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Rare Etiology of Reversible Hypokinetic Dilatated Heart Disease in Infants: Bland-White-Garland Syndrome (ALCAPA)

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Abstract

Bland-White-Garland syndrome or ALCAPA is an abnormality of birth of the left coronary artery from the pulmonary artery. It is a diagnostic and therapeutic emergency because it is a curable cause of hypokinetic dilated heart disease in infants. We report through this clinical case, the fourth case of infantile ALCAPA diagnosed in Senegal in a 7-month-old infant. The symptomatology began around the age of 2 months, with a grumpy state associated with more marked crying and moaning during feedings and bowel movements. The mother reported hospitalization for a severe lung infection when she was 6 months old. The examination noted an infant in poor general condition, retarded growth and weight, and a 3/6th holosystolic murmur at the apex. Troponinemia was positive at 43.90 ng/L. The electrocardiogram showed Q waves on the lower side, a sub endocardial lesion on the upper side and a ST segment elevation in aVR. Doppler echocardiography showed dilated cardiomyopathy with a mean alteration of systolic function of the left ventricle at 37%, a mean mitral insufficiency and a strong suspicion of a birth anomaly of the left coronary artery. The CT scan confirmed the diagnosis of ALCAPA. Surgical reimplantation of the left coronary artery at the aortic level was performed at 10 months of life with a favourable outcome at D50 postoperative.

Keywords

ALCAPA, Infant, Adult, Epidemiology, Physiopathology, Clinic, Diagnosis, Echocardiography, Treatment, Surgery, Evolution, Senegal

1. Introduction

Bland, White, and Garland reported the first case of infantile ALCAPA fully described [1] [2] [3]. It is an abnormal birth of the left coronary artery from the pulmonary artery [1] [2] [3]. This birth defect is more commonly known by the acronym ALCAPA (Anomalous of the Left Coronary Artery from the Pulmonary Artery). It is extremely rare and represents less than 0.5% of congenital heart diseases [1]-[7]. Its incidence would be 1 in 30,000 live births [2] [4] [6] [8]. In 2017, LEYE *et al.* reported the first three cases of ALCAPA diagnosed in Senegal [1].

There are two forms of ALCAPA: infant forms and adult forms [1] [3] [6] [9]. In the absence of surgical correction, nearly 90% of infantile forms of ALCAPA will die within the first twelve months of their extra uterine life [3] [5] [9]. According to the literature, there are four different surgical methods and the Neches technique is the gold standard in corrective surgery for ALCAPA.

The heavy mortality of infantile ALCAPA is due to myocardial infarction responsible for congestive heart failure [5] [6]. However, the adult forms can be asymptomatic and allow a long life expectancy or be responsible for stable angina with a risk of sudden death from serious arrhythmias [5] [6].

We report through this clinical case, the fourth case of infantile ALCAPA diagnosed in Senegal.

2. Observation

It was a 7-month-old infant, female and black. She is the result of a twin pregnancy. She had been brought for consultation by her mother, in the cardiology department of the Aristide LE DANTEC Hospital as part of an exploration of a staturo-ponderal and motor delay. The history of the disease found a symptomatology, which would have started around his 2 months of extra uterine life, by a grumpy state associated with more marked and more frequent crying and moaning than his twin would during feedings. Paediatric consultations find a tachycardia and especially a delay in weight and motor development.

Family and perinatal history was unremarkable. The follow-up of the pregnancy was well carried out. The delivery took place at 39 weeks of amenorrhea, vaginally, with expulsion of healthy twins. The birth weight of our patient was 2790 Kg. The APGAR was 8/10 then 9/10. No neonatal infections were reported. She received a mixed diet until her 6 months of extra uterine life. His vaccination follow-up is up to date. However, the only effective motor acquisition to date was holding the head. The mother reported hospitalization for severe lung infection when she was 5 months old. The examination noted an infant in poor general condition, pink with no dysmorphia, polypnea at 60 bpm with SaO_2 at 99% AA, HR at 160 bpm, height at 66 cm, weight at 4.89 kg, CP at 42 cm, PT at 39 cm, DB at 10 cm. The cardiac examination noted a peak shock deviated downwards and to the left, an infundibulum-pulmonary shock, a $3/6^{th}$ holosystolic murmur at the apex. Peripheral pulses were present and symmetrical. The lung fields were free. The examination did not note syncope, cyanosis, squatting,

or finger clubbing.

Biology found positive troponinemia at 43.90 ng/L. The blood count showed a leucocyte level at 14.5 giga/L, anemia at 9.7 g/dL, hypochromic (VGM: 76 fl) microcytic (TCMH: 23.3 pg) and thrombocytosis at 543 giga/L. Renal, hepatic function, blood ionogram, negative C-Reactive Protein.

The frontal chest X-ray (**Figure 1**) showed global cardiomegaly with sub-diaphragmatic tip with a cardio-thoracic ratio of 0.65, hyperconvexity of the left middle arch and pulmonary hypervascularization.

The 12-lead surface electrocardiogram in this 7-month-old infant (**Figure 2**)

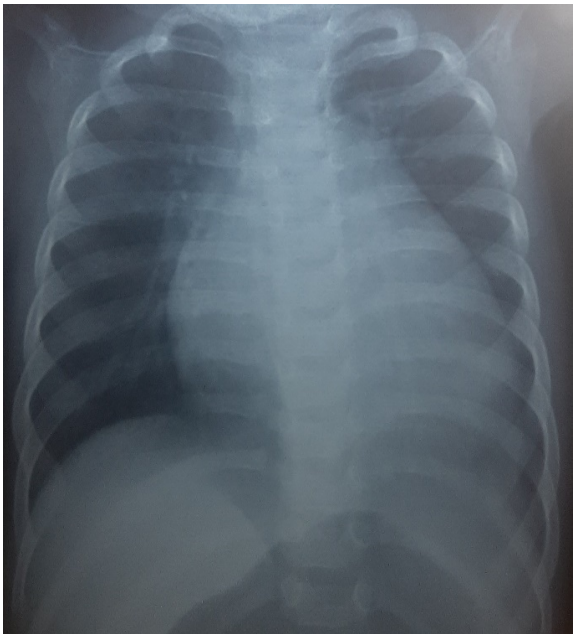

Figure 1. Frontal chest X-ray of our infant showing global cardiomegaly with a sub diaphragmatic tip, bulging of the left middle arch and pulmonary hyper vascularization.

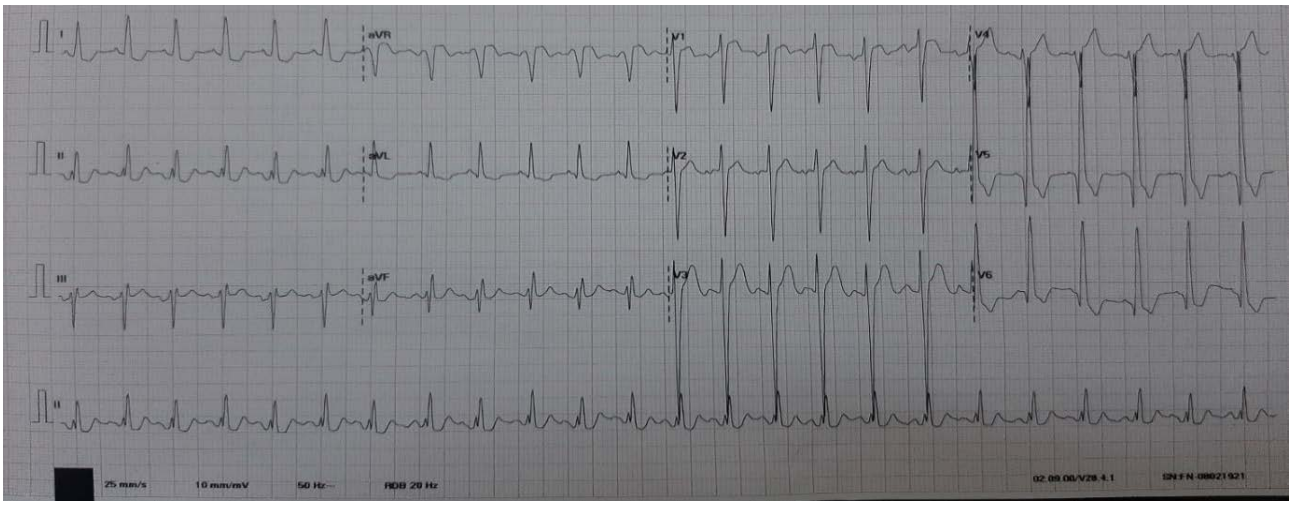

Figure 2. 12-lead surface electrocardiogram showing regular sinus rhythm, QRS axis at 0˚, left ventricular hypertrophy, Q wave of deep necrosis and sub epicardial ischemia on the lower side, sub endocardial lesion on the upper side and in D2. In addition, there was an elevation of the ST segment in aVR.

showed a regular sinus rhythm, a ventricular rate of 150 cycles per minute, a left axis at −10˚, left atrial hypertrophy, an rsR' aspect in DII, rSr' in DII, rSR' in aVF, left ventricular hypertrophy. In addition, there were Q waves of necrosis on the lower side, a sub endocardial lesion on the upper side and an elevation of the ST segment in aVR.

Transthoracic Doppler echocardiography (**Figure 3**) showed levocardia situs solitus with left aorta. Good veno-atrial, atrio-ventricular and ventriculo-arterial concordance with complete and tight septa. There was no persistence of the arterial duct or obstacle on the outflow tract. The pulmonary venous return was normal and the pericardium dry. There was a major dilation of the left heart chambers (left ventricle DVGd: +7.05 Z-score; DVGs: +8 Z-score; (**Figure 3(A)**); LA diameter = +5.15 Z-score (**Figure 3(B)**); an average deterioration of the systolic function of the left ventricle at 37% on the Simpson Biplane in connection with diffuse hypokinesia predominant at the level of the infero-septal and antero-lateral segments of the left ventricle. In addition, there was a dilation of the right (+2.78 Z-score) and left (+3.30 Z-score) pulmonary artery. In addition, there was moderate-to-moderate eccentric, mixed, ischemic mitral insufficiency due to restriction of the small mitral valve and dilatation of the mitral annulus (**Figure 3(C)**). The origin of the dilated right coronary artery was

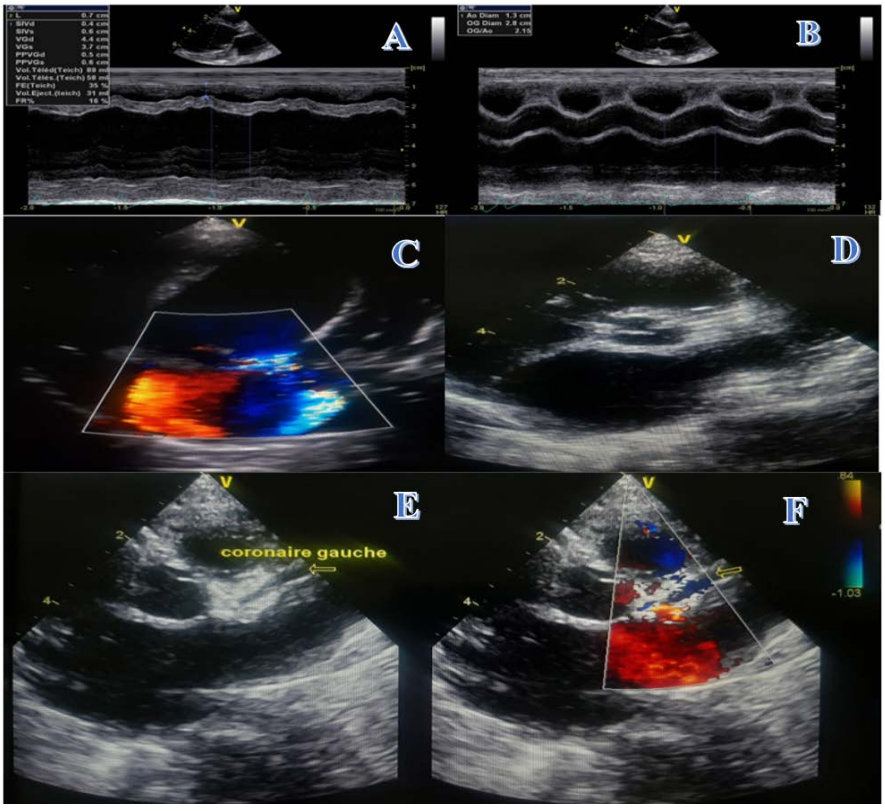

Figure 3. Doppler echocardiogram showing left ventricular dilation (A), left atrium dilation (B), eccentric mitral leak (C), right coronary occlusion in the right anterior cusp (D), the suspicious abutment of the left coronary from the pulmonary artery with the blue flow in its lumen (E), (F).

visible at the level of the antero-right sinus of the aorta (Figure 3(D)) and the left one was not visualized at the level of the antero-left sinus but rather suspected at the level of the pulmonary artery (Figure 3(E)). This suspicion was supported by the presence of a blue (retrograde) flow in its lumen (Figure 3(F)). All this evoked a birth defect of the left coronary artery of the pulmonary artery (ALCAPA) complicated by ischemic heart disease at the dilation stage with ejection fraction of the left ventricle altered to 37% Simpson Biplan and moderate mixed mitral insufficiency to medium.

The coro-scanner (Figure 4) carried out had made it possible to confirm the diagnosis by the demonstration of an anomaly of birth of the left coronary artery at the level of the lower face of the trunk of the pulmonary artery with a path between the left atrium and left ventricle.

The indication for surgery had been made. The patient received symptomatic medical treatment based on furosemide 5 mg × 3 per day, spironolactone 5 mg per day, captopril 5 mg × 3 per day, and digitalis 0.5 ml × 2 per day.

The corrective surgery was performed in the congenital and paediatric cardiology medical-surgical unit of the Necker Children's Hospital AP-HP. It consisted of an anatomical correction by reimplantation of the left coronary artery

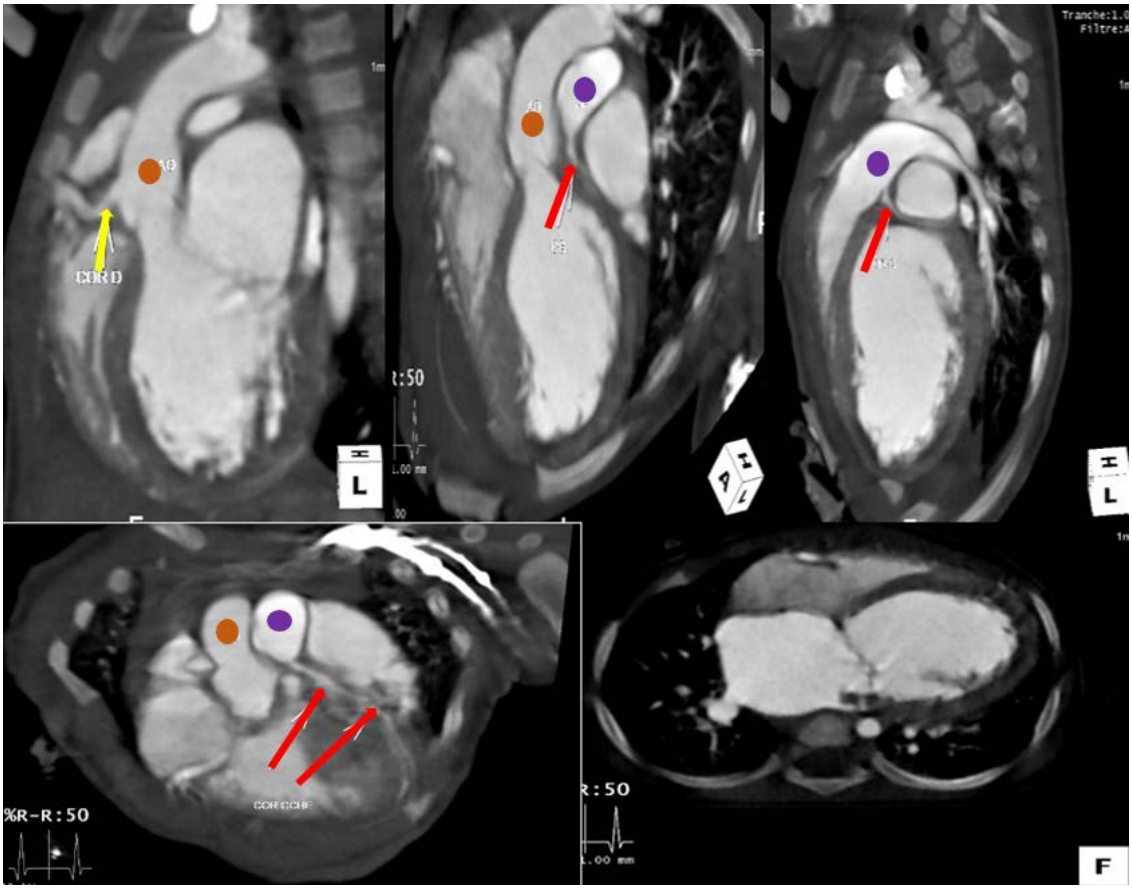

Figure 4. Coro-scanner images confirming ALCAPA by highlighting the abnormal birth of the left coronary (red arrow) from the pulmonary artery (violet point) and the modal birth of the right coronary (yellow arrow) from the aorta (brown point).

at the aortic level, under extracorporeal circulation for 2 hours 30 minutes. The patient did not need mechanical circulatory support postoperatively.

The immediate postoperative course was marked by pseudomonas aeruginosa pneumonia treated with cefepime and ceftazidime.

Echocardiography performed on D50 postoperatively shows a well-visualized left coronary artery from the aorta, with an anterograde flow (red) in its light, a left ventricular ejection fraction evaluated at 51% on the Simson Biplane, persistence of mixed mitral insufficiency. The follow-up of the patient has been decided to be done every two months and will include a physical examination and a Doppler echocardiography.

The chronology of the main events, from her birth to the surgery, has been summarized in Figure 5. The two main reasons for the loss of time were on the one hand the loss of sight of the patient for 3 months, on the other hand the search and the wait for surgery for 4 months.

3. Discussion

3.1. Epidemiology of ALCAPA

The first birth anomalies of the coronary artery by the pulmonary artery were observed by Krause in 1865 [10] [11] then by Brooks in 1886 [3] [4].

Bland, White, and Garland reported the first case of infantile ALCAPA fully described from clinical manifestations to arterial morphology at autopsy in 1933 in a 3-month-old extra uterine infant [12]. The average age of onset of symptoms in infantile forms of ALCAPA is between 2 to 3 months of extra uterine life

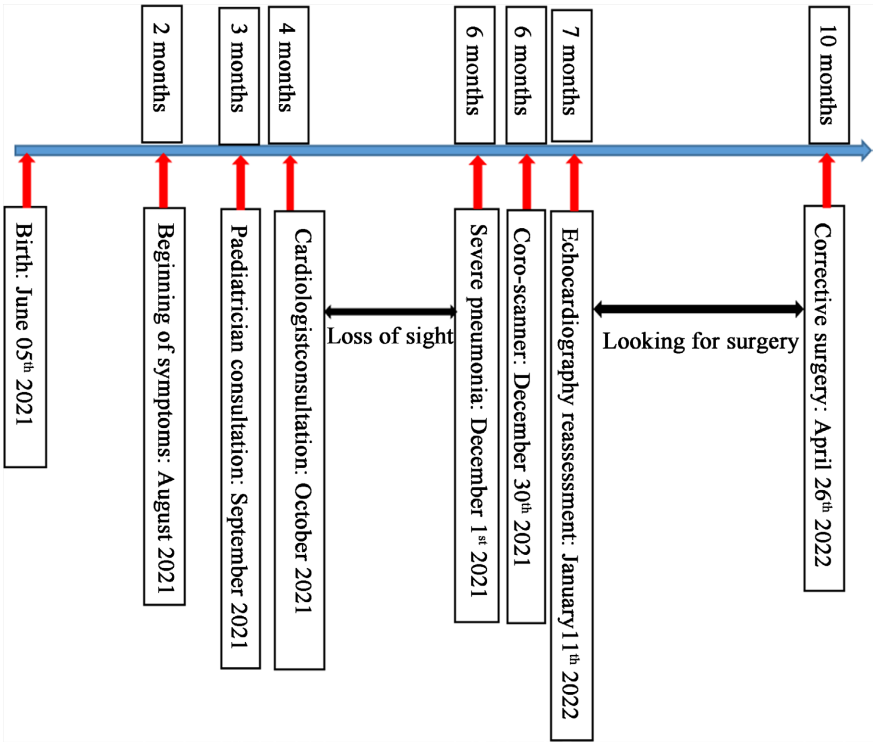

Figure 5. Chronogram of events.

[5] [9] [13] [14]. This was the case in our patient. Indeed the beginning of the symptomatology of our patient would have started around her 2 months of extra uterine life. In 1968, Wesselhoeft *et al.*, found in their series an age of onset of forms of infantile ALCAPA, which varied from two to 3 weeks to 6 months of extra uterine life, including the age group of 8 - 16 weeks of extra uterine life accounted for 58.33% of cases [9]. In 2019, in Benin, a male infant was also diagnosed at the age of 3 months [15]. In 2017, in the series of the first three cases of ALCAPA diagnosed in Senegal by Leye *et al.* there was a 3 month old male infant and two 6 and 10 month old female infants [1].

Regarding the adult forms of ALCAPA, it would represent 10 to 15% of ALCAPA [1] [3] [4] [5] [6] [16]. Maude Abbott has reported the first case in 1908 in a 60-year-old woman [11]. Yau *et al.* found an average age of 40.6 ± 15 years in a series of 151 adult ALCAPA cases collected from 1908 to 2008 [11]. The oldest subject was 83 years old [11]. Other cases diagnosed in old age have been reported: a 63-year-old woman [17] and a 70-year-old man in Italy [18], a 71-year-old woman in Malaysia [19], a 76-year-old in Estonia [13].

Through reported cases and some series in the literature, a female predominance seems to be the reality in ALCAPA. In their series, Yau *et al.* found a female predominance of around 69% [11]. The three cases of ALCAPA reported by Dahle were all female [4]. Among the nine patients operated on for ALCAPA in the series by Broks Holst *et al.*, seven (7) were female [20]. Similarly, of the 15 cases reported by Ramirez *et al.* there was an 86% female predominance [21]. This trend seems to be confirmed in Senegal. In fact, in the three cases reported by Leye *et al.* [1] and our present case, the female gender predominates.

3.2. Pathophysiology of ALCAPA

The first pathophysiological explanation of coronary circulation during ALCAPA was described by Jesse E. Edwards in 1964 [8] [22] (**Figure 6**).

During intrauterine life, pulmonary pressures are higher than systemic pressures [5] [22]. This ensures an anterograde flow in the abnormal left coronary

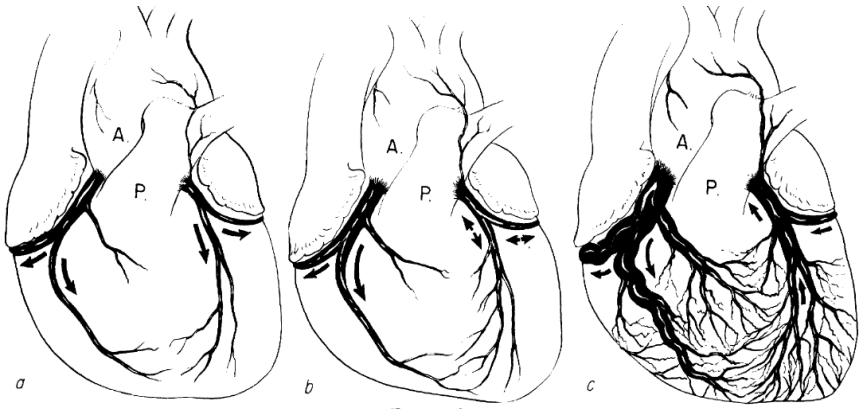

Figure 6. Illustration of the physiopathology of the circulation in the coronary arteries in case of ALCAPA. Extract from the article by Jesse E. EDWARDS in Circulation, Volume XXIX, February 1964 [22].

and in the normal right coronary [5] [8] [22]. At birth, pulmonary respiration of the neonate and umbilical clamping has the effect of progressively decreasing pulmonary pressures and raising systemic pressures [5] [8] [22]. This first follows a gradual equalization of pulmonary and systemic pressures in the early neonatal period. Thus, from D0 to D7 of extra uterine life there is less and less anterograde flow towards the left coronary artery. Then will come a definitive reversal of the pulmonary and systemic pressure ratio around the 8th week of extra uterine life [5] [8] [22]. This results in a flow reversal from the left coronary artery to the pulmonary artery [5] [8] [22].

Thus, during foetal and early neonatal life, the origin of the left coronary artery from the pulmonary artery is well tolerated because the pulmonary arterial pressure is greater than or equal to the systemic pressure, which leads to a flow anterograde in both the abnormal left coronary artery and the normal right coronary artery [5] [8] [22].

From the 8th week of extra uterine life, the complete reduction in pulmonary pressures reduces and reverses the flow in the left coronary artery which no longer manages to supply the myocardium and "drains" the fully oxygenated blood in the pulmonary artery [5] [8] [22] [23]. Thus, there is preferential blood flow in the low pressure pulmonary circulation rather than the high resistance myocardial circulation [5] [8] [22]. This left-to-right shunt is known as the "coronary steal" phenomenon [5] [8] [22]. This results in myocardial ischemia and left ventricular infarction [5] [8] [22]. It is the infantile form of ALCAPA (few or no collateral development inter coronary) with its consequences of severe myocardial ischemia of the left ventricle, dysfunction and dilation of the left ventricle and mitral insufficiency by ischemia of the papillary muscle and/or dilation of the mitral annulus [5] [8] [16] [22].

To survive beyond infancy and develop the adult form of ALCAPA, affected patients must have a dominant and perfectly patent right coronary artery, a restrictive opening of the left coronary artery at the level of the pulmonary artery and above all develop a large and extensive collateral circulation between the right coronary artery and the left coronary artery [5] [8] [12] [22] [24] [25] [26]. This is in the critical time interval between birth and the 8th week of extra uterine life [5] [8] [22]. Left ventricular myocardial perfusion then becomes dependent on these inter coronary collaterals [5] [8] [22] [24]. This is how some patients will survive to age 70 and beyond, with little or no symptoms of myocardial ischemia. Nevertheless, in the majority of cases, chronic sub endocardial ischemia of the left ventricle will persist, which is arrhythmogenic [5] [8] [22] [27].

3.3. Circumstances of discovery of infantile forms of ALCAPA

1) *Prolonged, recurrent or severe lung infection in an infant should suggest heart disease*. Indeed several observations of infantile ALCAPA report the existence in the history of the disease of a pneumopathy [1] [4] [15] [28] [29]. Our patient had also been hospitalized in her 6th month of life for severe pneumonia.

2) *Sudden growth retardation without obvious reason occurring around the* 2^{nd} *or* 3^{rd} *month of extra uterine life* may raise suspicion of infantile ALCAPA. Indeed Wesselhoeft *et al.* found that growth retardation was among the first symptoms in their series of 116 cases of infantile ALCAPA [9]. Similarly, Elena Peña presents the notion of growth retardation as being a sign generally present in infants in the event of ALCAPA [5]. Similarly, the patient of Gribaa *et al.* was underweight [30]. This was also the case in our patient who presented a break in her height and weight growth curves from the age of 2 months, in accordance with the charts and in comparison with those of her twin brother.

3) *A systolic apex murmur of mitral insufficiency* appearing after 2 months of extra-uterine life is another possible method of discovery. Wesselhoeft *et al.* found a proportion of 9.48% (11/116) of mitral insufficiency murmurs among infantile ALCAPAs [9]. This added breath noise was found in the cases of Adjagba *et al.* [15], Leye *et al.* [1], Trabelsi Sahnoun [31], Dahle [4], and Gribba [30]. The cardiac auscultation of our patient also noted this murmur.

4) *Congestive heart failure syndrome in infants:* It manifests itself in the form of choking during feedings, dyspnea with polypnea and sweating, tachycardia, left gallop sound. It is almost constant in the clinical picture of infantile ALCAPA and is generally ahead of the clinical picture [1] [4] [5] [9] [15] [30] [31]. One etiology of infant heart failure is indeed infantile ALCAPA [31].

5) *Cardiomegaly with a sub-diaphragmatic tip on the infant's chest radiograph* should systematically suggest an infantile ALCAPA (**Figure 7**). This anomaly is described in all reported cases of infantile ALCAPA [1] [4] [9] [15] [30] [31]. Our patient was no exception (**Figure 2**).

6) *Electrical signs of chronic left ventricular myocardial hypoperfusion on infant surface electrocardiogram* (**Figure 8**). These signs are of the type of ischemia, lesion and/or necrosis [11] [32]. They most often sit in the anterior territory, low antero-lateral, extended anterior [1] [4] [9] [15] [30] [31] [32]. In our

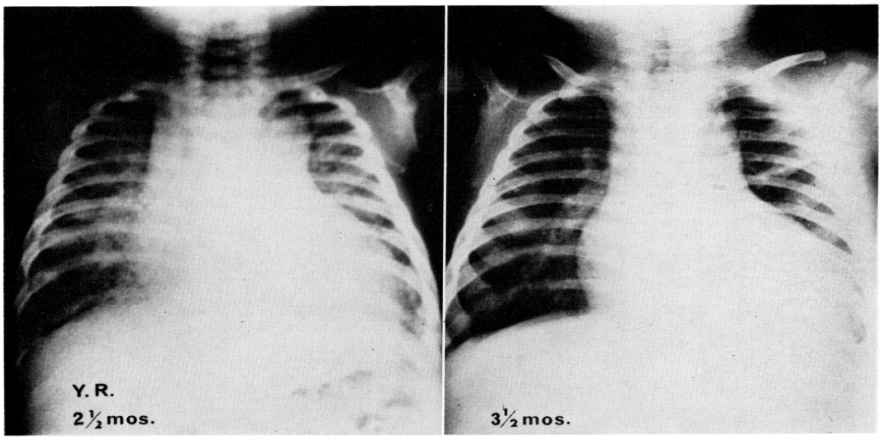

Figure 7. Example of a chest X-ray in 1968 of an infant (Y.R.) carrying infantile ALCAPA performed twice at one month intervals showing the worsening of global cardiomegaly with a sub-diaphragmatic peak. Excerpt from the article by HADWIG WESSELHOEFT *et al.* in Circulation, Volume XXXVIII, August 1968 [9].

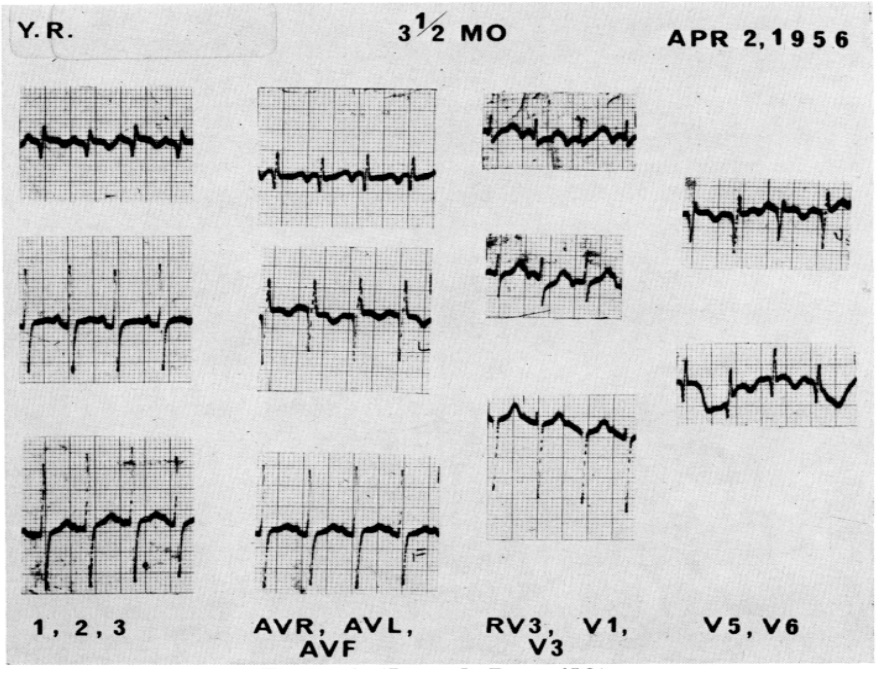

Figure 8. Example of an electrocardiographic tracing in 1968 of an infant (Y.R.) carrying infantile ALCAPA performed at the age of 3½ months showing Q waves of necrosis and subepicardial ischemia in the anterolateral direction. Excerpt from the article by HADWIG WESSELHOEFT *et al.* in Circulation, Volume XXXVIII, August 1968 [9].

patient, the Q waves were noted laterally in the lower lateral and the subendocardial lesion in the upper lateral. In addition, there was an ST segment elevation in aVR (**Figure 2**).

7) *Hypokinetic dilated cardiomyopathy on thoracic echocardiography in infants*. It must evoke and systematically search for an ALCAPA [1] [4] [9] [15] [30] [31] [32].

3.4. Circumstances of Discovery of Adult Forms of ALCAPA

They are dominated by stable angina, dyspnea [11]. Sudden death from malignant ventricular arrhythmias remains a formidable discovery modality [5] [8] [11] [16] [22] [27] [33] [34] [35]. It is estimated between 80% and 90% of sudden death at an average age of 35 years [16] [25] [26] [27] [36] [37]. ALCAPA in adults can be strictly asymptomatic until it is not the cause of death. The discovery is post-mortem at the autopsy [11].

3.5. Imaging Techniques for the Positive Diagnosis of ALCAPA

In addition to the autopsy which is a possible modality for the positive diagnosis of certainty, we also have invasive imaging techniques (supra-sigmoid aortography) and non-invasive (Doppler echocardiography, Coroscanner, cardiac MRI) for the diagnosis ALCAPA positive ante mortem [11] [38] [39]. Increasingly, the positive diagnosis relies on non-invasive imaging techniques to the detriment of coronary angiography (**Figure 9**). Indeed, nowadays, the strong suspicion is

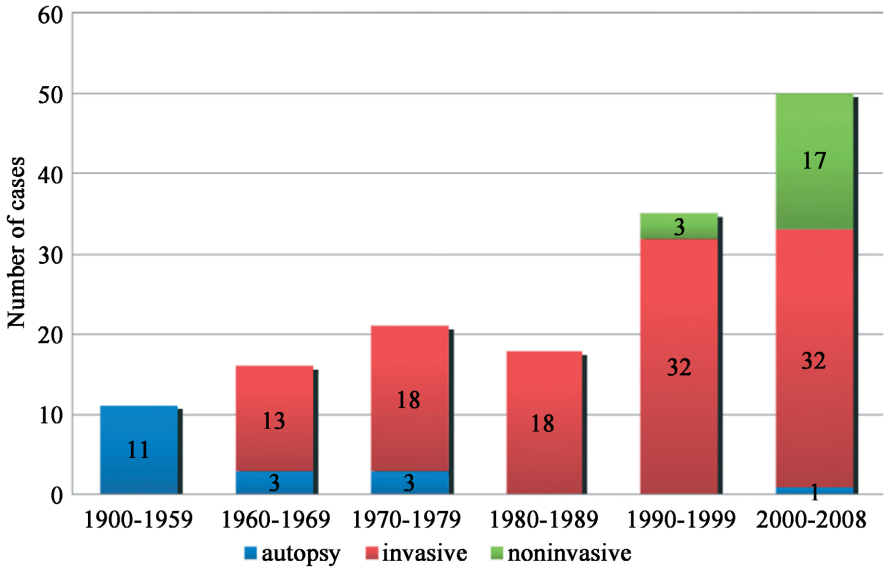

Figure 9. Periods and modalities of the ALCAPA diagnostic technique from 1900 to 2008, extract from the article by James M. Yau *et al.* [11].

brought by the echocardiography and the confirmation is made with the coro scanner. This diagnostic procedure was found in the cases reported by Leye *et al.* [1], and Adjagba *et al.* [15]. We also proceeded in the same way for the positive diagnosis of our present case.

The bundle of signs strongly suggesting ALCAPA on Doppler echocardiography are: right coronary artery dilation [33] [39] [40], a right coronary artery diameter to aortic annulus ratio greater than or equal to 0.14 [41], dilated coronary collateral arteries in the interventricular septum [33] [40], increased echogenicity of the anterolateral papillary muscle of the mitral valve [40] [41], predominance of systolic coronary flow at the ostium right coronary on pulsed Doppler [39], a visualization of the left coronary which does not connect to the aorta and with especially a blue flow in its lumen on color Doppler which testifies to the retrograde blood flow of the left coronary artery towards the pulmonary artery [1] [40].

Supra-sigmoid aortography (**Figure 10**) confirms that only the right coronary, which is large in caliber, originates from the aorta and that it perfuses the left coronary retrogradely [42] [43]. Opacification of the pulmonary artery is very transient [42] [43]. This technique has drawbacks; it requires an arterial puncture, does not provide good proof of the connection of the left coronary artery to the pulmonary artery and does not provide information on the level of connection on the pulmonary artery as well as the route of the left coronary artery [42] [43].

CT scan (**Figure 5**) confirms the diagnosis, showing a left coronary artery connected to the pulmonary artery [42]. It also offers extremely precise imaging of the connection level, of the path of the left coronary thanks to 3D reconstruction [42]. This information is of great help in view of corrective surgery. The

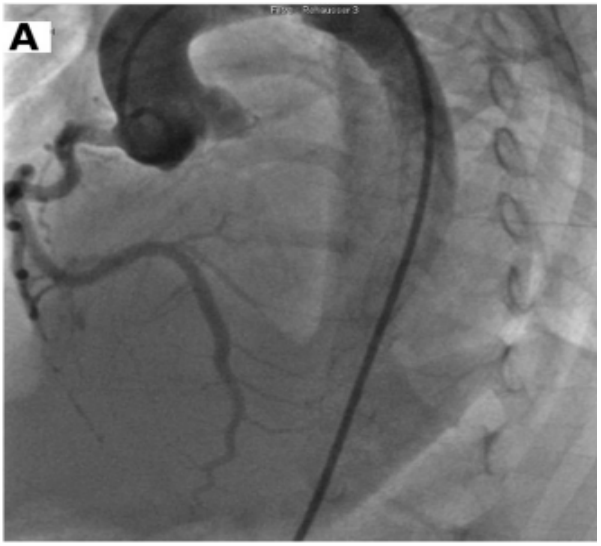

Figure 10. Supra sigmoid ALCAPA aortography image, showing that only the right coronary originates from the aorta. Excerpt from the article by Gribaa *et al.* [30].

major difficulty of the CT scan is the synchronization of the cardiac CT scan with the electrocardiogram [42]. This is particularly difficult in new borns and infants because of their high resting heart rate.

3.6. Possible Malformations Associated with ALCAPA

In the case of infantile ALCAPA, coarctation of the aorta is the most frequent associated malformation. Although of rare association, it should be systematically sought. Solomon *et al.* reported two cases in 1990 [44].

3.7. Treatment of ALCAPA

It involves medical treatment and surgical treatment. Medical treatment is symptomatic and consists of treatment of heart failure. On its own, it is associated with a low survival rate. Lethality varies between 45% and 100% [25] [26] [16] [45] [46].

Corrective surgery of the left coronary artery is the curative treatment and must urgently complete the medical treatment. In new-borns and infants, it should be done as soon as possible after diagnostic confirmation [25] [46]-[52]. In adults, surgery is urgent when there is a serious ischemic process, regardless of symptomatology, age or degree of inter coronary collateralization [47] [50]-[55]. Four techniques for surgical correction of ALCAPA have been described [8] [23]:

In 1959, Sabiston *et al.* [56] performed the first ALCAPA surgery: Through a left thoracotomy, the pericardium is opened and the left coronary is double ligated close to the pulmonary artery (**Figure 11(A)**).

In 1968, Meyer *et al.* [57] performed a second technique: Through a left thoracotomy, the pericardium was opened, the initial part of the left coronary artery

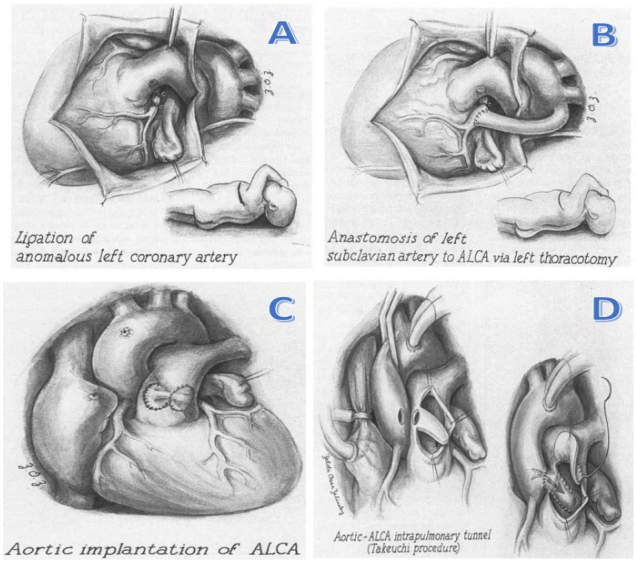

Figure 11. Illustrations of surgical techniques in the management of ALCAPA. Excerpts from the article by Backer *et al.* [23].

was ligated, and a bypass was made downstream with the left subclavian artery (**Figure 11(B)**).

In 1974, Neches *et al.* [58] tested a third technique. This is an anatomical correction of the ALCAPA: Through a median sternotomy with extracorporeal circulation, the left coronary is detached from the pulmonary artery with a button from the wall of the pulmonary artery. This coronary button is then implanted into the left lateral side of the ascending aorta (**Figure 11(C)**).

In 1979, Takeuchi *et al.* [59] tested a fourth technique. It serves as an alternative when the Neches technique is made uncertain by the fragility of the left coronary or an insufficiently long left coronary. It consists of a physiological correction of the ALCAPA: Through a median sternotomy with extracorporeal circulation and clamped aorta, the ostium of the left coronary is put in relation with the lumen of the antero left cusp of the aorta through a channel that passes into the pulmonary artery (**Figure 11(D)**).

Our patient had undergone anatomical correction (Neches Technique). This technique is the gold standard in corrective surgery for ALCAPA [21] [46] [47] [50] [60] [61] [62].

3.8. Postoperative Evolution of ALCAPA

Surgical repair of ALCAPA in symptomatic infants offers the greatest potential for recovery of left ventricular function, despite poorer initial presentation [46] [63] [64] [65] [66]. Indeed, the ejection fraction of the left ventricle on the Simson Biplane of our patient had increased from 37% to 51% in just 50 days after corrective surgery.

The Neches technique presents the least risk of progressive complications such as pulmonary supravalvular stenosis [46] [66] [67]. It is associated with a long-term survival superior to other techniques and is also associated with an

absence of re-intervention [46] [66] [67].

According to the literature, mitral valve repair or replacement is usually not performed at the time of corrective ALCAPA surgery [64] [65]. It may improve over time [64] [65]. On the other hand, if it persists and depends on its degree of severity, it may require remote mitral replacement surgery [64] [65].

4. Conclusion

ALCAPA is a rare birth defect. A distinction is made between infantile ALCAPA (very lethal in the absence of rapid surgical cure) and adult ALCAPA (ranging from asymptomatic forms to sudden death by serious ventricular arrhythmia through stable angina). Infantile ALCAPA is a reversible cause of hypokinetic ischemic dilated heart disease in infants. The most commonly used surgical technique in ALCAPA is the Neches anatomical correction (direct re-implantation of the left coronary artery at the aorta). Short- and long-term postoperative follow-up should watch for pulmonary supravalvular stenosis, aortic supravalvular stenosis, and left coronary ostium aneurysm. Finally, particular attention must be paid to mitral insufficiency to pose a possible indication for mitral valve replacement at the right time. The management of infantile ALCAPA requires the combined and complementary expertise of the pediatrician, cardiologist, radiologist, cardiothoracic surgeon and resuscitator.

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Conflicts of Interest

The authors declare no conflicts of interest regarding the publication of this paper.

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