Cardiovascular Surgery and Interventions

E-ISSN 2148-9211

CARDIOVASCULAR SURGERY and INTERVENTIONS

Official Electronic Journal of the Turkish Society of Cardiovascular Surgery

Türk Kalp Damar Cerrahisi Derneği 1988

Volume: 2 / Number: 2 / July 2015

Cardiovascular Surgery and Interventions

E-ISSN 2148-9211

CARDIOVASCULAR SURGERY and INTERVENTIONS

Official Electronic Journal of the Turkish Society of Cardiovascular Surgery

Türk Kalp Damar Cerrahisi Derneği 1988

Volume: 2 / Number: 2 / July 2015

E-ISSN 2148-9211

CARDIOVASCULAR SURGERY AND INTERVENTIONS

Volume 2 - Number 2 - July 2015

Cardiovascular Surgery and Interventions is the official and periodical journal of the Turkish Society of Cardiovascular Surgery. It is published three times a year.

Executive office:
Türk Kalp ve Damar Cerrahisi Derneği
Ataşehir Mah., Ataşehir Bulvarı, 48 Ada,
Mimoza 2/2, K: 2, D: 6,
34758 Ataşehir, İstanbul, Turkey
Tel: +90 216 - 456 14 54
Fax: +90 216 - 456 14 54
e-mail: info@tkdcd.org
URL: http://www.tkdcd.org

Editorial Contact Person
Ahmet Rüçhan Akar, MD.
e-mail: akarruchan@gmail.com

Publisher
Bayçınar Tıbbi Yayıncılık ve Reklam Hiz. Tic. Ltd. Şti.
Örnek Mah., Dr. Suphi Ezgi Sok., Saray Apt.,
No: 11, D: 6, 34704 Ataşehir, İstanbul, Turkey
Tel: +90 216 - 317 41 14
Fax: +90 216 - 317 63 68
e-mail: info@baycinartibbiyayincilik.com

Type of publication: Periodical
Publication date: August 03, 2015

The control of conformity with the journal standards and the typesetting of the articles in this journal, the control of the English abstracts and references and the preparation of the journal for publishing were performed by Bayçınar Medical Publishing.

Contents

PERSPECTIVE FROM the EDITOR IN-CHIEF
August 2015

Distinguished Colleagues and Readers,

It is 23 years since the *Turkish Journal of Thoracic and Cardiovascular Surgery,* the official journal of the Turkish Cardiovascular Surgery Society and Turkish Thoracic Surgery Society, was issued, which was first published in November 1991 under the leadership of Prof. Aydın Aytaç, MD., the president of the Turkish Cardiovascular Surgery Society and by Prof. Tayyar Sarıoğlu, MD., editor in-chief. As of the first issue, the journal has been published quarterly. Day by day, our journal has been gaining further strength in the national and international medical community as a result of its scientific content and independent philosophy.

It is a great honor to be the editor in-chief of the journal, since the Turkish Cardiovascular Surgery Society and Turkish Thoracic Surgery Society assigned me to this position this year. As it is well-known, re-assignments in each period may carry potential risks. To date, previous distinguished seven editors in-chief, have spent great effort to the current status of the journal despite severe difficulties. Past editors and the hardworking members of the editorial board can be deemed as fully responsible for the success of the journal with a growing number of manuscript submission and indexing the journal in the Science Citation Index Expanded (SCI-Expanded). We, all with the members of the recently re-organized editorial board, appreciate the responsibility and pride of this honorable task, by following the contemporary publishing principles and sticking to our institutional policies. I also would like to express my gratitude to the past editors, members of the editorial board, reviewers, and all authors who submitted their manuscripts and contributed to our prestigious heritage.

On the date of 12th June, 2015, we, including both editorial boards, convened to make a thorough evaluation of the journal and to identify further goals in the head office of the Turkish Cardiovascular Surgery Society. It was highly effective, as expected and I would like to introduce you the decisions and goals which were agreed upon during this meeting. Our first short-term goal is to increase the impact factor of the journal and to enable to be indexed in the PubMed. With the assistance of Dr. Suat Nail Ömeroğlu, editor, and Drs. Mehmet Kaplan and Atilla Gürses, administrative editors, we, therefore, are committed to make a full collaboration to achieve our goals.

Furthermore, I would like to express my thanks to all colleagues for their contributions to the journal for being indexed in the SCI-E. Of note, it is all our responsibility to sustain our success for SCI-E. Despite the high number of articles electronically downloaded, the number of citations, unfortunately, appears to be relatively low. Within this context, I would like distinguished members to highlight the importance of citation of the manuscripts published in our journal within the past two years in the international publications. I, in particular, advise using the official abbreviation of our journal, "Turk Gogus Kalp Dama" while referencing to a manuscript published in other journals.

One of the main principals of the board of the Turkish Cardiovascular Surgery Society and Turkish Thoracic Surgery Society is to maintain a homogenous administration for all tasks across the country. So far, we have spent great effort to establish the recent editorial board by prioritizing the efforts on our journals, considering the institutional dignity, and enabling regional representation. Furthermore, new national and international colleagues will be included in the editorial board according to the quality and the number of manuscripts evaluated as well as evaluation times.

Objectivity, ethic codes and confidentiality are the milestones for a peer-review process. We, as the member of the editorial board of the journal, are committed to follow these milestones. It is beyond any doubt that all colleagues' will to the full extent assist the editorial board working diligently.

Original research articles are the main type of papers in our journal. The manuscripts submitted are evaluated by three to five referees. Soon, the evaluation process will be shortened with a novel infrastructure and an option to recommend alternative reviewers. Unless the reviewer achieves his/her task, the final decision will be based upon the editor's discretion and the evaluation process will be shortened, if the number of evaluation is sufficient.

As a result of advances in science and technology, medicine is transforming. In this millennium, multidisciplinary approach for better health care is crucial. In parallel, the electronic journal of *Cardiovascular Surgery and Interventions,* which was first published in March 2014, has been rapidly extending its scope with new projects under the assistance of Dr. Şahin Şenay, editor, and the editorial board. E-CVSI aims to be the voice of multidisciplinary teams and will provide an active forum to advance our medical knowledge and creativity for our community as well as other disciplines. Fast dissemination of videos and podcasts on innovative surgical techniques and interventions and medical news will be provided. An interactive platform for relevant comments will soon be constituted. In addition, invited comment pages will be created. Android and Apple applications will be established to facilitate the access to our both journals.

I, also, would like to express my thanks to Bayçınar Tıbbi Yayıncılık for their diligent, timely, and high-quality works and Pleksus for their robust electronic infrastructure services and will be pleased to maintain our collaboration in the new period.

Finally, I would like to express my thanks to Prof. Anıl Z. Apaydın, MD. and the members of the board of directors for their great support and courage and to all for your efforts to establish and develop the journals within the hope of a sustainable commitment and interest.

Prof. A. Rüçhan Akar, MD.

Editor In-Chief,

Turkish Journal of Thoracic and Cardiovascular Surgery
Cardiovascular Surgery and Interventions

PERSPECTIVE FROM the EDITOR IN-CHIEF
August 2015

Distinguished Colleagues and Readers,

It is 23 years since the *Turkish Journal of Thoracic and Cardiovascular Surgery,* the official journal of the Turkish Cardiovascular Surgery Society and Turkish Thoracic Surgery Society, was issued, which was first published in November 1991 under the leadership of Prof. Aydın Aytaç, MD., the president of the Turkish Cardiovascular Surgery Society and by Prof. Tayyar Sarıoğlu, MD., editor in-chief. As of the first issue, the journal has been published quarterly. Day by day, our journal has been gaining further strength in the national and international medical community as a result of its scientific content and independent philosophy.

It is a great honor to be the editor in-chief of the journal, since the Turkish Cardiovascular Surgery Society and Turkish Thoracic Surgery Society assigned me to this position this year. As it is well-known, re-assignments in each period may carry potential risks. To date, previous distinguished seven editors in-chief, have spent great effort to the current status of the journal despite severe difficulties. Past editors and the hardworking members of the editorial board can be deemed as fully responsible for the success of the journal with a growing number of manuscript submission and indexing the journal in the Science Citation Index Expanded (SCI-Expanded). We, all with the members of the recently re-organized editorial board, appreciate the responsibility and pride of this honorable task, by following the contemporary publishing principles and sticking to our institutional policies. I also would like to express my gratitude to the past editors, members of the editorial board, reviewers, and all authors who submitted their manuscripts and contributed to our prestigious heritage.

On the date of 12th June, 2015, we, including both editorial boards, convened to make a thorough evaluation of the journal and to identify further goals in the head office of the Turkish Cardiovascular Surgery Society. It was highly effective, as expected and I would like to introduce you the decisions and goals which were agreed upon during this meeting. Our first short-term goal is to increase the impact factor of the journal and to enable to be indexed in the PubMed. With the assistance of Dr. Suat Nail Ömeroğlu, editor, and Drs. Mehmet Kaplan and Atilla Gürses, administrative editors, we, therefore, are committed to make a full collaboration to achieve our goals.

Furthermore, I would like to express my thanks to all colleagues for their contributions to the journal for being indexed in the SCI-E. Of note, it is all our responsibility to sustain our success for SCI-E. Despite the high number of articles electronically downloaded, the number of citations, unfortunately, appears to be relatively low. Within this context, I would like distinguished members to highlight the importance of citation of the manuscripts published in our journal within the past two years in the international publications. I, in particular, advise using the official abbreviation of our journal, "Turk Gogus Kalp Dama" while referencing to a manuscript published in other journals.

One of the main principals of the board of the Turkish Cardiovascular Surgery Society and Turkish Thoracic Surgery Society is to maintain a homogenous administration for all tasks across the country. So far, we have spent great effort to establish the recent editorial board by prioritizing the efforts on our journals, considering the institutional dignity, and enabling regional representation. Furthermore, new national and international colleagues will be included in the editorial board according to the quality and the number of manuscripts evaluated as well as evaluation times.

Objectivity, ethic codes and confidentiality are the milestones for a peer-review process. We, as the member of the editorial board of the journal, are committed to follow these milestones. It is beyond any doubt that all colleagues' will to the full extent assist the editorial board working diligently.

Original research articles are the main type of papers in our journal. The manuscripts submitted are evaluated by three to five referees. Soon, the evaluation process will be shortened with a novel infrastructure and an option to recommend alternative reviewers. Unless the reviewer achieves his/her task, the final decision will be based upon the editor's discretion and the evaluation process will be shortened, if the number of evaluation is sufficient.

As a result of advances in science and technology, medicine is transforming. In this millennium, multidisciplinary approach for better health care is crucial. In parallel, the electronic journal of *Cardiovascular Surgery and Interventions,* which was first published in March 2014, has been rapidly extending its scope with new projects under the assistance of Dr. Şahin Şenay, editor, and the editorial board. E-CVSI aims to be the voice of multidisciplinary teams and will provide an active forum to advance our medical knowledge and creativity for our community as well as other disciplines. Fast dissemination of videos and podcasts on innovative surgical techniques and interventions and medical news will be provided. An interactive platform for relevant comments will soon be constituted. In addition, invited comment pages will be created. Android and Apple applications will be established to facilitate the access to our both journals.

I, also, would like to express my thanks to Bayçınar Tıbbi Yayıncılık for their diligent, timely, and high-quality works and Pleksus for their robust electronic infrastructure services and will be pleased to maintain our collaboration in the new period.

Finally, I would like to express my thanks to Prof. Anıl Z. Apaydın, MD. and the members of the board of directors for their great support and courage and to all for your efforts to establish and develop the journals within the hope of a sustainable commitment and interest.

Prof. A. Rüçhan Akar, MD.

Editor In-Chief,

Turkish Journal of Thoracic and Cardiovascular Surgery
Cardiovascular Surgery and Interventions

Cardiovasc Surg Int 2015;2(2):21-26
http://dx.doi.org/DOI: 10.5606/e-cvsi.2015.406
www.e-cvsi.org

Cardiovascular Surgery and Interventions

Original Article Open Access

Possible causes of major pleural effusion in early period after cardiac surgery

Safa Göde[1], Mehmet Yeniterzi[1], Mehmet Kaya[1], Muhammet Hulusi Satılmışoğlu[1], Salih Güler[1], Mehmet Gül[2], Serkan Arslan[2], Mugisha Markior Kyaruzi[1], Murat Sünbül[3]

Received: June 23, 2015 ***Accepted:*** July 02, 2015 ***Published online:*** August 03, 2015

ABSTRACT

Objectives: This study aims to investigate possible causes of major pleural effusion in the early period after cardiac surgery.

Patients and methods: Between January 2012 and June 2012; 72 patients with major pleural effusion study group as confirmed by the chest X-ray two weeks after surgery were included. The control group consisted of 68 age- and sex-matched patients with minor or no effusion. Coronary artery bypass graft (CABG), valve replacement (VR), and CABG in combination with VR, the use of internal mammary artery, total perfusion time (TPT), and aortic cross-clamp time (ACCT) were compared between the two groups. The use of antiaggregants, anticoagulants, and diuretics was analyzed.

Results: The development of pleural effusion was found higher in CABG and CABG in combination with VR patients than only VR patients (p=0.007). Among CABG patients, the development rate of pleural effusion was higher in patients with a mammary artery than those with a non-mammary artery (p=0.043). In study group, TPT (p=0.007) and ACCT (p=0.042) were higher than those without pleural effusion. Logistic regression analysis showed that CABG was responsible for the development of major pleural effusion.

Conclusion: Based on our study results, CABG patients seems to be potential candidates for the development of major pleural effusion compared to VR patients possibly due to pleurotomy, atelectasis, impaired lymphatic drainage, and reduced sternal blood flow. Extended extracorporeal circulation time may also play a role in the development of pleural effusion through inflammatory responses.

Keywords: Cardiac surgery; chest-X-ray; pleural effusion.

It is estimated that about 66.000 cases of cardiac surgery are performed annually in Turkey.[1] After coronary artery bypass grafting (CABG), pleural effusion develops in 41 to 87% of patients, as confirmed by chest X-ray.[2-5]

Pleural effusions are usually minor and unilateral, asymptomatic, and resolve spontaneously or by conservative treatment.[6,7] Pleural effusion may lead to prolonged hospital stay and patient discomfort. It may also cause complications such as empyema and atelectasis. Despite of being an important issue, the reasons of pleural effusion which requires a therapeutic intervention after cardiac surgery still cannot be fully explained.

In this study, we aimed to investigate possible causes of major pleural effusion in the early period after cardiac surgery.

PATIENTS AND METHODS

Between January 2012 and July 2012, 74 patients (study group) who had major pleural effusion in the control chest X-ray two weeks after surgery were included. Major pleural effusion was defined as pleural effusion covering >25% of hemithorax in the control chest X-ray. The control group consisted of age- and sex-matched 68 patients with minor or no pleural effusion. The majority of the patients in control group were symptomatic and required intervention. Patients who were reoperated for bleeding revision, those who underwent complex cardiac surgery other than CABG and valve replacement (VR), and those who had an ejection fraction less than 35% were excluded. Six of 74 patients were excluded from the study group and, therefore, a total of 68 patients were included in the study.

Age, sex, diabetes mellitus (DM), hypertension (HT), chronic obstructive pulmonary disease (COPD), smoking history, ejection fraction (EF),

Departments of [1]Cardiovascular Surgery and [2]Cardiology, İstanbul Mehmet Akif Ersoy Thoracic and Cardiovascular Surgery Training and Research Hospital, İstanbul, Turkey
[3]Department of Cardiology, Medical Faculty of Marmara University, İstanbul, Turkey

Corresponding author: Safa Göde, MD. İstanbul Mehmet Akif Ersoy Göğüs Kalp ve Damar Cerrahisi Eğitim ve Araştırma Hastanesi Kalp ve Damar Cerrahisi Kliniği, 34303 Küçükçekmece, İstanbul, Turkey.
Tel: +90 532 - 603 73 46 e-mail: safagode@yahoo.com

Table 1
Pleural effusion levels of both groups

	Chest X-ray finding	Description
Control group	0	Effusion covering only costophrenic angle
	1	Effusion covering <25% of hemithorax
Study group	2	Effusion covering 25 to 50% of hemithorax
	3	Effusion covering 50 to 75% of hemithorax
	4	Effusion covering >75% of hemithorax

platelet count, urea and creatinine values, and whether or not emergency surgery were compared statistically between two groups. Also, CABG, VR and CABG with VR patients were compared in terms of development of pleural effusion. During CABG, patients with left internal mammary artery (LIMA), LIMA and right internal mammary artery (RIMA) being used and without mammary artery being used were also compared. The degree of hypothermia, total perfusion time (TPT) and aortic cross-clamp time (ACCT) were compared in terms of the impact on pleural effusion. The use of furosemide, aldactazide, furosemide with aldactazide and without use of diuretics during postoperative period was analyzed. Furthermore, patients who used only acetyl salicylic acid (ASA), ASA with clopidogrel, and ASA with warfarin postoperatively were studied. Logistic regression analysis was performed to analyze the operation, mammary artery use, TPT and ACCT.

Statistical analysis was performed using the NCSS 2007 for Windows statistical software program (Number Cruncher Statistical System, Kaysville, Utah, USA). Standard descriptive statistical calculations were made (mean and standard deviation) and unpaired t test was used to compare normally distributed data. The Mann-Whitney U test was performed to analyze abnormally distributed data. The chi square test was performed to evaluate the qualitative data. Significant univariate clinical variables were included in a multivariate logistic regression model which predicted the increased risk of effusion. A p value of <0.05 was considered statistically significant.

RESULTS

The mean age was 57.4±11 and M/F ratio was 59/13 in control group. In study group mean age was 60.1±11.3 and M/F ratio 48/20. The baseline characteristics of both groups in terms of pleural effusion levels are shown in Table 1.

There were 63 CABG patients, eight VR patients, and one CABG with VR patients in control group, while there were 52 CABG patients, five VR patients, and 11 CABG with VR patients in study group. The development of pleural effusion was significantly higher in CABG and CABG with VR patients compared to VR patients alone (p=0.007) (Table 2). During CABG, the number of patients who used LIMA, LIMA with RIMA and non-used both of them (only saphenous vein used) were 52, 2, and 18, respectively in the control group. These numbers were 60, 0, and 8, respectively in study group. The development rate of pleural effusion was significantly higher in LIMA and LIMA with RIMA used patients, than saphenous vein used patients alone (p=0.043) (Table 3).

In addition, TPT and ACCT in the control group were 85±27.5 min, and 47.8±23.6 min respectively,

Table 2
Comparison the groups in terms of operations

Operation	Control group		Study group		
	n	%	n	%	p
Coronary artery bypass graft	63	87.50	52	76.47	0.007†
Valve replacement	8	11.11	5	7.35	
Coronary artery bypass graft + valve replacement	1	1.39	11	16.18	

†: Statistically significant.

Table 3
Comparison of the groups in terms of grafts used

Greft	Control group		Study group		
	n	%	n	%	*p*
Only saphenous vein	18	25.00	8	11.76	0.043†
Left internal mammary artery (+)	52	72.22	60	88.24	
Left internal mammary artery + right internal mammary artery	2	2.78	0	0	

†: Statistically significant.

Table 4
Comparison of the groups in terms of peroperative variables

	Control group	Study group	
	Mean±SD	Mean±SD	*p*
Hypothermia °C	30.5±1.33	30.47±1.78	0.911
Total perfusion time (min)	84.97±27.45	101.53±42.76	0.007†
Aortic cross-clamp time (min)	47.76±23.61	57.91±34.26	0.042†

SD: Standard deviation; †: Statistically significant

while in the study group, these values were 101.5±42.8 min and 57.9±34.3 min, respectively. Also, TPT (p=0.007) and ACCT (p=0.042) were significantly lower in the control group, compared to the study group (Table 4). Although these parameters were found to cause the development of pleural effusion, there were no effects on the development of pleural effusion in the multivariate analysis in terms of TPT and ACCT.

There was no statistically significant difference between the two groups in terms of age, sex, DM, HT, COPD, smoking history, whether or not the emergency operation, EF, platelet count, urea and creatinine values (Table 5).

The mean hypothermia during surgery in the control group was 30.5±1.3 °C, while it was 30.5±1.8 °C in the study group. There was no statistically significant

Table 5
Comparison of the groups in terms of demographic characteristics

Demographic characteristics	Control group			Study group			
	n	%	Mean±SD	n	%	Mean±SD	*p*
Age			57.4±11.0			60.9±11.3	0.064
Gender							
Male	59			13			
Female	48			20			0.167
Body mass index	28.5	27.9					0.460
Diabetes mellitus	20	27.78		24	35.29		0.338
Hypertension	51	75.00		51	70.83		0.58
Chronic obstructive pulmonary disease	12	16.67		15	22.06		0.419
Smoking (pocket/year)			21.4±18.4			19.9±19.6	0.652
Ejection fraction (%)			53.7±9.4			52.6±9.7	0.516
Platelet count (n/mm^3)			265.9±113.4			267.2±79.9	0.938
Urea (mg/dL)			18.0±9.6			18.9±9.43	0.566
Creatinin (mg/dL)			1.1±0.8			1.0±0.3	0.399
Emergency	6	8.33		9	13.24		0.349

SD: Standard deviation.

Table 6
Comparison of the groups in terms of postoperative antiaggregant/anticoagulant use

Postoperative antiaggregant/anticoagulant use	Control group		Study group		
	n	%	n	%	*p*
Acetyl salicylic acid	51	70.83	40	58.82	0.189
Acetyl salicylic acid + warfarin	9	12.50	13	19.12	0.299
Acetyl salicylic acid + clopidogrel	12	16.67	15	22.06	0.401

difference in hypothermia between the two groups (Table 4).

When comparing the two groups in terms of using ASA, ASA with warfarin and ASA with clopidogrel which were prescribed during discharging, there was no statistically significant difference between the groups (Table 6). Also, there was no significant difference in the use of furosemide, aldactazide, and furosemide with aldactazide between the groups (Table 7). Also, CABG was found to be the main factor for the development of major pleural effusion (Table 8).

DISCUSSION

Most of the effusions after CABG are minor, left-sided, and regress spontaneously. A small part of pleural effusions become permanent. Patients with pleural effusion had significantly longer ICU and hospital stays and experienced higher rates of complications than those without pleural effusion.[8]

Yıldırım et al.[9] showed that four of 62 patients (6.45%) with pleural effusion after cardiac surgery required toracotomy and decortication operation for pleural thickening. It suggests the importance of pleural effusion after cardiac surgery. In addition, Labidi et al.[8] demonstrated that peripheral arterial disease, atrial fibrillation, heart failure, and some anticoagulants may lead to symptomatic pleural effusion. The authors reported that older patients and high serum creatinine level patients had symptomatic pleural effusion. In our study, however, we found no significant difference in these preoperative parameters between the patients with major pleural effusion and

Table 7
Comparison of the groups in terms of diuretics

Diuretic	Control group		Study group		
	n	%	n	%	*p*
No diuretic	64	88.89	60	88.24	0.229
Furosemide	1	1.39	5	7.35	0.209
Aldactazide	7	9.72	2	2.94	0.241
Furosemide + aldactazide	0	0	1	1.47	0.981

Table 8
Logistic regression analysis of variables

Surgical data	Odds ratio	95% CI		*p*
		Lower	Upper	
Coronary artery bypass graft	0.03	0.002	0.39	0.027†
Valve replacement	0.21	0.017	2.66	0.229
Coronary artery bypass graft and valve replacement	0.15	0.00	0.97	0.008†
Left internal mammary artery usage	0	0	0.001	0.297
Left internal mammary artery + right internal mammary artery usage	0	0	0.001	0.998
Total perfusion time (min)	1.02	0.99	1.05	0.998
Aortic cross-clamp time (min)	0.99	0.95	1.02	0.116

CI: confidence interval.

those with minor or no pleural effusion. Besides, elective surgeries were significantly less associated with pleural effusions in Labidi's[8] study. However, we were unable to detect any difference in the development of pleural effusion between elective and urgent patients. The differences between these two studies can be explained by the fact that Labidi's study included only symptomatic or patients requiring an intervention, while our patients with minimal pleural effusion were included in the same group with non-effusion patients. Therefore, some variables of the patients with pleural effusion might have changed the results in favor of the group with effusion.

In another study, Light et al.[2] performed chest radiographs 28 days after surgery and showed a significantly higher rate of pleural effusions among patients undergoing either CABG surgery (63%) or combined CABG and valve surgery (62%) than those undergoing VR alone (45%). Labidi et al.[8] concluded that VR was more strongly associated with postoperative pleural effusions than CABG. However, we found that there were significantly higher rates of pleural effusion in CABG and CABG with VR patients than VR patients alone, similar to the Light's study findings (p=0.007). Unlike our study results, Light's study included only pure non-effusion patients in the control group. However, it did not change the results. In addition, pleural effusion values in CABG patients may be contributed by TPT length and pleural trauma. There were five VR patients in the study group. This may be caused by an inflammatory response triggered by cardiopulmonary bypass (CPB). Moreover, in CABG patients whose LIMA or LIMA with RIMA was used had a high pleural effusion rate than those whose only saphenous vein was used in our study. In addition, Hurlbut et al.[5] reported an incidence of left pleural effusion of 84% for on the sixth postoperative day following internal mammary artery (IMA) grafting compared with an incidence of 47% after saphenous vein grafting. Similarly, Yıldırım et al.[9] obtained a strong correlation between pleural effusion and IMA harvesting. There is a number of studies reporting similar results in the literature.[5,10] Christakis et al.[11] revealed that there was no difference in the development of pleural effusion between the use of mammary and saphenous vein groups. The causes of pleural effusion may be atelectasis, impaired lymphatic drainage, decreased sternal blood supply, and pleurotomy in mammary artery harvested patients.[6]

Furthermore, Payne et al.[12] found no differences related to TPT and ACCT between pleural effusion and without pleural effusion. Similarly, we found that TPT and ACCT had statistically no effect on the development of pleural effusion, based on the logistic regression analysis. Wynne and Botti[13] showed that use of CPB had clear negative consequences on postoperative pulmonary function. They compared CPB patients with other types of major surgery in terms of pulmonary function and were found more frequent lung injury and delayed pulmonary recovery in CPB patients. Extracorporeal bypass circuit time may also contribute to the increased complication by causing an inflammatory response. Therefore, the pulmonary dysfunction is thought to be due to effects of an acute systemic and pulmonary inflammatory response commonly referred to as "pump lung" [14] or "post-pump syndrome."[15]

Additionally, there was no difference in the use of several medications including ASA, ASA with clopidogrel and ASA with warfarin between patients with major pleural effusion and minor or no pleural effusion who were prescribed with variable medication as such. As a result, antiaggregants and anticoagulants were shown to have no effect on developing pleural effusion. On the other hand, pleural effusion was not found to be less than the patients prescribed with several diuretics such as furosemide, aldactazide, furosemide with aldactazide. Therefore, diuretic treatment which was prescribed to a discharged patient had no effect on the prevention of pleural effusion.

In conclusion, clinicians should be more alert to the development of pleural effusion in CABG patients than VR patients, as CABG and CABG with VR patients are more potential candidate for the development of major pleural effusion than VR patients alone. Major pleural effusion plays also an important role for mortality and morbidity, as they are caused by complications such as atelectasis and empyema in CABG patients. Prevention of some complications related to major pleural effusion after cardiac surgery should be considered in some variables such as mammary artery harvesting.

Declaration of conflicting interests

The authors declared no conflicts of interest with respect to the authorship and/or publication of this article.

Funding

The authors received no financial support for the research and/or authorship of this article.

REFERENCES

1. Kervan U, Koç O, Ozatik MA, Bayraktar G, Şener E, Çağlı K, et al. Distribution and service quality of the cardiovascular surgery clinics in Turkey. Turk Gogus Kalp Dama 2011;19:483-9.
2. Light RW, Rogers JT, Moyers JP, Lee YC, Rodriguez RM, Alford WC Jr, et al. Prevalence and clinical course of pleural effusions at 30 days after coronary artery and cardiac surgery. Am J Respir Crit Care Med 2002;166:1567-71.
3. Vargas FS, Cukier A, Terra-Filho M, Hueb W, Teixeira LR, Light RW. Relationship between pleural changes after myocardial revascularization and pulmonary mechanics. Chest 1992;102:1333-6.
4. Peng MC, Hou CJ, Li JY, Hu PY, Chen CY. Prevalence of symptomatic large pleural effusions first diagnosed more than 30 days after coronary artery bypass graft surgery. Respirology 2007;12:122-6.
5. Hurlbut D, Myers ML, Lefcoe M, Goldbach M. Pleuropulmonary morbidity: internal thoracic artery versus saphenous vein graft. Ann Thorac Surg 1990;50:959-64.
6. Peng MJ, Vargas FS, Cukier A, Terra-Filho M, Teixeira LR, Light RW. Postoperative pleural changes after coronary revascularization. Comparison between saphenous vein and internal mammary artery grafting. Chest 1992;101:327-30.
7. Rolla G, Fogliati P, Bucca C, Brussino L, Di Rosa E, Di Summa M, et al. Effect of pleurotomy on pulmonary function after coronary artery bypass grafting with internal mammary artery. Respir Med 1994;88:417-20.
8. Labidi M, Baillot R, Dionne B, Lacasse Y, Maltais F, Boulet LP. Pleural effusions following cardiac surgery: prevalence, risk factors, and clinical features. Chest 2009;136:1604-11.
9. Yıldırım M, Dogusoy I, Demirbag H, Ustaalioglu R Management of pleural effusions developing after open heart surgery. Turk Gogus Kalp Dama 2013;21:982-6.
10. Landymore RW, Howell F. Pulmonary complications following myocardial revascularization with the internal mammary artery graft. Eur J Cardiothorac Surg 1990;4:156-61.
11. Christakis GT, Weisel RD, Buth KJ. Is body size the cause for poor outcomes of coronary artery bypass operations in women? J Thorac Cardiovasc Surg 1995;110:1344-56.
12. Payne M, Magovern GJ Jr, Benckart DH, Vasilakis A, Szydlowski GW, Cardone JC, et al. Left pleural effusion after coronary artery bypass decreases with a supplemental pleural drain. Ann Thorac Surg 2002;73:149-52.
13. Wynne R, Botti M. Postoperative pulmonary dysfunction in adults after cardiac surgery with cardiopulmonary bypass: clinical significance and implications for practice. Am J Crit Care 2004;13:384-93.
14. Conti VR. Pulmonary injury after cardiopulmonary bypass. Chest 2001;119:2-4.
15. Matthay MA, Wiener Kronish JP. Respiratory management after cardiac surgery. Chest 1989;95:424-34.

Cardiovasc Surg Int 2015;2(2):27-29
http://dx.doi.org/DOI: 10.5606/e-cvsi.2015.374
www.e-cvsi.org

Cardiovascular Surgery and Interventions

Case Report Open Access

Treatment of catheter entrapment during transradial approach in a patient with vasospastic angina

Ertuğrul Zencirci[1], Aleks Değirmencioğlu[1], Aycan Esen Zencirci[2], Gültekin Karakuş[1], Ahmet Akyol[1]

Received: March 25, 2015 ***Accepted:*** May 15, 2015 ***Published online:*** August 03, 2015

ABSTRACT

In this report, we present a 27-year-old male patient who was scheduled for an urgent left heart catheterization due to resting angina pectoris with ST segment elevation in anterior precordial leads. Catheter entrapment occurred due to severe vasospasm of radial artery during transradial coronary angiography which showed coronary spasm treated with intracoronary nitroglycerin. Reversal of severe vasospasm and release of entrapped catheter were unable to be achieved until forearm heating. This case report is presented to demonstrate that forearm heating may effectively reverse severe and resistant vasospasm of radial artery during a transradial intervention in patients with vasospastic angina.

Keywords: Heating; transradial approach; vasospastic angina.

Transradial approach has become increasingly popular and has been shown to decrease the incidence of access-site complications compared to the transfemoral approach.[1] Most of the previous studies revealed the risk factors and management of radial artery spasm, which is the most frequent complication during transradial approach.[2,3] However; radial artery spasm-related catheter entrapment during transradial cardiovascular interventions is extremely rare and its management has not been precisely defined.

CASE REPORT

A 27-year-old man was admitted to our emergency department with resting angina pectoris. He was a heavy smoker. Twelve-lead electrocardiography (ECG) showed, ST segment elevation in the anterior precordial leads. His physical examination findings were non-specific. His height was 172 cm and with a weight of 75 kg. He was scheduled for an urgent left heart catheterization. A 6F-short (7 cm) hydrophilic sheath Radiofocus Introducer II (Terumo, Tokyo, Japan) was introduced into the right radial artery. Nitroglycerin 200 µg and heparin 5,000 U were injected through the side port of the sheath. Guiding 6F left Judkins catheter (Launcher, Medronic, Minneapolis, USA) was inserted through the sheath to the aortic root over a hydrophilic guidewire without any difficulty. The left coronary angiography demonstrated left anterior descending coronary artery spasm causing %90 narrowing of the luminal diameter. After intracoronary injection of nitroglycerin 200 µg, coronary spasm was relieved and it was considered as vasospastic angina (Figure 1).

During removal of the guiding catheter, the patient suffered from severe right forearm pain distal to antecubital fossa. Further manipulation of the catheter was not possible. On fluoroscopy, the catheter was not looped or kinked. Severe vasospasm of radial artery was thought to cause catheter entrapment. Intravenous verapamil 5 mg waiting and re-trying, nitroglycerin infusion, and midazolam 2 mg all failed. After forearm heating by Warm Touch, model WT-5300 A convective air patient warming system (Covidien, Mansfield, USA), we removed the catheter without any difficulty. Finally, we performed a forearm angiography which showed mild vasospasm of radial artery persisting after catheter removal (Figure 2). Following the removal of the sheath, an inflatable hemostasis device was introduced. Radial artery was found to be patent the day after the procedure. The patient was free of angina throughout a six-month follow-up period.

[1]Department of Cardiology, Acıbadem Maslak Hospital, İstanbul, Turkey
[2]Department of Cardiology, Siyami Ersek Cardiovascular and Thoracic Surgery Training and Research Hospital, İstanbul, Turkey

Corresponding author: Ertuğrul Zencirci, M.D. Acıbadem Maslak Hastanesi, Kardiyoloji Bölümü, 34457 Maslak, İstanbul, Turkey.
Tel: +90 212 - 204 43 62 e-mail: ertuzencirci@gmail.com

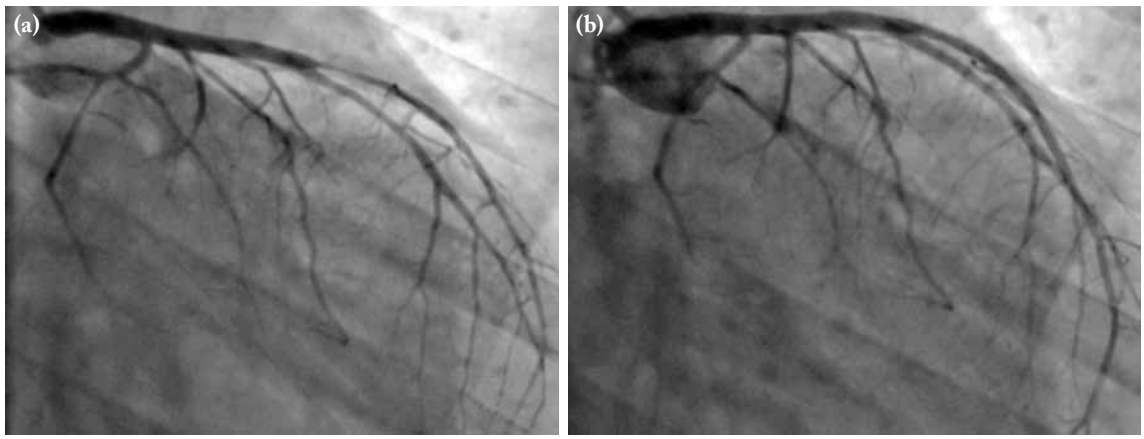

Figure 1. **(a)** Left coronary angiography image showing the left anterior descending coronary artery spasm causing 90% narrowing of the luminal diameter in the right caudal projection. **(b)** After intracoronary nitroglycerin injection, coronary spasm was relieved.

DISCUSSION

This case described the successful treatment of catheter entrapment due to severe radial artery spasm that accompanies vasospastic angina with a simple and effective non-pharmacological technique. Radial artery is mainly composed of smooth muscle cells together with alpha-adrenoceptor-predominance which explains the specific susceptibility to vasospasm.[4] Circulating catecholamines mainly activate alpha-1-adrenoreceptor. Patient-related factors such as small size and anomalous origin of the radial artery, female sex, younger age, short stature, and anxiety were defined as predisposing factors for radial artery spasm during transradial approach.[3] In the present report, our case was young and suffered from anxiety. The majority of previous reports advocated nitrate derivative or verapamil to prevent spasm of radial artery; however, there is currently no consensus on the optimal agents.[3] As in our case, we use intra-arterial nitroglycerin for such patients on a regular basis.

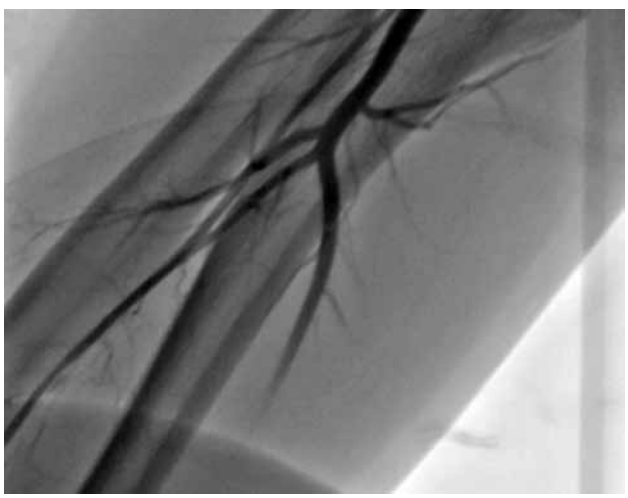

Figure 2. Recent forearm angiography image visualizing mild vasospasm of the radial artery following catheter removal.

In addition, heating has been shown to be a stimulus evoking the conduit artery dilatation. Previously, it has been demonstrated that heating may induce radial artery vasodilation through the flow-mediated dilatation.[5,6] Increased local temperature has been also documented to modulate alpha-1-adrenergic receptor-mediated vasoconstriction by increasing the release of endothelial cell-derived vasodilators.

Moreover, several studies have suggested hyperreactivity to adrenergic stimulation triggering coronary artery spasm in patients with vasospastic angina. Type A behavior pattern, severe anxiety and panic disorders were described as some of the predisposing factors for vasospastic angina. Previous studies have suggested that vasospastic angina is associated with migraine and Raynaud phenomenon.[7,8] These observations have stimulated the hypothesis that some of the patients with vasospastic angina have generalized vasospastic disorder.[7,8] The predisposing factors and hyperreactivity to adrenergic stimulation may account for the common underlying mechanism for vasospastic angina and radial artery spasm. Radial artery spasm may be also considered as a part

of the clinical presentation of generalized arterial vasospasm. However, definitive evidences supporting such an association are not available. As it may be a coincidental catheter-induced radial artery spasm. Further studies are warranted to establish a conclusion.

In conclusion, we suggest that forearm heating may effectively reverse severe and pharmaco-resistant vasospasm of radial artery during a transradial intervention in patients with vasospastic angina.

Acknowledgement

We would like to thank Osman Konencoglu, catheterization lab technician for his contribution to the present report.

Declaration of conflicting interests

The authors declared no conflicts of interest with respect to the authorship and/or publication of this article.

Funding

The authors received no financial support for the research and/or authorship of this article.

REFERENCES

1. Jolly SS, Amlani S, Hamon M, Yusuf S, Mehta SR. Radial versus femoral access for coronary angiography or intervention and the impact on major bleeding and ischemic events: a systematic review and meta-analysis of randomized trials. Am Heart J 2009;157:132-40.
2. Kiemeneij F, Vajifdar BU, Eccleshall SC, Laarman G, Slagboom T, van der Wieken R. Evaluation of a spasmolytic cocktail to prevent radial artery spasm during coronary procedures. Catheter Cardiovasc Interv 2003;58:281-4.
3. Ho HH, Jafary FH, Ong PJ. Radial artery spasm during transradial cardiac catheterization and percutaneous coronary intervention: incidence, predisposing factors, prevention, and management. Cardiovasc Revasc Med 2012;13:193-5.
4. He GW, Yang CQ. Characteristics of adrenoceptors in the human radial artery: clinical implications. J Thorac Cardiovasc Surg 1998;115:1136-41.
5. Joannides R, Costentin A, Iacob M, Compagnon P, Lahary A, Thuillez C. Influence of vascular dimension on gender difference in flow-dependent dilatation of peripheral conduit arteries. Am J Physiol Heart Circ Physiol 2002;282:H1262-9.
6. Bellien J, Thuillez C, Joannides R. Role of endothelium-derived hyperpolarizing factor in the regulation of radial artery basal diameter and endothelium-dependent dilatation in vivo. Clin Exp Pharmacol Physiol 2008;35:494-7.
7. Wayne VS. A possible relationship between migraine and coronary artery spasm. Aust N Z J Med 1986;16:708-10.
8. Miller D, Waters DD, Warnica W, Szlachcic J, Kreeft J, Théroux P. Is variant angina the coronary manifestation of a generalized vasospastic disorder? N Engl J Med 1981;304:763-6.

Cardiovasc Surg Int 2015;2(2):30-32
http://dx.doi.org/DOI: 10.5606/e-cvsi.2015.299
www.e-cvsi.org

Cardiovascular Surgery and Interventions

Case Report Open Access

An asymptomatic huge calcified intramyocardial hydatid cyst: a case report

Mehmet Beşir Akpınar, Veysel Şahin, Serkan Seçici, İhsan Sami Uyar, Mehmet Ateş

Received: November 13, 2014 ***Accepted:*** April 18, 2015 ***Published online:*** August 03, 2015

ABSTRACT

Hydatid cyst disease is rarely asymptomatic depending on its location. A 37-year-old man presented asymptomatically and was diagnosed with a hydatid cyst incidentally during regular check-up. Echocardiography and cardiac magnetic resonance angiography images demonstrated a calcified solid cystic lesion (90x60 mm) on the apicoposterolateral region of the left ventricle. The cyst was localized inside the myocardial fibers. The left ventricular cavity size was reduced due to the bulging of the mass. The cyst was evacuated with open heart surgery and the large calcified cavity was closed carefully.

Keywords: Cardiac hydatid cyst; *echinococcosis*; myocardial calcification.

Hydatid cyst is an endemic infestation disease in various regions in the world. *Echinococcus granulosus*, the causative agent of cystic hydatid disease, usually (60-70%) reaches the liver via intestinal veins or lymphatics. If embryos bypass the liver and the lung, they reach the systemic circulation and may affect any organ of the body. Cardiac involvement, which is rare, is between 0.02% and 2% of all hydatid diseases.[1,2]

The embryos can reach the myocardium via coronary circulation from the left side of the heart. The most common location of cardiac hydatid cysts are the left ventricle, interventricular septum, and right ventricle.[3-5] Cardiac symptoms (mostly chest pain, shoulder pain, dyspnea, and persistent cough) usually depend on the localization and size of the cyst.

The cyst may also grow between cardiac fibers without causing any symptoms. If it reaches a reasonable size, fever, palpitation, arrhythmias, and heart failure may develop. The most critical complication of a cardiac cyst is perforation with a high incidence ranging between 25% and 40%.[6] After perforation of the cyst, 75% of patients die from septic shock or embolic complications.[6]

CASE REPORT

A 37-year-old man presented with cardiac murmur without any clinical sign. He was asymptomatic with a non-specific medical history. Physical examination revealed 3/6 systolic murmur at the second right intercostal parasternal space.

Transthoracic echocardiographic examination revealed that an intramyocardial mass involved the inferior, posterior, and free lateral walls of the left ventricle, and severe inferoposterolateral myocardial hypertrophy. The nature of the mass was solid and there were necrotic tissues inside the mass. The left ventricular end-systolic and end-diastolic diameters were reduced. The rest of the echocardiographic variables were unremarkable.

Teleradiography showed an enlarged mediastinum and displaced cardiac apex superiorly and to the left side of the chest. Chest X-ray also revealed a calcified myocardial mass in the apical part of the heart (Figure 1). A smooth-surfaced calcified cystic mass of 90x60 mm at the left ventricular lateral wall was also seen on cardiac magnetic resonance angiography. The mass was localized inside the myocardial fibers. There was no contrast intake inside the mass; the left ventricular volume was severely decreased due to bulging of the mass (Figure 2). This huge mass was suspected for malignancy.

There was also an 18 mm diameter calcified cystic mass in the liver. No other cyst was detected in the body of the patient. His blood serology test results were negative. Based on these findings, the patient underwent open heart surgery.

Department of Cardiovascular Surgery, Medical Faculty of Şifa University, İzmir, Turkey

Corresponding author: Mehmet Beşir Akpınar, M.D. Şifa Üniversitesi Tıp Fakültesi, Kalp ve Damar Cerrahisi Anabilim Dalı, 35100 Bayraklı, İzmir, Turkey. Tel: +90 232 - 446 08 80 e-mail: mbakpinar@hotmail.com

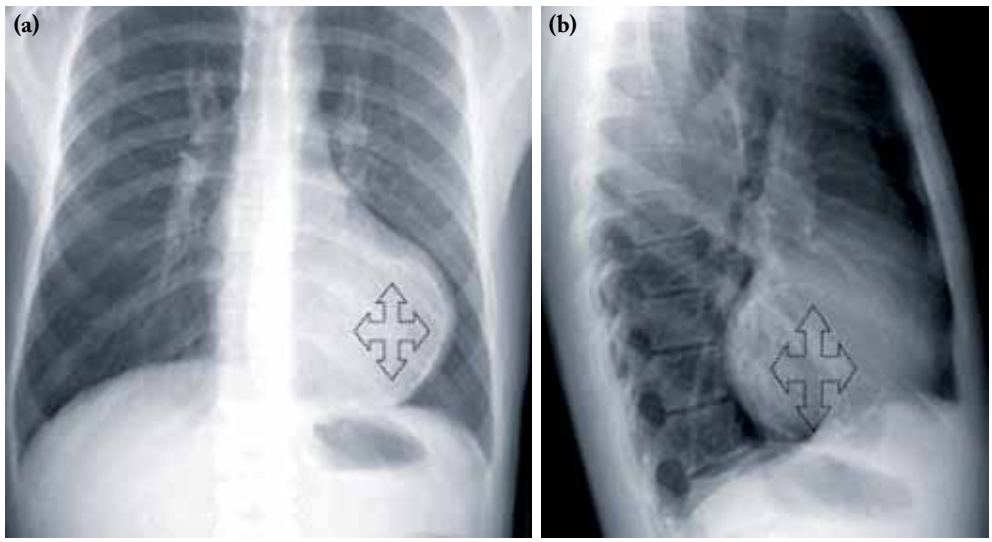

Figure 1. Preoperative X-ray of thoraces. **(a)** Anteroposterior, **(b)** left lateral X-ray images of the patient. The calcified borders of the cyst can be seen in apical part of the heart (two headed arrows).

The operation was performed through median sternotomy. Cardiopulmonary bypass through the aorto-bicaval cannulation was instituted. Cardiac arrest was induced by administration of intermittent cold blood cardioplegia. Systemic and topical cooling with cold saline slush were also performed. The mass was located at the apicoposterolateral wall of the heart. The cyst was opened by performing an incision into the apical region of the heart. 300 mL gel consistency, white necrotic tissue, and germinative membrane were evacuated. Samples were taken for pathological examination and culture inoculation. The cavity was washed thoroughly with 20% hypertonic saline solution and a 1% iodine solution. There was no communication with the ventricular cavity. A crater 90 mm in diameter was formed due to the calcified nature of the wall of the cavity (Figure 3). Its diameter and calcified wall were not convenient to capitonnage or primer closure. Therefore, the cavity was filled with three pieces of gelatin sponge (Clinisponge®; Yücel Medikal Ltd. Şti. Esenyurt, İstanbul, Turkey). Then, we used a Teflon patch (felt) and sutured the free edges of the cavity. The patient had an uneventful recovery (Figure 4).

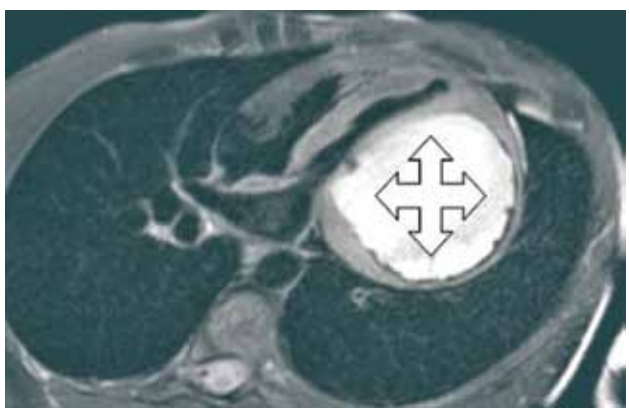

Figure 2. Cardiac magnetic resonance angiography image of the mass (two headed arrows).

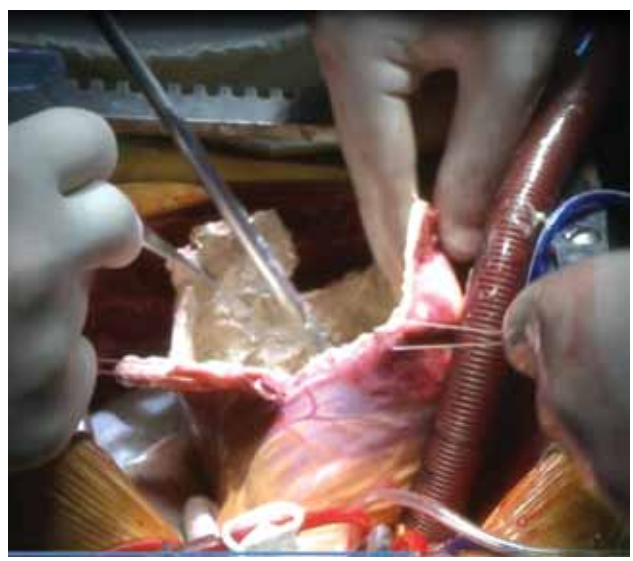

Figure 3. The crater of the huge cyst. The cyst was not connected with the left ventricular cavity.

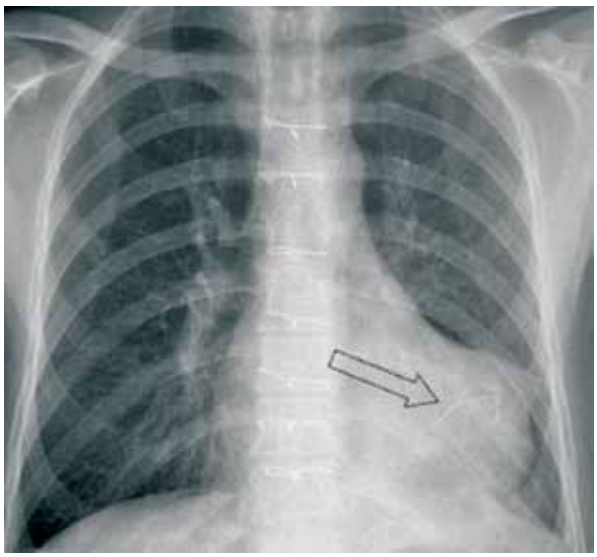

Figure 4. Postoperative X-ray. The calcified border of the cyst can be seen (arrow).

Gross and microscopic pathological examination confirmed the diagnosis of germinative membrane. Preoperative albendazole treatment (10 mg/kg/day) was continued for four weeks after surgery.

DISCUSSION

Cardiac hydatid cysts are often asymptomatic in the early stages. Our case had a 90x60 mm intramyocardial cyst and was surprisingly asymptomatic and diagnosed incidentally during the check-up. During surgery, we observed that the cyst was localized between the myocardial fibers and half of the left ventricular wall was pushed to the free edges of the mass.

Ideally, an echinococcal cyst should be aspirated, evacuated, and germinative membrane should be removed. Then, the remaining capsule can be closed with capitonnage technique.[5] However, in our case, the cyst was not alive; it did not contain any fluid inside the cyst. The content of the globe was necrotic, like a gel consistency. The borders of the globe were calcified. The wide base of the cyst was consisted from a tiny wall of the left ventricle. Removing of this calcified wall would severely reduce the ventricle volume due to the huge volume of the cyst. The cavity was not able to be closed with capitonnage technique (Figure 3). As a result, we filled the cavity with pieces of absorbable gelatin sponge to support cardiac geometry and reduce the risk of rupture from the base of the cavity. The patient recovered uneventfully and is still under follow-up.

Conclusion

During surgical excision of a huge, calcified, intramyocardial cardiac cyst hydatid with a wide base, left ventricular muscle mass should be preserved to avoid irreversible intraoperative left ventricular dysfunction. Therefore, the cavity of cyst after evacuation can be filled with gelatin sponge instead of excision to support left ventricular free wall.

Acknowledgements

We thank to Betül Doğan who is a college student for collecting data's and arranging the images of the case.

Declaration of conflicting interests

The authors declared no conflicts of interest with respect to the authorship and/or publication of this article.

Funding

The authors received no financial support for the research and/or authorship of this article.

REFERENCES

1. Shevchenko YL, Travin NO, Musaev GH, Morozov AV. Heart echinococcosis: current problems and surgical treatment. Multimed Man Cardiothorac Surg 2006;2006:810.
2. Eckert J, Deplazes P. Biological, epidemiological, and clinical aspects of echinococcosis, a zoonosis of increasing concern. Clin Microbiol Rev 2004;17:107-35.
3. Kardaras F, Kardara D, Tselikos D, Tsoukas A, Exadactylos N, Anagnostopoulou M, et al. Fifteen year surveillance of echinococcal heart disease from a referral hospital in Greece. Eur Heart J 1996;17:1265-70.
4. Kocogullari CU, Kocaturk H, Erkut B, Kocak H. An apical cardiac hydatid cyst. Turk Gogus Kalp Dama 2010;18:132-4.
5. Yildirim F, Tuncer B, Kurdal AT, Ozturk T, Iskesen I. A rare location for cardiac hydatid cyst in the interventricular septum. Cardiovasc Surg Int 2014;1:3-5.
6. Unlü Y, Ceviz M, Karaoğlanoğlu N, Becit N, Koçak H. Arterial embolism caused by a ruptured hydatid cyst in the heart: report of a case. Surg Today 2002;32:989-91.

Cardiovasc Surg Int 2015;2(2):33-35
http://dx.doi.org/DOI: 10.5606/e-cvsi.2015.230
www.e-cvsi.org

Cardiovascular Surgery and Interventions

Case Report Open Access

Iatrogenic diversion of superior vena cava to left atrium after surgical repair of an atrial septal defect

Muhammet Akyüz, Onur Işık, Fatih Ayık, Yüksel Atay

Received: July 18, 2014 ***Accepted:*** April 18, 2015 ***Published online:*** August 03, 2015

ABSTRACT

Iatrogenic diversion of the superior vena cava (SVC) into the left atrium during closure surgery for an atrial septal defect (ASD) is an extremely rare complication. Herein, we describe a 34-year-old woman with cyanosis who was surgically treated for ASD accompanied by partial anomalous pulmonary venous drainage one year prior. Following re-opening of the atrial defect, iatrogenic diversion of the SVC into the left atrium was intraoperatively identified. A Gore-Tex patch was inserted to divert the flow to the correct atrium.

Keywords: Atrial septal defect; iatrogenic; cyanosis; superior vena cava.

Partial anomalous pulmonary venous connection (PAPVC) to the right side of the heart often complicates surgery for atrial septal defects (ASD).[1] Although obstruction of the pulmonary venous drainage and superior vena cava (SVC) inflow are rarely seen after repair of ASD, iatrogenic diversion of the SVC into the left atrium (LA) has not been reported in the literature. Herein, we report an unusual case of iatrogenic diversion of the SVC to the LA after closure of a sinus venosus ASD of the SVC type.

CASE REPORT

A 34-year-old female patient was admitted to our clinic one year after surgical repair of the SVC type of sinus venosus ASD with complaints of mild exertional dyspnea and cyanosis of extremities with exertion. Her medical history revealed an open heart surgery at another center one year prior. On clinical examination, the patient had central cyanosis. Her clinical vital signs were normal and her arterial oxygen saturation was 85% on room air. Auscultation revealed no abnormality. Electrocardiography showed that the patient was in sinus rhythm with left axial deviation. A chest X-ray demonstrated cardiomegaly with clear lung fields. Transthoracic echocardiography (TTE) showed no leakage in the ASD patch. The inferior vena cava (IVC) was seen draining normally into the right atrium (RA). However, the SVC flow draining to the RA was unable to be definitely documented. Contrast echocardiography of the right antecubital vein was performed. The immediate contrast enhancement was achieved in the LA and the inter-atrial septum was intact. The SVC draining to the RA was not observed (Figure 1). Computed tomography (CT) angiography revealed SVC drainage to the LA with a non-obstructed flow and intact inter-atrial septum. The right upper pulmonary veins were also seen to drain to the SVC (Figure 2).

An informed consent was obtained from the patient. Re-median sternotomy and aortic-bicaval (selective SVC) cannulation were performed, and cardiopulmonary bypass was initiated. In the inspection of the venous anatomy, the right upper pulmonary veins drained into the SVC. After aortic cross-clamping and antegrade blood cardioplegia administration, right atriotomy was performed. The SVC ostium was unable to be visualized and a small residual ASD at the right side of the SVC into the RA was observed. After enlargement of the atrial defect, the pulmonary venous and systemic venous drainage abnormalities were identified. A Gore-Tex patch (W. L. Gore & Associates, Inc., Flagstaff, AZ) was inserted to divert the flow to the correct atrium. Contrasted transesophageal echocardiography (TEE) showed no residual defect (Figure 3).

The patient's oxygen saturation improved to 98-100% after surgery. Following an uneventful

Department of Cardiovascular Surgery, Medical Faculty of Ege University, İzmir, Turkey

Corresponding author: Muhammet Akyüz, M.D. Ege Üniversitesi Tıp Fakültesi Kalp ve Damar Cerrahisi Anabilim Dalı, 35100 Bornova, İzmir, Turkey.
Tel: +90 505 - 943 79 89 e-mail: drmak100@gmail.com

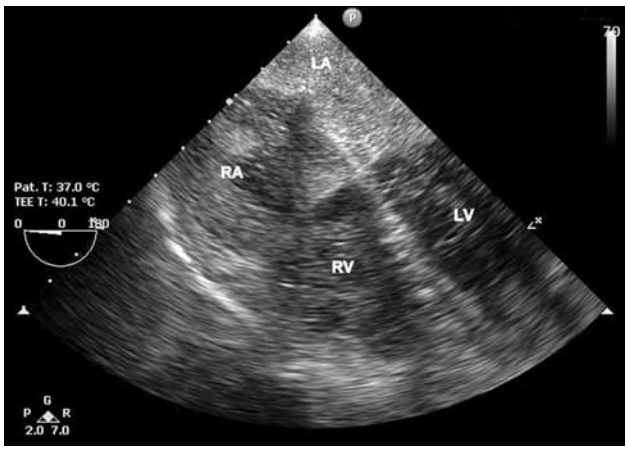

Figure 1. A preoperative transesophageal echocardiography image with contrast injected into the jugular vein. Contrast enhancement in the left atrium.

recovery, the patient was discharged on the postoperative fifth day. In the first follow-up visit at one month, she was symptom-free and in sinus rhythm and repeated electrocardiography (ECG) showed a normal right and left atrial anatomy with normal flow of SVC and pulmonary veins.

DISCUSSION

Partial anomalous pulmonary venous connection repair is a surgical procedure in which the pulmonary venous returns are separated from the systemic venous returns.[1] During this procedure, it is essential to consider the close association of the abnormal pulmonary veins with the IVC or SVC.[2] If it fails, iatrogenic systemic venous return anomalies and pulmonary venous return problems may occur. In addition, improper repair of those defects may result in significant surgical morbidity such as obstruction of the pulmonary vein orifices, SVC stenosis or obstruction, sinus node injury, and atrial bradyarrhythmias.[3]

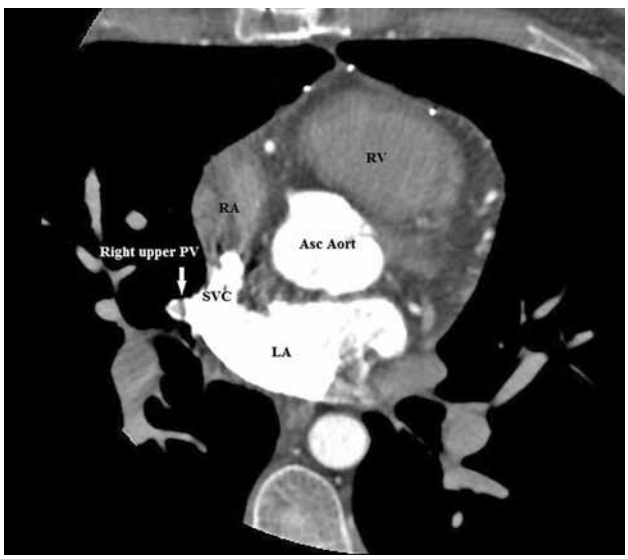

Figure 2. A computed tomography angiography image of the heart. The image showing an anomalous course of contrast from the superior vena cava to the left atrium. RV: Right ventricle; RA: Right atrium; PV: Pulmonary vein; Asc aort: Ascending aorta; SVC: Superior vena cava; LA: Left atrium.

Iatrogenic diversion of the IVC following repair of ASD is an uncommon complication of surgical repair of sinus venosus ASD with or without anomalous pulmonary venous drainage.[4] The presence of large secundum or sinus venosus type ASD or abnormal pulmonary venous return may increase the complication risk related to pulmonary or systemic venous return.[4]

Furthermore, reported cases with iatrogenic systemic venous anomaly presented primarily with cyanosis and hypoxia, which may occur immediately postoperatively or months to years after operation.[5-7] Until now, iatrogenic diversion of the SVC has not been reported in the literature. Our case presented with mild exertional dyspnea and cyanosis of extremities with exertion.

These types of serious complications can be prevented by some approaches in simple surgeries such as ASD with low morbidity and mortality rates. Therefore, it is critical to perform an effective preoperative evaluation, primarily by echocardiographic or cardiac imaging techniques.

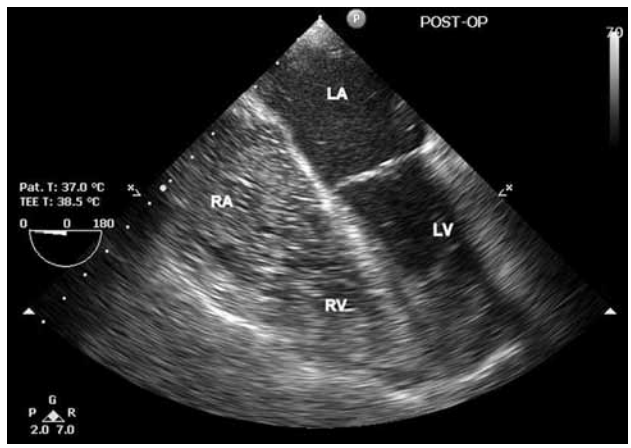

Figure 3. A postoperative transesophageal echocardiography image with contrast agent injected into the jugular vein. Contrast enhancement in the right atrium.

During ASD repair, cannulation can be selectively done above SVC or the right atrial appendix. In isolated ASD cases, there is no difference between these two cannulation strategies except surgical preference. Intra-atrial anatomy knowledge may further offer repair with both cannulation strategies. If pulmonary venous anomaly accompanies the defect, SVC should be carefully dissected and the draining pulmonary veins should be isolated. Selective cannulation above these pulmonary veins should be performed. As in selective caval cannulation in our case, it may produce pulmonary venous rerouting and provide convenience to the surgeon.

Moreover, sufficient exposure should be provided following right atriotomy in congenital cardiac defects requiring intracardiac repair. After right atriotomy, intra-atrial anatomy should be re-evaluated in detail. During the evaluation, the orifices of SVC and IVC, coronary sinus, tricuspid valve, and the margins of ASD should be identified. After the closure of ASD, the anatomical structures within the right atrium should be re-evaluated and it should be ensured that the repair is complete. After removal from cardiopulmonary bypass, intraoperative TEE should be done before decannulation. Any cyanosis identified during postoperative assessment is an indicator of a significant surgery-related complication (such as abnormal iatrogenic venous return).

In conclusion, PAPVC repair is a standard and common procedure which produces good treatment outcomes and low levels of associated morbidity. This case draws attention to this iatrogenic unusual complication secondary to surgical repair of sinus venosus ASD and anomalous pulmonary venous drainage. At each stage of the surgical procedure, cardiologists and cardiac surgeons should be aware of this extremely rare, but important complication to prevent a re-do surgery.

Declaration of conflicting interests

The authors declared no conflicts of interest with respect to the authorship and/or publication of this article.

Funding

The authors received no financial support for the research and/or authorship of this article.

REFERENCES

1. Gustafson RA, Warden HE, Murray GF, Hill RC, Rozar GE. Partial anomalous pulmonary venous connection to the right side of the heart. J Thorac Cardiovasc Surg 1989;98:861-8.
2. Alsoufi B, Cai S, Van Arsdell GS, Williams WG, Caldarone CA, Coles JG. Outcomes after surgical treatment of children with partial anomalous pulmonary venous connection. Ann Thorac Surg 2007;84:2020-6.
3. Trusler GA, Kazenelson G, Freedom RM, Williams WG, Rowe RD. Late results following repair of partial anomalous pulmonary venous connection with sinus venosus atrial septal defect. J Thorac Cardiovasc Surg 1980;79:776-81.
4. Staple TW, Ferguson TB, Parker BM. Diversion of the inferior vena cava into the left atrium following atrial septal defect closure. Am J Roentgenol Radium Ther Nucl Med 1966;98:851-8.
5. Thompson E, Moritz D, Perdue R, Cansino S. Diversion of the inferior vena cava following repair of atrial septal defect causing hypoxemia. Echocardiography 2004;21:329-32.
6. Beitzke D, Koestenberger M, Knez I, Beitzke A. Anomalous connection of the inferior vena cava to the left atrium: a surgical error in closing an atrial septal defect. Clin Res Cardiol 2008;97:191-3.
7. Desai M, Talwar S, Kothari SS, Jagia P, Airan B. Inferior vena cava to left atrium shunt presenting with polycythemia and stroke three decades following closure of atrial septal defect. Congenit Heart Dis 2013;8:102-5.

Cardiovasc Surg Int 2015;2(2):36-39
http://dx.doi.org/DOI: 10.5606/e-cvsi.2015.362
www.e-cvsi.org

Cardiovascular Surgery and Interventions

Case Report Open Access

Isolated insufficiency of the anterior accessory saphenous vein: should it be treated alone?

Hamit Serdar Başbuğ, Macit Bitargil, Kanat Özışık

Received: March 01, 2015 ***Accepted:*** June 09, 2015 ***Published online:*** August 03, 2015

ABSTRACT

Venous disorders of the lower limb are frequently seen in the general population. As the endovenous treatment of the venous disorders has evolved in the last two decades, our understanding on venous system anatomy has extended. Accessory saphenous vein is present in nearly half of the patients with lower limb venous insufficiency and should be taken into consideration before planning the treatment. In this article, we report a rare case of isolated reflux in an anterior accessory saphenous vein in the absence of a great saphenous vein insufficiency.

Keywords: Anatomy; saphenous vein; venous insufficiency.

Venous disorders of the lower limbs are common and may cause substantial morbidity in the general population, presenting a wide spectrum of clinical severity ranging from a minor varicosity to the chronic leg ulcers.[1] The prevalence of chronic venous insufficiency is as high as 17% of males and 40% of females.[2] The prevalence of the simple varicose veins is even greater in the population reaching 73% in females and 56% of males.[2] Irrespective of the clinical severity of the disease, chronic venous insufficiency almost always present with a superficial venous disease.[3] The great saphenous vein (GSV) reflux is the most common source of chronic venous insufficiency in up to 70% of the patients, followed by the small saphenous vein (SSV) (18%) and anterior accessory saphenous vein (AASV) (10%).[4] Anatomically, AASV begins at the distal thigh and courses upwards outside the saphenous compartment anterior to the GSV and drain into the femoral vein or GSV around the saphenofemoral junction (SFJ). It is present in 51% of the general population.[5]

During the last two decades, venous system has re-gained popularity with the advances in the endovenous treatments of venous diseases.[3] After the endovenous thermal ablations of the GSV mostly replaced with the conventional surgical practice, the anatomy and the variations of the venous system has been re-questioned and the nomenclature has been revised.[4,6] In this article, we report a rare case of isolated reflux in an anterior accessory saphenous vein in the absence of a great saphenous vein insufficiency which was treated with endovenous thermal ablation.

CASE REPORT

An healthy 24-year-old female patient was admitted to the outpatient clinic with complaint of numerous varicosities in her left leg. On physical examination, a large number of varicosities were seen over the anterior thigh. Duplex ultrasound (US) revealed a normal sized (3.8 mm) GSV with no reflux. However, an insufficient AASV with a diameter of 8.9 mm was present, which was feeding the other numerous venous packs (Figure 1). It was also extending down subcutaneously in parallel with the GSV through its distal course to the knee. Duplex US also revealed a severe reflux (>3 sec) in the common femoral vein segment above the level of SFJ. Reflux was due to the siphon effect and was directing back into the GSV, then leaking into the AASV as the pre-terminal valve of the GSV was intact. The GSV was anterolaterally anastomosed by AASV between the terminal and the pre-terminal valve of the GSV.

The patient was planned to be treated endovenously using radiofrequency thermal ablation (RFA). Under the Duplex US guidance, two 7F introducers were

Department of Cardiovascular Surgery, Medical Faculty of Kafkas University, Kars, Turkey

Corresponding author: Hamit Serdar Başbuğ, M.D. Kafkas Üniversitesi Tıp Fakültesi Kalp ve Damar Cerrahisi Anabilim Dalı, 36000 Kars, Turkey.
Tel: +90 474 - 225 11 90 e-mail: s_basbug@hotmail.com

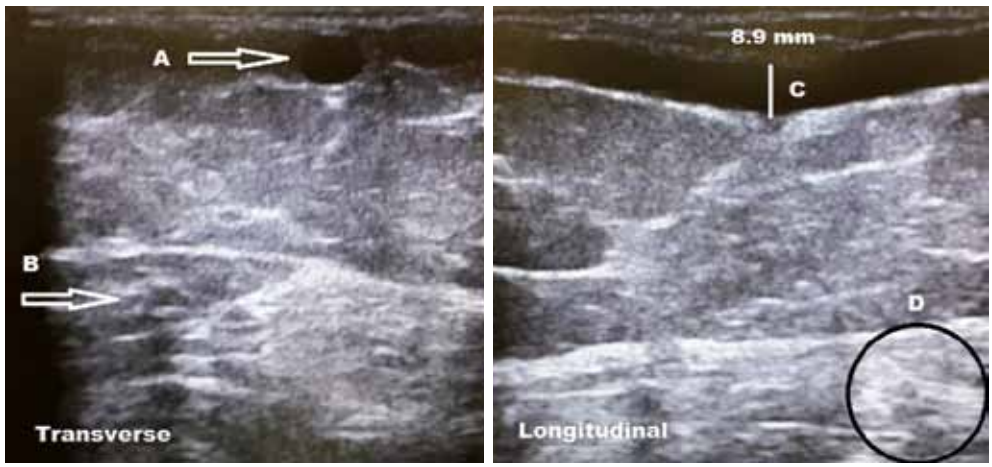

Figure 1. **(a)** The transverse view of the anterior accessory saphenous vein. **(b)** Great saphenous vein (and the saphenous compartment as 'Egyptian eye'). **(c)** The longitudinal image and measurement of the dilated anterior accessory saphenous vein. **(d)** Tip of the 7F catheter inside the great saphenous vein.

placed via Seldinger's technique inside the AASV and GSV side by side (Figure 2). The procedure was applied to the GSV and AASV consecutively with a separate perivenous application of the tumescent anesthesia. Operation was completed without any complication. The patient was discharged the other day with a venotonic and analgesic prescription and scheduled for a two week postoperative control.

DISCUSSION

Venous system anatomy constitutes the fundamental of the clinical phlebology and is essential for the accurate diagnosis and treatment of venous disorders.[6] The venous system anatomy of the lower limb are highly variable; however, it is within a systematic order.[7] The veins of the lower limb can be examined in three groups: deep veins, superficial veins, and perforating veins.[8] These are located in two compartments, namely the deep and superficial compartments. The deep compartment is delimited by the muscular fascia and contains deep veins, while the superficial compartment is delimited deeply by the muscular fascia and superficially by the skin containing the superficial veins.[6] Perforating veins can be only defined as the vascular communications between the veins of these two compartments, crossing through the holes in the muscular fascia. Within the superficial compartment, the separate saphenous compartment lies on the dorsum of the foot up to the inguinal ligament with a characteristic ultrasonographic view of the 'saphenous eye' or 'Egyptian eye' (Figure 1).[7,8] This saphenous compartment deeply bounded by the muscular fascia and superficially by the saphenous fascia.[9] The saphenous fascia, which was previously defined and then abandoned as Colles or Scarpa fascia, is the membranous layer of the subcutaneous tissue overlying the GSV and its roots, as well as the dorsal arch of the foot.[6,10] The saphenous compartment contains saphenous vein and accompanying nerves and arteries, whereas the tributaries and accessory veins lie externally.[6]

The accessory saphenous veins are the venous structures which lie in parallel and coursing superficially either anteriorly or posteriorly to the GSV outside the saphenous compartment.[6] Anterolateral and posterolateral veins of the thigh are the tributaries of the anterior and posterior accessory saphenous veins, respectively. Several types of anastomosis regarding the drainage of an accessory saphenous vein may be observed. It may drain directly into the femoral vein (below or above to the SFJ), GSV or into one of its tributaries (external pudendal vein, superficial epigastric vein, superficial circumflex iliac vein).

In the light of all these anatomic review and the setting of the nomenclature, some questions can be asked on the reported case. Firstly, what was the reason for the development of an isolated AASV insufficiency without affecting the GSV? Was AASV unprotected

compared to the GSV? What protected the GSV? Could GSV be left untreated, as it was normal?

As aforementioned, the AASV lies subcutaneously outside the saphenous compartment, making it more vulnerable to any venous hypertension rather than the GSV which lies within the saphenous compartment. Being buried in such a protective compartment may also explain the etymological origin of the saphenous vein which thought to be derived from the Arabic word 'al Safin' meaning 'hidden'. This connective sheath surrounding the GSV opposes the dilatation of the vein by serving a protective external cuff around the GSV. However, this surrounding sheat is not present around the AASV.[9] Additionally, contraction of the thigh muscles may modify the diameter of the GSV, as it happens in the deep veins.[9] Another possible reason for the development of an insufficiency in the AASV may be its fragile wall, compared to the relatively thicker saphenous type media layer. As the usual caliber of the GSV in our case without any visible reflux could not be left untreated, it was ablated. Otherwise, if the GSV was left untreated, the reflux would shift back into the GSV, instead of the AASV, soon after the ablation of the AASV, since the terminal valve of the SFJ was already insufficient. This insufficient valve would eventually damage the previously intact pre-terminal valve and would cause the GSV to become gradually insufficient and dilated. Therefore, to prevent further recurrences regarding the GSV, it should be treated prophylactically, if the accessory vein drains into the GSV at any level. However, the GSV may be left untreated, when an insufficient accessory vein drains individually into the femoral vein without any anastomotic relationship with the GSV.

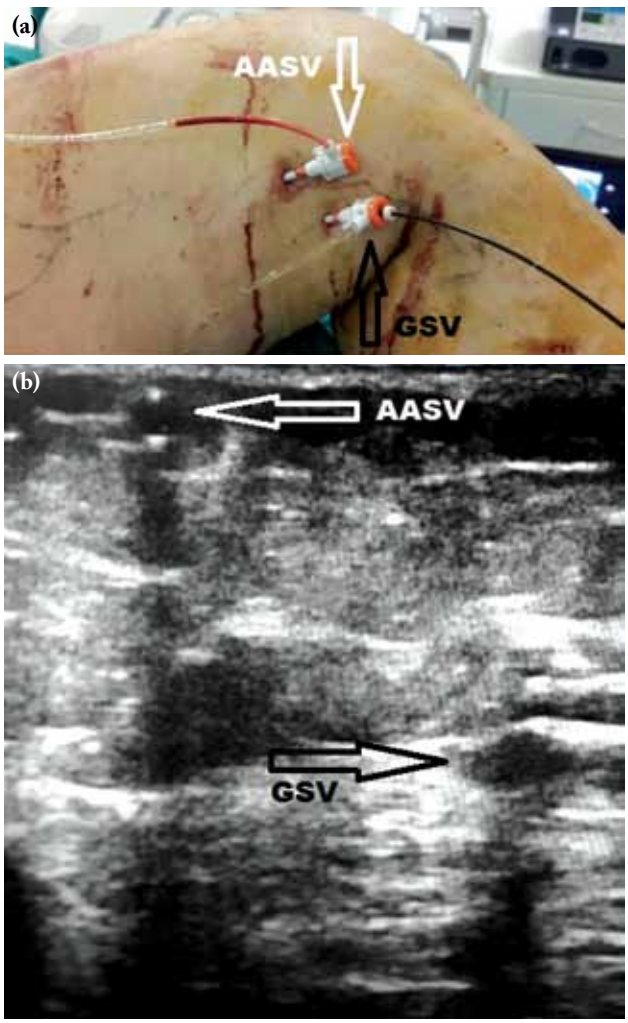

Figure 2. **(a)** Two 7F catheters placed together before tumescent anesthesia. **(b)** The transverse Duplex ultrasound image of both anterior accessory saphenous vein and great saphenous vein. AASV: Anterior accessory saphenous vein; GSV: Great saphenous vein.

In our case, two separate sheaths were introduced simultaneously into the GSV and the AASV, as seen in Figure 1a. If these two veins were cannulated separately instead of being simultaneous, the swelling effect of the tumescent anesthesia would deteriorate the Duplex US image, complicating the percutaneous access. In addition, endovenous thermal ablation procedure is performed under the tumescent anesthesia in which the gross amount of fluid containing local anesthetic is injected perivenously. If any of these veins was cannulated first and the other was remained uncannulated, the access to the remaining uncannulated vein might be difficult or even impossible after subsequent application of the tumescent anesthesia.[11] As a result, they were cannulated together before application any tumescent anesthesia.

Although the sequence of ablation is not critical, a particular interest should be given to the AASV, as it has a proximity to the skin.[12] The amount of the tumescent anesthesia should be kept high to protect the overlying skin from the thermal injury.[11] The venous tributaries are not necessarily extirpated, as they originate from the insufficient AASV. They will eventually fade away, as their primary feeding source is treated.

Anterior accessory saphenous vein is present nearly 50% of the patients and it is the third common cause of the chronic venous insufficiency.[5] In physical

examination and Duplex US investigation findings excluding the presence of the AASV may cause misdiagnosis, undertreatment, and possible recurrences.[13] Detailed preoperative Duplex US imaging is, therefore, necessary to figure out any anatomic description.[7]

In conclusion, we suggest that the isolated AASV insufficiencies should be treated together with the GSV. When they have a connection with each other, the endovenous thermal ablation procedure is effective in this treatment.

Declaration of conflicting interests

The authors declared no conflicts of interest with respect to the authorship and/or publication of this article.

Funding

The authors received no financial support for the research and/or authorship of this article.

REFERENCES

1. Evans CJ, Fowkes FG, Ruckley CV, Lee AJ. Prevalence of varicose veins and chronic venous insufficiency in men and women in the general population: Edinburgh Vein Study. J Epidemiol Community Health 1999;53:149-53.
2. Beebe-Dimmer JL, Pfeifer JR, Engle JS, Schottenfeld D. The epidemiology of chronic venous insufficiency and varicose veins. Ann Epidemiol 2005;15:175-84.
3. Min RJ, Zimmet SE, Isaacs MN, Forrestal MD. Endovenous laser treatment of the incompetent greater saphenous vein. J Vasc Interv Radiol 2001;12:1167-71.
4. Laredo J, Lee BB, Neville RF. Endovenous thermal ablation of the anterior accessory great saphenous vein. Endovasc Today 2010;March:36-9.
5. Mühlberger D, Morandini L, Brenner E. Venous valves and major superficial tributary veins near the saphenofemoral junction. J Vasc Surg 2009;49:1562-9.
6. Caggiati A, Bergan JJ, Gloviczki P, Jantet G, Wendell-Smith CP, Partsch H. Nomenclature of the veins of the lower limbs: an international interdisciplinary consensus statement. J Vasc Surg 2002;36:416-22.
7. Cavezzi A, Labropoulos N, Partsch H, Ricci S, Caggiati A, Myers K, et al. Duplex ultrasound investigation of the veins in chronic venous disease of the lower limbs--UIP consensus document. Part II. Anatomy. Eur J Vasc Endovasc Surg 2006;31:288-99.
8. Caggiati A. Fascial relationships of the long saphenous vein. Circulation 1999;100:2547-9.
9. Caggiati A, Ricci S. The long saphenous vein compartment. Phlebology 1997;12:107-11.
10. Wendell-Smith CP. Fascia: an illustrative problem in international terminology. Surg Radiol Anat 1997;19:273-7.
11. Alomari AI. Simple system for perivenous tumescent infusion for endovenous laser ablation. Phlebology 2012;27:242-4.
12. Shin S, Lee BB, Neville RF. Endovenous laser ablation of the anterior accessory great saphenous vein. Endovasc Today 2010;9:36-9.
13. Garner JP, Heppell PS, Leopold PW. The lateral accessory saphenous vein - a common cause of recurrent varicose veins. Ann R Coll Surg Engl 2003;85:389-92.

Cardiovasc Surg Int 2015;2(2):40-42
http://dx.doi.org/DOI: 10.5606/e-cvsi.2015.262
www.e-cvsi.org

Cardiovascular Surgery and Interventions

Case Report Open Access

Extracranial internal carotid artery stenosis associated with internal carotid artery aneurysm: a rare case in the literature

Süleyman Sürer, Yüksel Beşir, Orhan Rodoplu, Ömer Tetik

Received: September 21, 2014 ***Accepted:*** June 09, 2015 ***Published online:*** August 03, 2015

ABSTRACT

Although extracranial internal carotid artery aneurysms are rare, they may cause life-threatening complications. Typically, a pulsatile mass in the neck can be detected on physical examination; however, some cases may be asymptomatic and further investigations may be required. A 62-year-old male patient was admitted with complaints of head and neck pain and obscuration of vision with a history of peripheral arterial disease. Carotid angiography showed stenosis of the internal carotid artery and aneurysm of the internal carotid artery at the beginning of the carotid bulb. The patient was operated under general anesthesia. Following aneurysmectomy and endarterectomy, the artery was successfully closed with saphenous vein patch plasty.

Keywords: Aneurysm; internal carotid artery; stenosis.

Extracranial carotid artery aneurysms are rare vascular lesions. The most common causes include atherosclerosis, fibromuscular dysplasia, and trauma.[1] In addition, neck trauma, carotid dissection, previous carotid endarterectomy surgery, and infections including tonsillitis and peritonsillar abscess play a role in the etiology.[1] Marfan's syndrome, Behçet's syndrome, and Takayasu's arteritis are among the other rare causes.[1] Furthermore, rupture of aneurysm carries an important risk, while cerebral atheroembolism due to thrombus inside the aneurysm also poses a significant risk. Treatment of the proximal aneurysm surgery first began with the successful ligation of carotid artery aneurysms performed by Sir Astley Cooper in 1808.[2] Following this, the first successful resection and re-anastomosis surgery was conducted by Dimtza in 1956.[3] In this article, we present a case of a saccular aneurysm and stenosis of internal carotid artery successfully operated by saphenous vein patch plasty.

CASE REPORT

A 62-year-old male patient was admitted to our outpatient clinic with complaints of dizziness and obscuration of vision for nearly two years. The patient was hospitalized upon the detection of an internal carotid artery stenosis in Doppler ultrasound. Carotid digital subtraction angiography demonstrated an isolated and short-segment aneurysm at the level of the carotid bulb and a 80-90% stenosis due to a 20x6 mm plaque formation in the internal carotid artery, extending from the right carotid bulb to the proximal internal carotid artery (Figure 1). Coronary angiography showed a normal right coronary artery (RCA) and plaques in circumflex (CX) and left anterior descending (LAD) artery. The patient had a history of hypertension, smoking, and right superficial femoral artery stenting in 2005 for peripheral arterial disease. On physical examination, no pulsatile mass was detected in the neck. The patient was operated under general anesthesia. During surgical exploration, a saccular aneurysm, stenosis, muscular defect in the medial layer, and the ballooning of the vessel wall were detected in the internal carotid artery. Common carotid, internal, and external carotid arteries were controlled with vascular tapes. Carotid artery vascular clamp was placed after 5,000 IU of heparin. Internal carotid artery stump pressure was measured over 50 mmHg and surgery was continued without shunting. There was a long segment, ulcerated atherosclerotic plaque in the internal carotid artery. Following aneurysmectomy and endarterectomy, the artery was successfully closed

Department of Cardiovascular Surgery, Bursa Yüksek İhtisas Training and Research Hospital, Bursa, Turkey

Corresponding author: Süleyman Sürer, M.D. Bursa Yüksek İhtisas Eğitim ve Araştırma Hastanesi Kalp ve Damar Cerrahisi Kliniği, 16330 Yıldırım, Bursa, Turkey.
Tel: +90 224 - 360 50 50 e-mail: drsuleyman1@hotmail.com

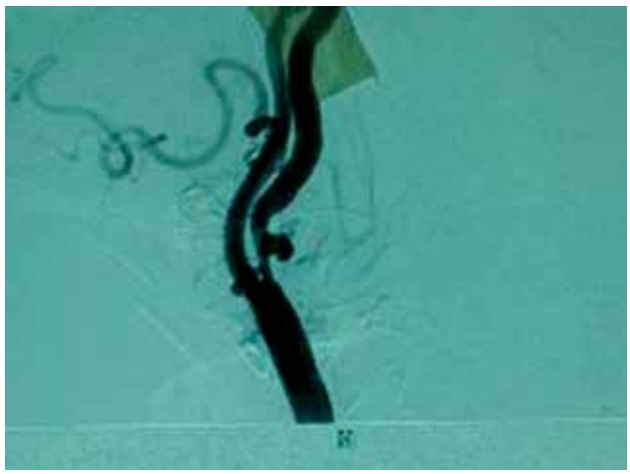

Figure 1. Angiographic appearance of the right carotid artery aneurysm and stenosis.

with saphenous vein patch plasty. The patient was discharged on the fifth postoperative day without any complication.

DISCUSSION

Aneurysm of extracranial carotid artery is a rare vascular lesion. Extracranial carotid artery aneurysms account for 0.1 to 2% of all carotid surgeries.[4] Moreover, the most common causes of intracranial aneurysms are atherosclerosis, fibromuscular dysplasia, and trauma. Radak et al.[5] showed aneurysms due to atherosclerosis (80.2%), trauma (6.6%), carotid surgery (6.6%), and fibromuscular dysplasia (5.5%). Additionally, Zhou et al.[6] showed carotid artery aneurysms due to atherosclerosis (50%), pseudoaneurysms (30%), and trauma (12%). Although extracranial carotid artery aneurysms are rare, they may cause potentially fatal complications including embolization, rupture, and local compression. Therefore, early diagnosis and treatment are of utmost importance for these patients. Sometimes, diagnosis can be challenging in patients without neurological symptoms, in particular. Radak et al.[5] showed that 31.9% of cases are asymptomatic at the time of diagnosis. Although the most common symptom is a pulsatile mass in the neck, it is not detected in calcified saccular aneurysms. However, Szopinski et al.[7] showed no pulsatile mass in six of 15 patients. Although rare, aneurysms can be dangerous and they should be considered in the differential diagnosis in patients with non-specific symptoms, such as dysphagia, speech disorders, headache, neck pain, anisocoria, even in the absence of findings of a pulsatile mass in the neck.[8] We did not detect any pulsatile mass in the neck at the physical examination in our case. El Sabrout and Cooley[9] showed a pulsatile mass in the neck in 59%, neurological symptoms in 43%, and signs of local compression in 10% in their study. In addition, most internal carotid artery aneurysms are hospitalized with embolic stroke.[10] Symptoms such as Horner's syndrome and dysphagia caused by cranial nerve lesions require careful research about underlying possible vascular pathologies. The first step in the diagnosis is Doppler ultrasound, which is a simple and noninvasive imaging modality; however, it may be inadequate to detect aneurysms which are small or close to the skull base distal internal carotid artery lesions. Contrast computed tomography, magnetic resonance imaging, and angiography are other methods used in the diagnosis of such aneurysms. Also, arteriography is the gold standard for aneurysms due to occurrence of thrombus location and collateral circulation.[11]

Furthermore, isolated and short-segment aneurysms at the level of the carotid bulb and a 80-90% stenosis due to a 20x6 mm plaque formation in the internal carotid artery was detected in our case angiographically. However, aneurysmal dilatation caused an atheroma plaque or ulcer. During surgery, muscular defect in the medial layer and the ballooning of the vessel wall brought us to the definitive diagnosis.

Moreover, open surgery is the preferred and accepted treatment method for extracranial internal carotid artery aneurysm.[6] The primary indication for surgery is the prevention of permanent neurological damage from thromboembolic events.[7] During surgery of large aneurysms, stroke due to distal embolization of atherosclerotic debris and damage due to cranial nerve traction are the potential risks.[12] In addition, aneurysm size, location, and etiology are the decisive factors in choosing the surgical procedure for a surgeon. Surgical treatment approaches include clipping, resection, and end-to-end anastomosis, resection and graft placement, extracranial to intracranial bypass, patch plasty and carotid artery ligation. Following aneurysmectomy and endarterectomy, we repaired internal carotid artery with saphenous vein patch plasty in our case. In some cases, endovascular treatment can be recommended as an alternative to surgery. However, intracranial carotid artery aneurysms and extracranial internal carotid artery stenoses in different localizations were published in the literature.[13,14]

In conclusion, the most characteristic feature of our case was the concurrence of an internal carotid artery stenosis with a saccular aneurysm. Review of the literature revealed a few reports of concurrence of an arterial aneurysm and an internal carotid artery stenosis.

Declaration of conflicting interests

The authors declared no conflicts of interest with respect to the authorship and/or publication of this article.

Funding

The authors received no financial support for the research and/or authorship of this article.

REFERENCES

1. McCann RL. Basic data related to peripheral artery aneurysms. Ann Vasc Surg 1990;4:411-4.
2. Cooper A. Account of the first successful operation performed on the common carotid artery for aneurysm in the year of 1808 with postmortem examination in the year 1821. Guys Hosp Pep 1836;1:53.
3. Dimtza A. Aneurysms of the carotid arteries; report of two cases. Angiology 1956;7:218-27.
4. Rosset E, Albertini JN, Magnan PE, Ede B, Thomassin JM, Branchereau A. Surgical treatment of extracranial internal carotid artery aneurysms. J Vasc Surg 2000;31:713-23.
5. Radak D, Davidović L, Vukobratov V, Ilijevski N, Kostić D, Maksimović Z, et al.Carotid artery aneurysms: Serbian multicentric study. Ann Vasc Surg 2007;21:23-9.
6. Zhou W, Lin PH, Bush RL, Peden E, Guerrero MA, Terramani T, et al. Carotid artery aneurysm: evolution of management over two decades. J Vasc Surg 2006;43:493-6.
7. Szopinski P, Ciostek P, Kielar M, Myrcha P, Pleban E, Noszczyk W. A series of 15 patients with extracranial carotid artery aneurysms: surgical and endovascular treatment. Eur J Vasc Endovasc Surg 2005;29:256-61.
8. Yetkin U, Yürekli İ, Gürbüz A. Extracranial internal carotid artery aneurysms. The Internet J Thorac and Cardiovasc Surg 2007;10:2.
9. El-Sabrout R, Cooley DA. Extracranial carotid artery aneurysms: Texas Heart Institute experience. J Vasc Surg 2000;31:702-12.
10. Hurst RW, Haskal ZJ, Zager E, Bagley LJ, Flamm ES. Endovascular stent treatment of cervical internal carotid artery aneurysms with parent vessel preservation. Surg Neurol 1998;50:313-7.
11. Windfuhr JP. Aneurysm of the internal carotid artery following soft tissue penetration injury. Int J Pediatr Otorhinolaryngol 2001;61:155-9.
12. Bardakçı H, Akgül A, Babaroğlu S, Kaplan S, Beyazıt M. Ekstrakranial internal karotis arter anevrizması; olgu sunumu. Damar Cer Derg 2006;15:49-52.
13. Cho YD, Jung KH, Roh JK, Kang HS, Han MH, Lim JW. Characteristics of intracranial aneurysms associated with extracranial carotid artery disease in South Korea. Clin Neurol Neurosurg 2013;115:1677-81.
14. Shumann MU, Mirzai S, Samii M, Vorkapic P. Xenon/CT CBF measurements as valuable diagnostic tool in a case of bilateral occlusive cerebrovascular disease associated with intracranial aneurysm. Acta Neurol Scand Suppl 1996;166:104-9.

Cardiovasc Surg Int 2015;2(2):43-46
http://dx.doi.org/DOI: 10.5606/e-cvsi.2015.373
www.e-cvsi.org

Cardiovascular Surgery and Interventions

Case Report Open Access

Critical lower limb ischemia in Leriche syndrome following acute myocardial infarction: limb salvage with an axillofemoral bypass

Hamit Serdar Başbuğ[1], Macit Bitargil[1], Ahmet Karakurt[2], Sefer Usta[3], Kanat Özışık[1]

Received: May 22, 2015 ***Accepted:*** June 09, 2015 ***Published online:*** August 03, 2015

ABSTRACT

The aortoiliac occlusive disease, known as Leriche syndrome, primarily merits an aortobifemoral bypass graft which is the standard surgical treatment of critical limb ischemia. However, axillofemoral bypass grafting can be also used as an alternative treatment in high-risk patients. The indications include intraabdominal graft infections, older age, and worsened overall status. In this article, a successful salvage of a limb with an axillofemoral bypass surgery in a critical leg ischemia subsequently developed soon after an acute myocardial infarction was reported.

Keywords: Acute myocardial infarction; arterial occlusive disease; axillofemoral bypass grafting; Leriche syndrome; limb salvage.

Aortoiliac occlusive disease, also known as Leriche syndrome, is an atherosclerotic obstructive disease involving the distal abdominal aorta prior to the bifurcation into the common iliac arteries.[1] It was first described by Leriche and Morel in 1948.[1] It is a relatively rare condition compared to the infrainguinal arterial obstructions.[2] The primary treatment is surgical revascularization. Aortobifemoral (ABF) bypass is the golden standard with a five-year patency rate of >80%.[3] However, axillofemoral (AXF) bypass was first introduced by Blaisdell and Hall, and Louw at the same time in 1963 as an alternative bypass technique for lower limb inflow revascularization.[3,4] Previously, use of AXF bypass for aortoiliac occlusive disease was limited due to its lower long-term patency rates, compared to ABF grafts. However, it has been, then, widely adopted as an alternative surgical treatment for aortoiliac occlusive disease more frequently with the recent improvements in structure of the prosthetic materials. With the introduction of externally supported grafts, the patency rates of AXF bypass increased up to 70% in five years.[5] Axillofemoral bypass is considered primarily as an alternative revascularization approach in patients with high-risk laparotomy or in whom an aortic approach is troublesome due to the previous abdominal infection or surgery.[6] It is also reserved as a more practical and relatively rapid procedure for elderly with worsened overall status and hemodynamic instability.

Coexistence of coronary artery disease and severe aortic occlusive disease is reported as 4 to 15% in different series.[7] In this article, we report a case of critical leg ischemia developed soon after an acute myocardial infarction and its rapid and efficient salvage with an AXF bypass surgery is presented.

CASE REPORT

A 65-year-old male patient was admitted to the emergency department with chest pain for the last four hours. No significant history was present except intermittent claudication on exertion without resting pain. Blood pressure was 113/76 mmHg. Electrocardiogram revealed tachycardia (116 bpm) without ST elevation. Blood biochemical test results were normal except increased serum troponin-T levels of 0.23 ng/mL (reference range: 0 to 0.02 ng/mL). Non-ST acute myocardial infarction (NSTEMI) was suspected and coronary angiography (CAG) was decided. On physical examination, bilateral femoral artery pulses were non-palpable. Thus, CAG was performed through the right brachial artery. It demonstrated a slow coronary flow in the left anterior descending (LAD) artery and three-vessel disease with a diffuse pattern. Left ventriculography

Departments of [1]Cardiovascular Surgery, [2]Cardiology, Medical Faculty of Kafkas University, Kars, Turkey
[3]Department of Cardiovascular Surgery, Ahi Evren Thoracic and Cardiovascular Training Hospital, Trabzon, Turkey

Corresponding author: Hamit Serdar Başbuğ, M.D. Kafkas Üniversitesi Tıp Fakültesi Kalp ve Damar Cerrahisi Anabilim Dalı, 36000 Kars, Turkey.
Tel: +90 474 - 225 11 90 e-mail: s_basbug@hotmail.com

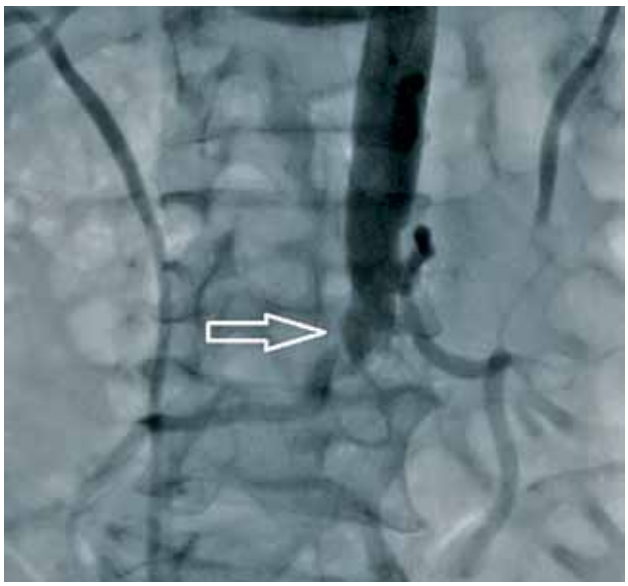

Figure 1. Aortography revealing the distal aortic occlusive disease.

showed an anterior segmental hypokinesia with slightly increased diameters and no mitral valvular regurgitation. Distal aortography demonstrated distal aortic occlusive disease (Figure 1). Coronary artery bypass graft (CABG) surgery and simultaneous aorta-distal revascularization were planned electively after troponin-T levels were decreased.

The patient was prepared for CABG and aorta-distal bypass operation in the cardiovascular intensive care unit. Low-molecular-weight heparin and coronary vasodilator treatment were initiated. During follow-up, the right lower limb demonstrated ischemic signs and symptoms on the following day after CAG. Pain and pallor progressed in a few hours to cyanosis and demarcation line below the right knee was almost settled. Blood gas analyses revealed slight acidosis (pH: 7.27) with compensatory respiratory alkalosis (pCO_2: 23.4). Blood urea nitrogen (BUN) and creatinine levels were increased (46 mg/dL and 1.6 mg/dL, respectively). Hemodynamic variables and cardiac enzyme levels remained high and the overall status of the patient was inconvenient neither for CABG nor aorta-distal revascularization with laparotomy. As a result, an extra-anatomic bypass was planned for the limb salvage. A contrasted computed tomography (CT) was performed to investigate the distal status of the vasculature (Figure 2). Computed tomography showed a distal aortic occlusion and antegrade re-filling of the femoral arteries through the collateral circulation. A right AXF bypass was decided as the best surgical strategy.

Under general anesthesia, the right common femoral artery and right subclavian artery were explored. Both were prepared for anastomosis by circumference with elastic vascular tapes. An 8 mm diameter ringed expanded polytetrafluoroethylene (ePTFE) graft was placed subcutaneously through the inserted steel tunneler. A mid-point cutaneous incision was made. The length of the graft was adjusted by trimming the both edges. Proximal part was passed under the pectoralis major muscle. Native arteries were clamped after an intravenous injection of 5,000 IU of unfractionated heparin. Proximal and distal anastomoses were performed with 6-0 polypropylene sutures with an end-to-side manner (Figure 3). Distal femoropopliteal embolectomy was performed before the termination of distal anastomosis. Anastomosed native arteries were de-clamped and de-aired. Circulation was successfully achieved. Intraoperative distal Duplex ultrasound showed triphasic flow pattern of the anterior and posterior tibial arteries. The right brachial artery flow rate was also triphasic and not affected.

Ischemic signs and symptoms of the right leg were totally regressed and returned to normal.

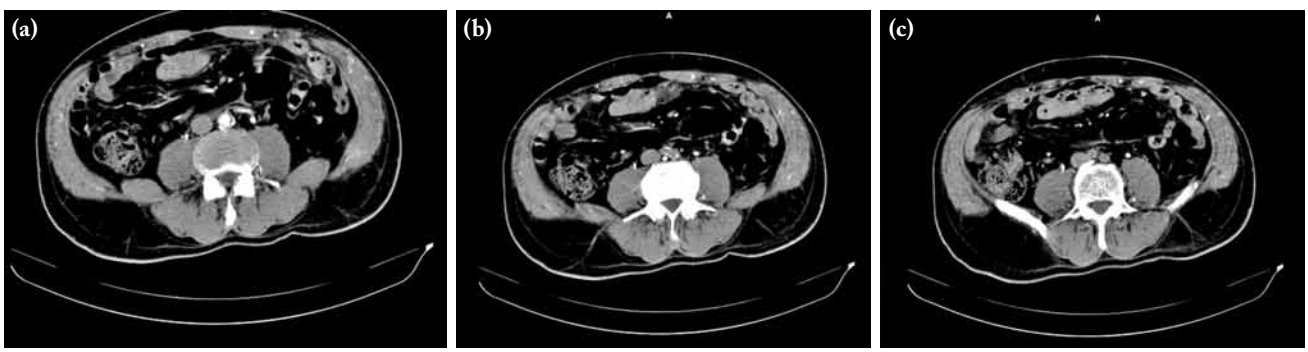

Figure 2. Computed tomography showing **(a)** distal aortic occlusive disease and **(b, c)** bilateral iliac occlusion.

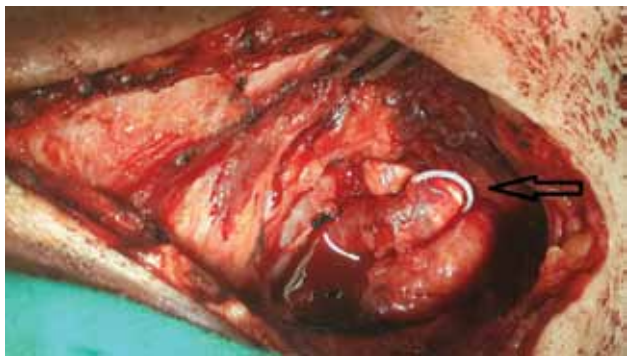

Figure 3. Proximal anastomosis of an expanded polytetrafluoroethylene graft to the subclavian artery.

Blood chemistry levels including blood gas analyses reversed to normal levels. The patient was followed for two weeks in hospital during the recovery period. Incision sites were recovered without any complication (Figure 4). Coronary artery bypass graft surgery was, then, performed successfully following cardiac enzymes were dropped to normal levels.

DISCUSSION

The management of distal aortic occlusive disease (Leriche syndrome) depends on surgical or non-surgical options.[6] The surgical treatment of Leriche syndrome can be performed either as direct anatomic (ABF) bypass or extra-anatomic (AXF) bypass.[8] Direct anatomic bypasses include aortoiliac and aortofemoral bypasses. Extra-anatomic bypasses include axillofemoral bypasses. The non-surgical treatment of Leriche syndrome mainly depends on endovascular revascularization.[9] Patients with aortoiliac occlusive disease can be treated with percutaneous techniques, such as balloon angioplasty or peripheral stenting. However, in the presence of a diffuse disease, angioplasty remains significantly ineffective when the distal run-off would not be warranted.[10] Therefore, these aforementioned limitations may make the AXF bypasses the primary therapeutic option for patients with significant comorbidities and diffuse aortoiliac occlusive disease.[11]

The simultaneous presentation of coronary artery disease (CAD) and peripheral arterial disease (PAD) are not uncommon. Recent studies have reported the rate of the concomitance about 40%.[7,12] In addition, the coexistence of aortoiliac occlusive was reported 4 to 15% in patients undergoing CABG surgery.[13,14] The concomitance of PAD and CAD

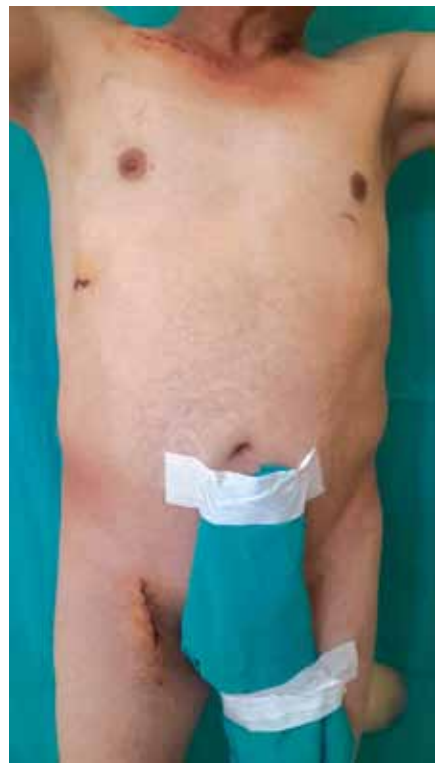

Figure 4. Total recovery of the incision sites (postoperative seventh day).

usually deserves a combined surgical approach, in which both pathologies are treated at the same session.[7] However, in case of deterioration in the overall status of the patient, procedural priority should be given to the more serious pathology. Briefly, life-threatening manifestation is initially treated and the other pathology is postponed, until the overall condition becomes convenient.[7]

In the presented case, a combined procedure was initially planned; however, the limb ischemia developed instantly and prompt intervention was needed. Thus, an AXF bypass was emergently performed to salvage the right lower limb. Also, CABG operation was postponed due to worsened overall status of the patient. If the combined procedure had been performed, multiple handicaps would have occurred. In addition, STEMI and elevated troponin-T levels, determinant factors of impaired cardiac contractility, would increase the intraoperative mortality. Ischemia period would be elongated due to CABG, leading to irreversible neurological complications such as dropped-foot, or even amputation. In addition, an intraaortic balloon pump insertion would be impossible, while it becomes crucial during CABG

surgery. As a result, the strategy was primarily built over the salvage of the leg.

Regarding the etiological progression of our case, what was the reason for the instant appearance of ischemic symptoms soon after the CAG? Although the brachial artery approach was selected for the percutaneous intervention, what caused the distal impairment in tissue perfusion? Was the leg ischemia following a CAG with brachial arterial access merely a coincidence?

In this case, severe occlusive disease affecting the distal aorta and both iliac arteries was considered as the primary pathology. This knife-edge condition was probably maintained, until an unusual physiological status developed. The CAG and the preceding NSTEMI were possibly directly or indirectly responsible for all the ischemic process affecting the right leg. Immobilization during the emergency room, CAG unit and intensive care unit might precipitate the symptoms of distal circulation. An excessive consumption of the radiopaque infusion during the CAG might also precipitate glomerulopathy causing dehydration or overhydration, both of which have the potential to change the hemodynamic variables. A slight increase in BUN levels after CAG might be a predictor of the probable opaque nephropathy which altered the blood composition, as perfusion in the microvascular network can be easily affected by the minute changes in blood rheology.[15]

In conclusion, coexistence of CAD and aortoiliac occlusive disease deserves a unique treatment strategy. Both pathologies can be treated at the same session, either by an endovascular or standard surgical approach. Regarding the surgical treatment, ABF bypass is the golden standard with high patency rates. However, if the laparotomy is unable to be performed due to various reasons, AXF bypass can be easily performed instead. With the evolution of the prosthetic vascular grafts, patency rates of AXF bypass have been also increased. Combined surgery should be considered in eligible patients with a good overall status. Otherwise, more serious pathology should primarily be treated, postponing the other entity to be considered electively.

Declaration of conflicting interests

The authors declared no conflicts of interest with respect to the authorship and/or publication of this article.

Funding

The authors received no financial support for the research and/or authorship of this article.

REFERENCES

1. Leriche R, Morel A. The Syndrome of Thrombotic Obliteration of the Aortic Bifurcation. Ann Surg 1948;127:193-206.
2. Szilagyi DE, Elliott JP Jr, Smith RF, Reddy DJ, McPharlin M. A thirty-year survey of the reconstructive surgical treatment of aortoiliac occlusive disease. J Vasc Surg 1986;3:421-36.
3. Blaisdell FW, Hall AD. Axillary-femoral artery bypass for lower extremity ischemia. Surgery 1963;54:563-8.
4. Louw JH. Splenic-to-femoral and axillo-to-femoral bypass grafts in diffuse atherosclerotic occlusive disease. Lancet 1963;29:1401-2.
5. Passman MA, Taylor LM, Moneta GL, Edwards JM, Yeager RA, McConnell DB, et al. Comparison of axillofemoral and aortofemoral bypass for aortoiliac occlusive disease. J Vasc Surg 1996;23:263-9.
6. Lee GC, Yang SS, Park KM, Park Y, Kim YW, Park KB, et al. Ten year outcomes after bypass surgery in aortoiliac occlusive disease. J Korean Surg Soc 2012;82:365-9.
7. Albeyoğlu ŞÇ, Filizcan U, Çınar B, Aydoğan H, Göksel O, Şahin V, et al. Simultaneous coronary artery bypass grafting and peripheral revascularization in generalized atherosclerotic patients. Turk Gogus Kalp Dama 2006;14:249-53.
8. Brewster DC. Current controversies in the management of aortoiliac occlusive disease. J Vasc Surg 1997;25:365-79.
9. Mwipatayi BP, Thomas S, Wong J, Temple SE, Vijayan V, Jackson M, et al. A comparison of covered vs bare expandable stents for the treatment of aortoiliac occlusive disease. J Vasc Surg 2011;54:1561-70.
10. Martin D, Katz SG. Axillofemoral bypass for aortoiliac occlusive disease. Am J Surg 2000;180:100-3.
11. Kashyap VS, Pavkov ML, Bena JF, Sarac TP, O'Hara PJ, Lyden SP, et al. The management of severe aortoiliac occlusive disease: endovascular therapy rivals open reconstruction. J Vasc Surg 2008;48:1451-7.
12. Hegde MV, Ajith S, Krishnan MG, Mohamed Faisal CK. Case report: rehabilitation of a patient with coronary artery baypas graft (CABG) with axillofemoral bypass. Int J Cur Res Rev 2013;5:16-23.
13. Reis RL, Hannah H. Management of patients with severe, coexistent coronary artery and peripheral vascular disease. J Thorac Cardiovasc Surg 1977;73:909-18.
14. Carrel T, Niederhäuser U, Pasic M, Gallino A, von Segesser L, Turina M. Simultaneous revascularization for critical coronary and peripheral vascular ischemia. Ann Thorac Surg 1991;52:805-9.
15. Alexy T, Pais E, Wenby RB, Mack WJ, Hodis HN, Kono N, et al. Abnormal blood rheology and chronic low grade inflammation: possible risk factors for accelerated atherosclerosis and coronary artery disease in Lewis negative subjects. Atherosclerosis 2015;239:248-51.

Cardiovasc Surg Int 2015;2(2):47-48
http://dx.doi.org/DOI: 10.5606/e-cvsi.2015.382
www.e-cvsi.org

Cardiovascular Surgery and Interventions

Interesting Image Open Access

Easily removal of a malappositioned coronary stent with a guidewire

Tevfik Güneş[1], Yusuf İzzettin Alihanoğlu[2], İhsan Alur[1], Bekir Serhat Yıldız[2]

Received: April 24, 2015 ***Accepted:*** June 01, 2015 ***Published online:*** August 03, 2015

Dislodgment of a stent during or after percutaneous coronary intervention (PCI) is a rare complication with an incidence ranging from 0.32 to 8%.[1,2] The main risk factors for stent dislodgment include extreme coronary angulations and tortuosity, diffuse long lesions, and highly calcified coronary arteries.[1,2] Also, direct stent deployment and the inadequate predilatation or debulking of the lesion may cause stent distortion and underexpansion, increasing the risk of dislodgement.[1,2]

Stent migration may give rise to serious clinical consequences; it may be embolized in the coronary circulation and cause cerebral or peripheral embolization. Coronary embolization may lead to coronary thrombosis, myocardial infarction, emergency coronary artery bypass graft surgery, or even death. Retrieval of a dislodged stent can be performed either percutaneously or surgically.[2,3]

With the advanced technology of today, stent dislodgement is less common. However, an ideal catheterization laboratory should be equipped with a set of instruments for intravascular foreign body retrieval and interventional cardiologists should be familiar with these retrieval techniques[2] in cooperation with the surgical team.

CASE REPORT

A 69-year-old man was admitted to our hospital due to the chest pain and diagnosed with inferior myocardial infarction (MI). In his medical history, a 2.75x24 mm everolimus-eluting-stent was inserted to the right coronary artery (RCA) due to stable angina a week ago in another health care center. He, then, urgently underwent a new coronary angiography. The RCA was fully occluded and previously deployed stent was malappositioned in the proximal portion (Figure 1a). During our attempt to cross the lesion, we detected that the tip of floppy guidewire (ChoICE™ Floppy - Boston Scientific, Natick, MA, USA) was coiled up and shrunk at the distal portion of the malappositioned stent. The malappositioned stent was easily coming out as we were cautiously pulling back the guidewire to push more distally the tip of guidewire. Therefore, the dislocated stent was immediately

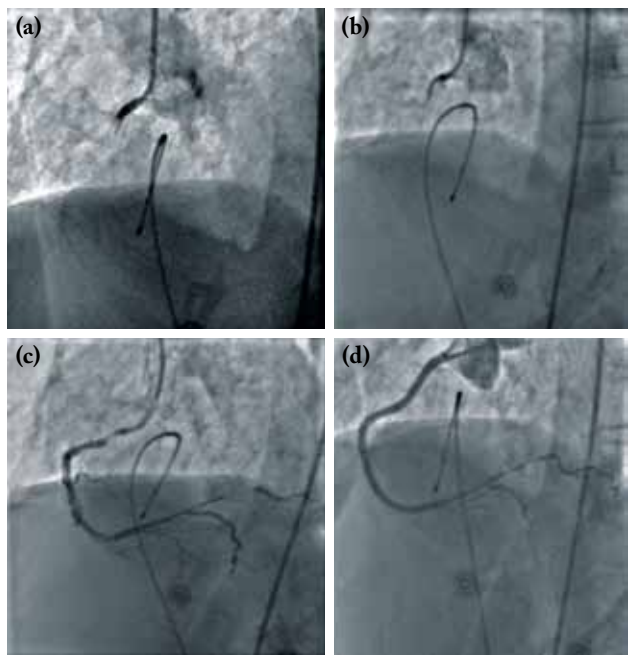

Figure 1. **(a)** Totally occlusion of the stent with a thrombus. **(b)** Native right coronary artery after the stent was spontaneously removed. **(c)** The view of right coronary artery after percutaneous transluminal angioplasty, before percutaneous coronary intervention. **(d)** The recent appearance of the right coronary artery after stenting of the dissected areas.

Departments of [1]Cardiovascular Surgery and [2]Cardiology, Medical Faculty of Pamukkale University, Denizli, Turkey

Corresponding author: Tevfik Güneş, M.D. Pamukkale Üniversitesi Tıp Fakültesi Kalp ve Damar Cerrahisi Anabilim Dalı, 20070 Kınıklı, Denizli, Turkey. Tel: +90 505 - 464 42 93 e-mail: tevfik04@yahoo.com

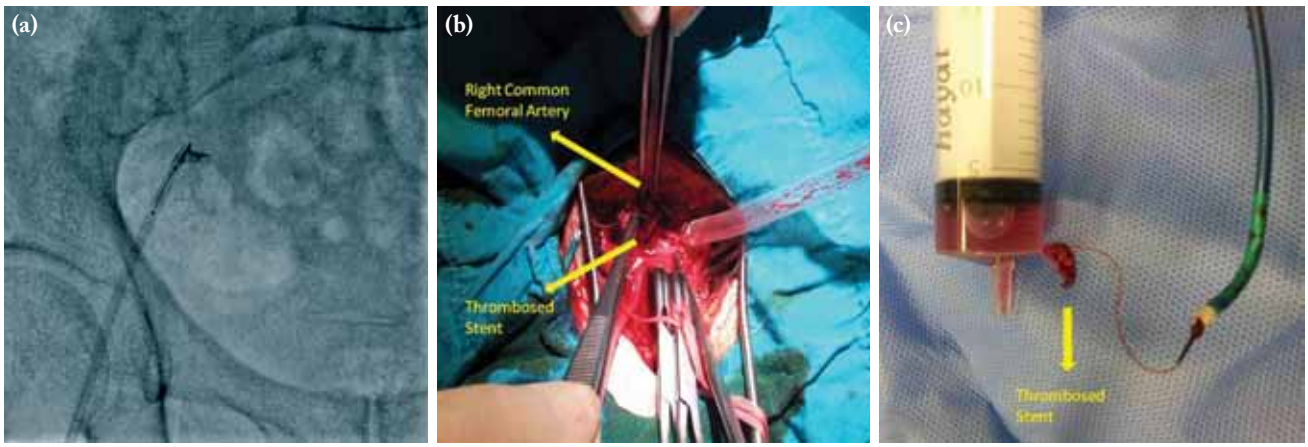

Figure 2. **(a)** Entrapped stent at the tip of the guidewire catheter. **(b)** Exploration of the right femoral artery and removal of the thrombosed stent. **(c)** A thrombosed stent.

removed and pulled back down. New introducer sheath was quickly placed in the other side. The RCA was passed with a new guidewire. A long dissection line was seen with spontaneous coronary flow (Figure 1b, c). After consecutive balloon predilatation with a 2.0x15 mm balloon, three everolimus-eluting-stents (2.75x24 mm, 3.0x24 mm, 3.0x28 mm, respectively) and finally one bare metal-stent (3.5x16 mm) were properly deployed. Eventually, RCA was repaired and Thrombolysis in Myocardial Infarction-3 (TIMI-3) coronary flow was completely achieved (Figure 1d). Ultimately, the right femoral artery was surgically explored and the dislocated stent removed through arteriotomy, as the stent was not able to be retrieved back into the right femoral sheath completely (Figure 2). Herein, we present an extremely rare case of PCI-related complication due to a malappositioned stent, which was totally shrunk and easily removed from the coronary system.

Declaration of conflicting interests

The authors declared no conflicts of interest with respect to the authorship and/or publication of this article.

Funding

The authors received no financial support for the research and/or authorship of this article.

REFERENCES

1. Porto I, Larosa C, Rosa I, Burzotta F, Trani C. Successful transradial removal of an inflated coronary stent dislodged from the right coronary ostium. Cardiovasc Revasc Med 2014;15:432-5.
2. Jang JH, Woo SI, Yang DH, Park SD, Kim DH, Shin SH. Successful coronary stent retrieval from the ascending aorta using a gooseneck snare kit. Korean J Intern Med 2013;28:481-5.
3. Kwan TW, Chaudhry M, Huang Y, Liou M, Wong S, Zhou X, et al. Approaches for dislodged stent retrieval during transradial percutaneous coronary interventions. Catheter Cardiovasc Interv 2013;81:245-9.