

REVIEW PAPER

Anomalous left coronary artery from the pulmonary artery – what we know about the disease 90 years after its discovery by Bland, White, and Garland

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ABSTRACT

Anomalous left coronary artery from the pulmonary artery (ALCAPA) is a rare congenital coronary anomaly associated with a mortality rate of 90% within the first year of life, with diagnosis occurring in only 10% to 15% of patients during adulthood. ALCAPA is subdivided into infant type and adult type. While angiography remains the diagnostic gold standard, echocardiography serves as a convenient, non-invasive, and highly reliable initial step in suspected cases of ALCAPA. Computed tomography and magnetic resonance imaging are also capable of providing a definitive diagnosis. Surgical management aims to establish a dual-coronary system through various techniques, with goals including gradual improvement in left ventricular function and reduction in mitral regurgitation. In selected cases, consideration may also be given to mitral valvuloplasty or placement of an implantable cardioverter defibrillator. Postoperatively, patients should undergo regular follow-up evaluations to monitor cardiac function and detect any complications.

KEY WORDS:

ALCAPA, coronary artery anomaly, anomalous left coronary artery from the pulmonary artery, Bland-White-Garland syndrome, adult congenital heart disease.

INTRODUCTION

Bland-White-Garland syndrome (BWGS), also referred to as White-Garland syndrome, is medically recognised as ALCAPA, which stands for anomalous left coronary artery from the pulmonary artery. This condition is characterised by an unusual origin of the left coronary

artery (LCA), either from the pulmonary trunk or from one of the pulmonary arteries (PA). It is a rare syndrome, occurring in approximately 1 out of 300,000 births, and it constitutes about 0.25–0.5% of congenital heart defects [1–4]. Our review seeks to present an overview and consolidate the latest insights into ALCAPA, as well as examine its diagnostic protocol and treatment approaches.

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HISTORICAL CONTEXT

The disease's historical narrative commences in 1885, when Brooks documented ARCAPA – an anomalous right coronary artery originating from the pulmonary artery [5]. The initial reports of anomalous left artery cases were credited to Konstantinovich in 1906 and Abrikosoff in 1911 [6, 7]. However, these early accounts remained relatively obscure until 1933, when 3 physicians from Massachusetts General Hospital in Boston – Paul Dudley White, William Franklin Bland, and Joseph Garland – outlined the case of a 3-year-old patient with pallor, wheezing, and sweating. The infant passed away after being hospitalised for 2 weeks. Upon examining the coronary arteries, a surprising discovery was made: the right artery was normal, but the left artery was found to originate from the pulmonary artery, supplying blood to the left ventricle. Microscopic analysis showed fibrosis, especially in the left ventricle, with no evidence of infarction. Bland, White, and Garland reviewed the current literature concerning anomalous coronary arteries and determined that these anomalies are linked to a negative prognosis [8].

PATHOGENESIS AND SYMPTOMS

During embryological development, a deficiency in vascular endothelial growth factor-C causes the cells of the capillary plexus around the pulmonary artery and aorta to fail to reach or penetrate the normal coronary origins. This deficiency results in an anomalous coronary origin from the pulmonary artery. ALCAPA may arise from the regression of the aortic buds, which are supposed to form the coronary arteries, while the pulmonary buds persist. In addition, abnormal separation of the conotruncus from the aorta and pulmonary artery also plays a role in this condition [9].

In cases of ALCAPA, the LCA, which arises from the PA, supplies the myocardium with low-pressure, deoxygenated blood. This results in ischaemia, mitral regurgitation, left ventricular dysfunction, and heart failure. Known as the infant-type ALCAPA, this condition carries a poor prognosis, with a 90% mortality rate within the first year of life if not corrected [10]. In a minority of patients, extensive collateralisation from the right coronary artery (RCA), originating from the aorta, supplies oxygenated, high-pressure blood to the left coronary system in a retrograde manner. The blood flow follows this sequence: aorta, RCA, collateral vessels, anomalous LCA, and finally shunts into the PA. These patients typically do not exhibit symptoms in childhood, corresponding to the adult-type ALCAPA, which manifests later in life with angina, heart failure, arrhythmias, or sudden cardiac death. The mechanisms involved in the adult form of ALCAPA include myocardial ischaemia due to blood flow diversion into the PA and left ventricular volume overload caused by left-to-right shunt [3, 11, 12].

The defect is usually isolated, sometimes coexisting with atrial septal defect, patent ductus arteriosus, tetralogy of Fallot, bicuspid aortic valve, aorto-pulmonary window, and coarctation of the aorta. Several conditions have also been described, including hypoplastic left heart syndrome,

scimitar syndrome, ventricular septal defect with aortic coarctation, ventricular septal defect with right pulmonary artery discontinuity, and double outlet right ventricle with mitral atresia [13–15]. In newborns, symptoms are usually absent; this is due to physiological high pulmonary resistance, which implies blood flow from the PA into the abnormal coronary artery. When the pressure in the pulmonary circulation is lowered in the second month of age, there occurs a reversal of flow resulting in a stealth syndrome: blood retreats from the LCA to the PA. The rapidity of patent ductus arteriosus closure and the development of collateral circulation between the coronary arteries are important in assessing the outcomes. Reduced inflow to the LCA causes perfusion disturbances in the area of the heart supplied by this artery. If the development of collaterals is inadequate, and the inflow of blood from the RCA to LCA is insufficient, the patient develops the symptoms of BWGS: pallor of the skin, restlessness, baby colic, and feeding difficulties [10, 16, 17].

Chronic ischaemia induces endocardial fibrosis and papillary muscle damage, culminating in the onset of mitral regurgitation and progressive dilation of the left ventricle. In severe cases, this can result in myocardial infarction. The presence of well-developed and efficient collateral circulation increases the likelihood of a clinically silent or mildly symptomatic disease course. Common symptoms in adults include syncope, dizziness, chest pain, as well as supraventricular and ventricular arrhythmias, sometimes leading to sudden cardiac death. Echocardiography typically reveals mitral valve regurgitation, regional contraction abnormalities, and left ventricular dilation [10].

Yau *et al.* [17] conducted a review of 151 adult cases of ALCAPA, finding that the average age at diagnosis was 41 years. Among these cases, 48 patients were older than 50 years, and there was a female-to-male ratio greater than 2 : 1. Symptoms of angina, dyspnoea, palpitations, or fatigue were observed in 66% of the patients. Meanwhile, 17% experienced ventricular arrhythmia, syncope, or sudden death, and 14% were asymptomatic.

DIAGNOSTICS

During a physical examination, findings are typically unremarkable. However, it may uncover a systolic heart murmur indicative of mitral regurgitation, pulmonary abnormalities such as crackles suggesting pulmonary oedema, and heart failure signs like elevated jugular venous pressure, poor perfusion, and peripheral oedema [18].

The gold standard for diagnosing ALCAPA is coronary angiography. Coronary angiography identifies several distinctive features: a tumour-like dilatation of the RCA, an empty left aortic sinus, and the flow of contrast medium from the tortuous RCA to the LCA via collateral circulation. The LCA then retrogradely fills the PA, confirming that the LCA originates from the PA [11, 19]. Coronary angiography images showing the RCA departing from the aortic bulb, and the abnormal LCA departing from the pulmonary artery, are shown in Figure 1.

Nevertheless, coronary angiography is often replaced by non-invasive diagnostic methods. CTA (computed tomography angiography) may also be used. This is

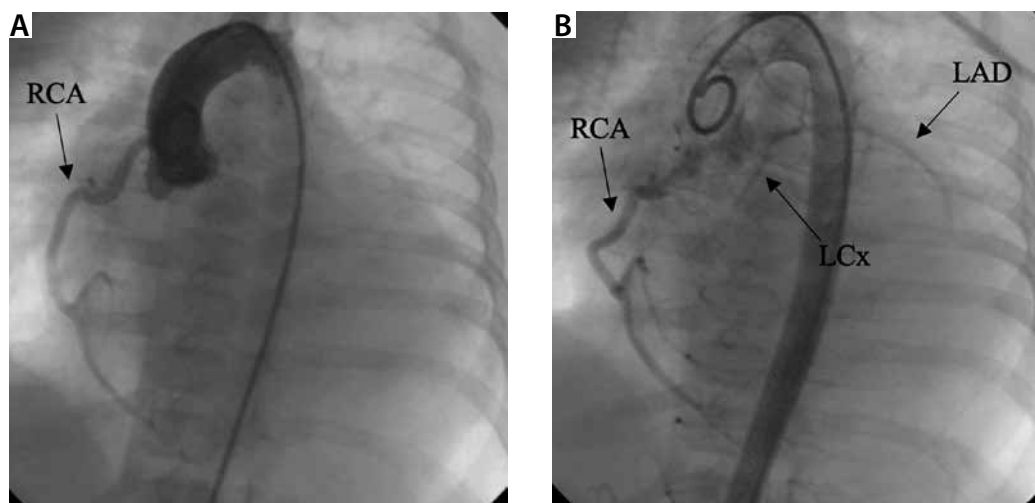


FIGURE 1. A) RCA departing from right aortic sinus, absent LCA departure. **B)** RCA departing from right aortic sinus, LCA originating outside of the aorta (departing from the pulmonary trunk) and subdividing into left anterior descending artery (LAD) and circumflex artery (LCx)

a non-invasive test that is safer than coronary angiography, while being less accurate. ALCAPA is indicated by direct visualisation of the LCA originating from the PA, a dilated RCA with extensive coronary collateral arteries, abnormal left ventricular wall motion, and dilated bronchial arteries serving as systemic collaterals. In this condition, the LCA originates from the left inferolateral aspect of the main pulmonary artery just behind the pulmonary valve. It then courses through the interventricular groove before dividing into the left anterior descending artery and left circumflex artery. However, in infants, the LCA and RCA may present without any abnormal appearance [11]. In recent years, various CTAs have become the preferred imaging modality for diagnosing coronary artery diseases, offering a fast and accurate method for identifying coronary abnormalities in infants and children [20]. However, diagnosing coronary artery disease with CTA presents technical challenges due to infants' rapid heart rates, their inability to hold their breath, and the small size of their coronary arteries. By employing a range of postprocessing techniques, a scanning method called dynamic volume CTA can directly and accurately visualise the ostium and course of ALCAPA, along with related indirect signs such as compensatory dilatation of the contralateral coronary artery, formation of collateral vessels, and ventricular enlargement. By completely eliminating the impact of heart-beat, respiration, and other factors on image quality, this method significantly enhances both the success rate of the examination and the quality of the images [21].

Despite the indisputable effectiveness of both above imaging techniques, transthoracic echocardiography is often sufficient for diagnosing ALCAPA. With transthoracic echocardiography, we can identify a dilated RCA and an abnormal origin of the LCA from the PA instead of the aortic sinus. Colour Doppler imaging has become the standard method for diagnosing ALCAPA because it can detect the characteristic reversal of flow in the LCA and its exit to the pulmonary artery [22]. LCA departing from the pulmonary trunk, with retrograde inflow on colour Doppler study, is shown in Figure 2.



FIGURE 2. LCA departure from the pulmonary trunk with retrograde inflow on colour Doppler

Moreover, echocardiography enables assessment of left ventricular dimensions (typically dilation) and function (including any contraction abnormalities), as well as estimation of mitral valve functionality. In BWGS, mitral valve regurgitation originates from progressive damage to the papillary muscles and dilation of the mitral annulus due to ventricular enlargement. Chronic myocardial ischaemia leads to increased echogenicity of papillary muscles and the presence of endocardial fibroelastosis [17, 23, 24]. Yu *et al.* [25] examined the echocardiographic features of 30 ALCAPA patients, 10 of whom were adults. They discovered that, in contrast to infantile ALCAPA patients, adult ALCAPA patients had abundant collateral circulation, no abnormal wall motion, no echo enhancement of the papillary muscle, normal or mildly dilated left ventricular diameter, normal ejection fraction, mild mitral regurgitation, and a significantly increased ratio of the RCA to the aortic annulus.

As for electrocardiography, the most common alterations typically manifest as signs of anterolateral myocardial infarction, characterised by the presence of Q waves, inverted T waves, and poor R-wave progression in V4–V6, I, and aVL leads. ST-segment elevation or depression in anterolateral leads has also been documented [12, 18, 26].

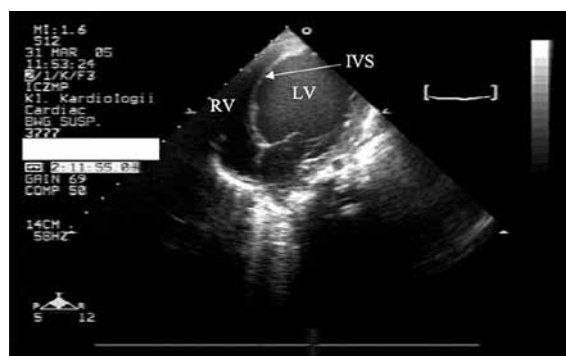


FIGURE 3. Expanded left ventricle (LV), overstretched interventricular septum (IVS) moved towards the right ventricle (RV) in a patient diagnosed with ALCAPA

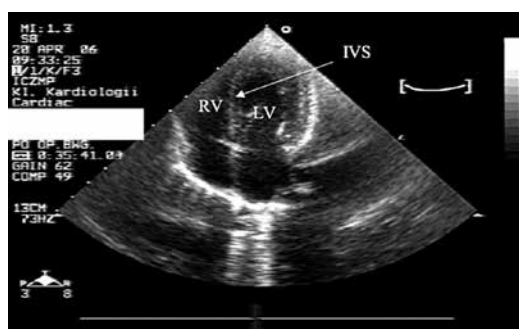


FIGURE 4. Improved left ventricular functionality in a patient after direct LCA implantation

Myocardial viability can be assessed using stress echocardiography, single photon emission computed tomography, positron emission tomography, and nuclear magnetic resonance. Magnetic resonance angiography offers superior functional visualisation of the coronary arteries compared to CTA. Particularly for paediatric patients, it stands out as the preferred choice due to its lack of ionising radiation exposure and the absence of a necessity for a low heart rate. Perfusion abnormalities typically impact the anterior and inferior walls, enabling scintigraphic imaging to differentiate between ALCAPA and dilated cardiomyopathy. In ALCAPA, these abnormalities contrast with those seen in dilated cardiomyopathy, where the greatly enlarged left ventricle shows severe contractile dysfunction [18, 27].

TREATMENT AND OUTCOMES

The 2018 American Heart Association/American College of Cardiology and 2020 European Society of Cardiology guidelines for congenital heart disease both recommend immediate surgery upon diagnosis of ALCAPA, regardless of the presence of symptoms, to restore dual coronary circulation (Class I) [28, 29]. Notable surgical procedures involve direct reimplantation of the coronary artery into the aorta (known as coronary button transfer), transpulmonary baffling (Takeuchi procedure), and coronary artery bypass grafting.

The earlier treatment approach consisted of ligating the abnormal LCA and depended exclusively on the RCA to maintain single-coronary circulation. Currently, this

method is rarely used and is applied only in a narrow group of patients, in the transitional period before surgery to restore the dual-coronary circulation.

Direct implantation provides a more physiologically accurate correction, leading to improved long-term outcomes. The procedure involves several critical steps: first, the main pulmonary artery is transected, followed by the excision of a circular section of the pulmonary artery surrounding the anomalous ostium of the LCA, known as the “button”. A hole is then created in the aorta, where the LCA is anastomosed in an end-to-side manner. Finally, the main pulmonary artery is reconstructed. This method has proven particularly effective in infants. However, this approach carries the risk of LCA rupture and bleeding due to its fragility and reduced elasticity when mobilising the coronary anomaly for repair.

In patients in whom direct aortic implantation is challenging due to the immobilisation or fragility of the LCA, alternative techniques are recommended. The Takeuchi method is typically employed in older patients and involves surgically creating an aorto-pulmonary window and constructing an internal tunnel that connects the coronary ostium in the pulmonary artery to the aorta. This approach is favoured when there is a significant distance between the abnormal coronary artery opening and the aorta, extensive collateral circulation around the LCA, or difficulties in mobilising the LCA. However, the Takeuchi method carries potential risks, including the development of supravulvar pulmonary stenosis, as well as patch leakage or narrowing of the constructed channel.

Coronary artery bypass grafting entails ligating the anomalous LCA at its origin and connecting the left anterior descending artery and left circumflex artery to the aorta using either an arterial graft (left internal mammary artery) or a venous graft (saphenous vein). This approach is preferred in adults and is generally well-tolerated, although it carries risks of graft occlusion and stenosis [11, 18, 30–33].

Corrective surgery is the established treatment for ALCAPA in both infants and adults. However, medications can be utilised alongside surgical intervention to achieve optimal outcomes, as recommended by Hu *et al.* [34]. Additionally, medications may serve as an alternative to surgery in cases where the risks associated with surgery are deemed high and outweigh the potential benefits, as outlined by Sinha *et al.* [35]. In contemporary practice, patients undergo surgery promptly upon diagnosis of the defect, typically identified in infants several months old. The outcomes, whether immediate or delayed, hinge on the severity and scope of myocardial injury. Reimplantation of the LCA into the aorta typically enhances cardiac function within a few months. Performing early surgery to restore double coronary blood flow can normalise left ventricular size and function, thereby reducing mitral regurgitation. Left ventricular volume expansion in a patient diagnosed with ALCAPA is shown in Figure 3. Enhancement of left ventricular function after direct implantation is shown in Figure 4.

However, despite surgical repair, chronic myocardial ischaemic changes such as segmental wall motion abnormalities, perfusion defects, and myocardial scars may persist [18, 30–33]. ALCAPA, which can occasionally lead to

severe and irreversible heart damage known as ischaemic cardiomyopathy, often necessitates consideration for heart transplantation [27].

After a 16-year follow-up, Kanoh *et al.* [36] conducted a comparison of the long-term postoperative outcomes between 2 groups of ALCAPA patients. They observed that adults with ALCAPA had a greater occurrence of major adverse cardiovascular events compared to infants, primarily attributable to irreversible left ventricular remodeling induced by chronic myocardial ischaemia over an extended period. Schmitt *et al.* [37], through late gadolinium-enhanced MRI findings, identified ischaemia-induced myocardial scarring in 65% of adult ALCAPA patients post-surgical correction. Nevertheless, all patients exhibited good left ventricle contractile function recovery [37]. Browne *et al.* [38] documented 2 instances of children diagnosed with ALCAPA who received orthotopic cardiac transplants. Pathological examination of their cardiac explants revealed extensive fibrotic tissue, corresponding to areas of abnormal delayed enhancement observed on their MRI scans.

Despite that findings, surgically corrected ALCAPA generally leads to positive long-term outcomes with low mortality rates. Most cases show improvements such as increased ejection fraction, recovered left ventricle function, improved mitral regurgitation, and reduced symptoms, regardless of the surgical method used [39]. However, patient outcomes largely depend on the extent of myocardial damage before surgery [40, 41].

In a multicentre review of 98 infant ALCAPA cases, Radman *et al.* [42] found that left ventricular function normalised in 98% of cases approximately 3 years after corrective surgery, though predicting the course of mitral regurgitation remained challenging. Mishra reported outcomes for 98 ALCAPA patients with a median follow-up of 5.9 years, all of whom underwent coronary reimplantation, revealing an in-hospital mortality rate of 8.5% and no late deaths [40]. Hu *et al.* [34] studied 80 paediatric ALCAPA patients who underwent various corrective procedures and found an 83.4% survival rate one year post-operation, with 13.8% hospital deaths and 1.3% late deaths at the one-year follow-up. In their multicentre study, Flores *et al.* [43] compared 258 patients with surgically corrected ALCAPA. Among these patients, 10 had complex ALCAPA and 248 had isolated ALCAPA. The heart defects associated with ALCAPA included hypoplastic left heart syndrome, scimitar syndrome, ventricular septal defect with coarctation of the aorta, ventricular septal defect with interruption of the right pulmonary artery, right ventricular double outlet with mitral atresia, and tetralogy of Fallot. Following ALCAPA repair, patients with complex ALCAPA were more likely to require extracorporeal membrane oxygenation (50% vs. 12%, $p = 0.002$), undergo cardiopulmonary resuscitation (30% vs. 6%, $p = 0.017$), or experience operative mortality (50% vs. 3%, $p < 0.001$) compared to those with isolated ALCAPA. Sundara Rao *et al.* [44] investigated the outcomes of surgical repair in 21 patients with ALCAPA, with a median follow-up period of 5 years. There were no deaths reported. However, 2 patients (9.52%) required a prolonged stay in the intensive care unit (over 7 days). By the final follow-up, the mean left ventricle ejection fraction had improved to $57.47 \pm 4.97\%$, and moderate mitral regurgitation had regressed in 4 patients (66.6%).

Straka *et al.* [45] compared 69 patients with early-onset ALCAPA (< 1 year) to those with late-onset ALCAPA (≥ 1 year). In the late-onset group, the median age was 7.6 years, with 25% of patients being older than 18 years. The late-onset group more often presented as an incidental finding (63%) and required fewer preoperative interventions compared to the early-onset group. Preoperative echocardiography revealed that patients in the late-onset group had significantly lower rates of moderate to severe mitral regurgitation (16.7% vs. 62%, $p < 0.001$) and left ventricular dysfunction (16.7% vs. 89%, $p < 0.001$) than those in the early-onset group. Reoperations were rare (12%), and both groups experienced almost complete improvement in mitral regurgitation and left ventricular dysfunction over time. One patient was listed for a heart transplant, which was carried out approximately one year after the initial surgery [45].

The use of ICD in patients with ALCAPA is a topic of ongoing debate because there are concerns about potential complications, including the risk of infection, that should always be considered prior to ICD implantation. However, it is sometimes recommended due to the presence of risk factors associated with sudden cardiac death post-surgical intervention. These factors include severe left ventricular dysfunction, fibrotic lesions potentially predisposing to ventricular arrhythmias, non-sustained ventricular tachycardia during exercise testing or Holter monitoring, and a positive programmed ventricular pacing test [46, 47].

Different levels of mitral regurgitation are commonly observed in ALCAPA patients, but there is ongoing debate regarding the simultaneous performance of mitral valve valvuloplasty or annuloplasty. Mitral regurgitation often shows improvement following ALCAPA correction, leading many to suggest avoiding mitral valve intervention. However, severe cases of mitral regurgitation may necessitate intervention [48].

Following surgical recovery, patients should undergo regular follow-ups to monitor cardiac function and detect any complications. In adults, ECG-gated multidetector CTA and cardiac magnetic resonance imaging are valuable tools for these follow-up assessments [11]. The American Heart Association/American College of Cardiology guidelines also advise regular noninvasive stress imaging every 3 to 5 years for patients who have undergone surgical repair for ALCAPA, ensuring ongoing assessment throughout their lifetime [49].

CONCLUSIONS

ALCAPA is a rare congenital defect in which the left coronary artery abnormally originates from the pulmonary artery. It presents differently in infants and adults, with subtypes identified by the degree of collateral blood supply and subsequent tolerance to coronary ischaemia. This abnormal origin causes ischaemia in the left coronary artery's territory, potentially resulting in left ventricular dysfunction, heart failure, mitral regurgitation, and sudden cardiac death. Coronary angiography is the gold standard for diagnosing ALCAPA. However, transthoracic echocardiography combined with other non-invasive tests like CTA or MRI can also provide a definitive diagnosis. The treatment for ALCAPA is surgical, aimed at

establishing a 2-vessel circulation. Techniques include direct coronary implantation, the Takeuchi procedure, and coronary artery bypass grafting. While early diagnosis and surgical correction generally lead to a good prognosis, chronic myocardial damage may persist. Post-surgery, patients should have regular follow-ups with serial cardiac imaging studies to detect any complications related to the type of surgical correction performed. In the future, the diagnosis of ALCAPA is likely to become more common due to the widespread availability of resuscitation techniques and advanced diagnostic tools. Highlighting the significance of coronary artery scanning in neonates is essential, particularly for those diagnosed with dilated cardiomyopathy. This testing is crucial to rule out the possibility of ALCAPA, ensuring accurate diagnosis and appropriate treatment. In conclusion, accumulating pathological and clinical data, along with long-term follow-up results after treatment, will enhance our understanding of this rare condition and help establish optimal treatment protocols.

DISCLOSURE

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