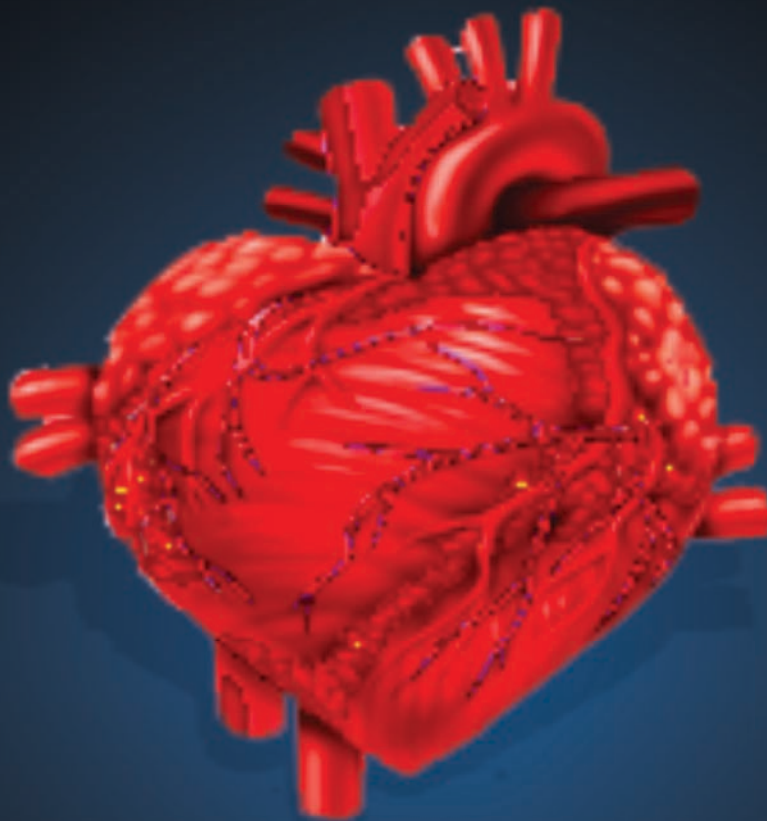


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Predictors of Premature Clopidogrel Discontinuation within 30 days of Successful Coronary Artery Stenting

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Abstract

Objective: We aimed to determine the prevalence, predictors, and mortality rate of premature clopidogrel discontinuation within 30 days of successful coronary stenting. **Methods:** All consecutive patients who underwent successful coronary stent implantation at our hospital between December 2006 and December 2007 were prospectively included in this study. Patients were interviewed by telephone 30 days after stent implantation. Premature clopidogrel discontinuation was defined as follows: patients who did not continue clopidogrel after discharge were defined as “never used” and patients who received clopidogrel for <20 days or interrupted therapy for at least 5 successive days within the first 30 days were defined as “partially used.” **Results:** Follow-up data were available for 381 patients and 58 (15.2%) patients reported premature clopidogrel discontinuation. No mortality and only 1 (0.3%) stent thrombosis occurred in adherent patients, whereas there were 2 (3.4%) mortalities and 6 (10.3%) stent thrombosis in the patients who prematurely discontinued clopidogrel. Those who discontinued clopidogrel therapy were older ($P = 0.02$), more likely to be female ($P = 0.02$), single ($P = 0.03$), of lower economic ($P < 0.05$) and educational status ($P < 0.01$), more likely to have chronic disease ($P = 0.04$), less likely to have undergone previous stenting ($P = 0.01$), and were more likely to be receiving a larger number of drugs ($P < 0.05$). In multivariate analysis, low- or intermediate-economic status, no history of previous stent implantation, and total number of prescribed drugs using were factors independently associated with premature clopidogrel discontinuation. **Conclusion:** This study demonstrates several predictors of premature clopidogrel discontinuation. This data may help clinicians pay particular attention to these patients in an attempt to improve the outcomes of coronary stenting.

Keywords: Clopidogrel, mortality, stent

INTRODUCTION

Despite improvements in coronary stents and interventional techniques, stent thrombosis remains a major problem following percutaneous coronary interventions. Several clinical, anatomic, and procedural factors associated with stent thrombosis have been identified.^[1,2] Premature discontinuation of clopidogrel therapy has been reported to be associated with stent thrombosis, cardiac rehospitalization, cardiovascular death, and all-cause mortality.^[3-5] Although most of the factors associated with stent thrombosis are not modifiable, premature clopidogrel discontinuation may be reduced by preventative measures.

Several previous studies have reported predictors of premature clopidogrel discontinuation.^[6-9] However, recently, Khalili *et al.* reported that the models used to predict nonadherence perform

poorly.^[10] In this study, we aimed to determine the prevalence, predictors, and mortality rates of premature clopidogrel discontinuation following successful coronary stent implantation.

METHODS

All consecutive patients who underwent successful coronary stent implantation at our hospital between December 2006 and December 2007 were prospectively included in this study. Patients who died because of reasons other than stent thrombosis during the same hospitalization, those who could not provide at least one telephone number for contact, and

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those who refused to participate were excluded from this study. Patients were interviewed by telephone 30 days after stent implantation. Detailed data regarding the adherence to the prescribed medications and rehospitalizations were obtained. Definite and probable stent thrombosis was also determined. Premature clopidogrel discontinuation was defined as follows: patients who did not continue clopidogrel after discharge were defined as “never used” and patients who received clopidogrel for <20 days or interrupted therapy by at least 5 successive days within the first 30 days were defined as “partially used.” The study complied with the principles of the Declaration of Helsinki, and the local Ethics Committee approved the study protocol.

Statistical analysis

Continuous variables were presented as mean \pm standard deviation or median (25%–75% percentiles), and categorical variables were expressed as number and percentage (%). The continuous variables were compared across the groups using the Student’s *t*-test or the Mann–Whitney U-test. The Kolmogorov–Smirnov test was used to identify normally distributed variables. The categorical variables were compared using the Chi-square or Fisher’s exact tests. Forward and backward binary logistic regression analysis was performed to determine the independent predictors of premature clopidogrel discontinuation. The results were presented as odds ratios (OR) and 95% confidence intervals (CI). Analysis of event-free survival was performed using the Kaplan–Meier method and the differences compared using the log-rank test. Event-free survival was defined as the time from enrollment in the study to the first event, which included death or stent thrombosis. All the data were analyzed with SPSS v14.0 for Windows (SPSS Inc., Chicago, IL, USA). A $P < 0.05$ was considered to be statistically significant.

RESULTS

We prospectively enrolled 398 consecutive patients in this study. Among these, 13 (3.2%) patients were lost to follow-up. A total of 6 (1.5%) patients died within 30 days and data on clopidogrel maintenance could not be obtained from 4 of these. Therefore, 30-day follow-up data were available for 381 patients. Among the 381 patients, 278 (73%) were men and 103 (27%) were women. Mean age was 57.8 ± 10.6 years. Overall, 58 (15.2%) patients reported premature clopidogrel discontinuation, while 323 (84.8%) reported adherence to clopidogrel therapy [Figure 1]. No patient reported that they prematurely discontinued clopidogrel due to allergy, operation, or bleeding. The baseline characteristics of the patients are summarized in Table 1. Those who discontinued clopidogrel therapy were older ($P = 0.02$), more likely to be female ($P = 0.02$) and single ($P = 0.03$), had lower economic ($P < 0.05$) and educational statuses ($P < 0.01$), were more likely to have chronic disease ($P = 0.04$) and using larger number of drugs ($P < 0.05$), and less likely to have undergone previous stenting ($P = 0.01$). The mortality, stent thrombosis, and nonadherence to other

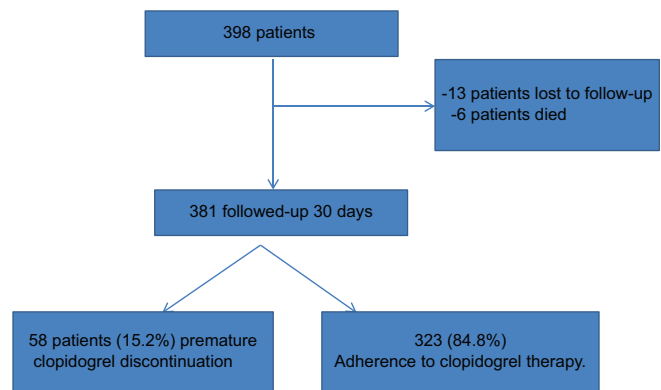


Figure 1: Flowchart of the study

cardiac drugs rates were significantly higher in patients who prematurely discontinued clopidogrel therapy within 30 days [Table 2]. Kaplan–Meier analysis showed that event-free survival within 30 days was higher in patients who adhered to clopidogrel therapy (log rank < 0.001) [Figure 2]. In multivariate analysis, low- and intermediate-economic status (OR: 7.299; 95% CI: 2.642–20.167; $P < 0.05$ and OR: 3.784; 95% CI: 1.352–10.593; $P = 0.0011$, respectively), no history of previous stent implantation (OR: 4.755; 95% CI: 1.339–16.896; $P = 0.016$), and total number of prescribed drugs (OR: 1.940; 95% CI: 1.437–2.619; $P < 0.05$) were factors independently associated with premature clopidogrel discontinuation [Table 3]. On the other hand, age, sex, education status, marital status, living alone, and having chronic disease were not associated with premature clopidogrel discontinuation.

In the subgroup analysis, the patients who had never used clopidogrel were slightly younger than the adherent patients, but the difference was not statistically significant (53.5 ± 10.3 vs. 57.10 ± 10.3 , $P = 0.4$). Conversely, the patients who had partially used clopidogrel were significantly older than the adherent patients (65.9 ± 9.8 vs. 57.10 ± 10.3 , $P < 0.005$).

DISCUSSION

In this study, we found that approximately 1 in 7 patients prematurely discontinued clopidogrel therapy within 30 days of coronary stent implantation. This prevalence is similar to previously reported rates.^[6,11,12] Except for one patient with stent thrombosis, all stent thrombosis and mortalities occurred among patients who prematurely discontinued clopidogrel therapy. A clear association between premature clopidogrel discontinuation and mortality/morbidity was apparent in concordance with previous reports.^[4,13-15] We found that a lower economic status is the strongest predictor of premature clopidogrel discontinuation. The majority of the patients had health insurance. However, patients have to pay a partial contribution toward the costs of their prescribed medications and doctor’s examination in Turkey. Even this minor contribution could have negative effects on economically disadvantaged patients’ ability to start and

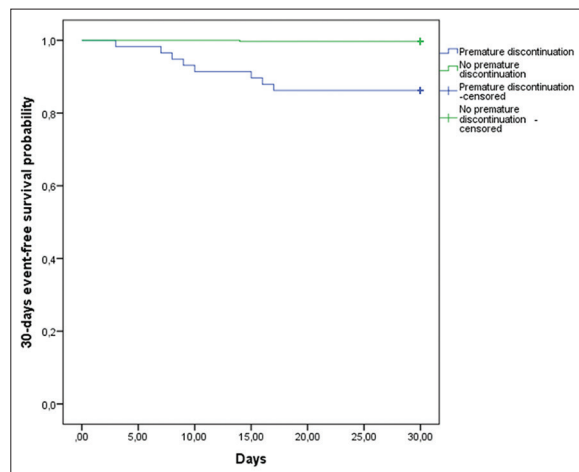
Table 1: Baseline characteristics of the patients

Characteristics	No premature discontinuation (n=323)	Premature discontinuation (n=58)	P
Age (years), mean±SD	57.10±10.3	61.8±11.5	0.02
Sex, male, n (%)	243 (75.2)	35 (60.3)	0.02
Married	266 (82.4)	38 (65.5)	0.03
Lifestyle, living alone, n (%)	26 (8.0)	10 (17.2)	0.28
Education, n (%)			
Illiterate	63 (19.5)	25 (43.1)	<0.05
Complete primary school	181 (56.0)	30 (51.7)	
Complete high school	56 (17.3)	28 (3.4)	
Complete university	23 (7.1)	1 (1.7)	
Economic status			
Low (%)	80 (24.8)	32 (55.2)	<0.05
Intermediate (%)	123 (38.1)	21 (36.2)	
High (%)	120 (37.2)	5 (8.6)	
No health insurance, n (%)	18 (5.6)	4 (6.9)	0.69
Heart failure, n (%)	52 (16.1)	9 (15.5)	0.09
Hypertension, n (%)	260 (80.5)	48 (82.8)	0.88
Diabetes mellitus, n (%)	68 (21.1)	11 (19.0)	0.86
Chronic kidney disease, n (%)	12 (3.7)	3 (5.2)	0.71
Chronic disease, n (%)	42 (13.0)	14 (24.1)	0.04
History of coronary stent, n (%)	56 (17.3)	3 (5.2)	0.01
Admission diagnosis, n (%)			
STEMI	111 (34.4)	21 (36.2)	>0.05
NSTEMI	126 (39.0)	30 (51.7)	
SAP	86 (26.6)	7 (10.1)	
Total number of medicines, mean±SD	5.2±0.9	6.1±1	<0.05

NSTEMI: Non-ST-segment elevation myocardial infarction, SAP: Stable angina pectoris, STEMI: ST-segment elevation myocardial infarction, SD: Standard deviation

Table 2: Stent thrombosis and mortality within 30 days and discontinuation of other cardiac medicines

	No premature discontinuation (n=323)	Premature discontinuation (n=58)	P
Stent thrombosis, n (%) (definite/probable)	1 (0.3)	6 (10.3)	<0.05
Mortality	0	2 (3.4)	0.01
Discontinuation of other cardiac medicines, n (%)	9 (15.5)	5 (1.5)	<0.05

**Figure 2:** Kaplan–Meier curves for 30-days event-free survival for patients with clopidogrel therapy

maintain treatment without interruptions. Moreover, these patients may experience other unpredictable circumstances that

may cause premature clopidogrel discontinuation. Similarly, Khalili *et al.* reported that although clopidogrel was provided at discharge and at nominal cost thereafter, a high rate of nonadherence was observed in a multiethnic, urban, and poor patient population.^[10]

No history of previous coronary stenting was another strong predictor of premature clopidogrel discontinuation. This finding shows that patients who did not receive a prior coronary stent were not aware of the hazards of premature clopidogrel discontinuation. On the other hand, this finding also underscores our failure to properly educate patients on the significance of clopidogrel. A short but comprehensive educational session given to patients and their families or providing a printed educational pamphlet on clopidogrel and other medications before discharge may increase adherence to treatment. Using a large number of medications was another significant predictor of premature clopidogrel discontinuation. According to a report by the World Health Organization, approximately 50% of patients do not adhere to the prescribed

Table 3: Predictors of premature clopidogrel discontinuation (backward logistic regression analysis)

Parameter	OR	95% CI	P
Low economic status	7.299	2.642-20.167	<0.05
Intermediate economic status	3.784	1.352-10.593	0.011
No previous stent	4.755	1.339-16.896	0.016
Total number of medicines	1.940	1.437-2.619	<0.05
Age	1.029	0.998-1.61	0.068

OR: Odds ratio, CI: Confidence interval

long-term therapies for chronic conditions.^[16] In their multicenter study reflecting daily practice in Turkey, Kilic *et al.* had shown that 20.7% of patients with ischemic heart disease do not receive antiplatelet therapy.^[17] Poor medication adherence is multifactorial. Broadly, it may be classified as patient-related, physician-related, and health-care system/team building-related factors.^[18-20] There are still several efforts that can be made to improve patient health literacy, patient-physician communication, and health-care system-related factors.^[21] In a review by Rashid *et al.*, the dominant impact of clinicians on patients' medication adherence was described. The authors claimed that clinicians often unintentionally influence patients' thoughts through their language and lack of accessibility.^[22]

In our study, we did not find the patients' education status to be an independent predictor of clopidogrel discontinuation. This result might be due to confounding effects of poor patient-physician communication and health-care system-related problems. Spertus *et al.* found that not completing high school was the only predictor of premature discontinuation of thienopyridine therapy.^[5] Age was also not an independent predictor of clopidogrel discontinuation in our study. In the subgroup analysis, while the mean age of patients who had never used clopidogrel after discharge was younger than the adherent patients, the difference was not statistically significant. Younger patients may be in denial about their illness, have a negative attitude toward taking medication, and/or may believe that they have been cured by the interventions.

Study limitations

We conducted a single-center study with a relatively few number of patients. Second, we obtained data by a telephone call and by patient's self-reporting on their adherence. The self-reported adherence data might be unreliable. An additional concern is that 3.2% of patients were lost to follow-up at 30 days and we were unable to obtain data on their adherence or other possible adverse events. Therefore, we may have underestimated the true rates of premature clopidogrel discontinuation. Although our study was not conducted recently, we believe that it still provides relevant and current information.

CONCLUSION

Improving patients' adherence to their medications is a significant task and an opportunity to decrease stent thrombosis

and related morbidity and mortality. Further, studies with more patients are necessary to determine the predictors of premature clopidogrel discontinuation.

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Conflicts of interest

There are no conflicts of interest.

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Percutaneous Intervention in Acute Pulmonary Embolism

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Abstract

Pulmonary thromboembolism (PTE) is usually caused by deep vein thrombosis (DVT) in the lower extremities; which can be as varied clinical spectrum as asymptomatic embolism detected incidentally to serious disease with massive embolism causing death. A 44 year-old female patient was admitted to emergency department with complaints of general condition impairment, hypotension and marked dyspnea. She had a fracture on the right femur proximal region after falling a month ago. Lower extremity Doppler ultrasonography revealed findings consistent with acute deep vein thrombosis in the right lower extremity. Emergency pulmonary CT angiography revealed bilateral massive pulmonary thromboembolism extending especially from the main pulmonary artery to the right pulmonary artery. IV thrombolytic was contraindicated as a result of head trauma and subdural hematoma history a month ago. The patient was taken to the catheter laboratory and we performed a selective thrombus aspiration and fragmentation. The vital signs and hemodynamics of the patient improved rapidly after the procedure. This case report is important for demonstrating rapid percutaneous management of a young female patient with a life-threatening condition and favourable outcome of percutaneous intervention despite many comorbid conditions.

Keywords: Percutaneous treatment, pulmonary thromboembolism, thrombectomy

INTRODUCTION

Pulmonary thromboembolism (PTE) is usually caused by deep vein thrombosis (DVT) in the lower extremities, which can be as varied clinical spectrum as asymptomatic embolism detected incidentally to serious disease with massive embolism causing death. Chronic thromboembolic pulmonary hypertension (CTEPH) is the chronic sequelae of the disease. The Virchow triad, which consists of local intimal damage to the vessel wall, hypercoagulability, and blood stasis, causes thrombus formation in the lower extremity, pelvis, or upper extremities' venules may cause embolism in pulmonary arteries. The most common source of pulmonary embolism is deep venules in the lower extremities and iliac veins.^[1,2] In this case, we presented a percutaneous treatment of a patient with massive acute PTE.

CASE REPORT

The patient who was then learned to have the right lower extremity splinted because of a fracture on the right femur proximal region after falling a month ago was admitted to emergency department with complaints of general condition impairment, hypotension, and marked dyspnea. The patient's

initial systolic blood pressure was 65 mmHg, and diastolic pressures could not be measured. On physical examination, radial pulse was palpated as filiform. In cardiac auscultation, S1-S2 was detected as rhythmic and tachycardic (120/min). Respiratory sounds were found bilaterally decreased. The patient's electrocardiogram showed sinus tachycardia and S1-Q3-T3 pattern. Arterial blood gas was detected as pH: 7.50, PCO₂: 25 mmHg, and PO₂: 65 mmHg. D-dimer value was 668 ng/ml and was found to be elevated. Troponin-I level was also elevated (313.2 pg/mL). Lower extremity Doppler ultrasonography revealed findings consistent with acute DVT in the right lower extremity. Emergency pulmonary computed tomography (CT) angiography revealed bilateral massive PTE extending especially from the main pulmonary artery to the right pulmonary artery [Figure 1]. The patient was referred to the chest diseases' consultation and reported that she could not get intravenous (IV) thrombolysis because of subdural hematoma history following the fall 1 month ago. After rapid progression of hemodynamic

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instability, the decision was made to manage the patient percutaneously in the cardiovascular surgery and cardiology council. The patient was taken to the catheter laboratory because of hemodynamic instability with acute massive PTE, and under the local anesthesia, the right femoral vein was cannulated using the Seldinger method. The main pulmonary artery was reached through the vena cava inferior, right atrium, and right ventricle. On pulmonary angiography, massive PTE was observed in the main and right–left pulmonary arteries compatible with CT [Figure 2]. The 0.038” hydrophilic guidewire first passed through the right pulmonary artery, then through the left pulmonary artery. We performed a selective thrombus aspiration from the main, right, and left pulmonary artery with thrombus aspiration catheter. However, because of the intense thrombus load, the thrombus was fragmented with the Cleaner rotational thrombectomy system on the launcher 8F guiding catheter, followed by selective thrombus aspiration to the right and left PAs and branches [Figure 3a & b]. The operation was repeated three times to get an optimal result. Although IV thrombolytic was contraindicated as a result of head trauma and subdural hematoma history a month ago, 15 cc thrombolytic was selectively administered into the pulmonary

artery due to the continuation of hypotension and general condition impairment. The vital signs and hemodynamics of the patient improved rapidly after the procedure. Control pulmonary CT angiography taken 5 days after admission to the Coronary Intensive Care Unit showed complete resorption of thrombus material in the main pulmonary artery and its branches [Figure 4a and b]. The patient was discharged with rivaroxaban scheduled to start at 15 mg 2 × 1 dose for 3 weeks followed by 20 mg 1 × 1 oral daily dose. No problems were observed during the 1st, 6th, and 12th months’ controls.

DISCUSSION

PTE is a clinical condition with high mortality rates that should always be considered as a possible diagnosis. PTE shows a wide spectrum of manifestations clinically. In the old sources, this distribution was in the form of massive, submassive, and nonmassive according to clinical severity. Serious hypotension, cardiogenic shock, or acute right ventricular failure associated with cardiopulmonary arrest may develop in the massive pulmonary embolism.

Deterioration and dilatation of the right ventricular free wall movement in transthoracic echocardiography against normal systemic blood pressure can be detected in submassive



Figure 1: Cross-sectional image of pulmonary computed tomography angiography compatible with marked thromboembolism in the right and left pulmonary arteries

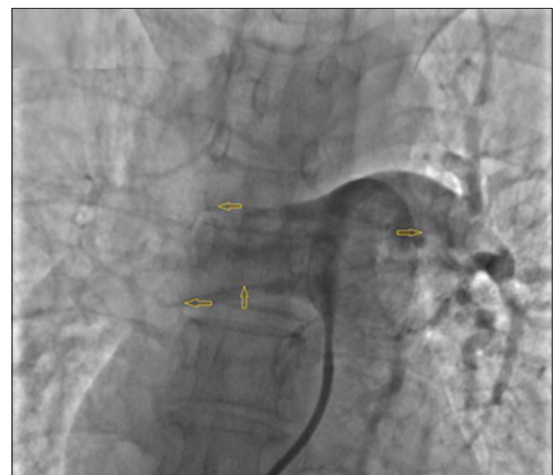


Figure 2: Pulmonary angiography showed marked thromboembolic images (arrows) in both pulmonary arteries and branches, especially in the right main pulmonary artery

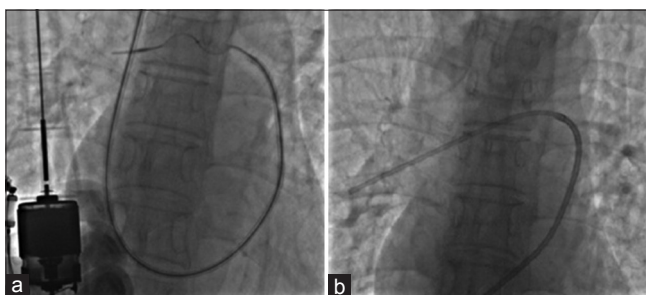


Figure 3: (a) Performing thrombus fragmentation with cleaner rotational thrombectomy system, (b) selective thrombus aspiration to pulmonary artery and branches

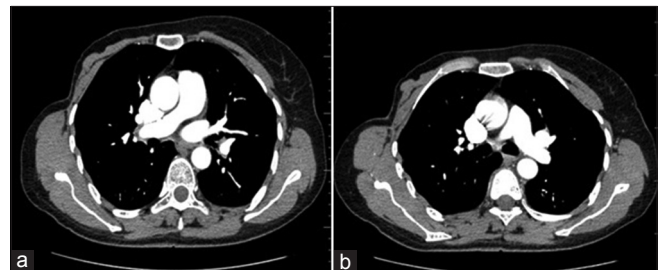


Figure 4: (a and b) On the 5th day of treatment, control pulmonary computed tomography angiography showed complete resorption of thrombus materials in the main pulmonary artery and its branches

pulmonary embolism. In the nonmassive pulmonary embolism, systemic blood pressure and right ventricular function are normal.^[3] The severity of PTE is considered according to the level of risk of premature mortality associated with PTE rather than the distribution, shape, and burden of intrapulmonary embolism. Therefore, current guidelines recommend that the estimation of the early mortality risk associated with PTE should be used instead of misleading terms such as “massive,” “submassive,” and “nonmassive”. According to this, if the clinical condition is accompanied by shock and hypotension, high-risk PTE is mentioned, while it is referred to as nonhigh-risk PTE if it is not.^[4] Mortality in mild pulmonary embolism is <5% in 3–6 months under anticoagulant treatment. The rate of recurrence of the disease is below 5%. However, the risk of recurrence of a 10-year pulmonary embolism is 30%.^[5]

ECG is a parameter that shows the electrical activity of the heart. Common findings in ECG in PTE patients are nonspecific ST/T wave changes and sinus tachycardia. However, a normal ECG is the most common finding. Rarely, ST segment elevation can also be observed. The classic S1-Q3-T3 pattern found also in our case is observed in only 20% of patients. It is not diagnostic, although it may suggest the possibility of PTE.^[6]

We believe that the immobilization of the patient due to the fracture in the right femur is the leading cause for the DVT, which is thought to be the cause of PTE. In the literature, it has been found that patients who have DVT in the lower extremity with Doppler ultrasonography are not always correspond with deep venous thrombosis in physical examinations.^[7] Having physical examination findings in the lower extremities is not a precondition for DVT diagnosis.

Mechanical thrombectomy catheters generally work on four different operating principles. These are

- a. Contact devices on the vessel wall: Arrow percutaneous thrombectomy device (PTD, Arrow International, Reading, PA, USA), Cleaner (Rex Medical, Fort Worth, TX, USA), etc.
- b. Hydrodynamic thrombectomy devices: Amplatz thrombectomy device (ATD/Helix, Microvena, White Bear Lake, MN, USA), Rotarex catheter (Straub Medical, Wangs, Switzerland), etc.
- c. Rheolytic (Flow-Based) thrombectomy devices: AngioJet (Possis Medical, Minneapolis, MN, USA), Oasis (Boston Scientific, Watertown, MA, USA), etc.
- d. Combination infusion catheter/isolated oscillation devices: Trellis Reserve (Bacchus Vascular, Santa Clara, CA, USA).^[8]

We had aspiration with thrombus aspiration catheter first because of the presence of severe thrombus material in the main pulmonary artery and in the proximal parts of the right and left pulmonary artery and then underwent thrombus fragmentation with a Cleaner rotating thrombectomy device rotating at 4000 rpm and then aspiration was done again. In such high-risk PTE cases, percutaneous thrombectomy may be lifesaving because of providing rapid hemodynamic

improvement due to performing thrombus fragmentation and concomitant pulmonary arterial thrombolytic infusion

One of the subclasses of pulmonary hypertension, CTEPH, is a disease with high mortality and morbidity, resulting from massive, recurrent, and/or organized thrombi or vessel wall remodeling blocking pulmonary vessels and slowing blood flow. In this disease, pulmonary vascular resistance increases, pulmonary artery pressure rises, disease progressively worsens, and results right heart failure and death. In the ESC 2015 pulmonary hypertension guidelines, pulmonary endarterectomy (PEA) is recommended as Class I, level of evidence C in the treatment of CTEPH; riociguat, which is a soluble guanylate cyclase stimulator, is recommended as Class I level of evidence B in case of persistent or nonspecific symptomatic CTEPH clinic after PEA. In our patient, CTEPH clinic was not observed in follow-up because of long-term effective anticoagulation with early intervention.^[4]

In conclusion, PTE is a health problem that can be lead to significant mortality and morbidity and the possibility of clinical diagnosis should always be kept in mind. In the treatment of this clinical entity, technics and devices are improving day by day in percutaneous interventions' era. This case report is important for demonstrating rapid percutaneous management of a young female patient with a life-threatening condition and favorable outcome of percutaneous intervention despite many comorbid conditions.

Declaration of patient consent

The authors certify that they have obtained all appropriate patient consent forms. In the form the patient(s) has/have given his/her/their consent for his/her/their images and other clinical information to be reported in the journal. The patients understand that their names and initials will not be published and due efforts will be made to conceal their identity, but anonymity cannot be guaranteed.

Financial support and sponsorship

Nil.

Conflicts of interest

There are no conflicts of interest.

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Complete Aortic Prosthetic Valve Dehiscence after Modified Bentall-De Bono Procedure

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Abstract

A 56-year-old male patient was admitted to our clinic due to persistent fever despite the use of antibiotics for 2 weeks, chest pain, and presyncope. His medical history revealed that the patient underwent modified Bentall-De Bono procedure 2 months ago due to ascending aortic aneurysm and severe aortic insufficiency. Transthoracic apical 5 chamber view showed that mobile vegetation prolapsed into the left ventricular outflow tract during ventricular diastole and that mechanical prosthetic valve was superior to the aortic annulus. Transesophageal echocardiography revealed normal aortic mechanical prosthetic valve function; however, the valve was positioned more superior to the annular plane and a dense vegetation was observed. Moreover, a complete dehiscence of the prosthetic valve was attached to aortic annulus with a single stitch in an area between noncoronary sinus and left coronary sinus. Dense thrombus formation was observed in the perivalvular region. Many cases with prosthetic valve endocarditis and partial dehiscence as its complication have been reported in the literature. However, to the best of our knowledge, there is no reported case of complete dehiscence secondary to infective endocarditis following complete ascending aortic graft and prosthetic aortic valve replacement (modified Bentall-De Bono procedure).

Keywords: Complete dehiscence, endocarditis, prosthetic valve

INTRODUCTION

Aortic valve prosthesis dehiscence is an uncommon complication of aortic valve surgery. It is usually recognizable at echocardiography due to an abnormal position of the prosthetic valve in relation to the native aortic annulus in conjunction with an abnormal periaortic space that fills with thrombus. We present a patient with complete aortic prosthetic valve dehiscence and periaortic thrombus.

CASE REPORT

A 56-year-old male patient was admitted to our clinic due to persistent fever despite the use of antibiotics for 2 weeks, chest pain, and presyncope. His medical history revealed that the patient underwent modified Bentall-De Bono procedure 2 months ago due to ascending aortic aneurysm and severe aortic insufficiency. On physical examination, his general condition was poor, he had tachypnea and tachycardia, his

body temperature was 39.3°C, and his skin was pale and clammy. His blood pressure was 110/45 mmHg, and pulse rate was 117 bpm; electrocardiography showed sinus rhythm. Laboratory examination revealed a white blood cell count of 26,000/mm³, hemoglobin level of 9.6 g/dL, and high-sensitivity C-reactive protein level of 119 mg/dL.

Transthoracic apical 5 chamber view showed that mobile vegetation prolapsed into the left ventricular outflow tract during ventricular diastole and that mechanical prosthetic valve was superior to the aortic annulus [Figure Panel 1a and Supplementary Video 1]. Transesophageal echocardiography revealed normal aortic mechanical prosthetic valve function; however, the valve was positioned more superior to the annular plane and a dense vegetation was observed [Figure Panel 1b and c, Supplementary Video 1]. Moreover, a complete

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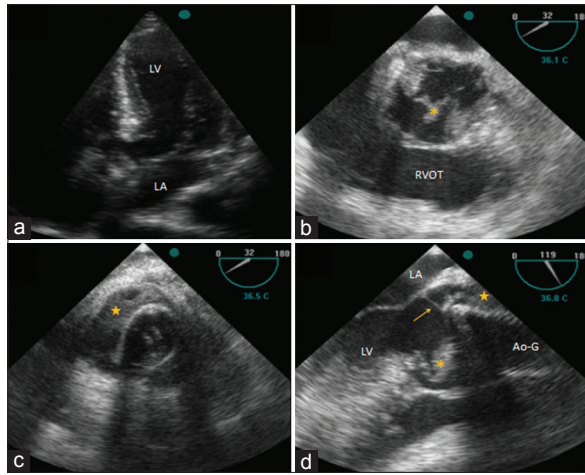


Figure 1: (a) Prosthetic valve was superior to the annular plane. (b) Vegetation at the aortic annulus (asterisks) and displacement of the prosthetic valve. (c) The prosthetic valve and dense thrombus formation (star) around it. (d) Vegetation (asterisk), complete dehiscence of the prosthetic valve attached to aortic annulus with a single stitch in between noncoronary sinus and left coronary sinus (arrow), and thrombus around aortic graft (star). LA: Left atrium, LV: Left ventricle, RA: Right atrium, RV: Right ventricle RVOT: Right ventricle outflow tract, Ao-G: Aortic graft, P-AV: Prosthetic aortic valve

dehiscence of the prosthetic valve was attached to aortic annulus with a single stitch in an area between noncoronary sinus and left coronary sinus [Figure Panel 1d and Supplementary Video 1]. Dense thrombus formation was observed in the perivalvular region. The patient underwent emergency operation due to complete dehiscence. Intraoperative macroscopic findings were consistent with the echocardiographic findings.

CONCLUSION

Infective endocarditis is a severe clinical problem, with considerable morbidity and mortality, characterized by microbial infection of intracardiac endothelial lining commonly involving one or more cardiac valves and less frequently mural endocardium, chordae, and myocardium. Intracardiac or endovascular devices or graft materials act as a reservoir for infection and also complicate the treatment.^[1,2] Prosthetic

valve endocarditis differs from native valve endocarditis in terms of prevalence, microbial agents, complications, and management. Today, significant portion of patients with infective endocarditis have prosthetic valve endocarditis. In prosthetic valve endocarditis, unlike native valve endocarditis, annular abscess, fistulization, severe valvular dysfunction, and dehiscence are the main severe complications; the presence of dehiscence being the case in our patient.^[1,3,4] Many cases with prosthetic valve endocarditis and partial dehiscence as its complication have been reported in the literature. However, to the best of our knowledge, there is no reported case of complete dehiscence secondary to infective endocarditis following complete ascending aorta and prosthetic aortic valve replacement (modified Bentall-De Bono procedure). Our purpose in the present paper is to discuss an undesired early complication following complete ascending aorta and prosthetic valve replacement with demonstrative images.

Declaration of patient consent

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Nil.

Conflicts of interest

There are no conflicts of interest.

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A Case Difficult to Diagnose in Adults: High Sinus Venous Atrial Septal Defect

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Abstract

Sinus venous atrial septal defect (SVD) is highly difficult to diagnose because of its location. Below, we report a case of SVD which is misdiagnosed as pulmonary hypertension and anomalous pulmonary venous return. A 57-year-old female patient was referred to congenital disease outpatient clinic of a tertiary center. She was admitted to the hospital with complaints of fatigue and exercise dyspnea which had started a year ago. She had transthoracic echocardiography (TTE) examination done in another hospital which showed dilated right heart chambers and pulmonary hypertension. She underwent transesophageal echocardiography (TEE) examination with the suspicion of atrial septal defect (ASD), but no defect was seen. As her symptoms persisted, we repeated the TTE and TEE examination in our center. TEE revealed 0.6 cm ASD on the upper side of the interatrial septum. All four pulmonary veins were draining into the left atrium. Right heart catheterization (RHC) confirmed the diagnosis. A left-to-right shunt was detected and localized by a significant step-up in blood oxygen saturation found between mid and upper segments of the right atrium. According to our TEE and RHC results, we planned the surgical closure of the defect. Sinus venous ASD is deficiency of the superior portion of atrial septum adjacent to superior vena cava. Diagnosis of SVD is often more difficult than other forms of ASD and may require special imaging such as TEE, magnetic resonance imaging, or computed tomographic scanning. In conclusion, cardiologists must be aware about the possibility of SVD patients who have unexplained exertional dyspnea and fatigue, dilated right atrium and ventricle, pulmonary hypertension, paradoxical embolism, or atrial arrhythmias in their respective populations.

Keywords: Adult congenital heart disease, atrial septal defect, sinus venous atrial septal defect, transesophageal echocardiography

INTRODUCTION

Atrial septal defect (ASD) is the most common congenital abnormality in adults characterized by defect on interatrial septum. The most common types of ASD include ostium primum ASD, ostium secundum ASD, and sinus venous ASD, respectively. Sinus venous atrial septal defect (SVD) is classified as high or low sinus venous type according to proximity to the superior vena cava (SVC) or inferior vena cava (IVC). SVD is highly difficult to diagnose because of its location. It requires surgical repair, and the outcome is much better if repair is done earlier.

Below, we report a case of SVD which is misdiagnosed as pulmonary hypertension and anomalous pulmonary venous return.

CASE REPORT

A 57-year-old female patient was referred to congenital disease outpatient clinic of a tertiary center. She was admitted

to the hospital with complaints of fatigue and exercise dyspnea which had started a year ago.

She had total thyroidectomy in her medical background, and she used 100 microgram oral levotirosin, daily. The patient's functional capacity was Grade III according to New York Heart Association functional classification. There was sinus rhythm and right bundle branch block on her electrocardiogram (ECG). Her laboratory tests were normal, and she walked 457 m on 6 min walk test [Figure 1].

She had transthoracic echocardiography (TTE) examination done in another hospital which showed dilated right heart chambers and pulmonary hypertension. She underwent

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transesophageal echocardiography (TEE) examination with the suspicion of ASD, but no defect was seen. Magnetic resonance imaging (MRI) and cardiac computer tomography (CT) were planned to confirm the diagnosis of ASD. MRI revealed dilated pulmonary truncus, right atrium, and right ventricle, and CT showed abnormal venous connections [Figures 2 and 3].

As her symptoms persisted, we repeated the TTE and TEE examination in our center. TEE revealed 0.6 cm ASD on the upper side of interatrial septum. All four pulmonary veins were draining into the left atrium. Right heart catheterization (RHC) confirmed the diagnosis. A left-to-right shunt was detected and localized by a significant step-up in blood oxygen saturation found between mid and upper segments of the right atrium. Cardiac output, pulmonary output, pulmonary vascular resistance (PVR), and systemic vascular resistance (SVR) was calculated as 5.0 L/min, 14.2 L/min, 1.5 Woods units, and 20.6 Wood units, respectively. Furthermore, Qp/Qs was calculated as 2.8.

The patient's RHC results are appropriate to the closure of ASD. According to our TEE and RHC results, we planned the surgical closure of the defect.

DISCUSSION

Sinus venous ASD is deficiency of the superior portion of atrial septum adjacent to SVC. ASDs account for about 10%–15% of all congenital cardiac anomalies and are the most common congenital cardiac lesion presenting in children and adults.^[1] SVD is relatively rare and is found in 2%–10% of patients with ASDs. Most of the patients are asymptomatic. Since many patients are asymptomatic at young age, the patients are diagnosed accidentally or when their symptoms become manifest at older ages. The patients most often present with congestive heart failure with pulmonary hypertension, atrial arrhythmias, right ventricular failure, or paradoxical embolization. Diagnosis of SVD is often more difficult than other forms of ASD and may require special imaging such as TEE, MRI, or CT scanning.^[2]

Shub *et al.* demonstrated 100% of ostium primum and more than 90% of ostium secundum ASD but only 44% of SVD in their trial with 154 adults and children who underwent TTE.^[3] Kronzon *et al.* designed a similar study to establish value of TEE in SVDs. Forty-one adult patients from 18 to 81 years old with the clinical diagnosis of ASD were studied by TTE and TEE. In 8 (20%) of 41 patients, the ASD was demonstrated by TEE and not by TTE. Six of the eight had a SVD; the other two patients had a secundum ASD (one of these two had a technically poor TTE and the other had a small ASD). TTE failed to demonstrate the SVD in 6 (75%) of eight patients.^[4]

Before correction of an ASD, an evaluation is made of the severity of the individual's pulmonary hypertension and whether it is reversible. Individuals with PVR more than 4.6 WU have increased mortality associated with closure of

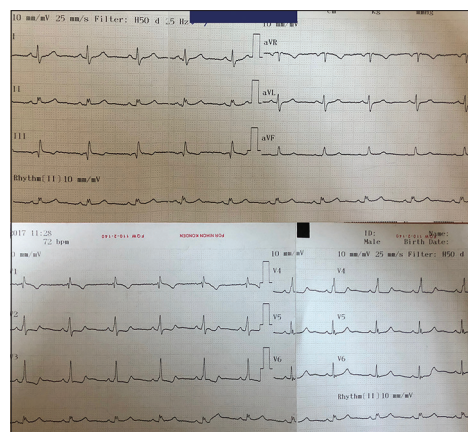


Figure 1: Electrocardiogram shows normal sinus rhythm

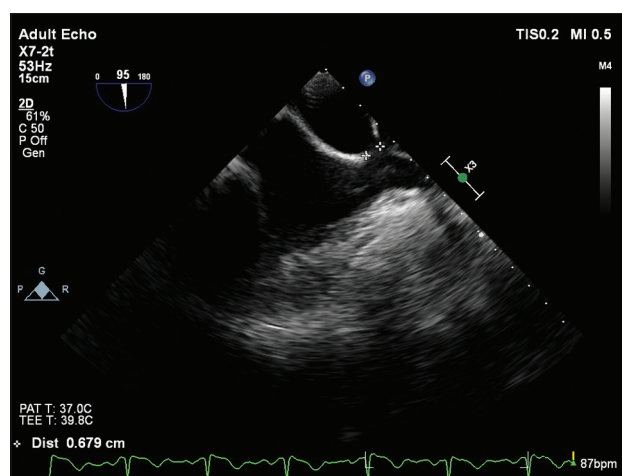


Figure 2: Transesophageal echocardiography shows 0.6 cm high sinus venous-type atrial septal defect

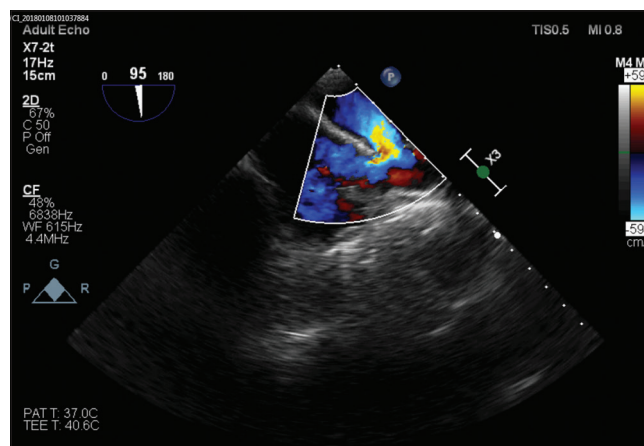


Figure 3: The color Doppler examination shows high sinus venous atrial septal defect

the ASD. While PVR is more than 2.3 WU but <4.6 WU, closure of ASD appropriates if Qp: Qs is more than 1.5, PVR: SVR <2/3, the defect is pretricuspid defect, and the patient is younger than 5 year old.

ECG, also, may be helpful in diagnosing and differentiating the type of ASDs. Patients with ASD may have a prolonged PR interval. A first-degree heart block is probably due to the enlargement of the right atrium and increased distance of internodal conduction from the SA node to the AV node.^[1] In addition, the mean QRS axis is important in differentiating the secundum from the primum defect. True right axis deviation was present in 81% of the secundum defects and in none of the primum defects. Left axis deviation was present in 82% of the ostium primum defects and in none of the secundum defects. Following surgical closure of the secundum defect, the R' usually decreases significantly within 4 months. There is often a normal rSr' or rS in Lead V1 within 1 year after closure. If regression does not occur, then there is cause for doubt as to the complete closure of the defect, or the presence of irreversible pulmonary vascular changes may be suspected.^[5] ECG of the patients with sinus venous ASD exhibits a left axis deviation of the P wave but not the QRS complex.

Both of percutaneous transcatheter closure or surgical closure strategies might be the treatment strategy. Catheter closure is only possible for the closure of secundum ASDs with a sufficient rim of tissue around the septal defect so that the closure device does not impinge on the SVC, IVC, or the tricuspid or mitral valves and aneurismal interatrial septum. Thus, the treatment of SVDs is surgical. All the complications must be treated with their optimal medical treatment according to their guidelines, respectively.

The prognosis is excellent for young patients who undergo repair of uncomplicated defects, particularly in the first 2 decades of their life. Repair delayed until the third decade of life is associated with a decrease in life expectancy.^[6]

CONCLUSION

Cardiologists must be aware about the possibility of SVD patients who have unexplained exertional dyspnea and fatigue, dilated right atrium and ventricle, pulmonary

hypertension, paradoxical embolism, or atrial arrhythmias in their respective populations. If there is any suspicion about the diagnosis, all the imaging facilities such as ECG, TTE, X-ray radiogram, TEE, CT scan, MRI, and RHC must be used to diagnose.

Declaration of patient consent

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Financial support and sponsorship

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Conflicts of interest

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Transjugular Closure of Secundum Atrial Septal Defect in a Patient with Interrupted Inferior Vena Cava

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Abstract

In this case, we report a successful closure of secundum atrial septal defect in a 32-year-old female patient with an interrupted inferior vena cava (IVC). Interrupted IVC was detected coincidentally during right heart catheterization. The defect was successfully closed through transjugular vein approach as an alternative to surgery.

Keywords: Amplatzer septal occluder device, inferior vena cava, jugular veins, ostium secundum atrial septal defect

INTRODUCTION

Atrial septal defect (ASD) is seen in 30%–33% of adults. Although ASD is usually asymptomatic, it can lead to exercise intolerance, atrial arrhythmia, right ventricular dysfunction, and pulmonary hypertension with aging and lifespan diminishes in adult patients with untreated large defects. As a potentially serious complication, the risk of developing pulmonary vascular disease is higher in adults with untreated defects. The main treatment option is transcatheter closure which is less invasive and more comfortable for patients. Percutaneous closure is routinely performed by the transfemoral route through inferior vena cava (IVC). However, operator rarely comes across unexpected vascular access problems such as interrupted IVC during the closure of ASD.

Congenital interrupted IVC with azygos continuation is a rare variation usually associated with other congenital anomalies. The incidence of this anomaly nearly 0.6% in patients with congenital heart defects additional to atrioventricular canal defect, anomalously connecting pulmonary veins, double outlet right ventricle, large ASD, pulmonary stenosis or atresia, and sick sinus syndrome.^[1,2] Usually, the presence of this vascular variation alone does not cause clinically problems, and it is found incidentally^[3,4] either during an examination or at postmortem dissection. However, in some cases, it can become clinically important.

CASE REPORT

A 32-year-old woman was admitted to our hospital with a complaint of dyspnea and palpitation. A fixed wide splitting of the second heart sound and a Grade 2/6 systolic murmur was auscultated at the second left intercostal space. Electrocardiogram was in sinus rhythm with an incomplete right bundle branch block. Transthoracic echocardiogram revealed mild right ventricular dilatation and secundum ASD. Transesophageal echocardiography (TEE) revealed a 12 mm secundum ASD with sufficient rhymes for percutaneous closure [Figure 1a]. Cardiac catheterization was performed under general anesthesia using TEE. A 7-F sheath was placed through the right femoral vein. Afterward; despite many attempts, the guidewire could not be passed through IVC. Venography revealed interrupted IVC [Figure 1b] and venous flow reached to the right atrium through collaterals and azygos continuation of interrupted IVC to superior vena cava (SVC) [Figure 1c]. Because of the sharp angulation of azygos continuation at SVC, percutaneous ASD closure was decided to be continued through transjugular approach. A 7-F sheath was placed to the right internal jugular vein. The initial attempt to cross the interatrial septum with an

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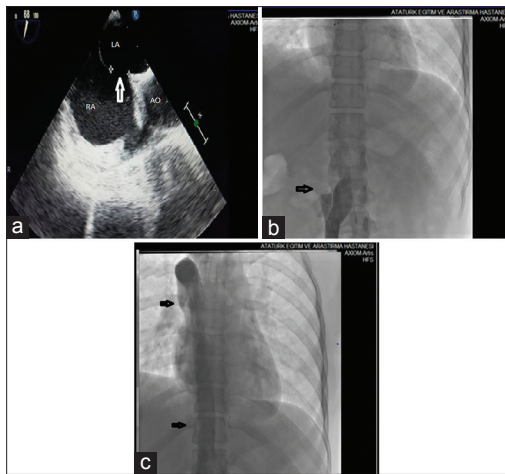


Figure 1: (a) Transesophageal appearance of the secundum atrial septal occluder before closure (white arrow), (b) Interrupted section of inferior vena cava, (c) The azygos continuation of interrupted inferior vena cava

6-F Multipurpose catheter was unsuccessful. In the next attempt, the tip of a Cobra (Terumo®) catheter was well oriented to the ASD and the catheter could be easily passed into the left atrium (LA) over a 0.35 j-tipped guidewire. Then, the 0.35 j-tipped guidewire was exchanged with a super stiff 0.38-inch guidewire which is placed in the left upper pulmonary vein. Afterward, an ASD sizing balloon was passed through the ASD over the stiff guidewire; however, with the introduction of the catheter of the sizing balloon, the stiff wire was displaced from the upper pulmonary vein. Therefore, the stiff wire was relocated into the left lower pulmonary vein where it was more stable and could better carry the sizing balloon catheter. The defect was measured 12 mm with the sizing balloon. After balloon sizing, an 8 F Amplatzer® delivery system (St. Jude Medical, St Paul, MN, USA) was introduced through the internal jugular vein. While negotiating the delivery system, the stiff wire was displaced from the lower pulmonary vein and fell into the left ventricular cavity [Figure 2a]. The delivery sheath and its dilator were gently advanced over the stiff wire until the sheath was placed in the left ventricular cavity. The stiff wire and dilator were removed from the delivery sheath while maintaining the tip of the sheath within the left ventricle. Under fluoroscopic guidance, a 14-mm Amplatzer® atrial septal occluder (ASO) (St. Jude Medical, St Paul, MN, USA) device was passed through the delivery catheter without exiting from the tip of the sheath [Figure 2b]. Then, the whole system was withdrawn into the left atrial cavity and the tip of sheath was placed above the mitral valve under TEE guidance. Left disc of the device was unsheathed within the LA and gently pulled against the interatrial septum. Using gentle tension on the delivery system, the sheath was pulled back, and the right atrial disc was opened [Figure 2c and d] Subsequent Minnesota maneuver of the delivery system ensured a safe device positioning across the ASD. Finally, the delivery system was unlocked, and device was released [Figure 2e]. Control TEE demonstrated

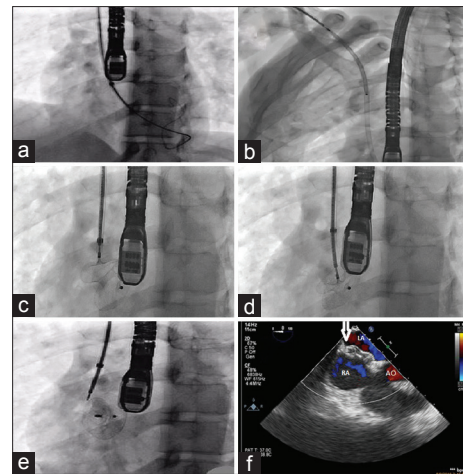


Figure 2: Transesophageal echocardiography and fluoroscopy-guided closure steps through internal jugular approach: (a) stiff wire in the left ventricle (b) atrial septal occluder device within the delivery system, tip of the delivery catheter is in the left ventricle (c and d) opening steps of the right atrial disc (e) released atrial septal occluder device (f) Cessation of the interatrial shunt (white arrow: atrial septal occluder device, RA: Right atrium, LA: Left atrium, AO: Aortic valve)

a stable occluder device with cessation of the interatrial shunt [Figure 2f] and the procedure was completed without any complications.

DISCUSSION

IVC is built of four segments during the embryonic progress and defect of the formation of hepatic segment. Interruption of the IVC with the continuation of azygos system represents the most common abnormality of these of embryonic veins.^[4] This malformation is a marker for the presence of atrial isomerism and the polysplenia syndromes.^[5] However, our patient had isolated secundum ASD and interrupted IVC without any other concomitant congenital abnormality. This finding can be important when an interventional procedure will be performed. The delivery systems for transcatheter closure are designed to be used from the femoral veins. However, there are some conditions impeding transfemoral venous approach such as IVC interruption. In these cases, transjugular, transhepatic approach, or surgery can be an alternative for closure. At some rare cases, transfemoral closure can be also possible through the azygos continuation of the interrupted IVC;^[6,7] however, in most cases, navigation of the with large caliber delivery sheaths through the azygos vein may be difficult and may result in vessel injury. Intrathoracic vein injuries can result in catastrophic hemorrhage, especially if the injury to the vein communicates with the pleural or mediastinal space. Because of the abrupt angulation of the azygos vein at its entry into the SVC, a stable guidewire and delivery sheath positioning may be extremely difficult as well as a proper sizing balloon placement. In suitable cases, transhepatic access can be used for percutaneous closure. This approach is particularly useful in infants, young children, and thin-built adults, in whom hepatic veins are larger than the

jugular veins. In these patients, transhepatic method permits easier venous sheath placement and provides better catheter stability during the procedure.^[8] On the other hand, transhepatic access may cause higher incidence of complications including retro or intraperitoneal bleeding, injury of the gallbladder, pneumothorax, pleural effusions, and liver abscess or peritonitis.^[9] As described previously in a few case reports, alternatively the transjugular approach is a potentially safer. Due to the previously explained difficulties, transjugular ASD closure has been reported only in two pediatric patients.^[9,10] On the other hand, review of the literature revealed 5 case reports of transjugular ASD closure in adult patients.^[11-15] These reports included patients with severe scoliosis,^[13] iatrogenic total occlusion of the IVC after an unsuccessful attempt with a CardioSEAL septal occluder,^[14] and postoperative residual ASD following surgical repair of total anomalous pulmonary venous connection.^[15] Transjugular ASD closure was reported in two adult patients with interrupted IVC,^[11,12] and the current case is the third similar case.

Transjugular approach for ASD closure is a relatively easier procedure in adults compared to the pediatric population. Due to the larger size of the internal jugular vein, venous puncture is easier and large caliber sheaths; delivery systems can be safely introduced in adult patients. Narin *et al.*, reported the hematoma in the muscles of the neck that was caused transient loss of strength after the procedure.^[10] Such complication was not observed in our case and previous transjugular ASD closures in adult patients.

Transjugular approach has some minor procedural differences compared to the transfemoral closure. Passing through the defect with a catheter and placing the guidewire in the pulmonary vein may be more difficult than the transfemoral route. In transfemoral approach, a multipurpose catheter is usually used to cross the defect. In transjugular approach, a right Judkins or cobra catheter can be preferred at this step since their tips are better directed toward the ASD.^[9,11] Placing and maintaining the guidewire in the left upper or lower pulmonary veins may be more difficult than the transfemoral route. In transjugular approach, the tip of the delivery system tends to point toward the left ventricular inflow or left atrial appendage and the tip of the catheter may easily fall into the left ventricular cavity before or after the guidewire removal. Similar to the current case, Bhargava *et al.*, have also demonstrated that the delivery sheath can be safely placed within the left ventricular cavity to provide good support for the ASD advancement through the catheter.^[11] However, TEE monitoring is essential during the procedure to prevent injury of the mitral valve and subvalvular structures. Echocardiographic guidance needs to ensure the opening of the left atrial disc away from the mitral valves in the left atrial cavity.

In transjugular approach, complications are quite a few. Bleeding is the most probable complications due to carotid, subclavian, or vertebral artery injuries. Compressing of artery is more difficult when compared with femoral vein

approach for controlling of bleeding. Other risk for the jugular approach is air embolism because of the upper position of the internal jugular vein from the level of the heart. The sheath manipulations must be performed under the level of the heart to avoid of air embolism. After removal of the dilator and guidewire, avoidance for wounding of the left atrial or left ventricular walls, the location of the delivery sheath tip should be in attention, and a good backflow from the catheter should be seen before the insertion of the occlusion device.

CONCLUSION

In experienced centers, percutaneous closure of ASD through transjugular access is a safe, feasible, and effective procedure in adult patients with interrupted IVC. Meticulous TEE guidance is essential during delivery sheath and device manipulations.

Declaration of patient consent

The authors certify that they have obtained all appropriate patient consent forms. In the form the patient(s) has/have given his/her/their consent for his/her/their images and other clinical information to be reported in the journal. The patients understand that their names and initials will not be published and due efforts will be made to conceal their identity, but anonymity cannot be guaranteed.

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Conflicts of interest

There are no conflicts of interest.

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