies are needed to reveal absolute cut off values for these markers.

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Received: 29/07/2017
Accepted: 24/11/2017
Published: 15/12/2017

Disclosure and conflicts of interest:
Conflicts of interest were not reported.

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Ertürk M., Turhan Caglar FN., Bıyık İ., et al. Correlations between hematological indicators and other known markers in acute coronary syndromes. EJCM 2017; 05 (4): 67-74. Doi: 10.15511/ejcm.17.00467.

EJCM 2017; 05 (4): 75-78 Doi: 10.15511/ejcm.17.00475

Successful surgical treatment of a ruptured giant atherosclerotic aneurysm of arcus aorta

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Abstract

Endovascular aortic repairs in hybrid theatres have been implemented as an alternative option for the treatment of aortic arch aneurysms. The improvements in synthetic graft technologies, cardiopulmonary bypass techniques, blood protection, myocardial protection and protection of the central nervous system have resulted in safer applications of surgical interventions, particularly to the arcus aorta. In this article, we present our successful emergency surgical therapeutic approach in a 74-year-old female patient case with a ruptured giant aortic arch aneurysm.

Key words: Endovascular aortic repairs, aortic arch aneurysms, arcus aorta.

Akça B., Erdil N., Dişli O. M., Çolak M. C., Battaloğlu B. Successful surgical treatment of a ruptured giant atherosclerotic aneurysm of arcus aorta EJCM 2017; 05 (4): 75-78. Doi: **10.15511/ejcm.17.00475.**

Introduction

Due to its high mortality and morbidity rates, the surgical treatment for aortic arch aneurysms is challenging,[1] although there is currently a lack of consensus on the optimal treatment modality in the high-risk patients. Recently, endovascular aortic repairs in hybrid theatres have been implemented as an alternative option for the treatment of aortic arch aneurysms.[1] The improvements in synthetic graft technologies, cardiopulmonary bypass techniques, blood protection, myocardial protection and protection of the central nervous system (particularly selective cerebral perfusion and hypothermic circulatory arrest) have resulted in safer applications of surgical interventions, particularly to the arcus aorta.[2-5] In this article, we present our successful emergency surgical therapeutic approach in a female case with a ruptured giant aortic arch aneurysm.

Case Report

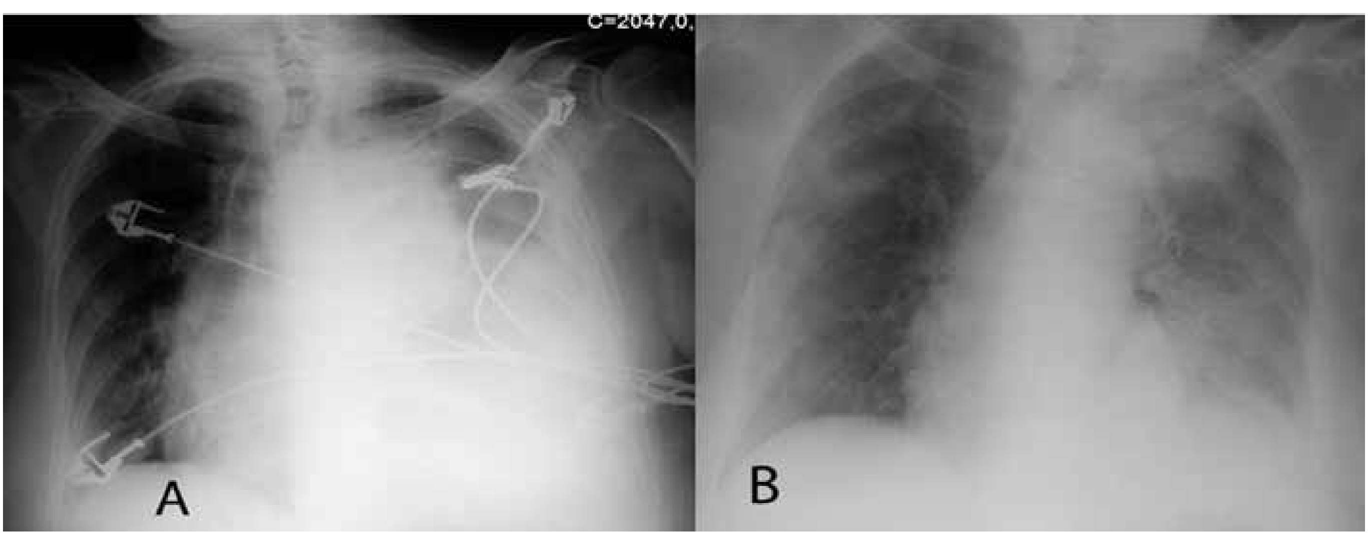
A 74-year-old female patient was admitted to our emergency clinic with complaints of blurred consciousness, chest pain and dyspnea. A physical examination of arterial blood pressure, pulse and oxygen saturation revealed values of 80/40 mmHg, 122 beats/min and 85–90%, respectively. On auscultation of the left hemithorax, breath sounds decreased, and a chest X-ray revealed a giant aortic aneurysm and opacity in the left hemithorax that was suggestive of a pleural effusion **(Figure 1A)**.

A true saccular aortic arch aneurysm that was measured 8x6 cm2 which had ruptured into the left hemithorax was detected in a contrasted thoracoabdominal tomography **(Figure 2A, 2B)**.

Written informed consent was obtained from the patient's relatives, and the patient who underwent emergency surgery. The left lung was adhered to the pleura, and the aortic arch was extremely atherosclerotic. A substantial amount of hematoma was present in the left thorax. A cardiopulmonary bypass was performed using the cannulation of the right axillary artery.

The patient was cooled to 25°C and aneurysmal sac opened by applying circulatory arrest under the right selective antegrade cerebral perfusion. An anastomosis of a 30-mm Hemashield graft (Maquet, France) was applied to the descending aorta, and the branches of the arcus were implanted onto the grafts cutting in the form of islets. The graft was then clamped, and a total bypass was applied. The total aortic arch replacement was completed through the graft anastomosis with the ascending aorta during the heating phase. The cardiopulmonary bypass was uneventful. The incisions were closed after bleeding management, and the patient was transferred to the Intensive Care Unit (ICU). She recorded no hemodynamic or neurological problems in her post-operative follow-up, although she was unable to tolerate extubation during the ICU stay (in the postoperative 10th hour), and she was re-intubated due to pulmonary complications.

Figure 1A - 1B



The patient was then transferred to the Anesthesiology and Reanimation Clinic and was extubated by administering a successful ICU program **(Figure 1B)**. She experienced no problems following extubation, and she was discharged on the 15th postoperative day.

Discussion

Aortic ruptures, as a complication of aortic aneurysms, leads to massive bleeding with a high rate of mortality. An emergent diagnosis and treatment following a multidisciplinary approach can lead to successful outcomes, despite high mortality rates, as in our case.

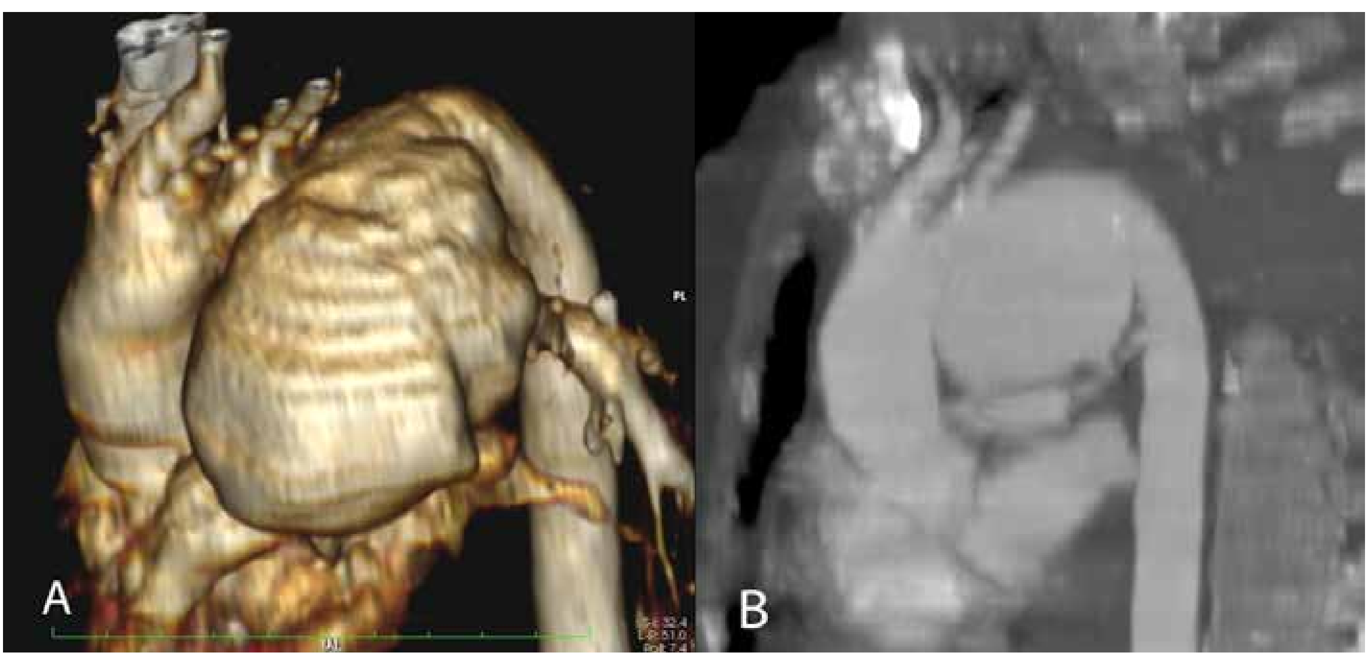
With the developments in technological methods and increased surgical experience over the past two decades, successful therapeutic results have been reported in patients with aortic aneurysms.(6) Applications of minimally invasive endovascular methods that can be performed via small inguinal incisions rather than thoracal aortic interventions through major surgical incisions have been rapidly increasing in recent years.(7) Hybrid methods and conventional open surgery have been compared in a study that investigated aortic arch interventions, and higher technical success rates with reduced mortality rates have been reported with the application of hybrid methods in a certain high-risk patient group.(1)

Previous reports of hybrid arch procedures showed an early mortality rate of 7.4–23.7% with an incidence of stroke seen in 0–13.1%.(1,8) In a meta-analysis with 463 patients who underwent hybrid arch surgery, the authors reported a 30-day mortality rate of 8.3% with an incidence rate of stroke and paraplegia of 4.4 and 3.9%, respectively.(9)

Endovascular treatments can be administered in cases of thoracic aortic disease that require a surgical approach if open surgery is considered to be a particularly high risk and if the anatomical appearance is appropriate. Our case was subjected to an open surgical intervention due to her presentation with hemodynamic instability and poor clinical status on emergency admission. Furthermore, there was no opportunity to make an endovascular intervention at the time of her admission, and an urgent intervention was needed.

In previous studies, it has been suggested that no definitive evidence exists that supports the superiority of hybrid thoracic endovascular aortic repair (TEVAR) over open arch repair,(10) although most cases of hybrid TEVAR are in high-risk patients who are ineligible for conventional open aortic repair. In this respect, comparing and interpreting the results can be challenging. Further prospective, randomized controlled trials are required to

Figure 2A-2B



compare the outcomes of both surgical strategies.

In conclusion, although the recent outcomes of open arch repair and hybrid TEVAR demonstrate acceptable results, particularly early after the procedure, open arch repair can bring more reliable outcomes during follow-up. These two surgical strategies, when properly selected based on the individual risk, can improve surgical outcomes in patients with aortic arch aneurysms, especially in ruptured cases.

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Received: 29/07/2017
Accepted: 18/11/2017
Published: 15/12/2017

Disclosure and conflicts of interest:
Conflicts of interest were not reported.

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EJCM 2017; 05 (4): 79-82
Doi: *10.15511/ejcm.17.00479*

Combined system usage for large neck anatomy in a high risk abdominal aortic aneurysm patient

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Abstract

Endovascular aneurysm repair (EVAR) is the first line approach in abdominal aortic aneurysm (AAA) repair. Open surgical repair of AAA is associated with high perioperative mortality and morbidity. A sufficient infrarenal aortic neck is one of the key points for successful outcome after EVAR. We presented in this case report a 75 year- old, male patient with AAA which maximal diameter is 60 mm. The aneurysm had a large neck that presents a hostile neck anatomy. He was unfit for conventional surgery because of comorbidities as chronic obstructive pulmonary disease, newly diagnosed lung cancer and coronary artery disease. He applied to the emergency service with severe and acutely onset back pain and he was admitted to our cardiovascular surgery clinic. Because of acute onset of the back pain, his serious comorbidities and we planned to use of thoracic and abdominal aortic endovascular stent-graft systems indwelling each other which is known as Funnel technique. Despite progress in endovascular therapy, large infrarenal necks remain a challenge for endovascular interventionists. It seems feasible to use combined thoracic and abdominal stent graft as an alternative treatment modality when the patient is not suitable for standard EVAR and open surgery.

Key words: Endovascular aneurysm repair, abdominal aortic aneurysm, large neck anatomy.

Yıldırım F., Amanvermez Şenarslan D., Kurdal A. T., Öztürk T., Bayram B., Cerrahoğlu M., İşkesen İ., Tetik Ö. Combined system usage for large neck anatomy in a high risk abdominal aortic aneurysm patient EJCM 2017; 05 (4): 79-82. Doi: 10.15511/ejcm.17.00479.

Introduction

Nowadays, endovascular aneurysm repair (EVAR) is the first line approach in abdominal aortic aneurysm (AAA) repair. Open surgical repair of AAA is associated with high perioperative mortality and morbidity. EVAR is a less invasive method than conventional surgery. A sufficient infrarenal aortic neck is one of the key points for successful outcome after EVAR. According to computed tomography angiography (CT), preoperative sizing and planning should be done precisely.(1)

The neck length, angulation, shape, existence of thrombus and calcification should be determined. When the anatomic features of the aneurysm are not favourable for standard EVAR therapies; it is termed "hostile neck anatomy". Proximal neck characteristics remain a challenge in endovascular treatments. Patients with hostile neck anatomy can be managed in some alternative ways; open surgery, chimney technique, fenestrated endovascular grafts and also Funnel technique described by Zachetta et al.(2,3)

Fenestrated endovascular abdominal aortic aneurysm repair (FEVAR) and chimney technique are more expensive, off the shelf fenestrated grafts are not available in emergency and in some countries health insurance may not cover the expense of these advanced techniques. In these circumstances funnel technique in which a thoracic stent-graft is placed through the main body of a bifurcated or uni-iliac endograft, must kept in mind.

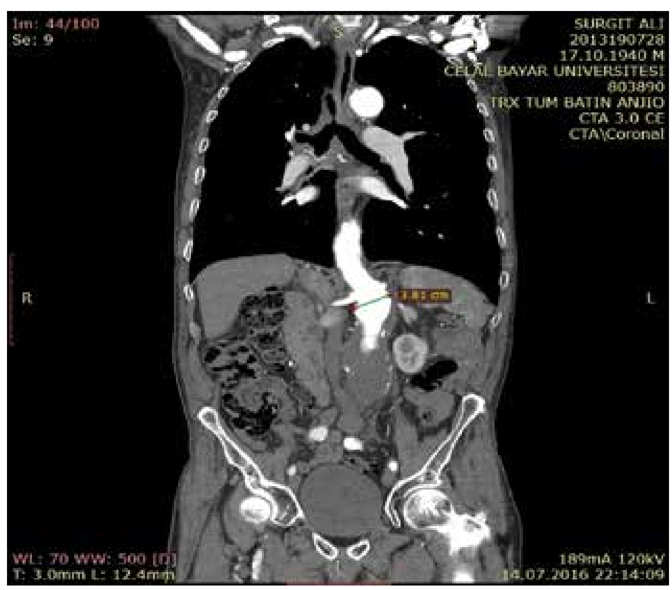
Case

We presented in this case report a 75 year- old, male patient with AAA which maximal diameter is 60 mm. The aneurysm had a large neck that presents a hostile neck anatomy. He was unfit for conventional surgery because of comorbidities as chronic obstructive pulmonary disease, newly diagnosed lung cancer and coronary artery disease. He applied to the emergency service with severe and acutely onset back pain and he was admitted to our cardiovascular surgery clinic.

In the CT Angiographic examination there was no dissection but he had an AAA with a large neck (38 mm internal diameter at just below the renal artery level) as shown in **Figure 1, 2, 3**. Because of acute onset of the back pain, his serious comorbidities and also because of insurance policies of our country we planned to use of thoracic and abdominal aortic endovascular stent-graft systems indwelling each other which is known as Funnel technique.

Figure 1. AAA with a large neck (38 mm internal diameter at just below the renal artery level), **axial** view of computed tomography anjiography.

Figure 2. AAA with a large neck (38 mm internal diameter at just below the renal artery level), **coronal** view of computed tomography anjiography.

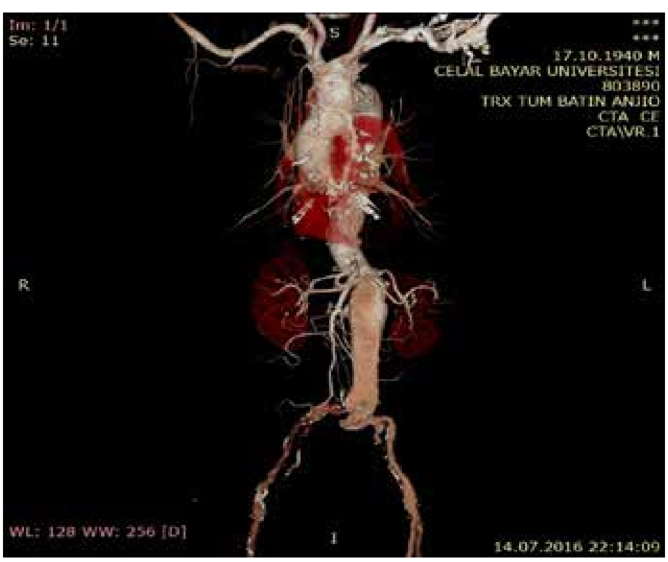


He was treated combining Valiant Captivia® (Medtronic, Minneapolis, MN) and Endurant® (Medtronic, Minneapolis, MN) endoprosthesis. We prepared the patient for intervention in hybrid operating room with epidural anaesthesia. Both femoral arteries were surgically exposed for arterial access and snared with purse-string sutures to control bleeding from access sites. Firstly, Valiant Captivia Thoracic Stent Graft ® 44-44 mm diameter and 100mm length stent graft was deployed just below the lowest renal artery. Then Endurant II ® aorto-uni-iliac 36-14 mm diameter and 102 mm length stent-graft was deployed inside of the previous endograft with an overlap of 50 mm.

Later Endurant II® 16-16 mm diameter and 82 mm length extension stent-graft was inserted to the left iliac arterial system. The right common iliac artery was coiled with endoluminal occluder® (OCL22, Medtronic Cardiovascular, Santa Rosa, CA). The control angiography showed a trace of endoleak from the proximal neck. The Reliant balloon catheter® was used for proximal landing zone fixation. The control angiography didn't show any type I endoleak after ballooning of the proximal neck.

At last, uni-iliac EVAR is accomplished by a left to right femorofemoral bypass operation with an 8-mm ringed PTFE graft. The operation was ended without any trouble. The patient was discharged from the hospital at postoperative third day. The patient has still no problem in his first and third month visits.

Figure 3. Preoperative 3-dimensional CT angiography reconstruction; hostile neck and hostile iliac artey anatomy.



Discussion

As many as 20-50% of AAA patients have anatomy that is not convenient for a standard endovascular therapy.[4] Funnel technique is an alternative way for patients with large hostile neck anatomy who are not suitable for open surgery and the other endovascular methods as FEVAR and chimney grafts. There are limited number of studies related to combined usage of thoracic and abdominal stent-grafts (Funnel Technique).[2-5]

Most of the literature consists of case reports. Zachetta et al. first described the method in a case report in 2006.[2] Ronsivalle S. et al. described Funnel technique for EVAR: "a way out" for AAA with ectatic proximal necks.[4] They both reported that the method was secure and effective in sealing to a large aortic neck. They did not observe type I endoleak or migration over a midterm follow up.

Many manufacturers latest-generation abdominal stent grafts have maximal 35-36 mm diameter neck for trunk and ipsilateral leg endoprothesis. But when the patient has a larger neck and given the 10-20% oversizing ratio for adequate sealing, the interventionist might need additional stents for endovascular treatment.

Secure fixation and adequate sealing zone at the proximal and distal AAA necks are one of the most important factors in predicting a successful outcome of EVAR.[2] Large stent grafts are more at the risk of proximal endoleak and reintervention in the long run.[6,7]

The authors emphasized the importance of at least 40 mm overlapping to the thoracic stent-graft, leaving more than 40 mm extending beyond the main body of the bifurcated device to ensure expansion to its nominal diameter and adequate sealing.[2,4] In our case, we also obeyed this protocol.

In a recent case report and literature review Pecoraro et al. compared large neck (24-34 mm) AAA treated by standard stent-grafts and wide neck (≥35mm) AAA treated by Funnel technique. He reports; surprisingly higher incidence of migrations in large neck group. Authors interpreted as Funnel technique may be a more valuable deployement methods than we thought.[5]

In some clinics, all patients who undergo EVAR are applied sac thrombization with intrasac biomaterials to prevent endoleaks.[2,4] But in our country, it is not possible because health insurance does not cover its expense. So, we did not apply sac thrombization. We have not yet mid-term and long-term follow-up results. We need further follow-up reports to be sure of its durability.

Conclusion

Despite progress in endovascular therapy, large infrarenal necks remain a challenge for endovascular interventionists. Different methods of endovascular therapies like "Funnel technique" enables us to treat high risk, old patients with multiple comorbidities in a short period of hospital stay. It seems feasible to use combined thoracic and abdominal stent graft as an alternative treatment modality when the patient is not suitable for standard EVAR and open surgery.

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Received: 16/06/2017
Accepted: 04/11/2017
Published: 15/12/2017

Disclosure and conflicts of interest:
The authors declare no conflict of interest.

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EJCM 2017; 05 (4): 83-86 Doi: 10.15511/ejcm.17.00483

Emergent operation of an extensive pulmonary damage after thoracal gunshot assault

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Abstract

Transthoracic gunshot injuries acquire immediate intervention with a correct approach as they have high mortality rates. The high mortality mainly depends on the presence of a probable concomitant injury of the cardiac, pulmonary and major thoracal vascular structures. In this case report, the emergent surgical management of a young female referred with a thoracal gunshot wound causing a massive pulmonary destruction is presented.

Keywords: Firearms; thorax; lung injury

Başbuğ H. Z., Göçer H., Özışık K. Emergent operation of an extensive pulmonary damage after thoracal gunshot assault
EJCM 2017; 05 (4): 83-86. Doi: **10.15511/ejcm.17.00483.**

Introduction

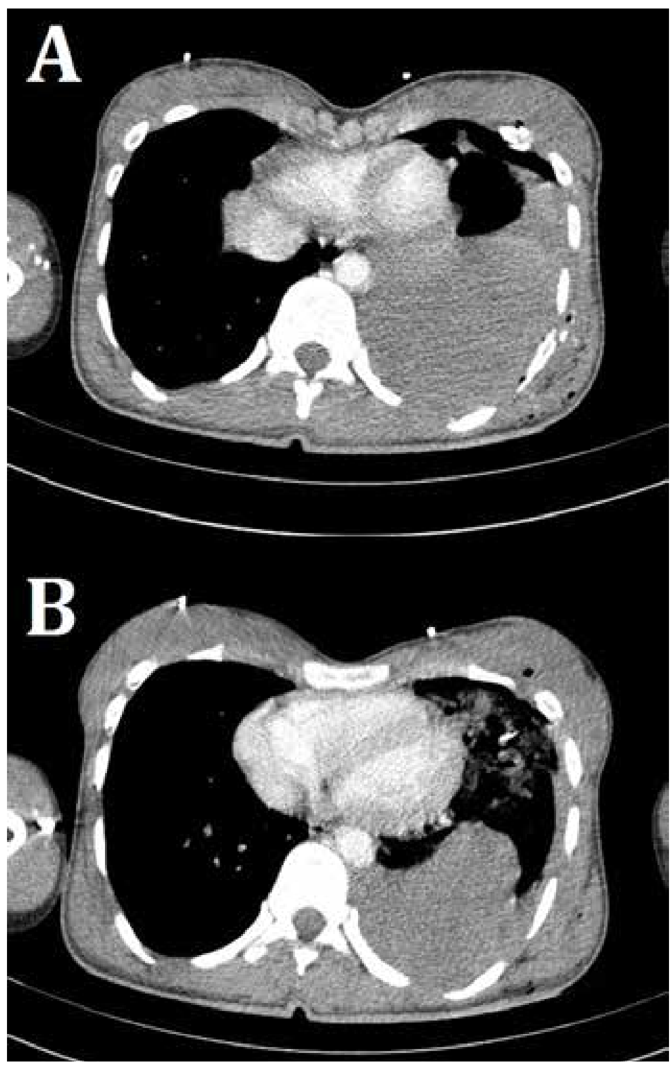
Thoracic gunshot injuries are associated with high mortality due to concomitant pulmonary, cardiac or major vascular injuries.[1] The treatment of hemodynamically unstable patients with thoracic gunshot wounds is emergent due to major cardiac or vascular injuries. These patients should immediately proceed to the operating room.[2] In hemodynamically stable patients, diagnostic tests are needed before urgent transfer to the operation.[3] In this paper, the emergent surgical management of a young woman with a transthoracic gunshot wound was presented. This case is diversified from others attributing to the presence of a pulmonary damage with a suspicious cardiac injury.

Case Report

A 22-year-old female referred with a thoracic gunshot wound. She was conscious with a blood pressure of 80/50 mmHg and heart rate of 124 bpm. She had firearm bullet entry wound just above her left nipple and an exit wound on her left posterior axillary line below the sixth intercostal space. Physical examination of the entry wound revealed a contact muzzle stamp over the nipple **(Figure 1)**. Posteriorly, there was a lateral bullet exit lesion between the sixth intercostal space and the fractured seventh rib. The Computerized Tomography (CT) Angiography of the thorax demonstrated the lack of great vessel injury and the presence of a massive left hemothorax consistent with the left pulmonary parenchymal injury **(Figure 1)**. There was an interesting patchy opaque extravasation around the pericardium near the cardiac apex which might be related to a cardiac injury **(Figure 2)**. Thus, the patient was taken to the operating room urgently. Following a median sternotomy, the pericardium was dissected 1 cm long to explore and see the pericardial

Figure 1. Morphological view of the penetrating firearm wound. Note the contact muzzle stamp around the bullet entry hole.

Figure 2. Computerized Tomography (CT) Angiography of the thorax demonstrates the presence of a massive left hemothorax with a contrast extravasation consistent with the left pulmonary parenchymal injury (A). The suspicious view in the CT Angiography implicating a cardiac injury (B).



aspirate. There was no hemopericardium consistent with a cardiac involvement that was suspected by the **Figure 1**. Left pleura was then widely opened beneath the sternum and 1200 ml of blood was aspirated from the left thorax. Upon examination of the left lung, the superior lobe was seriously injured. The parenchymal laceration was repaired with a 4-0 polypropylene by double running stitch **(Figure 1)**. Near the bullet exit site over the inferior lobe, there was another parenchymal laceration which was also repaired with the same suturing technique. No postoperative complication happened.

Discussion

Advances in prehospital care systems enabled shorter delivery times for the penetrating trauma patients to the hospitals. "Scoop and run" strategy have replaced the older "stay and play" manner. This resulted in more trauma victims become more able to reach the emergency departments in extremis.[4] Despite this advance in improvement in the transportation of the casualties to the hospital, overall survival after transthoracic gunshot injuries has not improved satisfactorily depending on the publications between 1966 and 2012.[1] From Bradley[5] who reported the first case series in 1966 with a 20% of mortality, up to Okeye et al.[1] who declared an overall mortality rate of 78%, the survival after transthoracic gunshot wounds is still inefficiently low.

Transthoracic gunshot wounds are almost always associated with a hemodynamic compromise and require urgent appropriate intervention.[6] However, hemodynamically stable patients with transthoracic gunshot wounds would also have a potential risk for significant occult injury.[7] Degiannis et al.[8] reported 42% occult injuries among stable patients. Moreover, Richardson et al.[9] observed occult injury in 63% of their patients who were initially stable. Thus, the traditional preoperative diagnostic tests in these instances become more crucial to determine the severity of the injury as well as to exclude a significant occult injury among the asymptomatic or less symptomatic cases. Intravenous contrast CT imaging is generally used for visualization the missile tract as well as to evaluate the vascular integrity.[10] The use of CT-imaging as a screening tool is generally sufficient to rule out significant thoracic and mediastinal injury.[11]

The operative approach in transthoracic penetrating trauma patients depends on the hemodynamic condition of the patient and the injury site.[12] In a hemodynamically stable patient with a mediastinal penetrating injury, the incision should be tailored meticulously. The majority of cardiac and thoracic injuries can comfortably be handled with a median sternotomy.[13]

However, for patients who arrived in extremis, lateral thoracotomy and aortic cross-clamping may be suitable to control the mediastinal bleeding rapidly.[14] The surgical approach must be directed to the optimal exposure and easiest access. Injuries of the structures located in the posterior mediastinum including the thoracic esophagus, descending aorta, distal tracheobronchial structures need lateral thoracotomy while the cardiac and ascend-

Figure 3. The intraoperative image is demonstrating the pulmonary parenchymal destruction. Note the hemorrhage through the laceration site.

Figure 4. The intraoperative image is demonstrating the pulmonary parenchymal repair. Note the hemorrhage was stopped.

ing aorta injuries need median sternotomy.[1] In this case, a median sternotomy was preferred to evaluate the suspicious cardiac involvement that was seen on CT image (Figure 3). CT image revealed a suspicious contrast extravasation that may implicate a possible apical involvement as the bullet was glanced off. Upon performing a tiny pericardial incision, no pericardial blood was seen. By excluding the cardiac involvement, the left lung was assessed and repaired through the sternotomy easily (Figure 4) and the drain tubes were successfully placed.

In conclusion, transthoracic firearm injuries are almost always associated with hemodynamic compromise and require rapid and appropriate resuscitative surgical exploration. Thus, depending on the injured structures and vital status of the patient, correct approach should be tailored in these life-threatening injuries.

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Received: 19/06/2017
Accepted: 21/11/2017
Published: 15/03/2017

Disclosure and conflicts of interest:
Conflicts of interest were not reported.

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EJCM 2017; 05 (4): 87-90
Doi: 10.15511/ejcm.17.00487

Which should be treated first; coronary artery disease or celiac artery compression syndrome?

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Abstract

Celiac artery compression syndrome is rare. The differential diagnosis and the choice of therapeutic intervention in this syndrome are controversial. Herein, we report of a case of celiac artery compression syndrome and concomitant coronary artery disease and discus differential diagnosis and treatment strategies.

Key words: Dunbar syndrome, celiac artery compression syndrome, coroner artery disease, intervention

Bıyık İ., Bahtiyar E., Işıksaçan N., Which should be treated first; coronary artery disease or celiac artery compression syndrome? EJCM 2017; 05 (4): 87-90. Doi: **10.15511/ejcm.17.00487.**

Introduction

Celiac artery compression syndrome (CACS) is a rare clinical entity. It has also known as median arcuate ligament (MAL) or Dunbar syndrome. The disease results from the external compression of the celiac artery by the diaphragmatic crura or the median arcuate ligament.(1) The incidence of this rare condition has been reported from 1.76% to 4.0% in different radiologic studies.(1) Approximately, 85% of patients with CACS are asymptomatic.(1) The leading symptoms of this entity has been reported as abdominal pain (94%), postprandial abdominal pain (80%), nausea and vomiting (55.6%), weight loss (50%), bloating (39%), and abdominal pain triggered by exercise (8%).(2) The differential diagnosis and the choice of therapeutic intervention in this syndrome are controversial.(2) Herein, we report of a case of CACS and concomitant coronary artery disease (CAD) and discus differential diagnosis and treatment strategies.

Case Report

Informed consent of presented patient has been taken for this report. A forty three years old male patient presented to our out-patient clinic of cardiology with the symptoms of long-standing epigastric pain, abdominal swelling and loss of appetite. His detailed history revealed that the symptoms increase especially after overdosed dinner, hiking and exercises. His habitual history revealed excessive alcohol and cigarette consumption. His physical examination was unremarkable except abdominal obesity. His body mass index was 32 kg / m2. His electrocardiogram and echocardiographic study were normal. His biochemical and laboratory investigations were in normal limits. Exercise electrocardiography showed horizontal ST segment depressions in leads DII, III and AVF at mid-level exercise.

Radionuclide myocardial perfusion scintigraphy also demonstrated reversible inferior wall hypo-perfusion and hypokinesia. Coronary angiography and selective celiac and mesenteric arteriography were performed for the differential diagnosis of symptoms. Coronary angiography displayed critical stenosis visually about 80 to 85% at the proximal level of well-developed obtuse margin branch of circumflex artery **(Figure 1)**.

In celiac arteriography, there were an external compression on celiac artery causing critical stenosis visually about 80 to 85% and post-stenotic dilatation of the artery **(Figure 2)**. Computed tomography angiography of abdominal arteries and reconstruction of data confirmed overhead external compression of celiac artery causing critical stenosis and post-stenotic dilatation of the artery **(Figure 3)**. The case was discussed in the council of the cardiology and cardiovascular surgery of the institution. We decided to treat coronary artery disease as first intervention with the stenting of the ob-

Figure 1. Selective coronary angiography

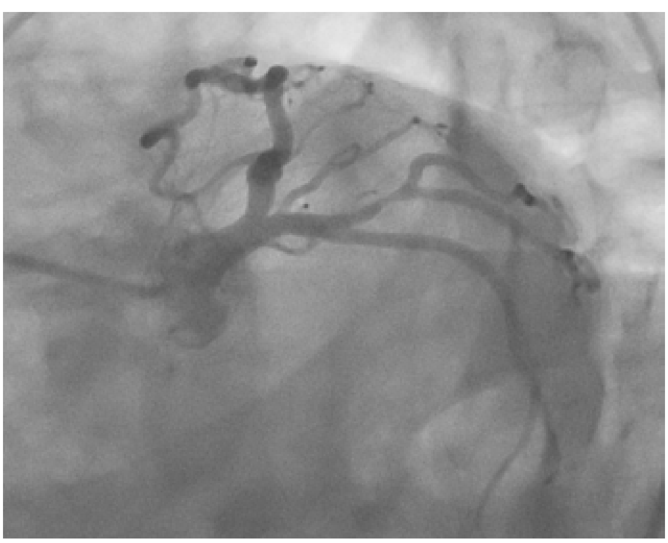
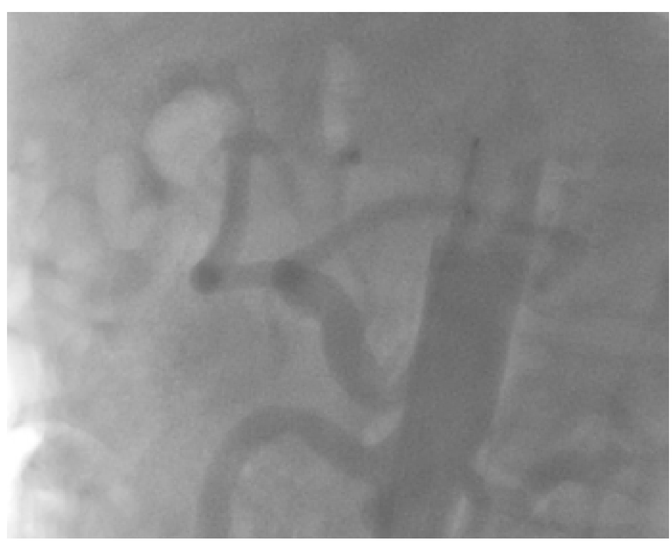


Figure 2. Celiac angiography



tuse margin branch lesion of circumflex artery and to observe the patient at least one month for the symptoms relief. After successful stenting procedure **(Figure 4)**, the symptoms of the patient quickly relieved, and he was discharged without symptoms. At first month visit, the patient was well condition and there were no his previous complaints.

Discussion

Symptomatic CACS is a very rare disease, which is clinically determined by the triad of postprandial abdominal pain, weight loss and an abdominal murmur because of the compression of the celiac artery.[3] Fibrous median arcuate ligament usually bestrides the celiac artery at the level of the first lumbar vertebra in 10% to 24% of the general population but this entity causes symptoms a few of these patients.[4]

There are a lot of reports on this syndrome in literature but its association with coronary artery disease has not been established to date. In the presented case, we took in consideration both CAD and CACS for the source and cause of complaints. Investigative work up was performed in two directions. Celiac arteriography added to coronary angiographic study revealed both critical coronary artery stenosis and celiac artery compression.

The case was discussed in details in institutional council of cardiology and cardiovascular surgery. Because CACS is frequently encountered but rarely causes symptoms, we decided to treat CAD at first. After successfully performed coronary intervention, patient's complaints and symptoms were disappeared.

CACS could be investigated with Doppler ultrasonography, computed tomography angiography, conventional angiography and magnetic resonance angiography. The traditional method for the treatment of CACS syndrome is open surgical intervention. Recent reports suggest that the laparoscopic surgery is best approach in the treatment of CACS.[5] Laparoscopic MAL release has some advantageous such as decreased postoperative pain, lower incidence of postoperative adhesions, shorter recovery time and hospital stay period.[5] Nowadays, this method has become standard surgical approach for the treatment of CACS.[2]

In addition, complex reconstruction procedures such as patch angioplasty, aorta-celiac bypass, reanastomosis of celiac artery to aorta have been performed when needed.[2] In patients having persistent symptoms and residual critical celiac artery stenosis after laparoscopic MAL release, percutaneous endo-

Figure 3. Computed tomography angiography of ab dominal arteries and reconstruction of data

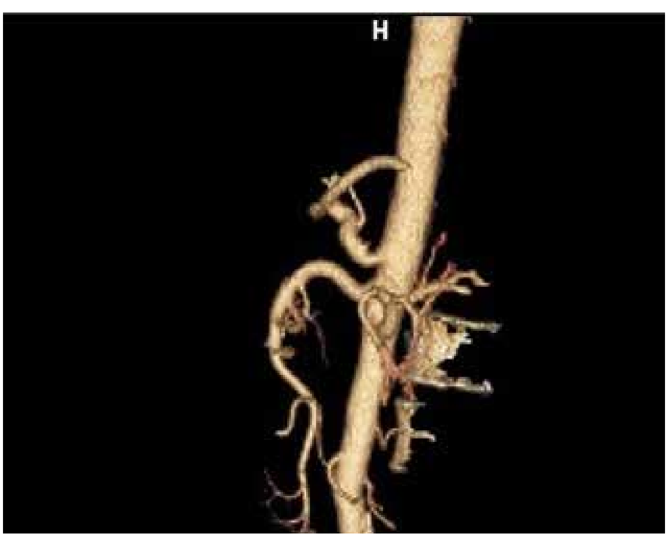
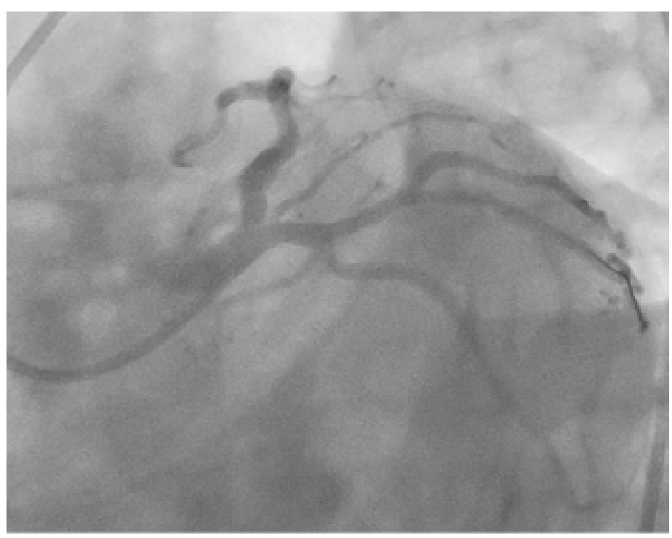


Figure 4. Stenting of obtuse marginal branch of cir cumflex artery



vascular intervention and stenting of persistent critical celiac artery stenosis may be performed.[6] CAD may mimic the symptoms of CACS especially such as associated with the exercise. Investigational work up should include both diseases.

Conclusion

Because symptomatic CACS is very rare entity, CAD should be taken into account. Diagnostic work up should include CAD. It may be a reasonable approach to treat coronary artery disease primarily in terms of clinical and prognostic aspects.

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Received: 16/05/2017
Accepted: 21/10/2017
Published: 15/12/2017

Disclosure and conflicts of interest:
Conflicts of interest were not reported.

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EJCM 2017; 05 (4): 91-94 **Doi:** 10.15511/ejcm.17.00491

It's not fair to compare transcatheter aortic valve implantation with surgical replacement

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Abstract

Surgical aortic valve replacement (SAVR) is still class I recommendation for the treatment of patients with symptomatic severe aortic valve stenosis(AS) according to recent guidelines. Although it assumed as one of the most succesful intervention of cardiac surgery; currently the transcatheter technique (transcatheter aortic valve replacement-TAVR) has came into notice as a feasible and effective option to treat high risk or inoperable aortic stenosis patients. New reports have been publishing comparison of these two techniques by numbers increasing with this technology1. On the stage, we would like to discuss about fairness of the comparison mentality in terms of a feature-by-feature style.

Key words: Fairness, TAV, SAVR, proscons

Adademir T., Çekmecelioğlu D., Çevirme D., Kalender M., Boyacıoğlu K., Büyükbayrak F., Alp H. M. It's not fair to compare transcatheter aortic valve implantation with surgical replacement EJCM 2017; 05 (4): 91-94. Doi: 10.15511/ejcm.17.00491.

As we all know, gold standart therapy should be surgical replacement for the patients with severe calcific aortic stenosis echocardiographically, either symptomatic or asymptomatic. Conventional SAVR yields excellent results after 1 year in lower-risk patients. Despite that, TAVR is also recommended as class I indication on recent guidelines for patients who inoperabl or have prohibitive high surgical risk with last decision given to Heart Valve Team.[2]

Nonetheless, constitutional complications of TAVR have been surfacing and limiting its use such as embolization of calcified debris and consequent cerebral infarction; complete AV block and need of pacemaker insertion; paravalvular leakage and its effect on long-term survival; coronary ostium closure and even aortic rupture.[3,4] Up to 42.5% complete AV block, 15% moderate-to-severe paravalvular leak, and 84% new cerebral infarction have been reported after TAVR.[5]

Despite higher complication rates in terms of major adverse cardiac and cerebrovascular events (MACCE) among TAVR patients; early studies those compared SAVR and TAVR revealed that TAVR is not inferior to surgical AVR in mortality rates. And even more recent trials claims superior mortality results with TAVR. Randomised and observational evidence adjusted on the baseline patients' characteristics finds a similar risk for 30 days mortality, 1-year mortality, stroke, myocardial infarct (MI) and acute renal injury in TAVR and SAVR.[6] Also Vernat et al[7] reported that mitral annular geometry is preserved better by TAVR than by SAVR. They concluded that TAVR might be more physiologic approach for aortic replacement. Kocaaslan et al[8] found that the increase in terms of quality of life parameters in the TAVR group was greater than the AVR group at the end of the 3rd month. Probably, the best attribution to TAVR is being non-invasive method that no need for cardiopulmonary bypass (CBP) during the procedure.

On the other hand, patients with high surgical risk did not have any significant difference for the first year mortality according to two large registries (Partner and GARY = German Registy). But reports showed a two-fold increased risk of cerebrovascular accident in the TAVR group. The PARTNER A and B trials showed that survival has been remarkably good, but stroke and perivalvular leakage require further device development.[9] These findings are far from being gold standard. However, progressing technology will continue to seek new answers to these problems. So, can not surgeons replace the aortic valve surgically which is gold standard with low mortality rates, even for high risk patients?

Fairness always needs feature-by-feature comparison. High technology should be compared with high technology. By using higher technology valves, for example suturless valves, we can speed up the surgery. By reducing cross clamp times, we would decrease operative mortality and morbidity. By using high technology, we can perform aortic valve surgery through minimal approaches via mini (CBP) circuits which would decrease morbidity of the pathology. Mini CBP circuits and cannulas may also decrease mortality rates by reducing inflammatory response which may yield sepsis condition.

Sutureless AVR exploitation self-expanding bioprosthesis is a new and favourable alternative to conventional surgical AVR in elderly and high-risk surgical patients. The advanced benefits of this technology involve improved implantability, shorter aortic cross-clamp and cardiopulmonary bypass (CPB) times, promised hemodynamic presentation and easier approach for minimally invasive surgery.[10]

Does faster replacement of the valve make a real significant effect especially in recent days which myocardial protection methods are well developed? There are several publications seeking answers to this question. Ranucci et al[11] showed that the aortic cross clamping time was an independent predictor of severe cardiovascular morbidity, with an increased risk of 1.4% per 1 min increase. Patients with a left ventricular ejection fraction < or = 40% showed the most relevant clinical benefits induced by reduction in cross clamp time.

Additionly, this technology admits entire removal of the diseased native valve and also consists a appropriate alternative to multiple valve procedures or associated coronary artery bypass grafting. Several European case series have demonstrate excellent early clinical and hemodynamic results.[12]

We can observe zero in hospital mortality and significantly lower paravalvular leak in high risk patients whom hospital mortality is 5.3% for TAVR group by adding only one high technology to surgical arm which is suturless valve. Comparative reports between sutureless valves and TAVR in intermediate and high risk patients have shown lower rate of perioperative complications in terms of MACCE and enhanced survival at 24-month follow-up with sutureless valves.[3,13] We can easily assume that the results would be even better if we can add mini CBP techniques into these technology. Consequently, sutureless aortic bioprostheses has been situated as an alternative to conventional surgical AVR or TAVR in elderly and high-risk patients. However, the need of multi-centered, randomized trials can not be ignored.

Cardiovascular surgeons involved in the treatment of valve diseases should be open to the introduction of innovative methods, technologies or hypothesis. Also he/she should not get carried off and should always examine the outcomes in the light of well-conducted short and long term clinical trials. Apperently that we must utilize the science strictly to keep patients' safety.[14]

TAVR is growing as a minimally invasive therapy for patients with severe AS. Today, it is completely acceptable that TAVR is already preferable in an increasing proportion of elderly and high risk patients; nevertheless, this procedure has potential for serious complications. The recent evidence and outcomes has showed that its feasibility and efficacy. They drawn the attention of industry and physicians to this new technology. There is little evidence on the long-term outcomes in spite of short-term efficacy is good. We aware that AVR treats the disease quite constantly and it is still considered the standard treatment for most of patients. The future is expected to be more wealthy as new developments and statistics got from continued trials. They will also provide the background to expand physicians' decision area and their applications. Lastly, Bavaria[15] et all found that not only were surgeons actively involved in the treatment decision-making process, but also played a significant role in the valve procedure, including deployment and post-procedural care of TAVR patients according the Society of Thoracic Surgerons(STS) survey. That is why cardiac surgeons need to have real and fair statement about TAVR.

Choice of SAVR, TAVR or suturless AVR and indications would be concisely established by fairness with a broader spectrum of patients.

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Received: 30/07/2017
Accepted: 09/11/2017
Published: 15/12/2017

Disclosure and conflicts of interest:
The authors declare no conflict of interest.

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EJCM 2017; 05 (4): 95-98 Doi: *10.15511/ejcm.17.00495*

A Quick Look at Ventricular Septal Defect Classification

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Introduction

I have read the well constructed review manuscript from the Authors Zeynep Eyileten, Adnan Uysalel with the title 'Isolated ventricular septal defect in infants ' published EJCM 2017;5(2):27-33 with great pleasure. There are two main classifications desribed by Richard Van Praagh and by Robert Anderson. I want to give information about the main differences between the classifications of these two cardiac morphologs.

Key words: Ventricular Septal Defect, infants, classifications of cardiac morphologs.

Defects between the ventricles are the commonest congenital cardiac malformations. As yet, however, there is no consensus as to how they can best be described and categorized. Although most of the cardiac structure have been extensively addressed, significant gaps continue to exist between the descriptions provided by morphologists and by those working in the clinical setting such as the cardiologists and cardiac surgeons.

Although there are several definitions depending on the localisation and the diamention of the VSD's the two modern anatomical descriptions were made by **Richard Van Praagh** and **Robert Anderson**.

Van Praagh classify the ventricular septal defects as:

- **Atrioventricular Canal**
- **Muscular**
- **Membranous**
- **Conoventricular**
- **Conal types.**

In **atrioventricular canal type** the VSD is located in the atrioventricular canal portion of the interventricular septum,under the tricuspid valve,and confined by the tricuspid annulus.This can ocur with or without a Common AV Canal.

Kutsal A. A Quick Look at Ventricular Septal Defect Classification. EJCM 2017; 05 (4): 95-98. Doi: 10.15511/ejcm.17.00495.

Muscular VSDs are localised within the anterior, mid-ventricular, posterior or apical portion of the ventricular septum.

Membranous defectes are usually small defects localised at the membranous septum.

Conoventricular VSD occurs due to hypoplastic or malaligned conal septumand is bordered by conal septum and the septal band. If thre is membranous septal involvement, it is descibed as paramembranous or juxtamembranous. According to Van Praagh as 'peri-'means around and the defect doesn't surround the membranous septum the name perimembranous is incorrect.

There are 4 subtypes:

- **Hypoplastic conal septum:** The conal septum is hypoplastic,r esulting in a large subaortic VSD, localized between the conal septum's inferior rim and the normally located septal band.
- **Hypoplastic and Anterosuperiorly Malaligned Conoventricular VSD:** The conal septum is hypoplastic and displaced at an anterosuperior direction leading to the hypoplasia of the pulmonary outflow tract (e.g.TOF)
- **Hypoplastic and Posteroinferiorly Malaligned Conoventricular VSD:** The conal septum is hypoplastic and displaced posteroinferiorly creating subaortic stenosis (e.g. IAA with subaortic stenosis)
- **Right Laterally Malaligned Conoventricular VSD:** The conal septum is displaced to the right (e.g. DORV Tauusig-Bing Type).

Conal VSD is located within the conal septum. Aortic and pulmonary valves are at the superior aspect of the defect. The remainder of the ventricular septal defect rim is bordered by the conal septal muscle.

Anderson classify the ventricular septal defecets as:

- **Perimembranous**
- **Muscular**
- **Doubly Committed Juxtaarterial**

Perimembranous defects bordered by the area of continuity between one or both AV valves and the arterial valve

There are 4 subtypes:

- **Inlet:** The defect mainly opens into the right ventricular inlet, seperating it from the left ventricular outflow tract.
- **Outlet:** The defect opens mainly into the right ventricularoutlet.
- **Central (Previously termed as Confluent):** The defect is so large that the left ventricular shunting extends to all parts of the right ventricle.
- **With overriding Tricuspid Valve:** The defect is due to malalignment of the atrial and ventricular septums, such taht the tricuspid valve overrides the interventricular septum.

Muscular ventricular defects are completely surrounded by muscular tissue.

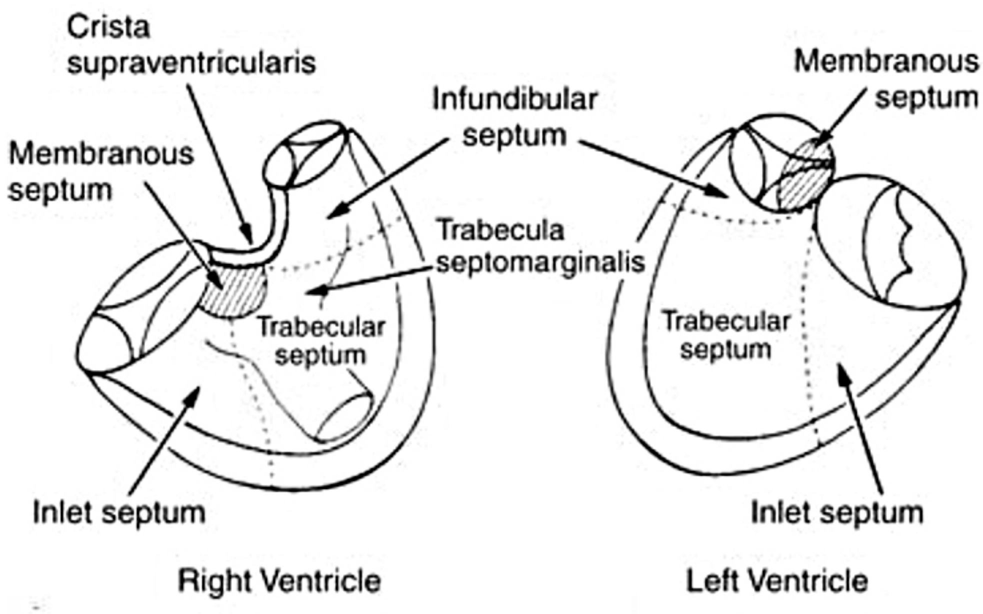
There are 3 subtypes:

- **Inlet:** The defect mainly opens into the right ventricular inlet.
- **Outlet:** The defect mainly opens into the right ventricular outlet.
- **Apical Trabecular:** The defect mainly opens into the right ventricular trabeculum.

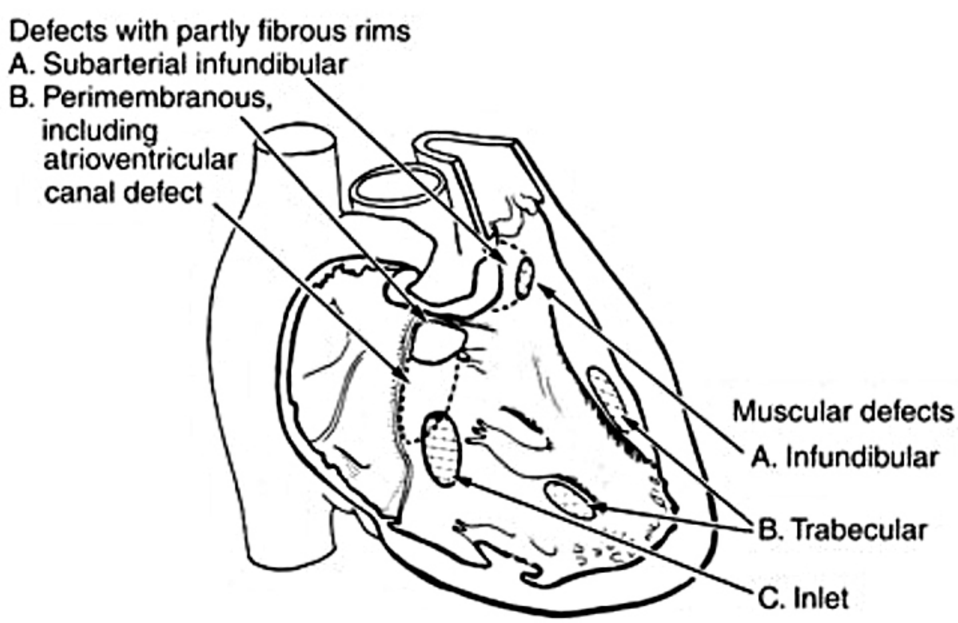
Doubly Committed Juxtaarterial defect is bordered by both arterial valves,and there is fibrous continuity of the leaflets of each of the arterial valves.

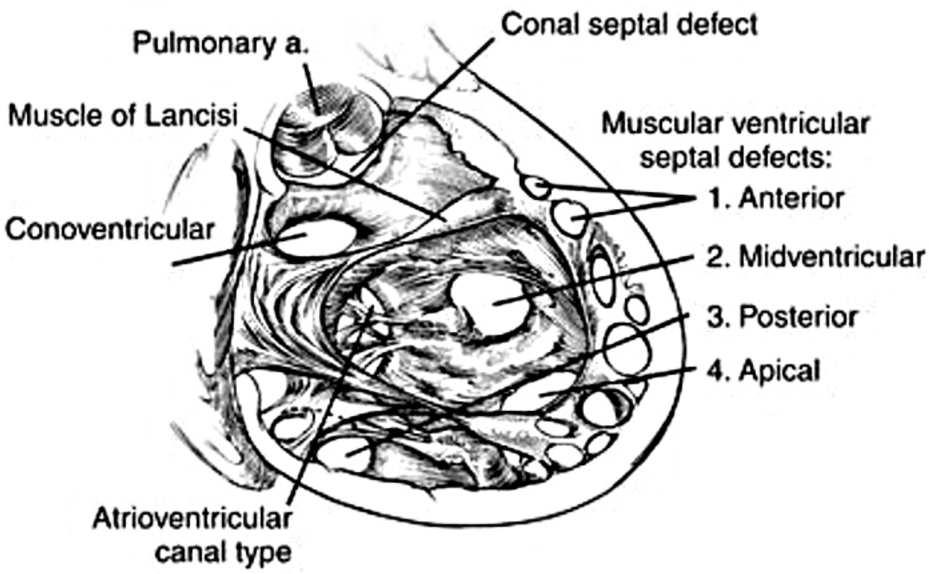
There are 2 subtypes:

- **With a Muscular Posterior-Inferior rim:** Extendes to the muscular septum,which seperates the aortic valve from the tricuspid valve.
- **With Perimembranous Extension:** Extends to the membranous septum,so that there is no seperation between the aortic and tricuspid valves.



The anatomy of the ventricular septum from the right and the left ventricular aspect.





The anatomic localisation of the ventricular septal defects.

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Received: 12/08/2017
Accepted: 28/11/2017
Published: 15/12/2017

Disclosure and conflicts of interest:
The authors declare no conflict of interest.

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Manuscripts should be prepared using a word-processing package (save in .doc, .docx or .rtf format). The font type and font size should preferably be Arial or Times New Roman 11 points. The manuscript should be double-spaced and should include line and page numbers. The lines of the reference list do not need to be numbered; include a section break before.

Manuscripts should be organized as follows:

(a) Title page; (b) Abstract and Key words; (c) Text with the following sections (not applicable for article types with unstructured abstracts): Introduction, Materials and Methods, Results, Discussion, Acknowledgement (optional), Funding statement, Conflict of interest statement, (d) Figure (and Video) legends; (e) Tables; (f) References.

Title page (1st page): Title: should be brief and descriptive (100 characters) - no abbreviations are allowed, even if well known.

Authors: list all authors by full first name, initial of or full middle name and family name. Qualifications are not required. Ensure the author names correspond (in spelling and order of appearance) with the metadata of the system

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Abstract (2nd page): An abstract should be a concise summary of the manuscript. Reference citations are not allowed. The abstract should be factual and free of abbreviations, except for SI units of measurement.

Keywords: Following the abstract, 3-6 keywords should be given for subject indexing.

Introduction: Should state the purpose of the investigation and give a short review of pertinent literature.

Materials and methods: Should be described in detail with appropriate information about patients or experimental animals. Use of abbreviations renders the text difficult to read; abbreviations should be limited to SI units of measurement and to those most commonly used, e.g. VSD, ASD, CABG (abbreviations should not be included in headings and extensions should be included at first mention).

Results: Results should be reported concisely and regarded as an important part of the manuscript. They should be presented either in tables and figures, and briefly commented on in the text, or in the text alone. Repetition of results should be avoided!

Discussion: The discussion is an interpretation of the results and their significance with reference to pertinent work by other authors. It should be clear and concise.

Acknowledgement: Acknowledgements and details of non-financial support must be included at the end of the text before the references. Personal acknowledgements should precede those of institutions or agencies.

Tables: All tables must be included in the manuscript file, should start on separate pages and be accompanied by a title, and footnotes where necessary. The tables should be numbered consecutively using Arabic numerals. Units in which results are expressed should be given in parentheses at the top of each column and not repeated in each line of the table.

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GENERAL RULES

Files should be prepared as a Word document using font size 12 Times New Roman characters, double-spaced and with 2.5 cm margins on each side, top and bottom. Only standard abbreviations should be used; other shortened phrases should be indicated in parentheses as used in the text. Generic or chemical names of drugs should be used instead of trade names.

ETHICAL ISSUES

Publishing responsibilities of authors and Ethics

The publication of an article in a peer-reviewed journal is an essential building block in the development of a coherent and respected network of knowledge. It is a direct reflection of the quality of work of the author and the institutions that support them. Peer-reviewed articles support and embody the scientific method. It is therefore important to agree upon standards of expected ethical behavior.

Reporting standards

Authors of reports of original research should present an accurate account of the work performed as well as an objective discussion of its significance. Underlying data should be represented accurately in the paper. A paper should contain sufficient detail and references to permit others to replicate the work. Fraudulent or knowingly inaccurate statements constitute unethical behavior and are unacceptable. Review and professional publication articles should also be accurate and objective, and editorial 'opinion' works should be identified as such.

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If the work involves chemicals, procedures or equipment that have any unusual hazards inherent in their use, the author must clearly identify these in the manuscript. If the work involves the use of animal or human subjects, the author should ensure that the manuscript contains a statement that all procedures were performed in compliance with relevant laws and institutional guidelines and that the appropriate institutional committee(s) has approved them. Authors should include a statement in the manuscript that informed consent was obtained for experimentation with human subjects. The privacy rights of human subjects must always be observed.

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Acknowledgement of sources

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Authorship should be limited to those who have made a significant contribution to the conception, design, execution, or interpretation of the reported study. All those who have made significant contributions should be listed as co-authors. Where there are others who have participated in certain substantive aspects of the research project, they should be acknowledged or listed as contributors. The corresponding author should ensure that all appropriate co-authors and no inappropriate co-authors are included on the paper, and that all co-authors have seen and approved the final version of the paper and have agreed to its submission for publication.

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This policy concerns the addition, deletion, or rearrangement of author names in the authorship of accepted manuscripts. Before the accepted manuscript is published in an online issue:

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Original Articles

Original articles should consist of sections titled as "Abstract, Introduction, Materials and Methods, Results, Discussion and Conclusion". For information about the abstract, refer to 'Manuscript Formatting' section.

The Introduction section of the manuscript should clearly state the purpose of the manuscript and include a brief summary of the most relevant national and international literature stating the main purposes and research question of the study. Contradictory aspects of the research, if present, should be mentioned. The expected contribution of this study to family medicine and practice should be highlighted.

The Materials and Methods section should describe the study population and the study design, with adequate information on the techniques, materials and methods used. The section should include information of the study type, population, sample, sample size and selection of the sample. Validity and reliability of scales and questionnaires used also should be referred to. A clear description of the statistical methods should also be given.

The Results section should include a detailed report on the findings of the study. All figures, tables and illustrations should be used in this section. Results should be presented either as text or figures and/or tables and not be replicated.

The Discussion section of the study should emphasize the importance of the results and compare them with the results of other authors with relevant citations from the most recent literature. Study limitations and strengths should be specified. Suggestions for further studies in this area should be added.

The Conclusion should include the main conclusions based on the results of the research, emphasize the contributions of the study to family practice and propose original suggestions. A brief revision of all the results and the discussion should be avoided.

Original articles excluding case reports and systematic reviews should not exceed 3000 words excluding the abstract, references and tables. Case reports should not exceed 1000 words excluding the abstract, references and tables. There are no restrictions for systematic reviews.

Short Reports

Short Reports are accepted when the research topic, aim and results of the study are in limited in scope and in cases that do not require writing a full original article. Short Reports can be described as a summarized version that have been prepared according to the structure of research articles. Publishing an article as a short report does not reflect a lower quality. The same rules as relevant to original articles apply to preparing a short report, but structured abstracts are not mandatory references and tables should not exceed 6 and 2 in number, respectively. Abstracts should not exceed 100 words and the text should be restricted to a maximum of 1000 words.

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Reviews are evidence-based articles about a specific topic using relevant citations from the most recent literature with the authors' conclusions on this subject. The author is expected to have conducted research on the subject and to have experience in order to discuss and analyze the subject. There is no obligation to follow a particular format and may contain subtitles depending on the subject. The text should not exceed 4000 words excluding the title, abstracts, references and tables. E Journal of Cardiovascular Medicine, only publishes review articles solicited by the editors.

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Letters to the editor or comments can be sent to provide commentary and analysis concerning an article published in the journal, to give information about ongoing research, to provide information in cardiology and cardiovascular-vascular-endovascular surgery, cardio-metabolic and vascular sciences. Letters to the editor or comments may include an optional title, tables and references. These articles should not exceed 1000 words.

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Title page

Abstract

Main text

References

Tables, figures and illustrations

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Abstract

Abstracts hould be exact in English, with a minimum of 150 and maximum of 350 words. Abstracts of original research articles should be structured under subheadings as follows: objectives, methods, results and conclusion. A maximum of 3 key words should be added to English abstracts.

Text

The text contains the rest of the manuscript. It is structured differently according to the type of manuscript (original research article, review, etc.). For example, original research articles should consist of aim and objectives, methods, results, discussion and conclusion.

References

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Tables and Figures

Legends should take place on the top of the page for tables, and bottom of the page for figures and placed on separate pages. Explain all nonstandard abbreviations in footnotes.

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