

Repair of Anomalous Left Coronary Artery from Pulmonary Artery (ALCAPA) beyond Infancy

Sachin Talwar, Aandrei Jivendra Jha, Shiv Kumar Choudhary, Saurabh Kumar Gupta, Balram Airan

Cardiothoracic Center, All India Institute of Medical Sciences, New Delhi, India

ABSTRACT

Between January 2002 and December 2012, five patients (4 female) underwent corrective surgery for anomalous left coronary artery from pulmonary artery (ALCAPA). They were older than 1 year (range, 3-56 years). One of the 2 patients younger than 10 years had presented with congestive heart failure, and the other had experienced repeated episodes of lower respiratory tract infection since childhood. Of the remaining 3 adult patients, 2 had experienced angina with effort, and 1 patient had had repeated respiratory tract infections since childhood, with mild dyspnea on effort of New York Heart Association (NYHA) class II. Three patients had the anomalous left coronary artery implanted directly into the ascending aorta via coronary-button transfer, and 2 patients underwent coronary artery bypass with obliteration of the left main ostium. Two patients underwent concomitant mitral valve repair procedures, and 1 patient underwent direct closure of a perimembranous ventricular septal defect. Four patients survived the surgery, and 1 patient died because of a persistently low cardiac output. Follow-up times ranged from 3 months to 4 years. All survivors are in NYHA class I and have left ventricular ejection fractions of 45% to 60%, with moderate (n = 1), mild (n = 1), or no (n = 2) mitral insufficiency. We conclude that a few naturally selected patients with ALCAPA do survive beyond infancy and can undergo establishment of 2 coronary systems with satisfactory results.

INTRODUCTION

Anomalous left coronary artery from pulmonary artery (ALCAPA) is a rare congenital anomaly affecting 1 in 300,000 individuals [Raghuram 2004]. It was first described by Brooks in 1886 and first reported in the literature by Bland et al [1933]. It usually occurs as a solitary lesion and is the most

common cause of myocardial ischemia and infarction in children. Without treatment, 90% of infants die before their first birthday [Wesselhoeft 1968]. Abbot [1908] was the first to describe an adult with ALCAPA, in Osler's Modern Medicine. This patient was an asymptomatic 60-year-old woman who had died an accidental death. The anomaly became known after autopsy.

The oldest person described in the literature with a diagnosis of this anomaly antemortem was a 69-year-old Korean woman treated surgically with a trapdoor implantation [Lim 2011]. The oldest patient with this anomaly diagnosed was 72 years old [Fierens 2000], and she was managed conservatively because of an increased risk-to-benefit ratio. This subgroup of patients with abundant collaterals is often referred to as the adult type, which constitutes approximately 10% to 15% of such patients, can survive into adulthood and beyond. Even though these patients may have less-severe symptoms (precipitated classically by exertion when the demand/supply mismatch overwhelms the collateral perfusion by the right coronary territory), they continue to be at very high risk for sudden death due to malignant ventricular arrhythmias, the substrate for which is the scarred ventricular myocardium (due to previous myocardial infarctions) and the normal ventricular myocardium subjected to stress during times of increased demand. Sudden death occurs in 90% of these patients by 35 years of age. The reason favoring survival of these patients beyond infancy is not known precisely. The most important factor allowing survival beyond infancy appears to be the volume of the collateral supply from the right coronary artery. Other factors that may be important are a right dominant coronary circulation [Wesselhoeft 1968] (a smaller area of the myocardium is at jeopardy), ostial stenosis of the ALCAPA [Wesselhoeft 1968] (prevents left coronary runoff into the low-pressure pulmonary circulation), development of collaterals from the systemic circulation supplying the ischemic myocardium, and development of pulmonary hypertension [Akbari 2007].

The other morphologic types of patients with ALCAPA do not have an extensive collateral network and are referred to as the infant type. Most of these infants (90%) die without treatment in the first year of life. Yau et al [2011] analyzed 151 adult ALCAPA cases (>18 years of age) reported in the literature between 1998 and 2008 and obtained a mean (SD) age at

Received January 13, 2013; received in revised form July 15 and July 29, 2013; accepted July 30, 2013.

Correspondence: Sachin Talwar, Additional Professor, Department of Cardiothoracic and Vascular Surgery, All India Institute of Medical Sciences, New Delhi 110029, India; 91-11-26594835; fax: 91-11-26588663 (e-mail: sachintalwar@hotmail.com).

Summary of Patients with Anomalous Left Coronary Artery from Pulmonary Artery (ALCAPA) Presenting after Infancy*

Patient No	Age, y	Sex	Presentation	Operation	Outcome	Preoperative LVEF, %	Postoperative LVEF, %
1	3	F	Recurrent LRTI	Coronary button transfer to aorta	Discharged on POD 8	40	60
2	22	F	Recurrent LRTI + DOE II since childhood	Coronary button transfer to aorta + direct closure of perimembranous VSD	Discharged on POD 11	35	50
3	5	M	CHF, syncope	Coronary button transfer to aorta + mitral valve repair	Died	25	
4	47	F	Chest pain, DOE IV	CABG ^{††} + mitral valve repair	Discharged on POD 11	25	45
5	56	F	DOE III, 3 y; AOE III, 3 mo; MI, 3 y	CABG [§]	Discharged on POD 9	45	60

*LVEF indicates left ventricular ejection fraction; LRTI, lower respiratory tract infection; POD, postoperative day; DOE, dyspnea on exertion; VSD, ventricular septal defect; CHF, congestive heart failure; CABG, coronary artery bypass grafting; AOE, angina on exertion; MI, myocardial infarction.

[†]The orifice of the ALCAPA was closed through the pulmonary artery with a pericardial patch to prevent runoff into the low-pressure pulmonary circulation.

^{††}Left anterior descending artery to the left internal mammary artery and reversed saphenous vein graft to the first obtuse marginal.

[§]Reversed saphenous vein grafts to left anterior descending and circumflex arteries.

presentation of 40.6 ± 15 years, with a skewed sex distribution toward female patients (female-to-male ratio, 2.23:1). They found that 14% of these patients were asymptomatic, 18% presented with life-threatening symptoms (ventricular arrhythmia, syncope, sudden death), and 68% had subacute symptoms (angina, dyspnea, palpitations, and fatigue). Of the 119 patients who were treated surgically, 21% had a simple ligation of the left main coronary stem; the remaining 78% had establishment of some form of dual coronary circulation. At present, no randomized controlled trials have compared outcomes of different surgical techniques that establish dual coronary circulation in these patients. Even the reports of adult ALCAPA patients in the literature are few and far between. In this retrospective series, we present an institutional review of patients older than 1 year who underwent corrective surgery for ALCAPA between January 2002 and December 2012 at the All India Institute of Medical Sciences, New Delhi, India.

PATIENTS AND METHODS

Between January 2002 and December 2012, five patients older than 1 year (4 female patients and 1 male patient underwent operation for ALCAPA at the All India Institute of Medical Sciences, New Delhi, India. The study protocol was duly approved by the institute ethics committee. The Table summarizes the presentation, investigations, surgery, and immediate postoperative outcomes of these patients. Additional features are summarized below.

The median age was 22 years. The initial diagnosis for patient 3 was congenital mitral insufficiency at 1 year, and she was being treated conservatively. At 5 years of age, however,

she presented with gross cardiac failure and received a diagnosis of ALCAPA. Four patients had cardiac enlargement with a left ventricular apex on a preoperative chest radiograph (defined as a cardiothoracic ratio [CTR] >0.50). Massive cardiomegaly (CTR = 0.75) was seen in patient 3. Pulmonary plethora was determined in this patient and in patient 2. Patient 5 had a normal chest radiograph. Electrocardiography evaluations revealed a nonspecific T-wave inversion in the right precordial leads in patient 1, and patient 3 had frequent ventricular extrasystoles and signs of left heart dilatation and volume overload. Patient 2 had significant Q waves in leads aVL and I that suggested an old left ventricular infarction. Patient 4 had evidence of atrial fibrillation, and patient 5 had an unremarkable electrocardiogram.

Echocardiography evaluations were performed in all 5 patients. The results were diagnostic of ALCAPA in patient 1 and were suspicious of ALCAPA in patients 2 (assumed to be a coronary arteriovenous fistula) and 3. The last 2 patients in the series were adults who had undergone coronary angiography elsewhere and were not suspected of having ALCAPA. They were referred to our center for coronary artery bypass grafting (CABG). Patients 1, 3, and 4 had severe mitral regurgitation with evidence of left ventricular chamber enlargement. The other 2 patients had either mild mitral regurgitation (patient 2, who also had a perimembranous ventricular septal defect causing left-to-right shunt; Qp/Qs = 2:1) or no regurgitation (patient 5). The preoperative ejection fraction ranged from 25% to 45% (mean, 34%). Dilated coronary arteries and heart chambers noted in echocardiograms and chest radiographs aroused suspicion for coronary arteriovenous fistula (patient 2) and dilated cardiomyopathy (patient 4).

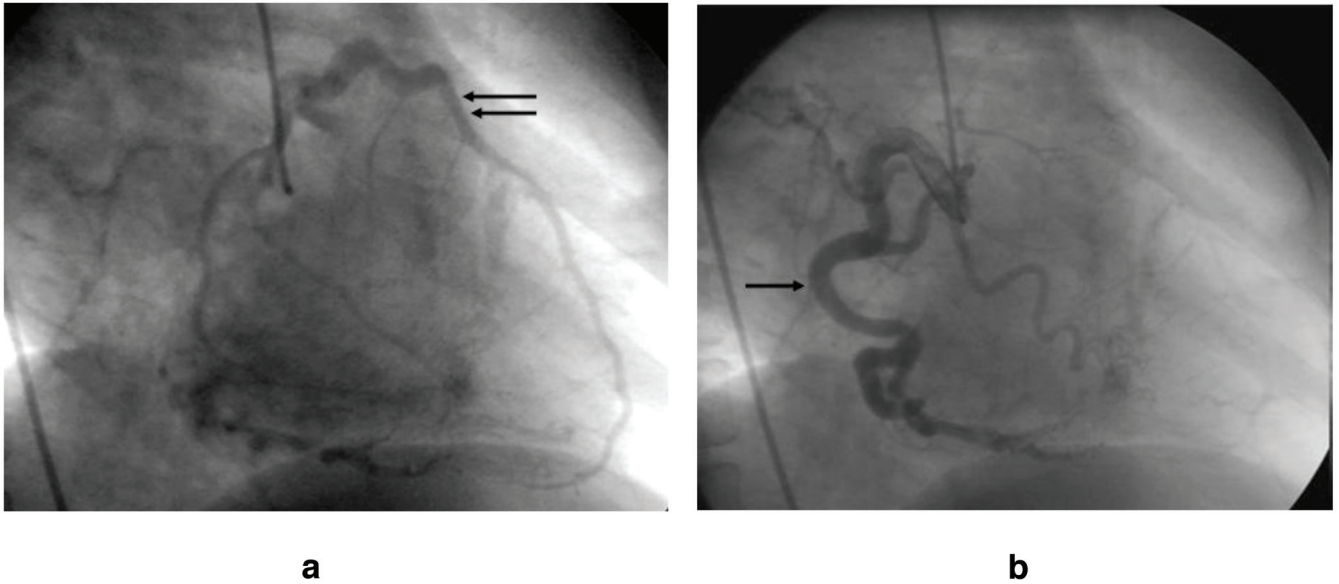


Figure 1. A, The left coronary system fills via collaterals in the later part of the same injection (double arrow). B, Right coronary injection shows a dilated and tortuous right coronary artery (arrow).

Cardiac catheterization and coronary angiography were performed in all patients and either confirmed ALCAPA (patients 1, 2, and 3) or led to its diagnosis. These examinations also confirmed moderate to severe mitral valve disease (patients 1, 3, and 4). Findings that confirmed the diagnosis of ALCAPA were multiple collaterals from a hugely dilated right coronary artery filling the anomalous left coronary artery retrogradely, with the dye reaching the pulmonary artery in

later phases (Figure 1). A significant step up in oxygen saturation (>10%) from the right atrium to the pulmonary artery was noted in patients 2 (by 12%) and 4 (by 18%). Computed tomographic angiography was required to accurately delineate the coronary anatomy in 1 patient (no. 5), who had been referred to this center for ostial left main disease, because the left main ostium was not visible in the coronary angiography images (Figure 2).

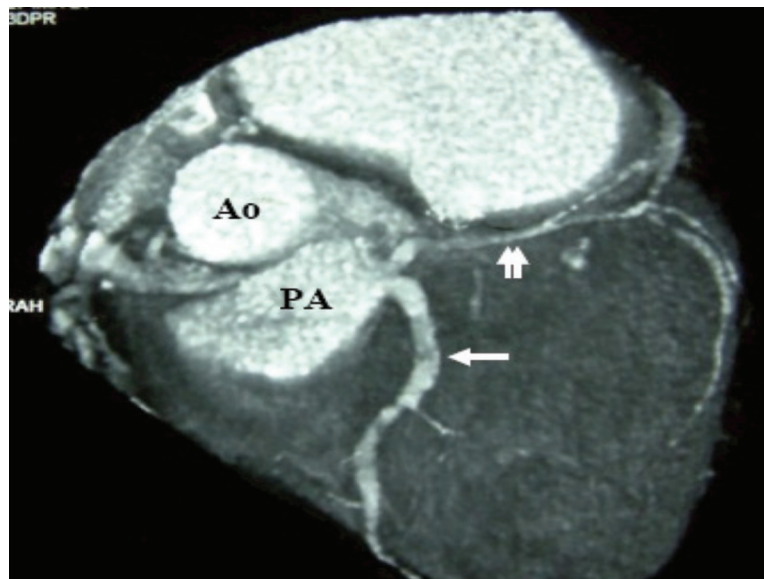


Figure 2. Computed tomography angiography image showing the anomalous coronary arising from the pulmonary artery (PA) and bifurcating into the left anterior descending artery (single arrow) and the left circumflex artery (double arrow). Ao indicates aorta.

Three patients underwent direct transfer of ALCAPA to the ascending aorta via the coronary button–transfer technique. Two adult patients presenting with features of coronary ischemia underwent CABG using the left internal mammary artery (LIMA) and reversed saphenous vein grafts. The LIMA was not used for patient 5 because the artery had been damaged during harvesting. One patient also required direct closure of a perimembranous ventricular septal defect. Two patients (nos. 3 and 4) with severe mitral insufficiency underwent concomitant mitral valve repair procedures. We did not perform mitral valve repair in patient 1, because this patient had undergone her operation early in our experience, when we believed that the mitral insufficiency would improve with adequate revascularization. We therefore preferred not to address the mitral valve to avoid prolonging the ischemia time. All patients thus received a dual coronary system. The ostium of ALCAPA was closed off directly (patient 5) or with a pericardial patch (patient 4) applied through the pulmonary artery in patients who underwent CABG. In those who underwent direct ALCAPA implantation into the ascending aorta via coronary-button transfer, the resulting defect in the pulmonary artery was closed with a patch of autologous pericardium that had been fixed in glutaraldehyde. None of the patients had the ALCAPA originating from the nonfacing pulmonary sinus, thereby making coronary-button transfer feasible. Adequate mobilization of the ALCAPA button permitted a tension-free anastomosis to the ascending aorta, which avoided the need to use coronary-elongation techniques or the Takeuchi procedure [Takeuchi 1979].

RESULTS

All patients were continued on mechanical ventilatory support after surgery until hemodynamic stability was achieved (mean, 56 hours; range, 25–170 hours). Inotropic support was gradually weaned over several days (mean, 63.2 hours; range, 32–170 hours). The length of stay in the intensive care unit averaged 68.8 hours (range, 38–170 hours). The mean hospital stay was 9.7 days.

There was one in-hospital death (patient 3). As we have detailed above, this patient was in a poor general condition before her operation. She had experienced cardiac arrest during induction of anesthesia, and cardiopulmonary resuscitation was continued for 40 minutes, after which she reverted spontaneously to cardiac rhythm; however, extracorporeal membrane oxygenation (ECMO) had to be instituted preoperatively because of her poor hemodynamics. The patient then underwent ALCAPA repair (implantation into the ascending aorta) and mitral valve repair. In the postoperative period, several attempts made to wean the patient from ECMO were aborted because of ventricular fibrillation. During one such attempt, on postoperative day 7, the patient developed refractory ventricular fibrillation, and a normal cardiac rhythm could not be restored.

Immediate and short-term follow-up results (3 months to 4 years) revealed that the 4 survivors experienced an improvement in the left ventricular ejection fraction of 15% to 20% (mean, 17.5%) above the baseline. Patient 1, who had

experienced severe mitral regurgitation preoperatively but had undergone no mitral valve repair, demonstrated a 20% increase in her ejection fraction postoperatively, with moderate mitral regurgitation at 3 months and a New York Heart Association (NYHA) class I status. Patient 2 was in NYHA class I at 57 months following surgery and had a left ventricular ejection fraction of 55% by the 4-year follow-up. Adult patients who underwent CABG grafting (patients 4 and 5) were in NYHA class I with an ejection fraction of 45% and 55%, respectively, at 3 months following discharge from the hospital. Patient 4 had moderate to severe mitral regurgitation preoperatively and only mild residual mitral regurgitation at 3 months.

DISCUSSION

As is discussed above, the presence and degree of the collateral circulation from coronary and noncoronary collaterals segregate ALCAPA patients into 2 distinct subgroups with different survival patterns. The “infant” variants have few collaterals, and without surgery most patients succumb to the onslaught of progressive myocardial ischemia, infarction, mitral regurgitation, and congestive cardiac failure before their first birthday. The “adult” variant of ALCAPA has abundant coronary and noncoronary collaterals, and patients with this condition survive into adulthood and beyond with a spectrum of symptoms ranging from asymptomatic and mild exercise intolerance to coronary ischemia and infarction. Sudden cardiac death due to malignant ventricular arrhythmias originating from ischemic or infarcted myocardium is the most common cause of death, which occurs by 35 years of age in 80% to 90% of these patients.

Shivalkar et al [1994] found peculiar subcellular adaptive changes in the left ventricular myocardium of ALCAPA patients. These changes consisted of variable fibrosis, loss of contractile elements, reduced organelle sizes, and significant glycogen deposition, as found in a “hibernating” myocardium. This adaptation enables complete recovery of function after several days to several weeks of revascularization as the cells improve their use of substrates and rebuild the normal contractile apparatus.

Contrary to the belief that human cardiac myocytes are not capable of regeneration after birth, Bergmann et al [2009] have demonstrated that slow regeneration does occur, decreasing from 1% annually at 25 years of age to 0.45% by 75 years. This finding perhaps explains why subendocardial scars that are present in asymptomatic adults with ALCAPA disappear after successful revascularization. Even severe ventricular dysfunction and mitral regurgitation always improve, albeit the latter occurs at a slower rate.

Asymptomatic or mildly symptomatic adults with ALCAPA have gone on to endure multiple pregnancies, and there are documented cases of patients remaining athletic until late adulthood and even remaining asymptomatic into old age. Therefore, the risk of sudden death in this population may be significantly overestimated, especially in those who have well developed systemic collaterals to supply the left coronary territory and do not show signs of myocardial ischemia.

As shown by patient 3, survival prospects seem particularly poor for younger patients with a decompensated cardiac reserve who present with congestive cardiac failure. This patient was the only death in our series and probably represents a case of compounded myocardial dysfunction secondary to ischemic damage that occurred during the prolonged cardiopulmonary resuscitation administered on the operating table before surgery. Despite the preoperative institution of ECMO and prolonged inotropic and ventilatory support, the patient did not survive.

Mechanical circulatory assistance (left ventricular assist device, ECMO, and intra-aortic balloon pump) has been used with varying success in ALCAPA patients who have severe ventricular dysfunction and difficulty weaning from cardiopulmonary bypass. ECMO is probably the most appropriate in this setting because it supports both ventricles. The right ventricle is also at risk, because the right coronary supply is shunted out into the pulmonary artery. Our patient (no. 3) had persistent refractory ventricular arrhythmias, and therefore ECMO was preferred to a left ventricular assist device. This patient likely had irreversibly damaged nonrecruitable myocardium following the prolonged resuscitation efforts, which compounded the preexisting myocardial injury and made him unweanable from ECMO.

The first successful attempt at ALCAPA repair was reported by Sabiston et al [1960], who ligated the ALCAPA at its pulmonary origin. Equally important was the demonstration of actual retrograde flow from the ALCAPA to the pulmonary artery and increase in ALCAPA pressure upon occlusion of the ostium. The proponents of this approach argue that it can be performed without cardiopulmonary bypass and can even be accomplished percutaneously [Collins 2007]. The main drawback of simple ligation is the creation of a "single coronary system." Eight years later, Wesselhoeft et al [1968] concluded that heart size did not return to normal following ALCAPA ligation, the electrocardiographic changes persisted, and the risk of sudden death due to silent ischemia remained. Recanalization, atherosclerosis, and ischemic dilated cardiomyopathy were other problems. Acceleration in the occurrence of atherosclerotic coronary artery disease as these patients grew into adults would lead to more severe consequences, ie, left main equivalent, given that the entire myocardium is supplied, in effect, by a single right coronary artery. Because of all the attendant concerns, the concept of a "two-coronary system" was promulgated over that of "single coronary system" and is definitely considered the superior of the two. None of the surgical approaches to establishing this 2-coronary system has been proved superior to another, however [Dodge-Khatami 2002].

Restoring a dual coronary system, regardless of the age group, has been the standard of care since Cooley et al [1966] first grafted the ALCAPA to the ascending aorta with a saphenous vein graft. Extrapolating from the usual rates of CABG vein graft patency, we conclude that patients require reoperation for revascularization as patency rates plummet to 80% at 5.8 years [Moodie 1983]. To achieve longer patency rates and avoid reoperation in these patients, who are significantly younger than the usual cohort of patients with atherosclerotic

coronary artery disease, cardiac surgeons soon came to prefer arterial revascularization with the LIMA.

The most remarkable development in the history of surgical treatment of ALCAPA was the successful direct implantation of an ALCAPA into the aorta with a button of the pulmonary artery [Neches 1974]. This modality is the most popular and is arguably the gold standard against which other modalities should be compared, at least in the pediatric population. It represents the most physiological and anatomically correct repair among all of the options available for establishing a dual coronary system, and it has the lowest mortality rate, ranging from 0% to 16% [Dodge-Khatami 2002]. Increasing experience with the coronary button-transfer technique among pediatric cardiac surgeons and the availability of improved cardioprotection techniques during cardiopulmonary bypass are responsible for its widespread acceptance in the current era. Other considerations that could make direct implantation technically challenging include insufficient length of the ALCAPA button (particularly if the ALCAPA arises from the nonfacing pulmonary sinus) and the presence of collaterals that make mobilization of the coronary button difficult.

ALCAPA button mobilization may be hazardous only in adult patients, because of increased adhesions, friability, diminished vessel elasticity due to the aging process, and multiple collaterals. In these cases, LIMA grafts should be used. If the LIMA is unavailable, as in our patient 5, we believe that use of a reversed saphenous vein graft is acceptable.

The left ventricular ejection fraction begins to improve weeks to months after the repair and returns to normal by 1 year, as the recruitment of hibernating myocardium is completed. Improvement in mitral insufficiency takes longer, because it is achieved through remodeling, which is a slower process than recruitment. Another significant contribution was made by Takeuchi et al [1979], who used native pulmonary artery tissue to make an intrapulmonary baffle from the ALCAPA ostium to the anticipated takeoff site on the ascending aorta, where a punch hole is made and an aortopulmonary window is fashioned. The defect in the pulmonary artery is patched with autologous pericardium or a homograft. Mortality rates are comparable to those of direct aortic implantation [Dodge-Khatami 2002], but more than one third of patients require a repeat intervention for complications, which include supravalvular pulmonary stenosis, aortic regurgitation, baffle obstruction, and baffle leaks causing coronary-pulmonary artery fistula. Owing to the high incidence of postoperative complications, this technique is used only in infants for whom direct coronary implantation is not possible because of short length or increased collaterals. As demonstrated in our series, we feel that the surgical procedures need to be individualized to the patient's anatomy. Direct implantation is always preferred in younger patients, because mobilization of the anomalous coronary artery is not difficult; however, CABG is a viable option for patients who are beyond the fourth decade of life, because it avoids unnecessary and hazardous mobilization of the anomalous coronary artery within an extensive collateral network and shortens the aortic cross-clamp time. The Takeuchi procedure is almost obsolete in current surgical practice.

Revascularization procedures are meaningful only if there is ischemic but salvageable myocardium. In cases with extensive myocardial infarction leading to nonrecruitable myocardium, little is to be gained by surgical revascularization, and cardiac transplantation is the last resort for such patients. In the current era, however, the diagnosis of ALCAPA is more frequent because of increased awareness and the availability of various noninvasive imaging modalities. An aggressive early surgical approach and the availability of ventricular support devices should make the need for this end-stage option very rare as more and more patients with severe preoperative myocardial dysfunction are saved.

Treatment of mitral valve regurgitation presenting with ALCAPA is a controversial issue for which there are no well-defined guidelines. Investigators who advocate leaving the mitral valve alone at the time of the initial procedure argue that it decreases the bypass time and that even severe ventricular dysfunction has been shown to regress in the majority of cases following repair with a dual coronary system [Schwartz 1997; Dodge-Khatami 2002; Brown 2008]. Others argue that some form of mitral annuloplasty is necessary for optimizing the patient's hemodynamics in the critical postoperative period [Isomatsu 2001]. Others prefer to address only severe degrees of mitral insufficiency during the initial revascularization procedure, especially in the absence of severe left ventricular dysfunction [Ali 2009]. This scenario indicates that besides ventricular dysfunction, other irreversible changes that need repair and are unlikely to improve with improving left ventricular function (e.g. annular dilatation, chordal elongation, papillary muscle fibrosis) are also important in causing mitral regurgitation.

CONCLUSION

A few naturally selected patients of ALCAPA survive beyond infancy; rarely, they survive into adulthood and beyond. The most important factor favoring this process of natural selection appears to be the density of the coronary and noncoronary sources of collateral circulation to the ALCAPA that develops over time. We conclude from our experience that a dual coronary system in these patients can be established with satisfactory results by using direct implantation into the ascending aorta or by using CABG with obliteration of the ALCAPA lumen when direct implantation is not feasible.

REFERENCES

- Abbot ME. 1908. Congenital heart disease. In: Osler W, ed. *Modern medicine: its theory and practice*. Philadelphia, PA: Lea and Febiger. p 420-1.
- Akbari J, Theodore S, Krishnamanohar SR, Neelakandhan KS. 2007. Pulmonary hypertension alters natural history of anomalous left coronary artery. *Asian Cardiovasc Thorac Ann* 15:23-4.
- Ali WB, Metton O, Roubertie F, et al. 2009. Anomalous origin of the left coronary artery from the pulmonary artery: late results with special attention to the mitral valve. *Eur J Cardiothorac Surg* 36:244-9.
- Bergmann O, Bhardwaj RD, Bernard S, et al. 2009. Evidence for cardiomyocyte renewal in humans. *Science* 324:98-102.
- Bland EF, White PD, Garland J. 1933. Congenital anomalies of the coronary arteries. Report of an unusual case associated with cardiac hypertrophy. *Am Heart J* 8:787-801.
- Brown JW, Ruzmetov M, Parent JJ, Rodefeld MD, Turrentine MW. 2008. Does the degree of preoperative mitral regurgitation predict survival or the need for mitral valve repair or replacement in patients with anomalous origin of the left coronary artery from the pulmonary artery? *J Thorac Cardiovasc Surg* 136:743-8.
- Collins N, Colman J, Benson L, Hansen M, Merchant N, Horlick E. 2007. Successful percutaneous treatment of anomalous left coronary artery from pulmonary artery. *Int J Cardiol* 122:e29-31.
- Cooley DA, Hallman GL, RD Bloodwell. 1966. Definitive surgical treatment of anomalous origin of the left coronary artery from pulmonary artery: indications and results. *J Thorac Cardiovasc Surg* 52:798-808.
- Dodge-Khatami A, Mavroudis C, Backer CL. 2002. Anomalous origin of left coronary artery from the pulmonary artery: collective review of surgical therapy. *Ann Thorac Surg* 74:946-55.
- Fierens C, Budts W, Deneff B, Van De Werf F. 2000. A 72 year old woman with ALCAPA. *Heart* 83:e2.
- Isomatsu Y, Imai Y, Shin'oka T, Aoki M, Iwata Y. 2001. Surgical intervention for anomalous origin of the left coronary artery from the pulmonary artery: the Tokyo experience. *J Thorac Cardiovasc Surg* 121:792-7.
- Lim JY, Chung CH, Ma DS, Lee SH. 2011. A 69-year-old woman with anomalous origin of left coronary artery from the pulmonary artery: surgical repair using a trap-door flap. *Korean J Thorac Cardiovasc Surg* 44:358-60.
- Moodie DS, Fyfe D, Gill, et al. 1983. Anomalous origin of the left coronary artery from the pulmonary artery (Bland-White-Garland syndrome) in adult patients: long-term follow-up after surgery. *Am Heart J* 106:381-8.
- Neches WH, Mathews RA, Park SC, et al. 1974. Anomalous origin of the left coronary artery from the pulmonary artery. A new method of surgical repair. *Circulation* 50:582-7.
- Raghuram AR, Krishnan R, Kumar S. 2004. Anomalous left coronary artery from pulmonary artery in an adult. *Ind J Thorac Cardiovasc Surg*.20: 213-15.
- Sabiston DC Jr, Neil CA, Taussig HB. 1960. The direction of blood flow in anomalous left coronary artery arising from the pulmonary artery. *Circulation* 22:591-7.
- Schwartz ML, Jonas RA, Colan SD. 1997. Anomalous origin of the left coronary artery from the pulmonary artery: recovery of left ventricular function after dual coronary repair. *J Am Coll Cardiol* 30:547-53.
- Shivalkar B, Borgers M, Daenen W, Gewillig M, Flameng W. 1994. ALCAPA syndrome: an example of chronic myocardial hypoperfusion? *J Am Coll Cardiol* 23:772-8.
- Takeuchi S, Imamura S, Katsumoto K, et al. 1979. New surgical method for repair of anomalous left coronary artery from pulmonary artery. *J Thorac Cardiovasc Surg* 78:7-11.
- Wesselhoeft H, Fawcett JS, Johnson AL. 1968. Anomalous origin of the left coronary artery from the pulmonary trunk. Its clinical spectrum, pathology, and pathophysiology, based on a review of 140 cases with seven further cases. *Circulation* 38:403-25.
- Yau JM, Singh R, Halpern EJ, Fischman D. 2011. Anomalous origin of the left coronary artery from the pulmonary artery in adults: a comprehensive review of 151 adult cases and a new diagnosis in a 53-year-old woman. *Clin Cardiol* 34;4:204-10.