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Retrospective Review on Anomalous Left Coronary Artery from Pulmonary Artery

LY ***Ko,*** AKT ***Chau,*** TC ***Yung,*** YF ***Cheung,*** KS ***Lun***

Abstract Anomalous left coronary artery from pulmonary artery (ALCAPA) is a rare but important congenital coronary anomaly. Patients with ALCAPA typically present with paroxysms of crying, pallor, diaphoresis, agitation and heart failure in infancy due to myocardial ischaemia. Clinical course thus very much depends on the presence and development of collateral circulation. We report the clinical presentation and method of diagnosis of ALCAPA in Chinese children in Hong Kong. We also study their long term outcomes after surgery and compare local data with overseas results. Early diagnosis and correction are important to avoid development of or progression of mitral regurgitation, which appeared to be associated with lower chance of requiring mitral valve annuloplasty/replacement. The majority of patients in Hong Kong presents as infantile form with heart failure, mitral regurgitation and dilated cardiomyopathy. Typical ECG changes are present in 5 cases (55.6%). Echocardiography confirms the diagnosis in 3 cases (33.3%). The remaining patients require cardiac catheterization to confirm the diagnosis. Early diagnosis therefore relies on a high index of suspicion. The diagnosis of ALCAPA should be considered as differential diagnosis of mitral regurgitation, endocardial fibroelastosis and dilated cardiomyopathy. Cardiac catheterization is warranted in doubtful cases. Long-term survival and outcome after surgical treatment appear to be favourable with good functional status. Residual lesions occur in 4 (44.4%) patients and re-operation on the mitral valve is required in 1 (11.1%) patient. Our local results are comparable to overseas reports.

Key words Anomalous left coronary artery from pulmonary artery; Diagnosis; Presentation; Surgical outcomes

Introduction

Anomalous left coronary artery from pulmonary artery (ALCAPA) is a rare congenital cardiac malformation in which the left coronary artery arises from pulmonary artery instead of the left coronary sinus. The typical clinical presentation of ALCAPA includes paroxysms of crying, pallor, diaphoresis, agitation and heart failure in infancy due to myocardial ischaemia. This rare congenital anomaly accounts for 0.5% of all congenital heart defects,[1] and yet, this is the most common congenital coronary artery defects presenting in childhood. This is also the most common congenital anomaly associated with myocardial ischaemia/infarction. The incidence was estimated to be 1 in 300,000 live births. To diagnose ALCAPA is not always easy, as sometimes only secondary diagnosis, such as mitral incompetence, dilated cardiomyopathy and endocardial fibroelastosis is made. Myocardial hypoperfusion, which leads to reduced fraction of contractile material with variable myocardial fibrosis, is dependent on the chronic collateral circulation. Clinical course thus very much depends on the adequacy of collateral circulation.

Department of Paediatric Cardiology, Queen Mary Hospital, 102 Pokfulam Road, Hong Kong, China

LY **Ko** (高利源)	*MRCPCH(UK), FHKAM(Paed), FHKCPaed*
AKT **Chau** (周啟東)	*FRCPCH, M Med(Singapore), FHKAM(Paed)*
TC **Yung** (翁德璋)	*MBBS, FHKAM(Paed), FHKCPaed*
YF **Cheung** (張耀輝)	*MD*
KS **Lun** (倫建成)	*FRCP(Edin), FHKAM(Paed), FHKCPaed*

Correspondence to: Dr AKT **Chau**

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In this review, we reported the clinical presentation and method of diagnosis of ALCAPA in Chinese children in Hong Kong. We also studied their long term outcomes after surgery and compare local data with overseas results.

Patients and Methods

All children with the diagnostic code of ALCAPA in Grantham Hospital from 1980 to 2007 were identified. All hospital records were reviewed retrospectively. Demographic and clinical data, the presence of heart failure, mitral incompetence, radiographic features, electrocardiogram (ECG) and echocardiographic findings were extracted. Data on the type of operation, follow-up duration, post-operative complications, recovery of mitral incompetence/left ventricular function and post-operative functional status according to the New York Heart Association functional class were obtained as well. Early mortality was defined as death within 30 days of operation.

For ease of comparison, we recruited, by a literature search on Medline, previous reports with 5 or more cases of ALCAPA for comparison.

Statistical Methods

Data were expressed as mean, median or percentages where appropriate.

Results

Clinical Presentation and Diagnosis

Nine children with ALCAPA were identified. The demographic and clinical data were shown in Table 1. The mean age of presentation was 9 months of age (range 1 month-4 years). Median age of presentation was 4 months of age. Male to female ratio was 6:3. Eight patients (88.9%) presented with heart failure symptoms and cardiomegaly on chest radiograph in early infancy. One (11.1%) patient presented at 4 years of age with incidental heart murmur, like the adult form of ALCAPA.

Five out of nine patients (55.6%) had typical ECG finding of anterolateral myocardial infarction. Eight patients (88.9%) presented with mitral regurgitation. One patient had evidence of left ventricular dysfunction but no mitral regurgitation. Clear echocardiographic evidence to confirm the diagnosis of ALCAPA was shown in three patients (Patients 2, 3, 8) (33.3%). Suspicious echocardiographic features of ALCAPA were noted in three patients (Patients 4, 5, 9) (33.3%). The remaining three patients (Patients 1, 6, 7) (33.3%) had no echocardiographic evidence of ALCAPA. Mitral regurgitation as primary diagnosis was made in five of these six patients. The diagnosis of endocardial fibroelastosis was made in one patient. Cardiac catheterisation was required to establish the diagnosis of ALCAPA in the six patients (66.7%) without echocardiographic confirmation. Three patients, Patient 4, 6 and 7, had delayed diagnosis of ALCAPA, 5 years, 8 years and 7 years respectively, after their initial presentation.

The Operation

The duration from presentation to corrective surgery ranged from 2 weeks to 8 years. Five out of nine patients had the operation within 6 months of presentation. Three patients had the operation more than 6 months from presentation and they had progressive mitral regurgitation and left ventricular dysfunction before operation. The asymptomatic patient (Patient 9) with adult form of ALCAPA had the operation 1 year after establishment of diagnosis.

All patients with ALCAPA underwent the Takeuchi operation, during which, an intrapulmonary artery tunnel of the left coronary artery (LCA) was constructed to direct oxygenated blood from aorta into the left coronary artery, aiming to bring about functional recovery. Patient 6, who had mitral regurgitation as the initial diagnosis, had a previous mitral valve annuloplasty. He underwent the Takeuchi operation and concomitant mitral valve replacement. Patient 7, who had mitral regurgitation as primary diagnosis, underwent the Takeuchi operation together with mitral valve annuloplasty to correct progressive severe mitral regurgitation. There was no early mortality.

Follow-up

The mean follow-up duration was 12 years 3 months (range 11 months-19 years 2 months). The outcomes of patients after operation were shown in Table 2. Mitral regurgitation and left ventricular function improved in five patients (55.6%) with Takeuchi operation. Reoperation was needed in one patient (11%). Mitral regurgitation did not improve in Patient 5 and he eventually required mitral valve replacement 4 months after Takeuchi operation but died of sepsis and intractable heart failure 7 months after the reoperation.

Mild supravalvular pulmonary stenosis was observed in three patients (33.3%). One patient had baffle leak with aneurysmal dilatation of the intrapulmonary tunnel, causing

Table 1 Demographic and clinical data

	Sex/ Age	Heart failure	Cardio-megaly from CXR	ECG features of myocardial infarction/ ischaemia	Echocardiographic findings	Method of diagnosis	Duration from presentation to surgery	Outcome after TO
1	F/1 m	+	+	+	MR	CC	4 m	Survived MR improved Mild acquired supravalvular PS
2	M/1 m	+	+	+	ALCAPA MR	Echo	2 m	Survived MR improved
3	M/3 m	+	+	+	ALCAPA LV dysfunction No MR	Echo	<1 m	Survived LV dysfunction improved Aneurysmal dilatation of intrapulmonary tunnel causing RVOT obstruction
4	M/3 m	+	+	–	EFE MR	CC	5 yr	Survived MR improved
5	M/4 m	+	+	+	LV aneurysm Echogenic papillary muscle suggestive of infarction or fibrosis MR	CC	5 m	Survived MR not improved Mild acquired supravalvular PS Reoperated with MVR 4 months after TO Died of intractable heart failure & sepsis 7 months after reoperation
6*	M/5 m	+	+	–	MR	CC	8 yr	Survived MR improved
7**	F/8 m	+	+	–	MR	CC	7 yr	Survived MR improved
8	M/1 yr	+	+	+	ALCAPA MR	Echo	1 m	Survived MR improved
9	F/4 yr	–	–	–	Dilated RCA, continuous flow in MPA MR	CC	1 yr	Survived MR improved Mild acquired supravalvular PS

Abbreviations:
+=present; –=absent; MR=mitral regurgitation, ALCAPA=anomalous left coronary artery from pulmonary artery; EFE=endocardial fibroelastosis, LV=left ventricular, RCA=right coronary artery; MPA=main pulmonary artery; Echo=echocardiography, CC=cardiac catheterization; TO=Takeuchi operation; PS=pulmonary stenosis; RVOT=right ventricular outflow tract; MV=mitral valve; MVR=mitral valve replacement
*MV annuloplasty before TO + MVR; **TO + MV annuloplasty done at the same time

Table 2 Outcomes of ALCAPA after surgery

	No. of patient	(%)
Improvement on mitral regurgitation and left ventricular function	5	(55.6)
Acquired supravalvular pulmonary stenosis	3	(33.3)
Baffle leak with aneurysmal dilatation of the intrapulmonary tunnel and atrial flutter	1	(11.1)

right ventricular outflow tract obstruction and atrial flutter. No reoperation was required for these patients thus far.

Up to the time of writing this manuscript, the survival rate was 88.9%. Of the eight surviving patients, seven patients were in NYHA (New York Heart Association) functional class I, while one patient has NYHA functional class II.

Comparison of Data with Other Reports

From the literature search, twelve reviews were obtained from different countries (Table 3). The number of patients in these reports ranged from 5 to 47. The median age of surgical repair of our patients was comparable to other countries. However, the mean age of surgical repair in our patients was higher than other studies. Various types of operation have been described in other reports, including Takeuchi operation, direct reimplantation of left coronary artery, subclavian interposition, coronary artery bypass grafting and aortic reconstruction. Many of the studies involved two or more types of surgical intervention. The type of operation was dependent on surgical expertise. None of our patients required mechanical circulatory support. On the contrary, some of the patients in Canada and UK (Report 4, 5) required this support in early postoperative period due to low cardiac output syndrome.[2,3] The overall mortality of ALCAPA after the surgical repair in Hong Kong was 11.1%, which was comparable to other reports which ranged from 0% to 20%. All our patients underwent Takeuchi operation. The rate of acquired pulmonary stenosis and baffle leak in our study were 33.3% and 11.1% respectively. In the reports from USA and Canada (Reports 3 and 4 respectively) where only direct reimplantation of the coronary artery was performed, the mortality rate ranges from 0% to 9% but the complication rates were not mentioned. As different surgical techniques were employed in different studies, it is difficult to make direct comparisons. Our patients had a higher rate of mitral valve repair/replacement compared with reports 2, 4 and 6-9 in Table 3.

Discussion

Normal coronary anatomy is a dual supply of blood to myocardium. The left main coronary artery arises from left sinus of Valsalva and divides into left anterior descending and left circumflex arteries. The right coronary artery arises from right sinus of Valsalva and gives rise to the posterior descending artery.

Infantile Type of ALCAPA

The majority of patients presents in early infancy. They develop myocardial necrosis shortly after birth, as hypoperfusion results in myocardial infarction. The perfusion of left ventricle is entirely collateral-dependent, resulting in poor global left ventricular function. It is not uncommon for these infants to present with unexplained congestive heart failure. If uncorrected, nearly 85% of these patient die within 1 year.[4] Survival of the untreated patient beyond infancy is uncommon due to severe left heart failure. Survivors are left with localised infarction.

Adult Type of ALCAPA

The minority of patients presents in adulthood. They have extensive collateral circulation, which allows them to reach adulthood, and may even lead a normal life. Myocardial ischaemia does not develop until adolescence/adulthood. Patients of this type may present with unexplained cardiomegaly, mitral incompetence (ischaemic damage to papillary muscle and chordae) or angina pectoris secondary to coronary steal phenomenon, incidental mitral incompetence murmur and ventricular aneurysm, which may be associated with ventricular arrhythmias. Many of them will have surgical correction as soon as the diagnosis is established. Other presentations include acute myocardial infarction, cardiomyopathy, left ventricle dysfunction and sudden death.

The infantile and adult forms are on the opposite ends of the spectrum. Presentation depends on the degree of collateral circulation between the right and left coronary artery systems. These collateral circulations may be formed in fetal life. It is difficult to know the timing and the extent of the collateral development. Those with good collateral circulation are likely to present later. During immediate newborn period, pulmonary artery resistance and pressure are increased, flow through the anomalous left coronary artery is antegrade from pulmonary artery and myocardial perfusion is adequate. Symptoms tend to occur when pulmonary vascular resistance and pulmonary pressure drop. The antegrade perfusion of the anomalous left coronary artery diminishes. If collateral is inadequate, myocardial infarction will likely occur. If collateral exists, myocardial infarction may or may not occur, depending on the degree of retrograde flow from right coronary system through the collateral into pulmonary artery (coronary steal phenomenon). The anomalous left coronary artery functions as a venous channel draining blood from myocardium, instead of supplying it. Supply-demand mismatch within myocardium will result in myocardial dysfunction.

Table 3 Comparison of our results with overseas results

	Reports	Patient number	Age range	Mean age of repair (months)	Median age of repair (months)	Type of operation	Mechanical circulatory support (%)	Overall mortality (%)	Acquired PS (%)	Baffle leak (%)	MVR/ Annulo (%)
1	Hong Kong	9	3 m-8 yr	39.2	12	TO	0	11.1	33.3	11.1	33.3
2	Michielon Italy 2003[6]	31 (ALCAPA 28)	–	–	7.1	TO DR MIS	–	10.1	9.7	8.3	3.2
3	Backer USA 2000[8]	16	–	–	5.4	DR	0	0	–	–	–
4	Azakie Canada 2003[2]	47	–	–	7.7	DR	ECMO 10.6	9	–	–	2.1
5	Pandey UK 2002[3]	5 (Infant)	–	3	–	TO DR	IABP 100	0	20	–	–
6	Malec Poland 2001[9]	8	2 m-10 yr	–	42	TO DR	0	0	–	–	0
7	Huddleston USA 2001[10]	17	–	0.5	–	TO DR	–	5.9	–	–	11.8
8	Lange Germany 2007[11]	56	–	16.2	–	DR MIS	–	14.3	–	–	5.4
9	Birk Israel 2000[7]	13	–	–	–	TO DR	–	15.4	–	–	7.7
10	Wu China 2000[12]	8	–	–	–	TO MIS	–	0	–	12.5	–
11	He China 2007[13]	10	–	–	–	TO DR MIS	–	20	–	–	–
12	Ando USA 2002[14]	13	–	–	46.8	TO DR	–	0	–	–	–
13	Isomatsu Japan 2001[15]	29	2 m-24 yr	–	29.3	TO DR	–	6.9	–	–	–

Abbreviations:

ALCAPA=anomalous left coronary artery from pulmonary artery; ECMO=extracorporeal membrane oxygenation; IABP=intra-aortic balloon pump; TO=Takeuchi operation; DR=direct reimplantation of left coronary artery to aorta; MIS=miscellaneous operation

Complications arising from myocardial infarction are mitral incompetence due to papillary muscle dysfunction or rupture, acquired atrial septal defect, ventricular aneurysm, dysrhythmias and even cardiogenic shock.

Eighty-nine percent of our patients belonged to the infantile type and presented with symptoms and signs of heart failure in early infancy. Fifty-six percent of our patients demonstrated ECG changes of myocardial infarction at presentation. The remaining patients had evidence of left ventricular straining pattern. ECG changes of ALCAPA usually include anterolateral myocardial infarction, pathological Q waves and T inversion in leads I, aVL extending to midprecordial & lateral precordial leads (V3 to V6). Therefore, presence of these ECG features are important clues to diagnose ALCAPA.

In our review, only 33.3% of the patients had clear echocardiographic evidence to allow us to confirm the diagnosis of ALCAPA, while another 33.3% had suspicious echocardiographic features of ALCAPA. In the remaining 33.3%, echocardiogram did not point to ALCAPA. These

patients were diagnosed as mitral valve disease causing mitral regurgitation or endocardial fibroelastosis instead. Therefore, ALCAPA should be considered as a differential diagnosis in infants with dilated cardiomyopathy, congestive heart failure, endocardial fibroelastosis and unexplained mitral regurgitation. It is also worth noting that sudden infant death may be an uncommon presentation.

The diagnosis of ALCAPA may still pose a challenge to paediatricians nowadays. Diagnosis of ALCAPA by echocardiogram is not fully satisfactory.[5] Echocardiography might be able to pick up the anomalous origin with high specificity, especially when doppler colour flow of LCA from pulmonary artery is elicited. But its sensitivity might not be adequate even in neonates, who have better echocardiographic window. The diagnosis of ALCAPA should be suspected in the case of failure to demonstrate the origin of LCA from left sinus of Valsalva in infants with unexplained congestive heart failure. Furthermore, increased right coronary artery diameter to aortic annulus ratio (>0.14) and increased papillary muscle echogenicity will also point to the likelihood of ALCAPA.

Coronary angiography remains the gold standard in diagnosing ALCAPA. Any clinical evidence of coronary insufficiency should prompt coronary angiography, which typically shows only a large right coronary artery from right aortic sinus of Valsalva, supplying an extensive collateral circulation to left coronary circulation. Injection of contrast in pulmonary artery may reveal the anomalous left coronary artery. However, most frequently it will not be apparent because of lack of antegrade flow. Left coronary artery may fill rapidly from right coronary artery and appear to originate from the left coronary cusp. Different standard views will be helpful in delineating the exact coronary anatomy.

ALCAPA patients require aggressive anti-failure treatment and surgical correction. Revascularization, which establishes a dual-coronary system, brings about functional recovery with good clinical outcome. The type of operation depends on the coronary anatomy. Dual-coronary system can be achieved by direct implantation of anomalous coronary artery to ascending aorta in proximity. However, left coronary artery, arising from posterior wall of pulmonary trunk, may be far from the ascending aorta. Takeuchi operation is an alternative by intrapulmonary tunneling of the left coronary artery. Gradual normalisation of right coronary size and angiographic disappearance of the collateral vessels are expected.

All of our patients underwent the latter operation. The rates of acquired supravalvular pulmonary stenosis and baffle leak were 33.3% and 11.1% respectively in our review. These are common post-operative complications from intrapulmonary tunneling.

Mitral incompetence and left ventricular function improved in 55.6% of our patients by Takeuchi operation. It was worth noting that five of the infantile ALCAPA patients had the operation less than 6 months from presentation as the diagnosis was made early. Early diagnosis and correction tended to be associated with lower chance of mitral valve annuloplasty/replacement. In fact, repair in younger age offers the best potential for recovery of left ventricular function.[6] Early repair is associated with faster myocardial recovery.[7] Complete recovery from myocardial dysfunction is expected. Those with severe left ventricular dysfunction have higher risk for repair and may require mechanical circulatory support such as left ventricular assist device. The degree of preoperative mitral incompetence may be predictive of outcome, as severe mitral incompetence is associated with increased mortality. Mild to moderate mitral incompetence tends to improve without the need of mitral valvuloplasty. Mitral valve repair is generally not necessary in the initial operation. Mitral incompetence usually improves after establishment of the dual-coronary system.

Mitral valve replacement may be required in some patients either at the time of Takeuchi operation or later in follow-up. Recurrence or persistence of mitral incompetence may suggest coexistent postoperative coronary stenosis. Cardiac catheterization is then indicated to assess the patency of the LCA before mitral valve surgery.

We had only one mortality after the Takeuchi operation. All survivors had satisfactory functional class. The long term outcome appeared to be satisfactory.

The outcomes of our patients are comparable to other reports. Of note here is that some reports showed low mortality and hence excellent results after the surgical repair. However, as different surgical techniques had been applied, it is difficult to make a direct comparison regarding the surgical outcome on Takeuchi operation alone. While Takeuchi operation is one of the early surgical techniques, direct reimplantation of left coronary artery to aortic root or coronary bypass grafting is increasingly performed with comparable results. The latter operations may be the preferred operation in patients with suitable anatomy. Regardless of the surgical techniques, the higher rate of mitral valve annuloplasty or replacement in our patients who had a delay in diagnosis emphasizes the importance of early diagnosis and treatment to avoid prolonged myocardial injury caused by ALCAPA.

Conclusion

ALCAPA is a rare but important congenital coronary anomaly. Early diagnosis and early correction are important to avoid development of or progression of mitral regurgitation, which appeared to be associated with lower chance of requiring mitral valve annuloplasty/replacement. The majority of the patients in Hong Kong presents as heart failure. The diagnosis of ALCAPA should also be considered in unexplained mitral regurgitation, endocardial fibroelastosis and dilated cardiomyopathy. Typical ECG changes of myocardial ischaemia/infarction should be actively looked for. Echocardiography may reveal the coronary anomaly and help confirm the diagnosis in some patients, but it has limitations. Cardiac catheterization is necessary to confirm the diagnosis in suspicious cases. Early diagnosis therefore relies on a high index of suspicion. Long-term survival and outcome after surgical treatment appear to be favourable with good functional status. However residual lesions may persist and re-operation may be required in a minority of patients, particularly on the mitral valve. Our local results are comparable to overseas reports.

References

1. Garson A. The science and practice of pediatric cardiology (2nd ed). Baltimore: Williams & Wilkins, 1998.
2. Azakie A, Russell JL, McCrindle BW, et al. Anatomic repair of anomalous left coronary artery from the pulmonary artery by aortic reimplantation: early survival, patterns of ventricular recovery and late outcome. Ann Thorac Surg 2003;75:1535-41.
3. Pandey R, Ciotti G, Pozzi M. Anomalous origin of the left coronary artery from the pulmonary artery: results of surgical correction in five infants. Ann Thorac Surg 2002;74: 1625-30.
4. Nicholson WJ, Schuler B, Lerakis S, Helmy T. Anomalous origin of the coronary arteries from the pulmonary trunk in two separate patients with a review of the clinical implications and current treatment recommendations. Am J Med Sci 2004;328:112-5.
5. Lee LF, Chang JS, Hung RS, Wu YL. Adult type anomalous origin of the left coronary artery from the main pulmonary artery: report of one case. Acta Paediatr Taiwan 1999;40:112-5.
6. Michielon G, Di Carlo D, Brancaccio G, et al. Anomalous coronary artery origin from the pulmonary artery: correlation between surgical timing and left ventricular function recovery. Ann Thorac Surg 2003;76:581-8; discussion 588.
7. Birk E, Stamler A, Katz J, et al: Anomalous origin of the left coronary artery from the pulmonary artery: diagnosis and postoperative follow up. Isr Med Assoc J 2000;2:111-4.
8. Backer CL, Hillman N, Dodge-Khatami A, Mavroudis C. Anomalous origin of the left coronary artery from the pulmonary artery: Successful surgical strategy without assist devices. Semin Thorac Cardiovasc Surg Pediatr Card Surg Annu 2000;3:165-72.
9. Malec E, Zajac A, Mikuta M. Surgical repair of anomalous origin of the coronary artery from the pulmonary artery in children. Cardiovasc Surg 2001;9:292-8.
10. Huddleston CB, Balzer DT, Mendeloff EN. Repair of anomalous left main coronary artery arising from the pulmonary artery in infants: long-term impact on the mitral valve. Ann Thorac Surg 2001;71:1985-8; discussion 1988-9.
11. Lange R, Vogt M, Hörer J, et al. Long-term results of repair of anomalous origin of the left coronary artery from the pulmonary artery. Ann Thorac Surg 2007;83:1463-71.
12. Wu QLD. Surgical treatment of anomalous origin of the left coronary artery from the pulmonary artery. Zhonghua Wai Ke Za Zhi Spe 2000;38:659-61.
13. He XH, Li Y, Huang MR, et al. [Anomalous origin of the left coronary artery from the pulmonary artery: report on 10 cases] Zhongguo Dang Dai Er Ke Za Zhi 2007;9:25-7.
14. Ando M, Mee RB, Duncan BW, Drummond-Webb JJ, Seshadri SG, Igor Mesia CI. Creation of a dual-coronary system for anomalous origin of the left coronary artery from the pulmonary artery utilizing the trapdoor flap method. Eur J Cardiothorac Surg 2002;22:576-81.
15. Isomatsu Y, Imai Y, Shin'oka T, Aoki M, Iwata Y. Surgical intervention for anomalous origin of the left coronary artery from the pulmonary artery: the Tokyo experience. J Thorac Cardiovasc Surg 2001;121:792-7.