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Letter to the Editor

Research Articles

Case Report

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Hypertension in elderly

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With increase in modern healthcare access thanks to the developments in technology, the advances in preventive and treating medicine and the improvements in health organization, the expected length of life is prolonged, and the population ages rapidly. The elderly people ≥60 years is the most rapidly growing segment in the population. In our country, the observation of increase in the aging population, was clearly revealed with the last two population censuses. In the last census, population ≥75 years nearly doubled compared to the previous census.

With the aging, both the prevalence and incidence of the cardiovascular events mainly stroke and myocardial infarction increase. Cardiac failure is a highly important problem in elderly that we face, and its incidence increases every passing day. Overall difficulty in blood pressure control in the society, is more significant in the elderly population, and the already low blood pressure control rates decrease to very low levels in elderly.

Isolated systolic hypertension (>140 mmHg systolic and >90 mmHg diastolic blood pressure values) is the most frequent form of high blood pressure seen in elderly. The frequency of high blood pressure increases with age, and isolated systolic hypertension is observed in 2/3 of the people over 60 years old and in 3/4 of the people over 75 years old. Isolated systolic hypertension is the most important parameter in elderly with regards to cardiovascular risk; thus the main blood pressure treatment target is the isolated systolic hypertension.

Progressive stiffness in the aortic wall emerging with aging, decrease in the balancing effect of aorta on flow volume, progressive increase in the systolic blood pressure as a result of these, and decrease in diastolic blood pressure, togethwer with the widened pulse pressure constitute the key pathophysiological steps of hypertension in elderly. In addition to this, impairment of renal functions, sodium retention, impaired baroreceptor function, comorbidities, multiple drug use and the difficulties coming with advancing age, are the important aspects making the antihypertensive therapy difficult in this age group. Also the decrease in diastolic blood pressure, is a condition that may impair coronary circulation perfused at dilation.

Secondary hypertension due to renal artery stenosis

should always be kept in mind particularly in elderly. In general, stage 2 hypertension with recent onset, hypokalemia without diuretic treatment of resistant hypokalemia under diuretic treatment, progressive creatinine elevation under proper antihypertensive treatment and hypertension resistant to triple medication treatment should direct physicians to secondary hypertension, and further investigation should be performed.

While there is a great body of evidence regarding the beneficial effects of antihypertensive treatment in elderly patients, there is conflicting information on the effectf of antihypertensive therapy in highly advanced age (>80). The most important reasons for this are that there is no study targeting this age group, the data is mostly based on subgroup analyses, and exclusion of advanced age group in many trials. Therefore, various recently published guidelines cannot provide a clear recommendation. Interestingly, the meta-analyses performed on this subject gave confusing results.

For example, in INDANA meta-analysis including 1670 patients, while 36% decrease is observed in the risk of stroke with antihypertensive treatment, mortality was reported to increase by 14%. When HYVET Pilot study gave a similar result, conducting a placebo-controlled, prospective trial became an ethical issue, and the beneficial results obtained with the indapamid and perindopril combination in PROGRESS trial led authors to conduct HYVET trial by using these two drugs. Conducted as an international, multicenter, randomized, double-blind and placebo-controlled trial, HYVET Trial included nearly 5000 patients over 80 years old and having 160-199 mmHg of systolic and >110 mmHg diastolic blood pressure value. First 2 mg and then 4 mg perindopril was added to 1.5 mg SR indapamid versus placebo, and the target blood pressure value was determined as 150-80 mmHg. At the end of a mean follow-up of nearly 2 years, in the stroke treatment group, 30% decrease in the risk of stroke was observed, and more importantly 21% decrease was obtained in all deaths. 64% decrease obtained in cardiac failure is highly dramatic.

Results coming from the subgroup analyses and the recently published meta-analysis results show that antihypertensive therapy in elderly have a beneficial effect in preventing the development of dementia. And this is an important clue supporting the benefit of antihypertensive therapy in elderly.

While there is no consensus on the first choice of treatment in older hypertensives, there is a recent tendency towards avoiding beta blockers, and preferring diuretics or calcium channel blockers. Blood pressure control in older hypertensive mostly requires 2-3 drugs. Fixed dose combinations may be appropriate because of their ease of use, and low side effects due to low-dose components.

In elderly patient, antihypertensive drug should be initiated at its low-dose, the dose should be increased with follow-ups in close intervals, however orthostatic hypotension and extreme decrease in diastolic blood pressure (>60 mmHg) should be kept in mind. During the antihypertensive drug choice process, comorbidities (COPD, BPH, DM, renal dysfunctions, etc.) and drug interactions should be considered. However, it should be remembered that nobody is too old to start antihypertensive treatment.

Important Notes

1. Hypertension is a serious problem seen ¾ of people over 60 years and older.

2. With the aging population, advanced age group segment rapidly grows; in fact in our day, it is the fastest growing part of the society.

3. Cardiovascular risk due to hypertension in elderly is related with elevation of systolic blood pressure, widening of pulse pressure and decrease in diastolic blood pressure. In this context, systolic blood pressure constitutes the key target for treatment.

4. Antihypertensive medication therapy in elderly has various characteristics.

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Percutaneous transcatheter closure of patent foramen ovale in patients with paradoxical embolism

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Summary

Aim: Cryptogenic stroke remains the final diagnosis in 40% of ischemic acute cerebrovascular events. Until now there are no clinical evidences that the percutaneous closure of PFO is able to prevent the recurrence of stroke or transient ischemic attack (TIA). The aim of this study was to evaluate the incidence of recurrence in patients successfully. Treated by percutaneous closure of PFO with different occlude devices by using TDC, TTE and clinical evaluation.

Material and Method: From June 2004 to February 2010, 72 PTS, (40 females and 32 males; everage age 46 yrs, range 14-66), admitted with diagnosis of recurrent ischemic neurologic events (58 stroke and 14 TIA)underwent percutaneous closure of PFO. Thirty-one (43%) of the 72 patients had a concomitant history of migraine, 16(52%) of whom with aura. Five different occlude devices were used, with a total amount of 74 implants. All pts were studied during the follow-up by clinical evaluation (Rankin modified scale), TCD and TTE.

Results: Successful device deployment is achieved in 100% of pts without any periprocedural major complication. Only in two pts atrial arrhythmia have occurred. All pts was discharged within 3 days in good overall conditions. In all pts a double antiplatelet regimen was adopted. The follow-up was complete in 100% of the cases (median 30, range 3-58 months). At five years, there was no recurrent stroke or TIA, and no new cerebral lesions developed by MRI in those patients with residual shunt. Moreover, in 65 (90%)of them the Rankin scale significantly ($P<0.0001$) reduced to 0 whereas only in 2 pts score 1 was reached. In 19 (61%) of the 31 pts with concomitant migraine, the intensity and the frequency of the attacks significantly ($P<0.0001$) decreased over time. At the TCD, 5 pts (7%) resulted positive for microembolic signals but, only 1 of them, was successfully treated for an associate defect. The TTE evaluation showed however an optimal sealing of all the devices without signs of erosion, incomplete closure and thrombus formation around the device.

Conclusion: Our experience suggests that percutaneous treatment of PFO is safe and beneficial at the medium term follow-up for secondary prevention since able to prevent the clinical recurrence of acute cerebrovascular events irrespective of the device used.

Keywords: Stroke, paradoxical embolism, percutaneous transcatheter.

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Introduction

Percutaneous transcatheter closure of patent foramen ovale in patients with paradoxical embolism. Cryptogenic stroke remains the final diagnosis in 40% of ischemic acute cerebrovascular events. Until now there are no clinical evidences that the percutaneous closure of patent foramen ovale (PFO) is able to prevent the recurrence of stroke or transient ischemic attack (TIA).[1,2] Stroke is the leading cause of disability and the third leading cause of death in the developed Countries. Stroke by cardiac source is provoked by various reasons, but PFO, especially if associated with atrial septal aneurysms (ASA) is among the most frequent and complex to investigate. Identifying the cause of neurologic ischemic syndromes is essential to any strategy intended to prevent the catastrophic consequences of cerebral infarction.

Since the initial reports of unexpectedly high prevalence of in younger patients with cryptogenic stroke appeared in 1998, there has been growing interest and experience in diagnosing and treating these patients both medically and/or with percutaneous closure in particular for the potential to eliminate paradoxical embolism via PFO that is a likely mechanism for stroke in these patients.[3,4] The aim of this study was to evaluate the incidence of recurrence of stroke and/or TIA in patients successfully treated percutaneously by using five different well established occlude devices through transcranial Doppler (TCD), transesophageal echocardiography (TEE) and clinical evaluation. Moreover we try to understand whether the selection of the device can influence the outcome in the middle and long term follow-up.

Material and Method

From February 2004 to November 2009, 72 consecutive patients, (40 females and 32 males), average age 46 years, range 14-66), admitted with diagnosis of recurrent ischemic neurologic events (58 stroke and 14 TIA) confirmed by cerebral imaging, underwent percutaneous closure of PFO by using five different occlude devices. Three PTS were scuba-divers and 31 (44%) of the 72 PTS had a concomitant history of migraine, 16 of whom (51%, that is the 22%of total amount) with aura. In 18 pts (25%) the PFO was associated with ASA. According our protocol, the gold standard method for diagnosing PFO in symptomatic patients was been TEE[5] associated with TCD evaluation. The TEE was performed during intravenous injection of agitated saline as a contrast agent, usually accompanied by provocative maneuvers as Valsalva. TCD was performed with intravenous agitated saline in all patients generally without sedation according to the Venice Consensus Conference in 1999 and showed the appearance of reflective bubbles in the intracranial circulation with or without “curtain” effect. Provocative measures are imployed, and the timing of the appearance of contrast after such maneuvers allowed to distinguish between intracardiac and extracardiac (e.g. pulmonary arteriovenous fistula) shunts.

In all patients a permanent from moderate to severe right-to-left shunt was found. PFO length generally range from 4 mm to 25 mm(average 12-13 mm). patients with alternative or additional sources of thromboemboli(atrial fibrillation, deep vein thrombosis) 6 were exclude from the percutaneuos treatment. The detection of inherited thrombophilic disorders, in particular factor V Leiden and prothrombin gene mutation in six patients of our opulation study since is a risk factor of paradoxical embolism in subjects with PFO, in our thought, did not represented a contraindication for percutaneous closure.

The selection of the appropriate occlude device was made taking in account the morphology of the defect (septum primum and septum secundum characteristics) and the association with an aneurysms of a fossa ovalis (ASA) defined according the definition of Mugge et al.[7] As an interatrial septum of abnormal mobility with protrusion of the septum into the left or right atrium of leatst 10 mm beyond baseline, or with other atrial defects. Altogether, 74 occluder device s were implanted. In particular, we implanted 27 Premere Occlusion System (St Jude Medical Inc., Maple Grove, MN, USA), 28 Amplatzer Occluder, 12 Atriasept, 5 Biosatar (NMT Medical Inc, Boston, MA, USA), 2 Solysafe System.

In two patients implantation of a double devce was performed due to the persistence of a moderate right to left shunt after the procedure. The Amplatzer PFO or cribiform device was selected in case of highly redundant and floppy septum primum, associated ASA,

or when septum primum tissue was plentifully defective or showed multifenestrated aspect. The Premere device, due to its low profile, was chosen in case of tunnel–like morphology especially with very long channels (>15mm), in absence of ASA, in presence of a PFO with minimal distance between the two septa and a medium or long longitudinal diameter.

The Atriatsept was selected in those patients presenting a septum secundum very thick or complex connections between the two septa. The introduction of intracardiac echocardiography[8] (ICE) Acunav 8 fr, Acuson Siemens) has made intraprocedural TEE necessary only in the first 12 treated patients of our series, so the procedure was performed using local anesthesia under mild conscious sedation in less than 20 minutes in almost all patients. Bilateral venous access was gained via the right and left femoral vein and the PFO was catheterized using a multipurpose catheter under ICE guidance. The difference occluder devices were implanted in accordance to the specific Company implantation recommendations.

All patients received 100IU/Kg heparin i.v. at the time of the procedure and antibiotic prophylaxis. All treated patients were studied during the follow-up by clinical neurologic evaluation (Rankin modified scale) and TCD with microbubbles test in basal condition and after Valsalva manoeuvre at 6 months and by transthoracic echocardiography at 1.6 and 12 months. If at 6 months a moderate shunt is detected (>10 microbubbles), transesophageal echocardiography was performed. The follow-up was 100% complete in all treated patients.

Results

Successful device deployment is achieved in 100% of patients without any intra or periprocedural major complication. Only in two patients atrial arrhythmia have occurred: in one case within the first hours after catheter-based therapy and and spontaneously cessed, while in the second successful DC shock cardioversion was performed at the end of the procedure. In the follow-up period only one patient developed some episodes .

In the follow-up period only one patient developed some episodes of paroxysmal supraventricular tachycardia that convert back to sinus rhythm spontaneously. Significantly, the use of a double vein approach for ICE probe introduction and implantation procedure did not increase the overall incidence of periinterventional vascular complications (1 patient with a small arteriovenous fistula which didn't require surgical treatment but only pressure bandage). All pts were discharged within 3 days in good overall conditions.In all pts a double antiplatelet regimen was adopted for 3-6 months. All patients received a scheme for prophylaxis of bacterial endocarditis as usually recommended for all adult congenital heart diseases.

The follow-up was complete in 100% of the cases (median 30, range 3-58 months). At five years of follow-up there was no recurrent episode of stroke and/or TIA documented by nuclear magnetic resonance (NMR) and neurological examination, and no cases of recurrence of acute cerebrovascular events was observed in older patients irrespective of the device used.

Moreover, in 52 of them (89.6%), the Rankin scale reduced to 0 whereas only in 4 patients score 1 was reached. In 19 of the 31 patients with concomitant migraine (61%), the intensity and the frequency of the attacks decreased over time in particular in those forms of migraines preceded by aura, while the others form of headache don't seems to improve particularly after the closure. At 6 months TCD, 3 patients resulted slightly positive for microembolic signals (10-12 HITS detected one of them after Valsalva manoeuvre), while in 2 patients a moderate right-to-left shunt (22 and 34 HITS without curtain effect and only after Valsalva manoeuvre) was detected despite the optimal sealing of the implanted device and the absence of any device-related thrombotic formation. All these patients received an Amplatzer PFO or cribriform device.

However, no recurrent neurological event occurred documented at the MRI evaluation performed in these patients and the contrast-TEE performed confirmed the presence of residual shunt in all five patient except one. In our experience it's important to perform TCD starting from the 6 month from the procedure because the endothelisation process is almost complete. The TTC evaluation at 1,6 and 12 month showed an optimal sealing of all device, without any signs of erosion,

malapposition, dislodgement or incomplete closure and thrombus formation around the device.

Discussion

Stroke represents the major cause of disability in most European populations, contributing in large part to the escalating costs of health care. In particular cryptogenic stroke is a devastating experience since affected young, full force, healthy persons and their familial entourage.[9,10] Form this point of view, it's important to offer a safe and less invasive therapeutic chance to these kind of patients that minimizes the treatment and allows the patient to timely undergo rehabilitation program. Our experience suggests that percutaneous treatment of PFO appears safe and beneficial for secondary prevention since it is able to prevent the clinical recurrence of acute cerebrovascular events reducing the incidence of these latter nearly to 0% and making our results comparable or less frequent to others surveys.[11,12] This is true also in elderly treated patients.

Moreover, the remarkable improvement of the Rankin scale in patients admitted with stroke diagnosis (who are the majority), demonstrated that the catheter-based therapy is effective not only for secondary prevention but also in terms of improvement of considerable improvement in migraine attacks frequency and or intensity after PFO closure (61%), especially in those patients whose migraine is preceded by aura, is in line with the literature data[13,14] and can offers in some cases a cure since migraine represents today an important health problem accounting for a high social cost due to the fact that heavily affect the quality of life, not to mention that, some observational studies reported that migraine may be for itself a risk for subclinical brain lesions and stroke.[15] The principal determinant of a successful procedure is the choice of the appropriate device for the specific morphology of the defect.

Our experience suggests that the effectiveness of each kind of occluder device is likely to depend form different anatomy of the defect. The foramen ovale has a various anatomic configuration, and therefore the transesophagel echocardiographic evaluation prior of treatment is essential for the catheter-based strategy. The morphology of foramen ovale show a remarkable variability from case to case, both from a pathological and echocardiographic point of view. Some aspects can have great relevance form a clinical and or technical point view in relation to a possible closure procedure and therefore must be carefully investigated by transesophageal evaluation. When the defect has a tunnel-like morphology especially with very long channels (>15 mm), in absence of ASA, or when minimal distance between the two septa and a medium or long longitudinal diameter are found, the most performing device in terms of lower mechanical impact over interatrial septum is, the Premere occluder device; this latter has demonstrated its validity also during the follow-up period since no residual shunt was detected at TCD evaluation in all 27 patients that received it and all the patients remained asymptomatic.

Similar good performance showed the Atriasept and Biostar device even thought a small number of patients received them. Notably the Atriasept device due to its high flexibility is especially suitable for very thick septum secundum until some pictures of "lipomatous septum" observed, in which the thickness is more than 1 cm. On the other hand, five patients revealed in the follow-up period a residual right-to-left shunt, remaining however asymptomatic, and all received an Amplatzer device. In all these patients the PFO was associated with ASA. This finding can be partially explained by the consideration that the complexity of the atrial defect, the larger PFO diameter, a multifenestrated anatomy and the associaton with ASA and remnants structures as Eustachian valve and Chiari's network founded in these patients prior to closure has required the use of a device[3], as the Amplatzer, with an higher mechanical impact and a more ability to stabilize the atrial septum between the two discs of the device.

The presence of ASA is often associated with large tunnel PFO and relevant right-to-left shunt and this seems to increase the risk of stroke in these patients. Infact the coexistence of PFO and ASA results in a two- to fourfold increase in the risk of recurrent thromboembolic events in patients with cryptogenic stroke as compared to patients with PFO alone.[16] Moreover the aneurysm morphology is more often associated with persistent remnants venous valves that, according with what was recently reported, seems for itself increase the risk for stroke.[17] The Chiari's network is a remnant

of the right valve of the sinus venosus and is constitutes from a network of thin filaments poorly echoic originates from a region of the eustachian valves with attachment the upper wall of the right atrium or atrial septum, floating inside the right atrium.

The eustachian valve is a membranous structure, with high eco-reflectivity that extends from the junction between the inferior vena cava and right atrium, with in the right atrium, toward the entrance of PFO. The Eustachian valve is considerate reduntant when his lenght exceed 10 mm. A recent study using contrast enhanced TEE found a frequent association between Chiari's network and PFO and between large right-to-left shunting and Chiari's network. This study also detected Chiari's network in 24% of patients with ASA.

Moreover the Chiari's network is more common in cryptogenic stroke patients than in patients evaluated for other indications and it may facilitate paradoxic embolism.[18] In two patients with residual shunt treated with cribrioform Amplatzer device, the analysis performed prior to closure by TEE, showed a septum primum widely defective containing multiple fenestrations: they are small interatrial communications in the context of septum primum, more frequently associated with ASA. They are located in contiguity with the entrance of PFO in the right atrium, or near to the entrance of inferior vena cava in the right atrium, in the inferoposterior portion of the septum primum. They must be carefully sought at TEE since can be a cause of significant residual right-to-left shunt after transcatheter closure of PFO.

In follow-up, we observed that if TCD is negative, no further examination may be necessary; whereas in all cases in which TCD showed a residual shunt, it is advisable to perform a TEE investigation. In conclusion in patient with history of cryptogenic thrombo-embolic events potentially correlated to the presence of PFO a carefully anatomic and functional evaluation of the morphology of the defect with TEE is mandatory for any strategy intended to close the PFO and the associated defects.

On the other hand transcatheter -based therapy in symptomatic patients is a safe and effective procedure and has demonstrated on the medium term follow-up the potential to reduce the risk of recurrent neurological events irrespective of the five devices used appeared basically superior over the others. The present study showed that beside the extensive use of consolidates devices a reasonable good outcome, that required further studies with a larger patient population, was observed in particular with the use of Premere occluder device.

References

1. Sacco RL, Ellenbergh JH, Mohr JP, Tatemichi TK, Hier DB, Price TR et al. Infract of undetermined cause: the NINCDS Stroke Data Bank. Ann Neurol 1989;25:382-90.
2. Sherman DG. Prevention of Cardioembolic Stroke. In: Norris JW, Hachinski VC,editors. Prevention of Stroke. New York, NY: Springer Verlag; 1991.p.149-59.
3. Lechat P, Mas JL, Lacault G, Loron P, Theard M, Klimczac M et al. Prevalence of patient foramen ovale in patients with Stroke. N Engl J Med 1988;318:1148-52.
4. Webster MW, Chancellor AM, Smith HJ, Swift DL, Sharpe DN, Bass NM et al. Patent foramen ovale in young stroke patients. Lancet 1988;2:11-2.
5. Cabanes L, Mas JL, Cohen A, Amarenco P, Cabanes PA, Oubary P et al. Atrial septal aneurysm and patent foramen ovale as risk factors for cryptogenic stroke in patients less than 55 years of age: a study using transesophagel echocardigraphy. Stroke 1993;24:1865-73.
6. Cramer SC, Rordor G, Maki JH, Kramer LA, Grotta JC, Buring WS et al. Increased pelvic vein thrombi in cryptogenic stroke: results of the Paradoxical Emboli formLarge Veins in cryptogenic stroke (PELVIS) study. Stroke 2004;35:46-50.
7. Mugge A, Daniel WG, Angermann C, Spes C, Khanderia BK, Kronzon I et al. Atrial septal aneurysm in adults patients. A multicenter study using transthoracic and transesophageal echocariography. Circulation 1995;91:2785-92.
8. Zanchetta M, Rigatelli G, Onorato E. Intracardiac echocardigraphy and transcranial Doppler ultrasound to guide closure of patent foramen ovale.J Invas Cardiol 2003;15:93-6.
9. Di Tullio M, Sacco RL, Gopal A, Mohr JP, Homma S. Patent foramen ovale as a risck factor for cryptogenic stroke. Ann Intern Med 1992;117:461-5.
10. Bogousslavsky J,Garanzi S,Jeanrenaud X, Aebischer N, Van Melle G. Stroke recurrence in patients with patent foramen ovale. The Lausanne Study. Lausanne Stroke with Paradoxical Embolism Study Group. Neurology 1996;46:1301-5.
11. Windeker S, Wahl A, Chatterjee T, Grachemani A, Eberli FR, Seiler C, Meier B. Percutaneous clouse of patent foramen ovale in patients with paradoxical embolism: long-term risk of recurrent thromboembolic events. Circulation 2000;101:893-8.
12. Martin F, Sanchez PL, Doherty E, Colon-Hernandez PJ, Delago G, Inglessis I et al. Percutaneous transcatheter closure of patent foramen ovale in patients with paradoxical embolism. Circulation 2002;106:1121-6.
13. Schwerzamann M, Wiher S, Nedelttchev K. Percutaneous closure of patent foraman ovale reduces the frequency of migraine attaks. Neurology 2004;2:1399-401.
14. Giardini A, Donti A, Formigari R, Salomone L, Prandstraller D, Bonvicini M et al. Transcatherter patent foramen ovale closure mitigates aura migraine headaches abolishing spontaneous rigth-to-left shunting. Am Heart J 2006;151:922. E 1-5.
15. Ferrarini G, Malferrari G, Zucco R, Gaddi O, Norina M, Pini LA. Hight prevalence of patent foramen ovale in migraine with aura. J Headache Pain 2005;6:71-6.
16. De Castro S, Cartoni D, Fiorelli M,Rasura M, Anzini A, Zanette EM et al. Morphological and functional characteristic of patent foramen ovale and their embolic implications. Stroke 2000; 31:2407-13.
17. Rigatelli G, Dell'Avvocata F, Braggion G,Giordan M, Chinaglia M, Cardaioli P. Persistent venous valves correlate with increate shunt and multiple preceding embolic events in patients with patent foram ovale: an intracardiac echocardiographi study. Cathete Cardiovasc Interv 2008;72:973-6.
18. Schneider B, Hofmann T, Justen MH, Meinertz T. Chiari's network : normal anatomic variant or risk factor for arterial embolic events? J Am Coll Cardiol 1995;26:203-10.

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Surgical treatment of patients with coronary heart disease and mild stenosis of the aortic valve

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Summary

Objective: Management of asymptomatic mild aortic stenosis at the time of coronary artery bypass grafting remains controversial. Therefore we have reviewed our experience with such patients.

Material and Method: We have retrospectively analyzed a cohort of 42 patients (group A) with asymptomatic aortic valve lesion subjected to isolated coronary artery bypass (38 men and 4 women with a mean age of 56.4 ± 6.5 years) and compared their with a group of 67 patients (group B) receiving coronary artery bypass grafting and aortic valve replacement simultaneously at the first operation (61 men and 6 women with a mean age of 63.4 ± 7.3 years). No significant differences in the characteristics of patients in both groups were noted.

Results: Acute heart failure, perioperative myocardial infarction, heart arrhythmia, respiratory failure, most often observed among patients of group B. Hospital mortality among these patients was also higher (6.3% versus 0, $p > 0.05$). However, these differences were not significant. 11 (26.1%) patients, who had undergone CABG an average of 30.6 ± 13.3 months previously, were subsequently re-operated due to progression of aortic stenosis. We noted that mean preoperative aortic gradient (34.8 ± 4.4 versus 26.3 ± 7.5 mm. Hg $p < 0.05$) and calcification of the aortic valve (1.6 ± 0.5 versus 0.8 ± 0.7 $p< 0.05$) in these patients was significantly higher than the remaining patients of group A. Progression of valvar calcification has led to a decrease in the area of the aortic opening an average of 0.25 ± 0.04 cm ^{2}/year and an increase in the gradient, an average of 13.3 ± 9.3 mm.Hg/ year.

Conclusion: Thereby, calcification of the aortic valve with mild to moderate aortic stenosis and a mean pressure gradient within 25-40 mm Hg is sufficient to carry out combined operations. Execution only isolated coronary artery bypass grafting determines the greater likelihood of re-operation because of the inevitable progression of aortic valve calcification.

Keywords: Aortic valve disease, coronary bypass surgery, aortic stenosis.

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Introduction

The combined atherosclerotic lesion of coronary arteries and aortic valve is a typical and common condition especially among elderly patients. Management of asymptomatic mild aortic stenosis at the time of coronary artery bypass grafting remains controversial. Some surgeons advocate aortic valve replacement (AVR) on the assumption that many of these patients will develop significant valve disease within a few years time.[1]

Thus they would otherwise be exposed to the risks of a redo operation to replace the valve. On the other hand, critics of this position believe that prophylactic AVR for mild aortic stenosis in a patient whose primary symptoms are coronary insufficiency unjustifiably increases the operative mortality as well as the risk of subsequent valve-related events, and such patients should not have valve replacement until hemodynamically significant aortic stenosis develops.[3] Therefore we have reviewed our experience with such patients.

Materials and methods

We have retrospectively analyzed a cohort of 42 patients (group A) with asymptomatic aortic valve (AV) lesion subjected to isolated coronary artery bypass (38 men and 4 women with a mean age of 56.4 ± 6.5 years) and compared their with a group of 67 patients (group B) receiving coronary artery bypass grafting and aortic valve replacement simultaneously at the first operation (61 men and 6 women with a mean age of 63.4 ± 7.3 years). New York Heart Association (NYHA) functional class, history of myocardial infarction, prior operation was recorded. Angina was graded using the Canadian Cardiovascular Society (CCS) classification.

Cardiac catheterization findings recorded included number of diseased vessels and left ventricular (LV) function. LV end-diastolic pressure, state of the AV and systolic gradient across the AV at first and subsequent catheterizations were also recorded. Aortic stenosis was considered mild when the aortic valve area was 1.0 cm^2 or greater, moderate when the area was less than 1.0 cm^2 but greater than 0.7 cm^2, and severe when the area was 0.7 cm^2 or less. Progression of aortic stenosis was considered to have occurred after stenosis increased from mild to moderate, mild to severe, or moderate to severe on the basis of the calculated aortic valve area.

The general conduct of all surgical procedures was similar. The operations were performed using a membrane oxygenator, systemic normothermia (34-36°C), hemodilution and cardioplegic solution.

Results

Both groups were similar with respect to the risk factors for heart disease. The extent of coronary artery disease and the frequency of preoperative myocardial infarction were similar. The majority of patients in both cohorts had mild left ventricular dysfunction. No significant differences in the characteristics of patients in both groups were noted (Table 1).

Table 1. Patient characteristics

Variable	Group A (n-42)	Group B (n-67)	p Value
Angina class (CCS):			
III	**(83.4%)**	**54 (80.5%)**	**NS**
IY	**7 (17.1%)**	**13 (19.4%)**	
NYHA class:			
II	**5 (11.9%)**	**7 (10.4%)**	**NS**
III	**28(66.7%)**	**49 (73.1%)**	
IY	**9 (21.4%)**	**11 (16.2%)**	
Myocardial infarction	**19 (45.2%)**	**34 (50.1%)**	**NS**
Ejection Fraction < 0.5	**17 (40.4%)**	**31 (46.2%)**	**NS**
Triple and more Coronary lesion	**23 (54.7%)**	**38 (56.7%)**	**NS**
NYHA = New York Heart Association;	*CCS = Canadian Cardiovascular Society.*		

Table 2. Cardiac catheterization data

Variable	Group A (n-42)	Group B (n-67)	p Value
Mean AV gradient (mm Hg)	28.4 ± 8.1	56.2 ± 11.7	< 0.05
Aortic valve area (cm2)	1.5 ± 0.4	0.7 ± 0.3	< 0.05
Aortic valve regurgitation	0.9 ± 0.5	2.1 ± 1.3	< 0.05
LVEDP (mm Hg)	10.8 ± 2.6	17.5 ± 8.5	< 0.05
Calcification of the AV	0.9 ± 0.8	1.7 ± 0.7	< 0.05

LVEDP = left ventricular end-diastolic pressure; AV = aortic valve.

None of the patients in group A were considered to have hemodynamically significant aortic stenosis at the time of the initial myocardial revascularization. Patients of group B had a significant stenosis and calcification of the aortic valve (Table 2).

In both groups the use of internal mammary artery (IMA) and the number of bypass grafts inserted were similar. Patients in group B were undergoing combined (CABG and AVR) procedures and not unexpectedly incurred a significantly greater global myocardial ischemic and extracorporeal circulation time (Table 3).

Postoperative complications occurred with a significantly greater frequency in those patients having AVR and myocardial revascularization. The most frequent postoperative complications were acute heart failure, perioperative myocardial infarction, heart arrhythmia, (Table 4). Hospital mortality among these patients was also higher. There were 4 patients (group A - 0; group B - 5.9%; not significant) who died in the hospital.

The most common cause of death was low cardiac output. (n – 2). The remaining 2 hospital deaths were caused by multiorgan system failure (n-1) and cerebrovascular accident (n-1). Five-year survival in both groups was similar (Fig.3).

Figure 3. ***Survival***

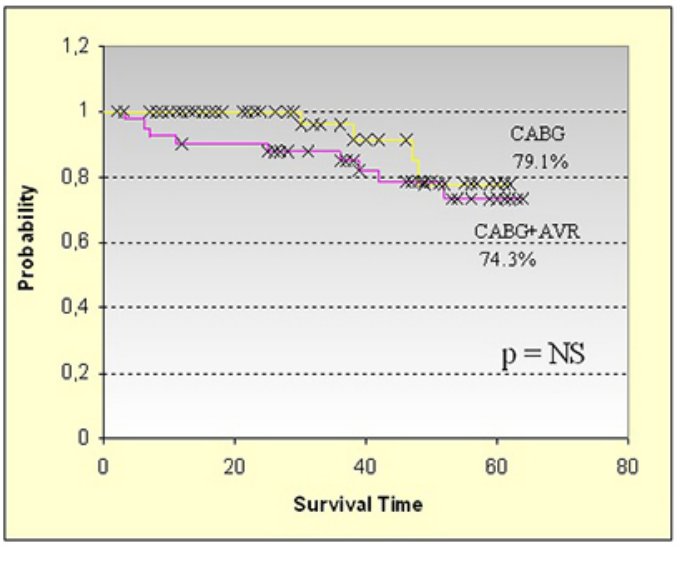

Table 3. Operative data

Variable	Group A (n-42)	Group B (n-67)	p Value
IMA	40 (95.2%)	61 (91.1%)	NS
No. of CAB grafts	3.2 ± 1.1	2.8 ± 0.8	NS
CPB time (min)	79 ± 26.4	147 ± 31.2	< 0.05
Cross-clamp time (min)	40.2 ± 18.8	98 ± 22.3	< 0.05

CAB = coronary artery bypass; CPB = cardiopulmonary bypass; IMA = Internal mammary artery NS = not significant.

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Table 4. Postoperative complications

Variable	Group A (n-42)	Group B (n-67)	p Value
Acute cardiac failure	3 (7.1%)	11 (16.4%)	NS
Arrhythmia	5 (11.9%)	19(28.3%)	< 0.05
Myocardial infarction	2 (4.7%)	4 (5.9%)	NS
Stroke	-	1 (1.4%)	NS
Postoperative bleeding	-	5 (7.4%)	NS
Mortality	-	4 (5.9%)	NS

Discussion

We emphasize that our review does not provide information on the incidence of need for subsequent valve replacement in the category under consideration, ie, concomitant mild aortic stenosis and coronary artery disease requiring reoperation. Our review does confirm that in at least a portion of such cases, the valve stenosis progresses and reoperation causes a burden of increased risk.

This observation has been reported by Collins and associates. According to their research appearance of symptoms and signs of severe AS occurred in 16% by 3 years; 45% by 4 years; and 75% by 5 years after CABG surgery. They demonstrated a 23.5% operative mortality for reoperative AVR after CABG compared with 7.6% for reoperative AVR without CABG and 6.6% for primary AVR with CABG.[2] The reasons for this high mortality are multifactorial. At the time of reoperation the patients are older and the procedure takes longer. Age, prolonged bypass time, and prolonged cross-clamp time are the strongest independent predictors of mortality after AVR.[4,5]

Patients requiring AVR subsequent to CABG have progressive native and graft coronary atherosclerosis, which may need to be addressed at reoperation.[6] 11 (26.1%) patients in our study, who had undergone CABG an average of 30.6 ± 13.3 months previously, were subsequently re-operated due to progression of aortic stenosis. The average age was 66.8 ± 5.2 years. We analyzed the patients subjected to re-operation, and noted that mean preoperative aortic gradient (34.8 ± 4.4 versus 26.3 ± 7.5 mm. Hg $p < 0.05$) and calcification of the aortic valve (1.6 ± 0.5 versus 0.8 ± 0.7 $p< 0.05$) in these patients was significantly higher than the remaining patients of this group (**Table 5**).

Progression of valvar calcification has led to a decrease in the area of the aortic opening an average of 0.25 ± 0.04 cm^2/year and an increase in the gradient, an

Table 5. Cardiac catheterization data re-operated and the remaining patients of Group A.
(before the first operation)

Variable	Re-operated patients (n-11)	Non-operated patients (n-31)	p Value
Mean AV gradient (mm Hg)	34.8 ± 4.4	26.3 ± 7.5	< 0.05
Aortic valve area (cm2)	1.4 ± 0.3	1.6 ± 0.2	NS
Aortic valve regurgitation	1.1 ± 0.3	0.9 ± 0.4	NS
LVEDP (mm Hg)	12.3 ± 2.3	11.2 ± 2.5	NS
Calcification of the AV	1.6 ± 0.5	0.8 ± 0.7	< 0.05

LVEDP = left ventricular end-diastolic pressure; AV= aortic valve

Table 6. Dynamics of Cardiac Catheterization Data Re-operated Patients

Variable	before the first operation	before the second operation	p Value
Mean AV gradient (mm Hg)	34.8 ± 4.4	68.2 ± 22,9	< 0.05
Aortic valve area (cm2)	1.4 ± 0.3	0.8 ± 0.2	< 0.05
Aortic valve regurgitation	1.1 ± 0.3	1.9 ± 0.9	NS
LVEDP (mm Hg)	12.3 ± 2.3	17.1 ± 7.6	< 0.05
Calcification of the aortic valve	1.6 ± 0.5	2.6 ± 0.4	< 0.05

LVEDP = left ventricular end-diastolic pressure.

average of 13.3 ± 9.3 mm.Hg/ year (Table 6). Changes in the aortic valve were atherosclerotic in nature, which manifested significant calcification valves and fibrous ring (Fig 1).

Main difficulties encountered during re-operation were: access to the heart, myocardial protection, high probability of damage to the grafts, the limited space on the aorta (Fig.2).

Hospital mortality (n-2) among these patients was higher than in the group with combined operation (CABG and AVR) and was 18.2% versus 5.9% (p>0.05). 1 patient died as a result of myocardial infarction due to injury IMA, and the second cause of death was cerebrovascular accident.

Conclusion

These data observations suggest that mild, asymptomatic valve deformity may progress to symptomatic, hemodynamically severe AS within a short time after CABG surgery, well before recurrent symptoms of coronary obstructive disease. The analysis of our results in comparison with other studies shows that a moderate stenosis of the aortic valve leaflets with calcification and mean pressure gradient within 25-40 mm Hg is sufficient reason for the audit of the aortic valve and combined operations. Execution only isolated coronary artery bypass grafting determines the greater likelihood of re-operation because of the inevitable progression of aortic valve calcification.

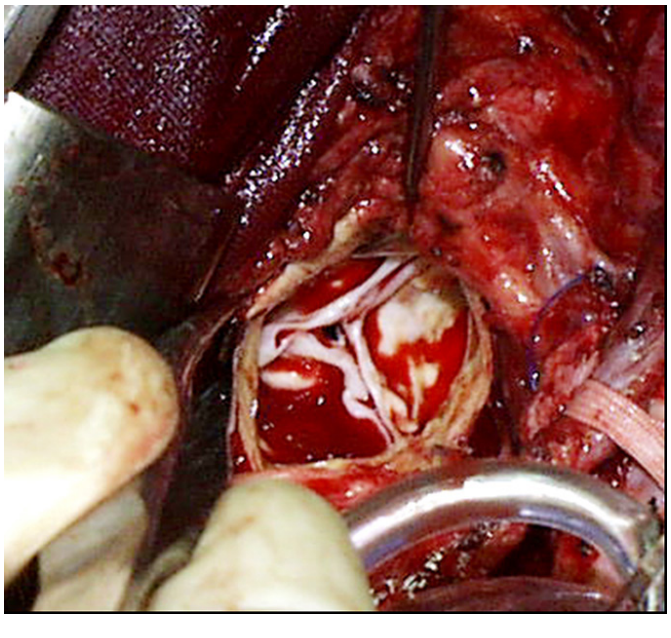

***Figure 1.** Aortic valve at re-operation*

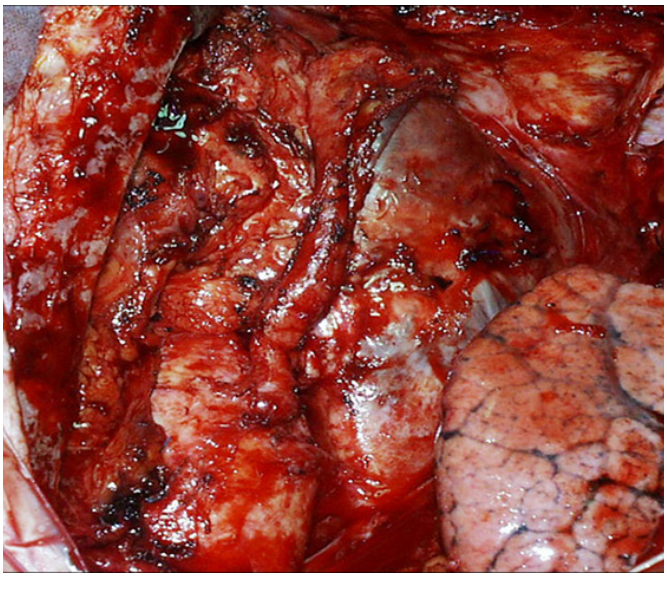

***Figure 2.** The heart and ascending aorta with re-operation*

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References

1. Alsir A.M. Ahmed, Alastair N.J. Graham, Deirdre Lovell, Hugh O. O'Kane Management of mild to moderate aortic valve disease during coronary artery bypass grafting. *Eur J Cardiothorac Surg* 2003; 24: 535-540.
2. Collins JJ Jr, Aranki SF. Management of mild aortic stenosis during coronary artery bypass graft surgery. *J Cardiac Surg* 1994:9 (Suppl):145–7.
3. Davies S.W., Gershlick A.H., Balcon R. Progression of valvar aortic stenosis: a long-term retrospective study. *Eur Heart J* 1991; 12(1): 10-14.
4. Jacob P. Dal-Bianco, MD. Management of Asymptomatic Severe Aortic Stenosis *J Am Coll Cardiol,* 2008; 52: 1279-1292.
5. James W. Tam, MDa, Roy G. Masters, MDa, Ian G. Burwash, MDa, Alain D. Mayhew, BSa, Kwan-Leung Chan, MD. Management of Patients With Mild Aortic Stenosis Undergoing Coronary Artery Bypass Grafting. *Ann Thorac Surg* 1998;65:1215-1219
6. T. Mihaljevic, M. R. Sayeed, S. C. Stamou, and S. Paul. Pathophysiology of aortic valve disease. Card. Surg. Adult, 2008; 3 (2008): 825 - 840.

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Surgical repair of diversion of inferior vena cava into the left atrium nine years after surgical repair of atrial septal defect

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Summary

Although surgical repair of ASD is fairly safe and a routine procedure very rarely it may be complicated to diversion of inferior vena cava (IVC) into the left atrium. In this report we present a 30 year old male patient who had undergone surgical repair of ASD nine years before he came to our clinic. An echocardiogram was performed and revealed diversion of inferior vena cava into left atrium. Surgical repair of this complication was performed by reconstructing the opening of the IVC and redirecting it into the right atrium.

Keywords: Atrial septal defect, ciyanosis, diversion of inferior vena cava.

Introduction

Atrial septal defect (ASD) is the third most common congenital cardiac malformation.[(1)] Although surgical repair of ASD is fairly safe and a routine procedure very rarely it may be complicated to diversion of inferior vena cava (IVC) into left atrium. Diversion of inferior vena cava into left atrium; although unusual; remains a cause of morbidity following repair of ASD. This leads to dyspnea and hypoxemia which may present immediately postoperative or several years later. In this report we present a 30 year old male patient who started suffering from mild dyspnea and cyanosis on effort one year after surgical repair of ASD. He was brought to the emergency clinic when he underwent a trauma nine years after the closure of ASD, and on performing complete blood count his hematocrit was %65 and hemoglobin 20 mg/dl. An echocardiogram

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was performed and revealed diversion of inferior vena cava into left atrium. Surgical repair of this complication was performed by reconstructing the opening of the IVC and redirecting it into the right atrium.

Case

A 30 year old male had undergone surgical repair of ASD in another hospital nine years ago before he presented to our outpatient clinic. One year after the first surgical repair of his ASD he started suffering from perioral cyanosis and dyspnea on effort. The patient neglected these complaints till he fell down from a tree nine years after the first surgery. He was brought to the emergency clinic of another hospital and on performing complete blood count (CBC) his hematocrit was %65 and hemoglobin 20 mg/dl.

An echocardiogram was performed and revealed diversion of inferior vena cava into left atrium. Then he was referred to our outpatient clinic. His blood pressure was 110/70 mmgh and heart rate was 87/min. On physical examination of the patient, mild central cyanosis and clubbing of the fingers of his hands were observed. There was 2/6 systolic ejection murmur with a fixed split S2. Cardiac catheterization was performed as well and confirmed the diagnosis of diversion of inferior vena cava into left atrium. Surgical repair of this complication was performed by reconstruction of the opening of the IVC and redirecting it into the right atrium.

Surgical technique:

After re-median sternotomy and dissection the adhesions, aortobicaval cannulation was performed. The purse sutures were place on the IVC and SVC so the tow caval cannules were inserted directly into the IVC and SVC. The Superior and inferior vena cava were snared by the tapes. Cardiopulmonary bypass started and the ascending aorta cross clamped. Ante grade blood cardioplagia was administered and moderate hypothermia established, thus physiological cardiac arrest performed. We stained the inferior and superior vena caval snares. On opening the right atrium we found that the orifice of the IVC had completely occluded with a scar tissue which we thought that it had been formed by the healing process. On making an incision over the scar tissue we observed the diversion of the IVC into the LA and there was a residual ASD which was closed by primary sutures (**fig.1a**). We dissected the upper part of the IVC and found its opening into the left atrium as seen in **fig.1b**. The cannules of the IVC was removed and total circulatory arrest established. The upper part of the IVC was reconstructed by a Dacron graft patch and redirected into the right atrium as seen in **Figure 2**.

The cannule was re-inserted into the IVC and total circulatory arrest finished. The period of total circulatory arrest was 8 minutes. The right atriotomy was closed and the cross clamp removed. After recovering from the hypothermia, weaning from CPB was uneventful and the sterontomy closed. Postoperative Po2, So2, Hct

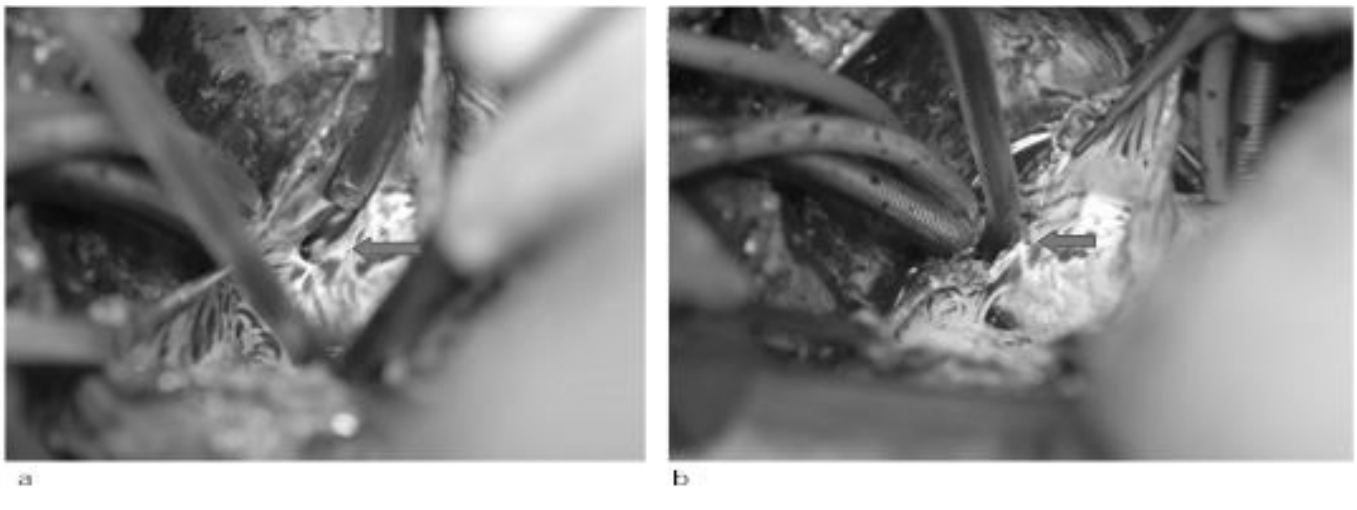

Figure 1. ***A residual ASD (arrow) and the disappearance of the orifice of the IVC in the right atrium surface. B the upper part of the IVC after being dissected and observing the diversion to the left atrium (arrow).***

and, Hb were 87, %98 %32 and 10mg/dl respectively. The patient was followed up and no complication was reported and the cyanosis disappeared. He was discharged on the fourth day postoperatively. Two months later he came to our outpatient clinic for control. He had no complaints. Echocardiogram revealed no pathology. His Hct and Hb were %37 and 11.5mg/dl respectively.

Discussion

Diversion of inferior vena cava into left atrium complicating surgical repair of ASD is a rare condition. As far as we know few cases were published in the recent years, yet it was more frequently seen before the use of cardiopulmonary bypass because time limitations were imposed by only hypothermia and no inflow occlusion.[2]

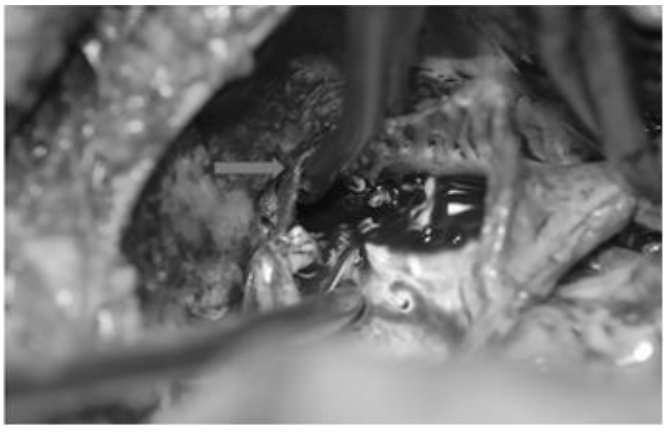

Figure 2. ***The orifice of the IVC after being reconstructed by a Dacron graft (arrow).***

This is an unusual case was discovered nine years after the first operation. Usually the symptoms of this complication appear soon after surgery when the patient becomes cyanotic and hypoxic however some factors such as the relief of pulmonary venous congestion and right ventricular strain by the correction of the left-to-right shunt, the occurrence of only partial diversion of the IVC flow to the LA, and the occurrence of stenosis of the IVC, with collaterals draining to the superior vena cava through an azygos vein may contribute to the appearance of the symptoms of this complication later in life.[3] Reported factors associated with this complication include a large secundum defect or sinus venosus defect, and anomalous pulmonary return into the RA.[4]

In our case we observed that the inferior rim of the defect was not included in the first surgical closure. In addition; there was a scar tissue covering the upper part of the IVC on the RA surface so we believe that there was partial diversion of the IVC at first and by healing process complete diversion of the cava occurred gradually. In conclusion, although unusual; diversion of IVC to LA must be considered in cyanotic patients who underwent surgical repair of ASD.

References

1. Murphy JG, Gersh BJ, McGoon, Mair, Porter CJ, Ilstrup. Long-term outcome after surgical repair of isolated atrial septal defect. Follow-up at 27 to 32 years. *N Engl J Med* 1990; 323:1645-50.
2. 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: 8518.
3. Muath Alanbaei, Luc Jutras, Judith Therrien, and Ariane Marelli, Iatrogenic cyanosis and clubbing: 25 years of chronic hypoxia after the repair of an atrial septal defect. *Can J Cardiol.* 2007; 23(11): 901–903.
4. Ellen Thompson, Dennis Moritz, Romaine Perdue, and Silvestre Cansino. Diversion of the Inferior Vena Cava Following Repair of Atrial Septal Defect Causing Hypoxemia. Echocardiography: *A Jrnl. of CV Ultrasound & Allied Tech.* Vol. 21, No. 4, 2004.

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A rare cause of ischemic chest pain: Congenital Ostial Atresia of the right coronary artery

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Summary

The prevalence of congenital coronary anomalies is reported in about 0.5% to 1.5% of patients undergoing coronary angiography. Tüccar E. And Elhan A. Just reported coronary artery anomalies in adult Turkish population. They analysed 5000 coronary angiograms. Twenty-five (0.5%) coronary arteries with anomalous origins were found. We are presenting an extremely rare right coronary origin abnormality. In this case right coronary artery originates from left anterior descending coronary artery. According to general opinion, coronary segments with an anomalous course are no more susceptible to coronary atherosclerosis than normal segments in the same individual. Only 10.1% of patients with congenital coronary artery anomalies (CCAA) identified during cardiac catherization, had another congenital heart defect. Coronary artery anomalies are very important because coronary artery anomalies rank second as a cardiovascular cause of sudden death in the young, behind hypertrophic cardiomyopathy.

Keywords: Congenital coronary anomalies, ostial atresia, coronary atherosclerosis.

Introduction

The prevalence of congenital coronary anomalies is reported in about 0.5% to 1.5% of patients undergoing coronary angiography.[(1)] Tüccar E. And Elhan A. Just reported coronary artery anomalies in adult Turkish population. They analysed 5000 coronary angiograms. Twenty-five (0.5%) coronary arteries with anomalous origins were found.[(2)] We are presenting an extremely rare right coronary origin abnormality. In this case right coronary artery originates from left anterior descending coronary artery. According to general opinion, coronary segments with an anomalous course are no more susceptible to coronary atherosclerosis than normal segments in the same individual. Only 10.1% of patients with congenital coronary artery anomalies (CCAA) id-

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entified during cardiac catherization, had another congenital heart defect.[3] Coronary artery anomalies are very important because coronary artery anomalies rank second as a cardiovascular cause of sudden death in the young, behind hypertrophic cardiomyopathy.[4]

Case:

Initial Presentation: 55 year-old male with chest pain without any radiation, diaphoresis, and nausea.

History of Present Complain: Two months ago, after his first physical examination, he was treated by an emergency room physician at a local hospital. His history was very likely to myocardial infarction.

Transthoracic echocardiography showed: LV global hypokinesia LV EF: %50, LV diastolic dysfunction, LV consantric hypertrophy, mitral regurgitation (1-2 plus), trivial aortic regurgitation, aortic root: 47 mm,

ECG: SR, HR: 61 beat per min, V1-4 R amplitude missing, LAHB

Past medical history: DM (+), HT (+), DL (+), AÖ (+), MI (?)

Coronary Angiography: The right coronary artery originating from the proximal segment (after diagonal-1) of left anterior descending artery. There were no obstructive lesions.

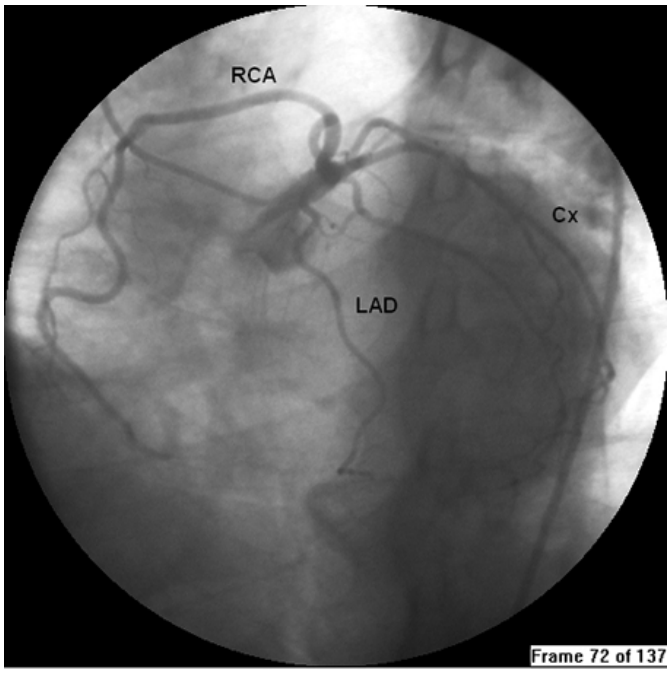
On physical examination, patient has a BP of 130/70 and a heart rate of 61 bpm regular. He has no jugular venous hypertension. The point of maximal impulse is not palpable. He has a grade 3/6 systolic ejection murmur in the mitral area. He had no diastolic murmur and his lungs were clear, good peripheral pulses, and no bruits.

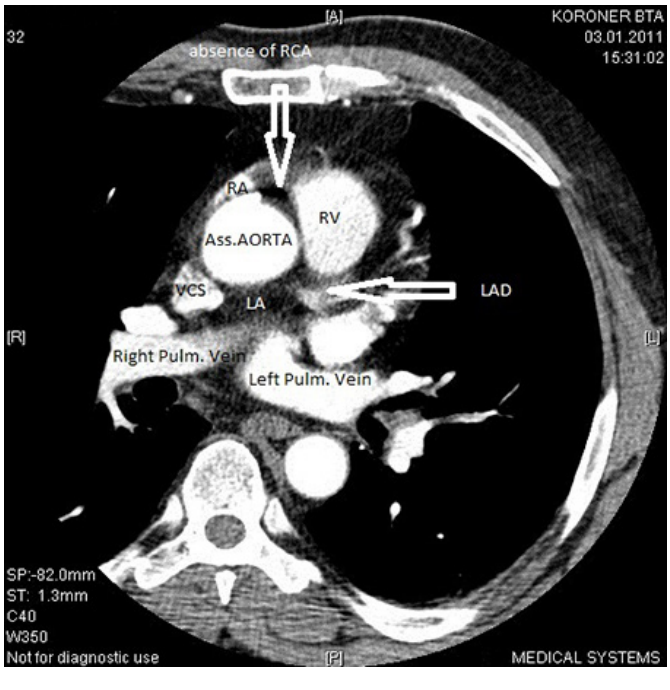
Discussion

In large retrospective series coronary anomalies were categorized as either anomalies of origin and distribution. The prevalence of coronary anomalies in Hobbs and Yamanaka series were reported as 1.55% and 1.3%.[5] There have been at least 37 published reports of an anomalies artery arising from the LAD, coursing anterior to right ventricular outflow tract to supply the usual RCA territory. The segment of origin from the LAD is either proximal or mid LAD. When you review the literature the frequency of this originating segment changes according to the used nomenclature.

In our case the anomalous RCA was originating from the mid LAD just after the 1.st diagonal branch.

There is no clear agreement of the clinical significance of an anomalous RCA from the LAD. While some authors suggest such an anomaly is usually benign and there appears to be no increased incidence of coronary atherosclerosis in such patients. On the other hand some authors suggest the opposite to be true. The

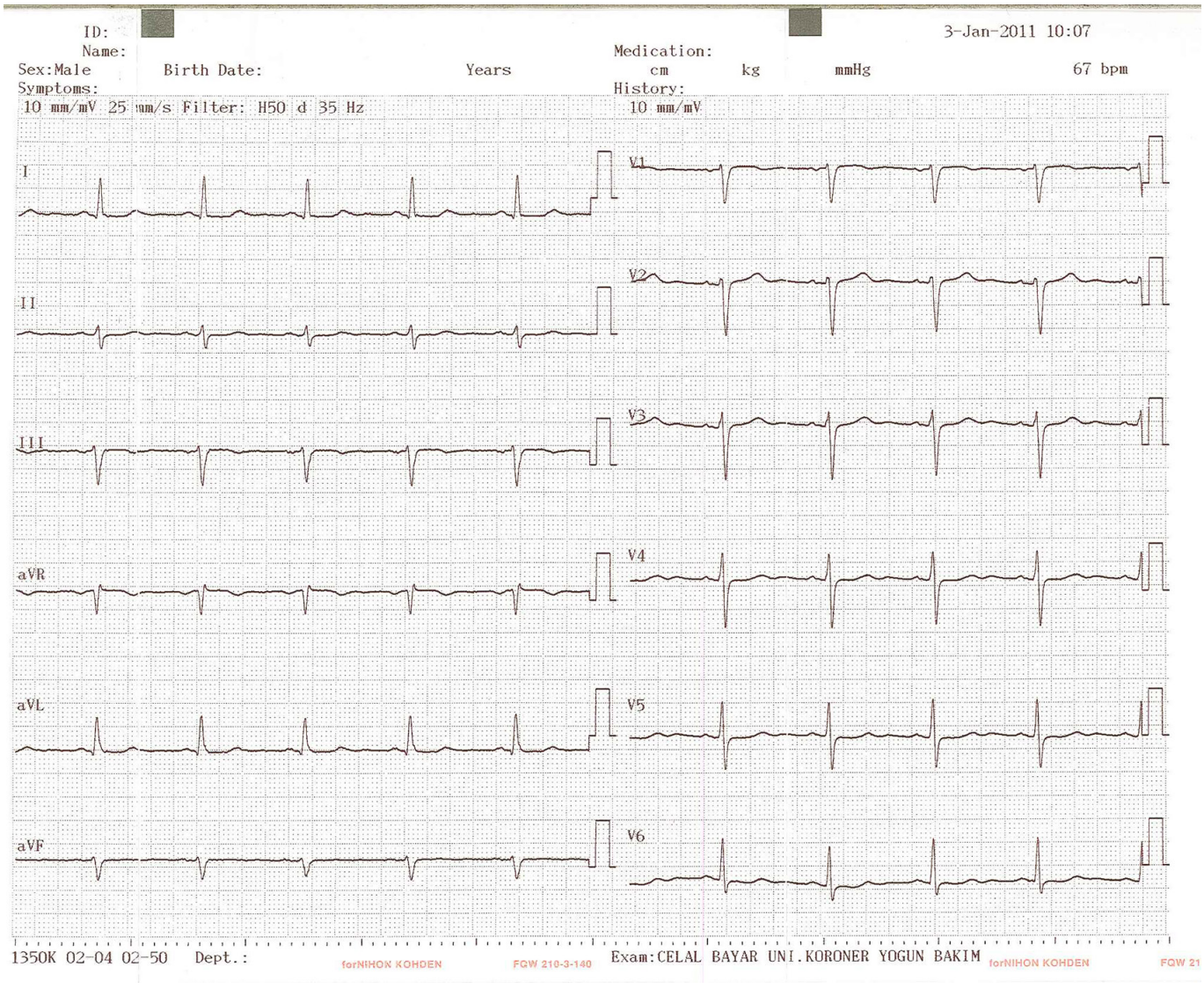




presence of an additional major artery near the first diagonal may increased to the tendecy of narrowing at this point. Another possibility is the presence of ischemia without overt coronary atherosclerosis due to reduced flow velocities in the anomalous vessel. In case of a sharp angle of take-off for the anomalous vessel, flow velocities may be reduced.

In our case there was no obstructive coronary artery lesion despite a an acute angle greater than 90 degrees.

In conclusion, majority of anomalous right coronary arteries have their origin from the mid LAD segment as defined by CASS (Coronary Artery Surgery Study) nomenclature.[6] There is some disagreement regarding the association of this anomaly and increased coronary atherosclerosis.



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References

1. Poulis M. Anomalous Origin of the Right Coronary Artery. *Clin. Cardiol.* 1999; 22: 815
2. Tüccar E, Elhan A. Examination of Coronary Artery Anomalies in an Adult Turkish Population. *Turk J Med Sci.* 2002; 32: 309-312.
3. Tuzcu EM, Moodie DS, Chambers IL, Kayser P, Hobbs RE. Congenital heart diseases associated with coronary artery anomalies. *Cleve Clin J Med* 1990; 57: 147-152.
4. Taylor AJ, Virmani R. Coronary Artery Anomalies In: Crawford. M. H. Di Marco J.P, ed. *Cardiology Mosby* 2001; 2: 10-11.
5. Yamanaka O, Hobbs RE. Coronary artery anomalies in 126.595 patients undergoing coronary angiography. *Cathet Cardiovasc Diagn* 1990; 21: 28-40
6. Click RL, Holmes DR Jr, Vliestra RE, Kosinski AS, Kronmal RA. Anomalous coronary arteries;location, degree of atherosclerosis and effect on survival a report from the Coronary Artery Surgery Study. *J Am Coll Cardiol* 1989; 1;13: 531-7.

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