E Journal of Cardiovascular Medicine

Volume 4 | Issue 2

April-June 2016

www.ejcvsmed.org

E Journal of Cardiovascular Medicine

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Publisher

Medikal Akademi Yayıncılık ve Prodüksiyon Tic. Ltd. Sti.
Halaskargazi Cad. No: 172, D: 134 - Şişli / İstanbul
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EJCM 2016; 04 (2): 27-32

Doi: 10.15511/ejcm.16.00227

Hypertrophic obstructive cardiomyopathy and the cost of treatment

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Abstract

Objective: To evaluate the demographics of patients admitted with Hypertrophic Obstructive Cardio-Myopathy (HOCM) and the financial burden of this disease on the health care system.

Methods: The Healthcare Cost and Utilization Project (HCUP), sponsored by The Agency for Healthcare Research and Quality's (AHRQ), includes the largest collection of longitudinal hospital care data in the United States of America. HCUP creates the National In-patient Sample data (NIS) to help conduct national and regional analyses of inpatient care. Using the NIS data (2013), we performed a retrospective cohort study that involved patients who were admitted and treated for HOCM.

Results: A total of 2605 patients were admitted for the principal diagnosis of HOCM in 2013.Mean hospitalization was 4.9 days. In our total population, 33% of the patients were above 64 years of age. Mean cost of admission was 25,433$. Private insurance and Medicare or Medicaid paid for 43% and 47% admissions respectively. 76%, 3.5%, 4.6% and 13% patients were discharged to routinely home, another short term hospital, nursing home and for home health care, respectively.

Conclusions: HOCM admissions are relatively uncommon but effects all ages. Most of these patients were treated at a private hospital, and the hospital costs were very high. Large number of patients required rehabilitation services after discharge which increase financial burden on health care system.

Keywords: Healthcare research, hypertrophic obstructive cardiomyopathy, nursing and rehabilitation, healthcare cost

Acknowledgements: *The authors acknowledge all of the HCUP Data Partners that contribute to HCUP. List of these State organizations can be accessed at (www.hcup-us.ahrq.gov/hcupdatapartners.jsp).*

Jan A, Anwar Shah M., Rehman S., Rungatscher A., Ahmed N, Faggian G. Hypertrophic obstructive cardiomyopathy and the cost of treatment. EJCM 2016; 04 (2): 27-32. Doi: 10.15511/ejcm.16.00227.

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Introduction

Hypertrophic cardiomyopathy (HCM) is a complex but relatively common form of genetic heart muscle disease and has been under investigation since more then last 50 years.[1-4] Prevalence of the disorder in the general population is estimated to be 0.2%5. It is often identified by clinicians later on in the disease course. A subset of patients with HCM has hypertrophic obstructive cardiomyopathy (HOCM), in which systolic septal bulging into the LVOT, malposition of the anterior papillary muscle, with enlarged posterior mitral leaflet and hyperdynamic LV contraction and drag forces, through a Venturi effect, provoke systolic anterior motion of the anterior leaflet of the mitral valve (SAM), contributing to the creation of the LVOT gradient.[6] HOCM is the most common cause of heart-related sudden death in people under 30 years of age 7, and it can also be responsible for exercise intolerance at almost any age. HOCM occurs in both genders with female dominance and has been reported in many races.[8] Although HOCM is a chronic disease without a known cure, a number of treatments options are now available to alter its course.

In 2014, U.S. health care spending increased 5.3 percent to reach $3.0 trillion, or $9,523 per person. The share of the economy devoted to health care spending has been rising to 17.3 percent in 2013. Further spending for hospital care increased by 3.5 percent in 2013. Spending growth for freestanding home health care agencies accelerated in 2014, increasing to $83.2 billion.Similarly total private health insurance expenditures increased 4.4 percent (33 percent of total health care spending) to $991.0 billion in 2014, faster than the 1.6 percent growth in 2013 along with nursing home and rehabilitation expenses.

In this age of exponentially rising costs of health care, we wanted to assess the management cost of patients admitted to hospitals with the lethal diagnosis of Hypertrophic Obstructive cardiomyopathy(HOCM). We wanted to further look at their demographics and quantify the financial burden of this disease on the individual patient and health care system across USA.

Methods

The Healthcare Cost and Utilization Project (HCUP), sponsored by The Agency for Healthcare Research and Quality's (AHRQ), includes the largest collection of longitudinal hospital care data in the United States of America. HCUP creates the National Inpatient Sample (NIS) to help conduct national and regional analyses of inpatient care. Using the NIS (2013), we performed a retrospective cohort study that involved patients who were admitted and treated for Hypertrophic Obstructive Cardiomyopathy. To identify these patients we used ICD-9-CM principal diagnosis code 425.11. ICD-9-CM stands for the "International Classification of Diseases - 9th revision - Clinical Modification." The "principal diagnosis" is that condition established after study to be chiefly responsible for occasioning the admission of the patient to the hospital for care. The principal diagnosis is always the reason for admission. The unit of analysis for HCUP data is the hospital discharge (i.e., the hospital stay), not a person or patient. This means that a person who is admitted to the hospital multiple times in one year will be counted each time as a separate "discharge" from the hospital.

Using statistical analysis we compared the demographics, geographical distribution and the cost of treating these patients. Unweighted, HCUP contains data from more than 7 million hospital stays each year. Weighted, it estimates more than 36 million hospitalizations nationally taken from more than 4,000 HCUP participating hospitals.

Results

A total of 2605 patients were admitted for the principal diagnosis of HOCM in 2013.Mean Length of hospital stay was 4.9 days. 55% of the patients were women.33% of the patients were above 64 years of age, whereas 18% patients were aged below 45 **(Table 1)**.

Hospital "charges" is the amount the hospital charged for the entire hospital stay. It does not include professional (MD) fees. "Costs" tend to reflect the actual costs of production, while charges represent what the hospital billed for the case. Total charges were converted to costs using cost-to-charge ratios based on hospital accounting reports from the Centers for Medicare and Medicaid Services (CMS).Mean cost of admission was 25,433$ and Median cost of admission 19,422 $. But the mean hospital charge for the admission was

Table 1-1: Outcomes for hypertrophic obstructive cardiomyopathy ICD-9-CM code 425.11

Variables		Total number of discharges		Charges $ (mean)	Costs, $ (mean)	Routine Discharge
All discharges		2,605	100.00%	88,646	25,433	76.39%
Age group	1-17	*	*	82,642	33,230	*
	18-44	470	18.04%	117,034	32,057	89.36%
	45-64	1,165	44.72%	86,340	24,942	78.97%
	65-84	790	30.33%	81,157	22,976	65.19%
	85+	80	3.07%	41,650	8,993	*
Sex	Male	1,170	44.91%	87,848	25,628	82.91%
	Female	1,435	55.09%	89,287	25,276	71.08%
Payer	Medicare	955	36.66%	73,647	19,681	64.40%
	Medicaid	260	9.98%	71,918	18,967	76.92%
	Private insurance	1,120	42.99%	103,823	31,370	82.59%
	Uninsured	125	4.80%	*	*	88.00%
	Other	*	*	61,735	21,848	*
	Missing	*	*	*	*	*
Median income for zipcode	Low	625	23.99%	97,783	27,506	81.60%
	Not low	1,915	73.51%	86,378	24,938	74.41%
	Missing	65	2.50%	64,491	19,387	*
Owner	Government	320	12.28%	68,846	20,961	82.81%
	Private, not-for-profit	2,140	82.15%	91,094	26,608	75.23%
	Private, for-profit	145	5.57%	97,738	18,687	79.31%
Location/ teaching status	Rural	85	3.26%	24,175	9,567	*
	Urban nonteaching	405	15.55%	95,875	23,499	74.07%
	Urban teaching	2,115	81.19%	89,921	26,478	77.54%

Weighted national estimates from HCUP National Inpatient Sample (NIS), 2013. Statistics based on estimates with a relative standard error (standard error / weighted estimate) greater than 0.30 or with standard error = 0 are not reliable. These statistics are suppressed and are designated with an asterisk (*).Significant at p<.05it

88,646 $ and median Hospital charge 58,460$. Highest charges were for the patients with the following factors below 44 years of age, private insurance being the payer, West region of USA and Pacific census division.47% of admissions were paid by either Medicare or Medicaid and 43 % of admissions were paid by private.5% patients were uninsured.82% patients were taken care at private hospitals. 74% patients belonged to a high income area in the country **(Table 1)**.

Bedsize categories are based on hospital beds and are specific to the hospital's location and teaching status. The definitions of small, medium, and large hospitals vary by region . A hospital is considered to be a teaching hospital if the American Hospital Association (AHA) Annual Survey indicates it has an American Medical Association approved residency program, is a member of the Council of Teaching Hospitals (COTH), or has a ratio of full-time equivalent interns and residents to beds of 25 or higher. Teaching hospitals took care of 82% of these admissions **(Figure 1)**.

Discharge status indicates the disposition of the patient at discharge from the hospital, e.g., routine (home), to another short term hospital, to a nursing home, to home health care, or against medical advice

Table 1-2: Outcomes for hypertrophic obstructive cardiomyopathy ICD-9-CM code 425.11

Variables		Total number of discharges		Charges $ (mean)	Costs, $ (mean)	Routine Discharge
Bedsize	Small	245	9.40%	72,282	22,155	73.47%
	Medium	445	17.08%	86,702	24,507	67.42%
	Large	1,915	73.51%	91,305	26,094	78.85%
Region	Northeast	570	21.88%	90,831	22,463	62.28%
	Midwest	*	*	83,300	29,230	*
	South	805	30.90%	74,287	21,079	82.61%
	West	405	15.5%	134,577	31,947	72.84%
Census division	New England	*	*	59,281	21,945	*
	Middle Atlantic	420	16.12%	102,099	22,648	64.29%
	East North Central	*	*	85,936	24,199	*
	West North Central	*	*	80,727	34,139	*
	South Atlantic	430	16.51%	56,105	16,775	83.72%
	East South Central	*	*	*	33,259	*
	West South Central	225	8.64%	74,986	21,184	84.44%
	Mountain	120	4.61%	105,473	27,079	83.33%
	Pacific	285	10.94%	152,040	34,867	68.42%

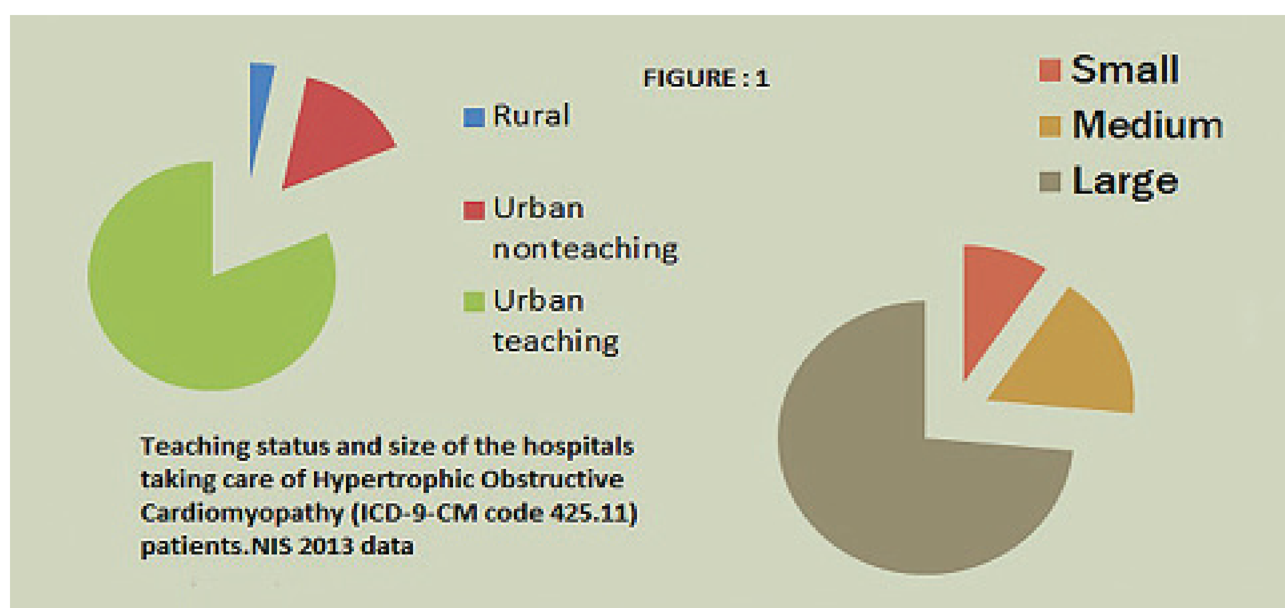
Weighted national estimates from HCUP National Inpatient Sample (NIS), 2013. Statistics based on estimates with a relative standard error (standard error / weighted estimate) greater than 0.30 or with standard error = 0 are not reliable. These statistics are suppressed and are designated with an asterisk (*).Significant at p<.05it

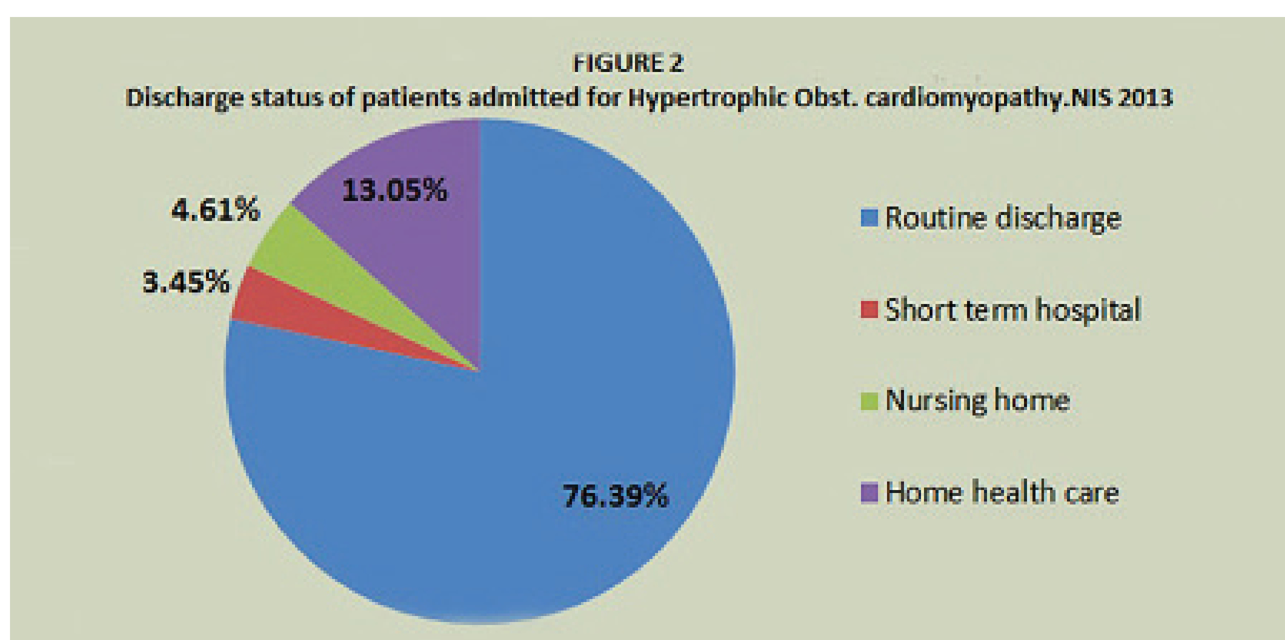
(AMA).Increase in age has a inverse relation with routine discharge i.e from 90% to 65% decline as age goes from 18 to 85 years.**(Table 1)** 20% of the patients require further institutional care after discharge **(Figure 2)**.

Conclusion

Our study presents valuable real-world information regarding HOCM from the largest available inpatient care database. HOCM admissions are relatively uncommon but effects all ages. Its admissions are relatively more common in South of USA and in high income population.

Most of these patients were treated at a private hospital, and the hospital charges were very high. Large number of patients required care after discharge adding to the cost of already very expensive treatment.





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Received: 17/04/2016
Accepted: 11/05/2016
Published: 20/06/2016

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

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EJCM 2016; 04 (2): 33-40
Doi: 10.15511/ejcm.16.00233

Echocardiographic findings among children with pectus excavatum

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Abstract

Objective: Pectus excavatum (PE) is characterized by the posterior displacement of inferior sternum and adjacent cartilages and is the most common congenital chest wall deformity. We aimed to investigate right and left ventricular functions and its correlation with pectus severity index in children.

Methods: Echocardiography was performed in 32 children with PE and 40 healthy controls. The following parameters were monitored: Left and right ventricular (LV, RV) ejection fraction (EF), ejection time (ET), stroke volume (SV), shortening fraction (SF), mitral and tricuspid early (E) and late (A) ventricular filling velocities and deceleration time (DT), median pulmonary arterial pressure (PAPm), aorta and pulmonary acceleration time (Ao-AT, PA-AT), RV work index (RVWI), isovolumetric myocardial acceleration (IVMA), and LV pulmonary ejection period (PEPLV). To assess the severity of pectus, Haller Index (HI) was calculated by thorax computed tomography.

Results: There was no significant difference regarding averages of the body surface area (BSA) between the groups. The arithmetic averages of the EF-Left, ET-Left, ET-Right, SV-Left, TV-DT, PA-AT, and PEPLV were higher in children with PE than in controls, but SF-Left, SF-Right, MV-A, and IVMA were found to be lower. Haller Index (HI) value in children with PE was 2.00-4.93(2.62±0.56). We failed to demonstrate any statistically significant relationship between the cardiac data of children with PE and HI.

Conclusion: Despite the fact that most children had only a mild or moderate form of PE, the RV and LV functions were affected.

Keywords: Children, echocardiography, left ventricular function, pectus excavatum, right ventricular function

Akyüz Özkan E. Echocardiographic findings among children with pectus excavatum. EJCM 2016; 04 (2): 33-40. Doi: 10.15511/ejcm.16.00233

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Introduction

Pectus excavatum (PE), or funnel chest, is one of the most common congenital skeletal deformities. Characterized by an inward depression of the sternum, it is seen in approximately 1 in every 400 male births.[1] Pectus anomalies can be diagnosed at an early age and are three times more common in boys.[2]

The etiology of PE is unknown; it is often seen as isolated anomalies. The primary deformity is dystrophic costal cartilage growth along with the sternum depression. Pectus deformity manifests as subtle abnormalities associated with collagen morphology of children's costal cartilages, but the causal significance of this is not precisely known. Approximately one-third of cases have a positive family history of chest wall deformity.[3]

In addition to cosmetic problems, posterior angulation of the sternum and rib cartilages may cause changes in the rotation and location of the heart and may lead to cardiorespiratory function abnormalities.[4] Some studies demonstrated a significant compromise of cardiac or pulmonary functions[5, 6], whereas other studies showed no change in cardiac functions.[7] Decreased cardiac output, mitral valve prolapse (MVP), and dysrhythmias are considered the primary cardiac effects of PE. Compression of the heart, in particular of the right atrium and ventricle by the chest wall, results in incomplete filling and decreased stroke volume, and eventually in decreased cardiac output.[5, 6] Similarly, compression of the right ventricle (RV) by the chest wall can lead to patient symptoms including dyspnea and chest pain with exertion.[8,9] The chest deformity may also cause compression on the vena cava inferior.[10]

There are conflicting studies about the effects of PE on cardiopulmonary functions. Malek et al.[11] demonstrated that the oxygen pulse and maximum oxygen uptake were low in PE patients. Yalamanchili et al.[12] showed in a case with PE that SV-Rt (RV stroke volume) was reduced. Haller et al.[13] reported that cardiopulmonary functions improved after surgery. Conversely, it is reported that since these patients do not participate in social events and sports, the cardiopulmonary symptoms are related to psychological disorders.[14, 15]

We aimed to investigate right and left ventricular (LV) functions more comprehensively and determine their correlation with pectus severity index in children with PE.

Materials and methods

We studied 32 pediatric patients selected randomly from those with PE but without any other congenital anomaly or disease and 40 healthy subjects. Individuals with pulmonary, renal diseases and a history of diabetes, hypertension, obesity or other systemic diseases were excluded. A full history was taken and a complete physical examination was performed by the same physician. The body height and weight of all children were recorded. Body surface area (BSA) was calculated by Mosteller formula.[16]

At the time of the other tests, an electrocardiogram was recorded for all patients. Transthoracic echocardiography was performed by a single experienced pediatric cardiologist and the following parameters were monitored:

Ejection fraction (EF) was calculated using the standard dimension cubed formula:

EF = (LVDD3 - LVDS3) / LVDD3, where LVDD and LVDS stands for LV dimension in diastole and systole respectively.

SV was calculated as: ***SV = (LVOT / 2)2 × VTIAo × 3.141.*** The LV outflow tract (LVOT) diameter was measured at the base of the aortic leaflet at the parasternal long axis view in echocardiography. Time velocity integral for aortic valve (VTIAo) was obtained with continual wave Doppler immediately below the aortic valve in the apical long axis view.

Aortic Doppler was used to calculate the time intervals—the pre-ejection period (PEP), i.e. the time interval from Q wave of ECG to the onset point of aortic Doppler flow, and the Q-T offset interval, i.e. the time interval from Q wave of ECG to the offset point of aortic Doppler flow.

Mitral and tricuspid early (E) and late (A) filling velocities were recorded from the apical four-chamber view with the pulse-wave Doppler during diastole. E,

A, and deceleration time (DT) were used as both ventricular diastolic function parameters. Ejection time (ET) was calculated from the beginning to the end of the pulmonary and aortic flow.

Isovolumetric myocardial acceleration (IVMA) was calculated by dividing isovolumetric volume (IVV) by the time interval from onset of IVV to the time at peak velocity of this wave.

Mean pulmonary arterial pressure (PAPm) = 0.65 × (PAPs + 0.55);

RV work index (RVWI) = 0.136 × (PAPm - RAP) × SV;

Shortening fraction (SF) = LV end-diastolic - LV end-systolic / LV end-diastolic diameter;

Haller Index (HI) = A / B where A stands for transverse diameter at the deepest level of deformity, and B for anterior-posterior diameter of the same level. An HI <2.5 was considered as mild, HI = 2.5-3.2 as moderate, and HI>3.2 as severe deformity. The A and B diameters of our PE subjects were calculated by thorax computed tomography.

Statistical analyses

The statistical analyses were performed using Statistical Package for the Social Sciences (SPSS). Data were analyzed by independent t test and Analysis of covariance (ANCOVA) test. The arithmetic means of cardiac measurements of children with pectus deformity and of the control group were compared according to independent t test. Important parameters between groups as determined by independent t test were subjected to ANCOVA multiple analyses. Cardiac parameters can be affected by age and the BSA, so these two factors are taken as covariate variables to multi ANCOVA analysis. In ANCOVA analysis, pectus and control group were taken as fixed factors; age and BSA were taken as covariate variables. HI arithmetic average and correlation values of HI and the cardiac parameters were investigated.

Results

32 children (23 male, 9 female) with PE deformity and 40 (17 male, 23 female) healthy controls were included in the study. There was no significant difference regarding average of the age and body surface area (BSA) between the groups.

The arithmetic averages of the EF-Lft, ET-Lft, ET-Rt, SV-Lft, TV-DT, PA-AT and PEPLV were found to be higher in children with PE than in controls **(Table 1)**.

ANCOVA analysis revealed that, when age and BSA were taken as covariate variables and pectus and control group as fixed factors, all parameters mentioned above were found to be statistically significant **(Table 2)**.

The arithmetic average of the SF-Lft, SF-Rt, MV-A and IVMA were found to be lower in children with PE than in controls **(Table 1)**. According to ANCOVA analysis, when age and BSA were taken as covariate variables and pectus and control group as fixed factors, all parameters except for MV-A were found to be statistically significant. These differences in SF-Lft of 48.5%, in SF-Rt of 19.4%, in IVMA of 8.2%, and in EF-Rt of 7.2% can be attributed to pectus disease **(Table 2)**.

HI value among children with PE ranged between 2.00 and 4.93 (2.62±0.56). Of the total of 32 patients with PE, 14 showed mild, 15 moderate, and 3 severe deformities. We found no statistically significant correlation between HI and cardiac parameters among children with PE.

Discussion

Although PE can be viewed as a slight problem, it can lead to much more than a cosmetic deformity. Volume reduction and cardiac chest compression can lead to a reduction in cardiopulmonary function and physical capacity. Symptoms rarely appear in early childhood but increase with age.[17] The chest wall elasticity decreases, stiffness increases, heart deviation to the left decreases, and pressure on the heart increases, with corresponding increase of symptoms with age in PE patients.

RV dysfunction can be seen in patients with PE. Ventricles share a common septum and are within the same pericardial cavity. This relationship between the ventricles also causes similar changes in both systolic and diastolic functions.[18] The compression on the RV

also can cause changes in the size and function of the LV. Cardiac output and SV were demonstrated to be reduced in pectus patients and improved after corrective surgery.[19] Gürsu et al.[20] found lower EF-Lft in the PE group and also revealed that there was an inverse relationship between EF-Lft and SF by HI. EF-Lft and SF-Lft were significantly reduced by increasing HI, but LV end-diastolic volume showed no significant change. Another study[21] demonstrated, using transesophageal echocardiography, that RV end-diastolic size and EF-Lft were increased after the surgery. Bawazir et al.[22] showed that after the pectus corrective surgery, the LV cardiac output and index improved and was maintained thereafter. In their meta-analysis, Malek et al.[23] indicated that the LV function was increased after surgery. Lyons et al.[24] reported the pattern of the RV pressure in

Table 1-1. Echocardiographic findings of children with pectus excavatum compared to the control group

Cardiac parameters	Groups	N	Mean	Std. Deviation	t	p
Age	Pectus	32	11.16	3.15	-1.215	.228
	Control	40	12.08	3.21		
BSA	Pectus	32	1.248	.301	-1.435	.156
	Control	40	1.359	.347		
ET-Lft (msec)	Pectus	32	292.968	21.159	7.275	<.001
	Control	40	245.800	30.898		
ET-Rt (msec)	Pectus	32	317.161	26.303	11.171	<.001
	Control	40	241.100	30.006		
SV-Lft (ml)	Pectus	32	61.398	20.000	2.541	.114
	Control	40	65.989	23.084		
SV-Rt (ml)	Pectus	32	66.545	22.769	.334	.739
	Control	40	64.700	23.655		
SF-Lft (%)	Pectus	32	.222	.074	-8.243	<.001
	Control	40	.342	.049		
SF-Rt (%)	Pectus	32	.289	.092	-4.206	<.001
	Control	40	.387	.101		
MV-A (cm/sec)	Pectus	32	53.000	9.837	-2.000	.049
	Control	40	58.600	13.163		
MV-E (cm/sec)	Pectus	32	103,56	11,528	-.244	.808
	Control	40	104,28	12,926		
MV-DT (msec)	Pectus	32	207,88	54,289	1.306	.196
	Control	40	192,50	45,608		

SD: Standard Deviation. BSA: Body surface area, EF: Ejection fraction, ET: Ejection time, SV: Stroke volume, SF: Shortening fraction, MV: Mitral valve, TV: Tricuspid valve, DT: Deceleration time, PAPm: median pulmonary arterial pressure, PA-AT: Pulmonary artery acceleration time, Ao: aorta, Acc: acceleration, RVWI: Right ventricular work index, IVMA: isovolumetric myocardial acceleration, PEPLV: Left ventricular pulmonary ejection period, Rt: right, Lft: left.

patients with PE. Peterson et al.[25] found significant improvement in RV end-diastolic volume and EF-Rt and also increments in LV end-diastolic volume index and SV index after pectus surgery. Saleh et al.[26] found that both EF-Lft and EF-Rt were lower and RV end-systolic volume was significantly higher in PE patients.

They did not detect any significant correlation between the EF-Rt and EF-Lft and pectus index. They also did not find any differences in EF-Lft, SF-Lft, aorto-pulmonary circulation time, or pulmonary flow indices between PE patients and controls. They suggested that the high end-systolic volume caused reduced EF-Rt. To assess ventricular global functions, EF is the commonly used measurement. Reduced EF generally reflects decreased contractility. Because EF depends on loading

Table 1-2. Echocardiographic findings of children with pectus excavatum compared to the control group

Cardiac parameters	Groups	N	Mean	Std. Deviation	t	p
TV-A (cm/sec)	Pectus	32	47,84	10,913	-1.595	.117
	Control	40	51,48	7,646		
TV-E (cm/sec)	Pectus	32	82,97	15,233	-1.476	.145
	Control	40	87,78	12,413		
TV-DT (msec)	Pectus	32	214.938	59.976	1.993	.050
	Control	40	189.875	46.765		
PAPm (mm/Hg)	Pectus	32	12,996	3,526	.475	.638
	Control	40	12,700	,000		
PA-AT (msec)	Pectus	32	129.513	19.639	3.252	.002
	Control	40	115.600	16.400		
PA-Acc	Pectus	32	8.912	2.072	-1.075	.286
	Control	40	9.473	2.301		
Ao-Acc	Pectus	32	13,289	4,152	-.377	.708
	Control	40	13,616	3,102		
Ao-AT (msec)	Pectus	32	94,067	17,031	1.203	.233
	Control	40	89,425	15,152		
RVWI (gm-m/m2)	Pectus	32	4.181	2.011	1.189	.238
	Control	40	4.721	1.839		
IVMA (cm/msec2)	Pectus	32	.067	.027	-2.374	.020
	Control	40	.083	.029		
PEPLV (msec)	Pectus	32	71.813	12.238	3.195	.002
	Control	40	63.050	11.001		

SD: Standard Deviation. BSA: Body surface area, EF: Ejection fraction, ET: Ejection time, SV: Stroke volume, SF: Shortening fraction, MV: Mitral valve, TV: Tricuspid valve, DT: Deceleration time, PAPm: median pulmonary arterial pressure, PA-AT: Pulmonary artery acceleration time, Ao: aorta, Acc: acceleration, RVWI: Right ventricular work index, IVMA: isovolumetric myocardial acceleration, PEPLV: Left ventricular pulmonary ejection period, Rt: right, Lft: left.

Table 2. The cardiac measurements were analyzed by ANCOVA according to age, BSA vs. groups

Cardiac measurements	Variables	F	Sig.	Partial Eta Squared	R2	Adj. R2
EF-Rt (%)	Age	1.56	.216			
	BSA	.48	.490			
	Groups	5.29	.025	.072	.108	.069
ET-Lft (msec)	Age	4.12	.046	.058		
	BSA	.35	.554			
	Groups	63.06	<.001	.485	.507	.485
ET-Rt (msec)	Age	.249	.620			
	BSA	.222	.629			
	Groups	130.62	<.001	.661	.662	.647
SF-Lft (%)	Age	.16	.694			
	BSA	.03	.867			
	Groups	63.47	<.001	.483	.495	.472
SF-Rt (%)	Age	2.10	.152			
	BSA	.75	.391			
	Groups	16.34	<.001	.194	.233	.199
MV-A (cm/sec)	Age	5.11	.027	.070		
	BSA	4.29	.042	.059		
	Groups	3.84	.054	.054	.121	.082
TV-DT (msec)	Age	2.022	.160			
	BSA	.590	.445			
	Groups	4.527	.037	.062	.093	.053
PA-AT (msec)	Age	1.178	.282			
	BSA	.052	.820			
	Groups	12.229	<.001	.154	.173	.136
PEPLV (msec)	Age	3.525	.065			
	BSA	1.319	.255			
	Groups	11.268	<.001	.142	.182	.145
IVMA (cm/msec2)	Age	1.501	.225			
	BSA	.579	.449			
	Groups	6.005	.017	.082	.101	.061

Groups: *Children with Pectus and normal children.* **BSA:** *Body surface area.* **EF:** *Ejection fraction,* **ET:** *Ejection time,* **SF:** *Shortening fraction,* **MV:** *Mitral valve,* **TV:** *Tricuspid valve,* **DT:** *Deceleration time,* **PA-AT:** *Pulmonary artery acceleration time,* **IVMA:** *isovolumetric myocardial acceleration,* **PEPLV:** *Left ventricular pulmonary ejection period,* **Rt:** *right,* **Lft:** *left.*

conditions, and changes in preload and afterload affect the contractility, EF is not considered an accurate measurement[27, 28] Similarly, regional EF is also load-dependent and does not reveal contractility.[29] IVMA is a measurement of ventricular contractile function that is unaffected by preload and afterload changes in a physiological range. Vogel et al.[30] showed in a pig model that IVMA was less load-dependent than EF.

They demonstrated that IVMA was unchanged with the decrease in the preload and increase in the afterload conditions and therefore can be used to assess RV myocardial function. In the current study, EF-Rt was found to be significantly lower in pectus children than in controls, although EF values were within normal limits and EF-Lft was similar between the two groups. At the same time, IVMA was found to be lower in patients with pectus than in controls, showing that RV contractility is reduced in pectus children. RV functions using quantitative echocardiographic parameters were assessed by Gurkan et al.[31] They reported that the preoperative tricuspid annular plane systolic excursion, SV, and IVMA values were significantly improved in the postoperative period.

We showed that SF-Rt and SF-Lft were lower in pectus children than in controls. A decrease in the SF usually precedes a noticeable decrease in the EF and, similar to EF, is related to ventricular function. Besides RV functions, LV contractility was also affected. E, A, and DT were used for both ventricular diastolic function parameters. Tricuspid valve DT that was found to be prolonged in patients with PE and PAPm was similar in both groups.

Conclusion

We found that PE can lead to major cardiac problems, not limited only to the RV just below the defect; the LV systolic function also can be affected. We failed to demonstrate a relationship between the severity of PE and cardiac function. This possibly was due to the limited number of severe cases in our PE group. Clinicians should pay more attention even in mild PE cases due to the markedly affected cardiac functions in these individuals.

Study limitations: ***In the current study most of the children had a mild and moderate form of the PE. Further studies with larger sample size and a greater number of children with a severe form of PE are warranted to better elucidate the cardiac functions in patients with PE.***

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Received: 27/04/2016
Accepted: 24/05/2016
Published: 20/06/2016

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

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EJCM 2016; 04 (2): 41-44 Doi: 10.15511/ejcm.16.00241

A rare existence of paragangliomas Bilateral carotid body tumors: A case report

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Abstract

Carotid body tumors are rare paragangliomas and generally they have benign characters. Only 3% of all paragangliomas occur in the head and neck region. Surgical procedure of carotid body tumor has a high rate neurological morbidity because of a close comminication with neurovascular formations. We report here a very rare case of glomus tumor threated surgically.

Keywords: Carotid body tumor, bilateral, neurologic complication.

Kaplan S., Avcı T., Songur Ç.M., Ceylan C. A rare existence of paragangliomas; Bilateral carotid body tumors: A case report. EJCM 2016; 04 (2): 41-44. Doi: 10.15511/ejcm.16.00241.

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Introduction

Carotid body tumors are rare paragangliomas and generally they have benign characters. They are originating in the paraganglionic cells of the carotid bifurcation. Their incidences are between 0.06 and 3.33 per 100,000 patient.[1-2] In most cases, paragangliomas are sporadic (75%) and %25 of them are associated with hereditary paraganglioma syndrome.[3] M ultifocal paragangliomas are rarely seen in sporadic cases (%10-20) but they have increasing tendency to be multifocal in familial form (%80).[4-5]

Generally cervical paragangliomas grow slowly, but if they left untreated, they grow rapidly due to rich vascular supply and expand encircling the vascular structure and cranial nerves. Surgery is the only curative treatment in this cases however surgical management of carotid body tumor has a high rate neurovascular morbidity because of a close comminication with neurovascular formations. For the surgical planing and prognostic purposes, Shamblin classification was described for these tumors' size and invasion degree in 1971.[6] We report here a very rare case of glomus tumor treated successfully with surgery.

Case Report

48 years-old male patient refered to us with the complain of bilateral painless huge neck masses. He had also obstructive sleep apne and hypopne syndrom leading to significant cyanosis for 2 years which was concurrent with neck masses. The masses diameters were 10,5x8cm on the left **(Picture 1)** side and 3x3cm on the right side.

Radiologic examination with USG, two and three dimential CT **(Picture 2-3)** and MRI demonstrated glomus tumor in which were class 3 in Shamblin Classification. There was no hormonal activity or neurologic deficit about mass compression.

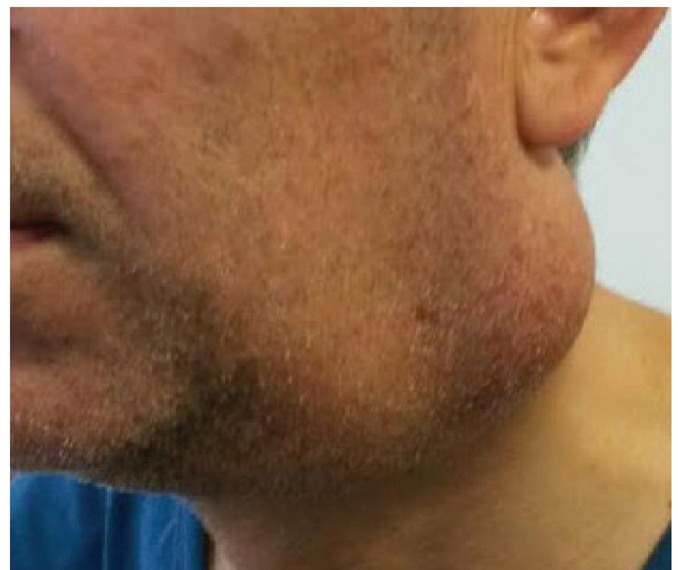
Material and Method

Three days after the embolization procedure he was operated on his left side. A huge tumoral mass which was thightly encircling the releated vessels was excised from all around the internal and external carotid arteries also like hypoglossus, vagus and laryngeus recurrens. There was no residuel mass. Histology confirmed the diagnosis of glomus tumor.

Surgery was uncomplicated. He was moved to service bad on the next day and discharged 10 days after the surgery. A mild apathy and aphasia were occured on second day of the surgery.he was cosultated to the neurology department. With corthicosteroid theraphy, the symptoms were regressed on 4. day and disappeared on 8. day. He was checked on the first month of the surgery. There were no permanent operative cranial nerve injuries and other problems about surgery. His right side surgery will be applied in a few months.

Conclusion

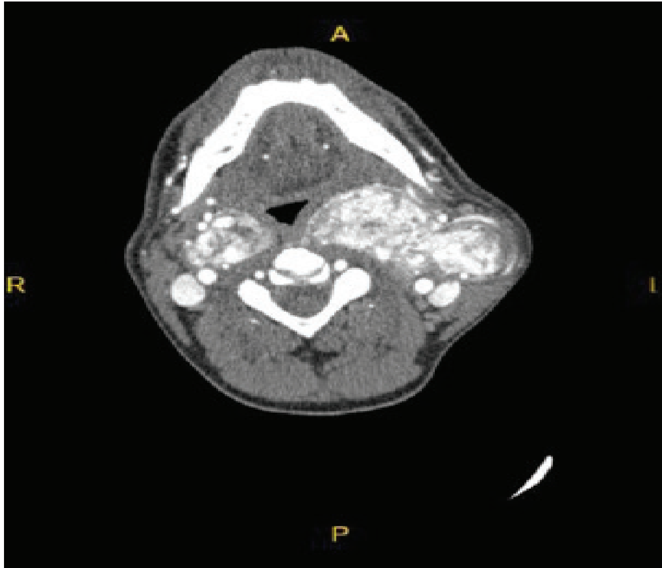
Carotid body tumors are very rare neoplasms and rarely seen in neck resion. In this cases, generally, clinical follow-up is advised untill the compression or hormonal activation signs due to mass were occured. Reported nerve injury rates range from 11% to 50% [7,8, 9,10] and has high risks with higher Shamblin class. [1] Neuronal injuries (transient or permanent) of the vagus nerve, hypoglossal nevre and other communicated nerves and vascular injuries of the carotid artery and it's branches and surrounding veins have been associated with surgical procedure[8], especially in larger tumors with close proximity to critical structures requiring a more complex procedure for removal.



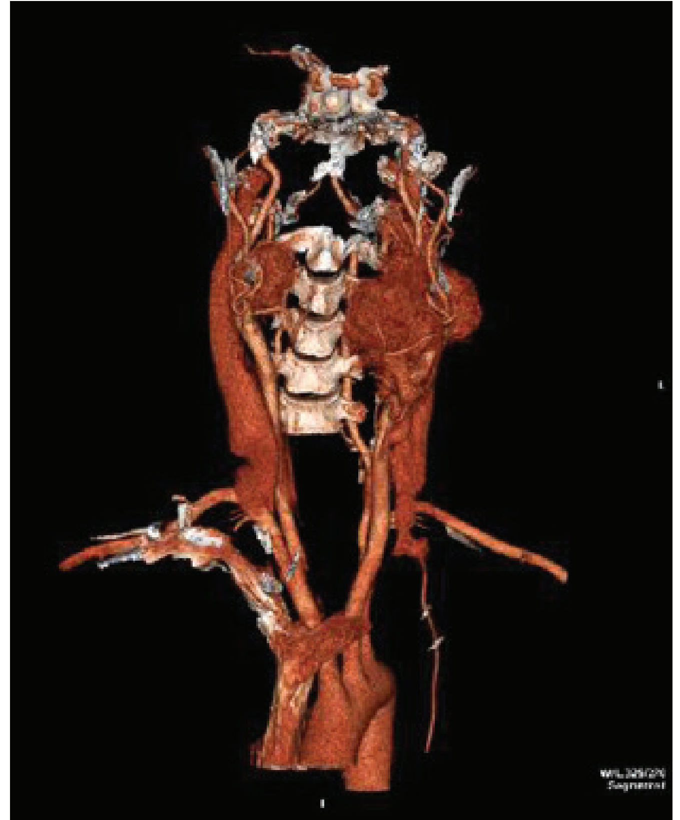
Picture 1

Results

Surgery is the only curative therapy and accepted as a gold standart despite high risks for neurovascular complications.



Picture 2



Picture 3

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Received: 29/03/2016
Accepted: 08/05/2016
Published: 20/06/2016

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

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EJCM 2016; 04 (2): 45-48 Doi: 10.15511/ejcm.16.00245

May the extraction of fresh thrombus prevent no-reflow phenomenon in coronary artery bypass surgery?

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Abstract

The most frequent reason of acute coronary syndrome is the occlusion of coronary artery with thrombus due to the rupture of the plaque in it. Treatment involves medical treatment, percutaneous coronary interventions and coronary artery bypass grafting (CABG). In CABG the revascularization of the coronary artery and, if necessary, the extraction of thrombus may be lifesaving.

Keywords: Thrombus, acute coronary yndrome, coronary artery bypass

***Demirdaş E., Atılgan K., Çiçekçioğlu F. May the extraction of fresh thrombus prevent no-reflow phenomenon in coronary artery bypass surgery? EJCM 2012; 04 (2): 45-48. DOI:* 10.15511/ejcm.16.00245.**

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Introduction

The most frequent reason for the acute coronary syndrome (ACS) is the occlusion of the coronary artery with the thrombus due to the rupture of the plaque in the coronary artery.[1] The cure involves medical treatment, percutaneous coronary inteventions (PCI) and coronary artery bypass grafting (CABG).[2-4] Concisely, medical treatments are anti-ischaemic, anti-thrombotic and fibrinolytic treatments. PCI has a very important position in ACS as a well accepted and spreading process. PCI enables both the stabilization of the ruptured plaque and the resolution of the thrombus in the coronary artery. Revascularization of the totally occluded coronary artery due to the rupture of a plaque and the extraction of the thrombus, during CABG being performed under emergent circumstances, provides us to have pleasing results in morbidity and mortality.

Case History

A 37-year old male without a history of an accompanying illness or drug utilization was referred to our clinic with a severe chest pain. Having the results of inferior ST elevations in the electrocardiogram and troponin I:21 ng/ml in biochemical tests, 600 mg of Clopidogrel per orally and 10000 Units of Enoxaparine subcutaneously were applied to the patient. Three vessel disease and a filling defect in the right coronary artery compatible with thrombus were observed in coronary angiography. The patient with unstabil angina underwent an emergent CABG on the fourth hour of chest pain.

Operating Procedure

The patient was operated under emergent circumstances. Following median sternotomy, saphenous vein grafts and left internal mammarian arteries (LIMA) were harvested. After having cardiac arrest with ategrade and retrograde cold crystalloid cardioplegia, arteriotomy was applied on the distal body of the right coronary artery, just before the bifurcation of the posterior descending artery and the posterolateral artery.

We observed a fresh thrombus and a thrombectomy process was applied with a 2F Fogarty catheter (**Figure 1, 2**). Then, right coronary arter bypass was applied with the saphenous vein graft. LIMA was anastomosed to the left anterior descending artey and the other part of the saphenous vein graft was anastomosed to the optional diagonal and optus marginalis arteries sequatially. After decannulation and the neutralization of heparin with prothrombin, tranexamic acid was infused with a dose of 15 mg/kg. The patient

Figure 1. **Fresh thrombus is seen after coronary incision**

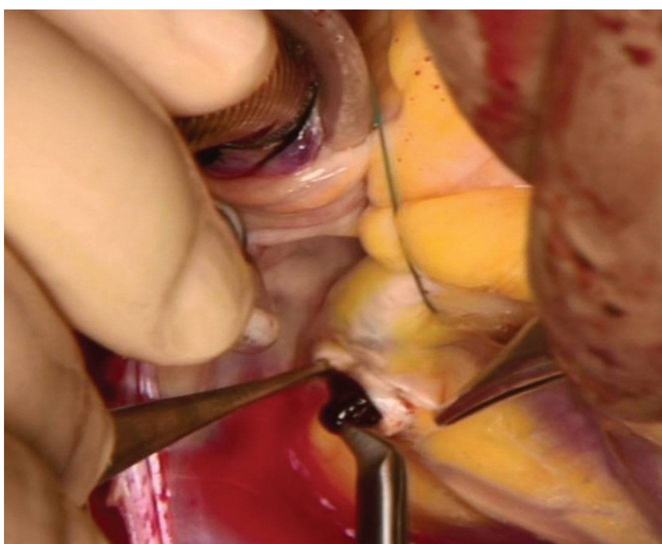
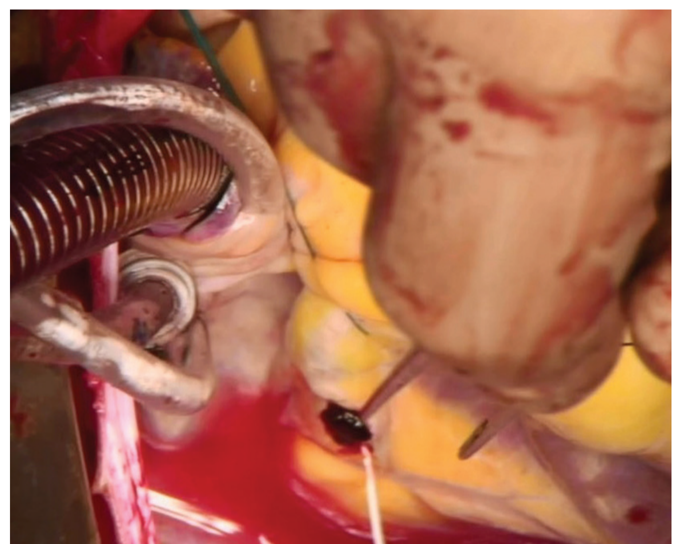


Figure 2. **The embolectomy of fresh thrombus**



was discharged on the postoperative fourth day.

Discussion

Soft plaques are pathologies with a high risk of rupture resulting with the acute occlusion of the coronary artery and, hence, ACS compeling the patient to face serious fatal complications. Thus, ACS has an important place among the corornary artery diseases. Despite the early diagnosis and the provision of TIMI 3 flow after the revascularization with even CABG or PCI, sometimes, it may be possible to observe the persistence of the myocardial ischaemia.

It is not uncommon having unsufficient myocardial response, haemodynamic instability resulting with the initiation of positive inotropic drug and intraaortic ballon pump treatments despite the succesfull coronary artery bypassing and effective heart protecting techniques. This situation is called as “No-relow phenomenon”, which is a conclusion of multiple factors as microvascular spasm, thrombus load, endothelial dysfunction, inflammatory neutrophil activation, interstitial edema and embolization.[5-7]

The distal embolization of the microparticulas of fresh thrombus in the coronary artery having a soft plaque anatomy is the main point of the pathophysiology in this process. The mortality rate has a ten fold increase in the patients having no-reflow phenomenon in comparison with the ones not having no-reflow phenomenon.[8-10] Thus, in this case we prefered to extract the fresh thrombus in the coronary artery.

As a result, we believe that among the patients undergoing emergent CABG due to ACS inflicting because of the acute rupture of soft palque and total occlusion of the artery with fresh thrombus, bypassing the coronary arteries after the extraction of the fresh thrombus would save better mortality and morbidity rates and survey.

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Received: 15/03/2016
Accepted: 22/05/2016
Published: 20/06/2016

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

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