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Case Report
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Double Aortic Arch Associated with Bilateral Pulmonary Artery Agenesis: A Fatal Neonatal Malformation and Embryological Considerations

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ABSTRACT

Introduction: congenital cardiovascular anomalies represent a major cause of neonatal mortality. Double aortic arch constitutes the most common form of complete vascular ring, while bilateral pulmonary artery agenesis is an exceptionally rare and uniformly fatal malformation. The association of these two anomalies has not been extensively documented in the literature.
Case Report: we report a unique case of a full-term male newborn who died immediately after delivery. Autopsy revealed the presence of a double aortic arch forming a complete vascular ring, associated with complete bilateral pulmonary artery agenesis and severe bilateral pulmonary hypoplasia. The cause of death was attributed to respiratory failure secondary to the absence of pulmonary perfusion.
Conclusion: this case represents an extremely rare association of two distinct cardiovascular malformations resulting from developmental abnormalities of the aortic arch system. Understanding the embryological basis of these anomalies is essential for accurate diagnosis and highlights the importance of prenatal imaging in detecting severe cardiovascular malformations.
Keywords: Double aortic arch; Pulmonary artery agenesis; Congenital heart defects; Vascular ring; Embryology.

Introduction

Congenital anomalies of the cardiovascular system constitute a significant proportion of neonatal morbidity and mortality, accounting for approximately 25-30% of all congenital malformations[1]. Among these, variations involving the aortic arch system and pulmonary arteries, although relatively uncommon, carry substantial clinical significance due to their profound impact on cardiopulmonary physiology and overall infant survival[2]. The complexity of cardiovascular embryological development, particularly during the critical period between the fourth and eighth weeks of gestation, provides numerous opportunities for developmental errors that may result in life-threatening malformations[3].

Double aortic arch represents the most frequent form of complete vascular ring, accounting for approximately 40-65% of all symptomatic vascular rings in pediatric populations[4]. This anomaly results from the persistence of both the right and left fourth aortic arches, which normally undergo asymmetric regression during fetal development. The resulting anatomical configuration creates a complete vascular ring that encircles both the trachea and esophagus, potentially leading to varying degrees of airway compression and dysphagia[5]. The clinical presentation of double aortic arch is highly variable, ranging from severe respiratory distress in the neonatal period to incidental findings in asymptomatic adults, depending on the degree of tracheal and esophageal compression[6].

Pulmonary artery agenesis (PAA) represents a distinct spectrum of cardiovascular anomalies characterized by the complete absence or severe hypoplasia of one or both main pulmonary arteries[7]. This rare condition results from abnormal development or premature regression of the sixth aortic arches during embryogenesis. The incidence of unilateral pulmonary artery agenesis (UPAA) is estimated at 1 in 200,000-300,000 live births[8], while bilateral pulmonary artery agenesis (BPAA) is extraordinarily rare and invariably fatal. In bilateral agenesis, the complete absence of blood supply to both lungs leads to severe pulmonary hypoplasia and renders survival beyond the immediate neonatal period impossible[9]. In stark contrast, unilateral pulmonary artery agenesis, while serious, can be compatible with life due to compensatory mechanisms including systemic collateral vessel development and redistribution of pulmonary blood flow[10].

The clinical spectrum of unilateral pulmonary artery agenesis demonstrates remarkable variability. Several case reports have documented patients remaining

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completely asymptomatic until adulthood, with the diagnosis made incidentally during imaging studies performed for unrelated conditions[11]. However, other patients with UPAA present with significant clinical manifestations including recurrent pulmonary infections affecting the ipsilateral lung, progressive pulmonary hypertension in the contralateral lung due to increased blood flow, hemoptysis from bronchial collateral vessels, and exercise intolerance[12,13]. The prognosis and clinical course of UPAA largely depend on the presence of associated cardiac anomalies, the extent of collateral vessel development, and the degree of pulmonary hypertension in the contralateral lung[14].

The association of double aortic arch with bilateral pulmonary artery agenesis is extraordinarily rare, with very few cases reported in the medical literature. This combination represents a catastrophic failure of normal aortic arch development, affecting both the fourth and sixth aortic arch systems simultaneously. We present this unique case to highlight the embryological mechanisms underlying these combined malformations, discuss the invariably fatal nature of bilateral pulmonary artery agenesis, and contrast this with the more favorable prognosis of unilateral pulmonary artery agenesis. Furthermore, this case emphasizes the critical importance of comprehensive prenatal cardiovascular screening and the need for improved diagnostic capabilities to detect such severe malformations before delivery[15].

Case Report

A full-term male neonate was delivered at 38 weeks of gestation following an apparently uncomplicated pregnancy. The prenatal period had been unremarkable, with routine ultrasound examinations failing to identify any structural abnormalities. The mother, a 28-year-old primigravida, had received adequate prenatal care and had no significant medical history. There was no family history of congenital heart disease or other genetic disorders. The pregnancy proceeded without maternal complications such as diabetes mellitus, hypertension, or exposure to known teratogens.

Labor and delivery occurred spontaneously at term. The infant was delivered vaginally with clear amniotic fluid and normal placental appearance. However, immediately upon delivery, the neonate demonstrated profound cyanosis and complete absence of respiratory effort despite appropriate resuscitative measures. Apgar scores were 0 at one minute and 0 at five minutes. Advanced neonatal resuscitation including endotracheal intubation, positive pressure ventilation, and cardiac compressions were initiated immediately but proved unsuccessful. The neonate was pronounced dead approximately 15 minutes after delivery. Given the unexpected nature of the infant's death and the absence of any prenatal diagnosis, the case was referred to the pathology department for complete autopsy examination to establish the underlying cause of death.

Gross autopsy examination revealed multiple severe cardiovascular malformations (Figure 1):

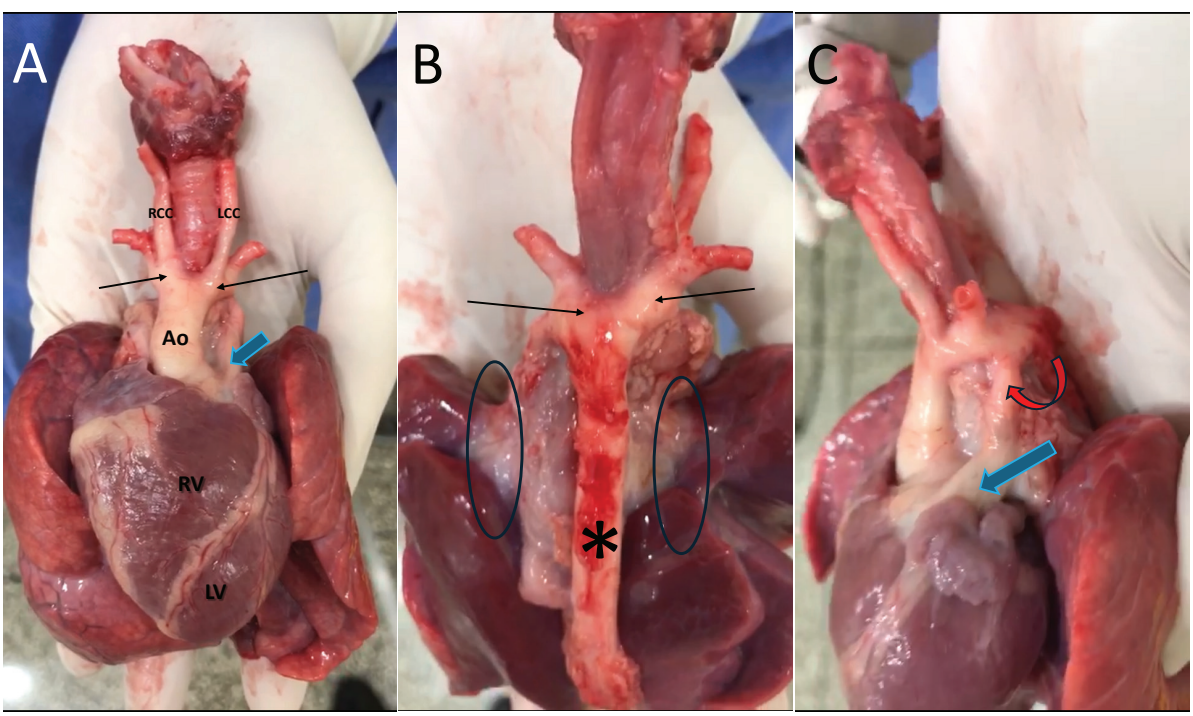

Figure 1. Anterior (A), posterior (B) and left lateral (C) views of the thoracic block removed during autopsy. In A, we identify the structures of the sternocostal surface of the heart: RV = right ventricle, LV = left ventricle, Ao = ascending aorta. Note the bilateral pulmonary hypoplasia. In B, note that the ascending aorta bifurcates into two brachiocephalic trunks (aortic arches, arrows) that join forming a complete arterial circle around the trachea and esophagus, giving rise to the descending aorta (*). The circles indicate the pulmonary hila with confirmed absence of any pulmonary arterial structures bilaterally. In C we indicate the pulmonary trunk (blue arrow) ending blindly and the ductus arteriosus of large caliber (red curved arrow) that communicates the pulmonary trunk with the left arm of the aortic ring.

The cardiovascular examination demonstrated the presence of a double aortic arch configuration. Both the right and left fourth aortic arches had persisted, forming a complete vascular ring that encircled the trachea and esophagus. The right aortic arch was dominant and larger in caliber than the left arch. Both arches ascended from a common ascending aorta and reunited posteriorly to form a single descending aorta at the level just below the tracheal bifurcation. The vascular ring was complete and compressed both the trachea and esophagus, although the degree of compression appeared insufficient to independently account for the neonatal death.

Most significantly, complete examination of the pulmonary arterial system revealed total bilateral pulmonary artery agenesis. There was complete absence of both the right and left main pulmonary arteries. The pulmonary trunk was present but ended blindly without continuation into right or left branches. No pulmonary artery branches could be identified extending to either lung. Dissection of the pulmonary hila confirmed the absence of any pulmonary arterial structures bilaterally.

Both lungs demonstrated severe hypoplasia with markedly reduced size and weight compared to expected values for gestational age. The right lung weighed 8 grams (expected: 25-30 grams) and the left lung weighed 7 grams (expected: 20-25 grams). The lungs appeared dark red and firm in consistency, with absent normal aeration. Histological examination of lung tissue revealed markedly decreased alveolar development with thickened interalveolar septa, consistent with severe pulmonary hypoplasia secondary to absent arterial perfusion throughout fetal development. Bronchial structures were present but hypoplastic.

The heart demonstrated normal four-chamber anatomy with intact atrial and ventricular septa. The cardiac valves appeared structurally normal. Both ventricles were of normal size and wall thickness. The coronary arteries arose normally from the aortic root. The foramen ovale was patent, as expected in a newborn. The ductus arteriosus was patent but connected only to the blind-ending pulmonary trunk.

No other significant congenital anomalies were identified in other organ systems. The brain, gastrointestinal tract, genitourinary system, and musculoskeletal system were grossly and microscopically normal.

The cause of death was definitively attributed to severe respiratory failure resulting from complete absence of pulmonary arterial perfusion throughout fetal development, leading to critical pulmonary hypoplasia incompatible with extrauterine life. The bilateral nature of the pulmonary artery agenesis rendered any possibility of survival impossible, even with immediate medical intervention.

Discussion

Embryological Basis of the Observed Malformations:

The cardiovascular malformations observed in this case reflect complex developmental errors occurring during the critical period of aortic arch system formation. Understanding the normal embryological sequence (Figure 2) is essential to comprehend the pathogenesis of these anomalies[16].

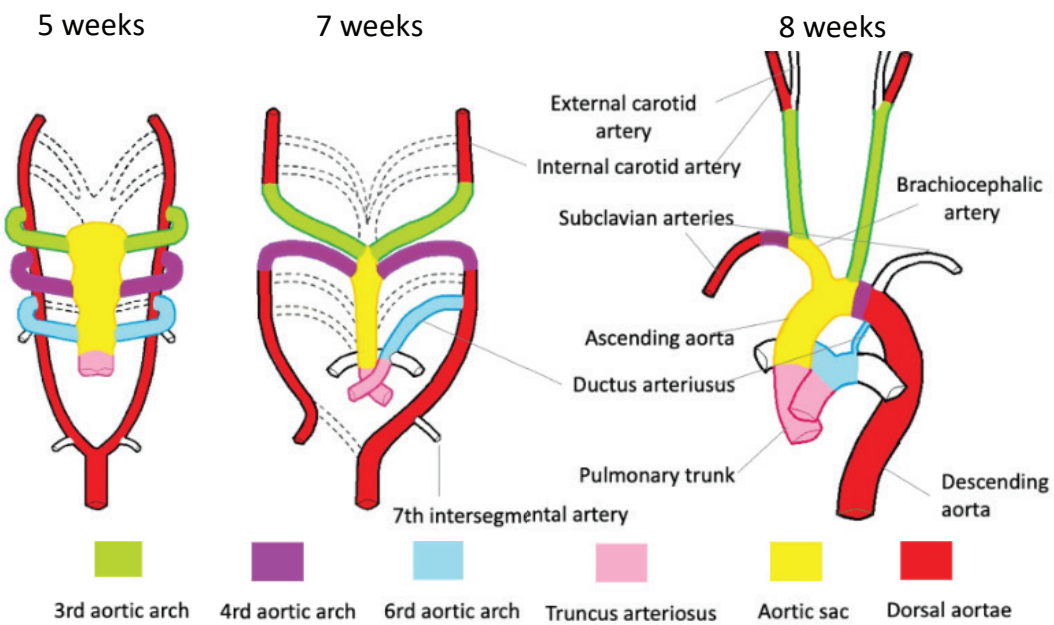

Figure 2. Scheme of the embryologic development of the branchial arches into the normal pattern of the thoracic aorta and its branches. Dashed lines represent the regressed vessels in each of the represented periods. By the 5th week of development, the six arches from the aortic sac, the 1st and 2nd arches have regressed. The truncus arteriosus is now partially divided by the conotruncal septum and will give rise to the ascending aorta and the pulmonary trunk. At 7 weeks of development, a large arterial remodeling occurs, and the 6th arch disappears on the right side and gives rise to the ductus arteriosus and the pulmonary trunk on the left side. At 8 weeks, the thoracic aorta and related large arteries have almost completed their development, being the ductus arteriosus patent at this stage. Figure modified from https://encyclopedia.pub/entry/46273

During normal human embryonic development, a series of paired aortic arches develop in the pharyngeal region between the fourth and eighth weeks of gestation[3]. Six pairs of aortic arches form sequentially, connecting the ventral aortic sac to the paired dorsal aortae. These arches undergo a precisely orchestrated pattern of persistence, modification, and regression to ultimately form the mature aortic arch and its major branches, as well as the pulmonary arterial system[17].

The double aortic arch anomaly results from abnormal persistence of both the right and left fourth aortic arches[4]. Normally, during fetal development, the right fourth aortic arch persists to form the proximal right subclavian artery, while the right dorsal aorta between the fourth arch and the seventh intersegmental artery regresses. Simultaneously, the left fourth aortic arch normally persists to form the definitive aortic arch, while the left dorsal aorta distal to the left fourth arch forms the descending aorta[18]. In double aortic arch, the normal regression of the right dorsal aorta fails to occur, resulting in persistence of both fourth arches and bilateral dorsal aortae, creating a complete vascular ring around the trachea and esophagus[5].

The sixth aortic arches normally contribute to formation of the pulmonary arteries and ductus arteriosus. The proximal portions of both sixth arches form the respective right and left pulmonary arteries. The distal portion of the right sixth arch typically regresses, while the distal portion of the left sixth arch persists as the ductus arteriosus[19]. Complete bilateral pulmonary artery agenesis results from either failure of the sixth aortic arches to develop initially or from premature complete regression of both sixth arches during early embryogenesis[7]. The precise molecular mechanisms underlying sixth arch development involve complex interactions between transcription factors, growth factors including vascular endothelial growth factor (VEGF), and signaling pathways that remain incompletely understood[20].

In the present case, the simultaneous occurrence of double aortic arch and bilateral pulmonary artery agenesis indicates a catastrophic failure affecting multiple components of the aortic arch system. The fourth arches abnormally persisted bilaterally while the sixth arches either failed to form or regressed completely and prematurely. This suggests a fundamental disturbance in the regulatory mechanisms controlling aortic arch development, possibly involving genetic factors, although no specific genetic testing was performed in this case. Recent studies have implicated various genetic mutations including those affecting TBX1, GATA6, and other transcription factors in aortic arch anomalies, though most cases remain sporadic without identified genetic causes[21].

Unilateral Pulmonary Artery Agenesis: Clinical Spectrum and Outcomes:

In marked contrast to the uniformly fatal bilateral pulmonary artery agenesis, unilateral pulmonary artery agenesis (UPAA) demonstrates a remarkably diverse clinical presentation and is compatible with survival, sometimes for many decades[11]. Several comprehensive case series and individual case reports have documented the wide spectrum of clinical manifestations associated with UPAA[10,12,13].

A subset of patients with UPAA remains completely asymptomatic throughout childhood and into adulthood, with the diagnosis made incidentally during chest radiography or computed tomography performed for unrelated indications[11]. These asymptomatic patients typically develop extensive bronchial collateral vessels that provide some degree of perfusion to the affected lung, preventing complete lung collapse and allowing partial maintenance of lung function. The contralateral normal lung undergoes compensatory enlargement and accommodates the entire cardiac output through the pulmonary circulation[23].

However, many patients with UPAA develop significant clinical symptoms[23]. Common presentations include recurrent pulmonary infections affecting the ipsilateral lung due to poor clearance of secretions and reduced local immune surveillance. Progressive dyspnea on exertion develops as pulmonary hypertension evolves in the contralateral lung, which must accommodate the entire pulmonary blood flow. Some patients experience hemoptysis from rupture of enlarged bronchial collateral vessels. Physical examination may reveal asymmetric chest expansion, and auscultation often demonstrates decreased breath sounds on the affected side[12].

Diagnostic evaluation of suspected UPAA typically involves multiple imaging modalities[10]. Chest radiography may show asymmetric lung volumes with elevation of the ipsilateral hemidiaphragm and mediastinal shift toward the affected side. Computed tomography angiography provides definitive diagnosis by demonstrating absence of the pulmonary artery on the affected side and often reveals enlarged bronchial arteries serving as collateral vessels. Ventilation-perfusion scanning shows absent perfusion to the affected lung with maintained ventilation. Cardiac catheterization can confirm the diagnosis and assess the degree of pulmonary hypertension in the contralateral lung[13].

Management strategies for UPAA vary based on patient age, symptomatology, and presence of associated cardiac anomalies[24]. In asymptomatic patients, conservative management with regular clinical and imaging surveillance is often appropriate. Symptomatic patients may require various interventions. In pediatric patients diagnosed early, surgical creation of a systemic-to-pulmonary artery

connection using a prosthetic graft can establish blood flow to the affected lung and may prevent progressive pulmonary hypoplasia and reduce the risk of pulmonary hypertension in the contralateral lung[3]. Adult patients with established pulmonary hypertension may benefit from pulmonary vasodilator medications such as phosphodiesterase-5 inhibitors or endothelin receptor antagonists[14]. Patients with severe recurrent hemoptysis may require embolization of bronchial collateral vessels[13].

Long-term prognosis for patients with UPAA is generally favorable when diagnosis is made early and appropriate management is instituted. Many patients survive into adulthood with acceptable quality of life, particularly those without associated cardiac anomalies[25]. However, long-term complications including progressive pulmonary hypertension, right heart failure, and recurrent respiratory infections can impact survival and quality of life, emphasizing the importance of ongoing specialized follow-up[14].

Contrast with Bilateral Pulmonary Artery Agenesis:

The profound difference in clinical outcomes between unilateral and bilateral pulmonary artery agenesis underscores the critical importance of pulmonary blood flow for lung development and postnatal survival. In unilateral agenesis, several compensatory mechanisms allow survival. The contralateral normal pulmonary artery accommodates the entire cardiac output, and the affected lung receives limited perfusion through collateral vessels arising from bronchial arteries and other systemic sources. While this perfusion is inadequate for normal lung growth, it prevents complete lung collapse and allows some degree of gas exchange[22].

In bilateral pulmonary artery agenesis, these compensatory mechanisms cannot develop. Both lungs are completely deprived of arterial blood supply throughout fetal development, resulting in severe bilateral pulmonary hypoplasia[9]. The lungs fail to develop adequate alveolar structures, and the pulmonary vasculature is virtually absent. At birth, when the transition from fetal to neonatal circulation occurs and the infant attempts to breathe independently, no gas exchange can occur due to the combination of structural lung hypoplasia and absence of pulmonary blood flow. This creates an insurmountable physiological barrier to survival, as there is no mechanism to oxygenate blood or eliminate carbon dioxide.

The present case exemplifies this fatal pathophysiology. Despite the presence of a structurally normal heart capable of generating adequate cardiac output, the complete absence of pulmonary arteries rendered survival impossible. Even the most aggressive resuscitative measures including mechanical ventilation and extracorporeal membrane oxygenation (ECMO) would have been futile, as there was no anatomical substrate to allow pulmonary blood flow or gas exchange.

The additional finding of double aortic arch in this case, while anatomically significant, was not the primary cause of death. While a double aortic arch can cause clinically significant tracheal compression and respiratory symptoms in surviving infants[4,5], in this case the degree of tracheal compression was moderate and would not independently account for the immediate neonatal death. The lethal outcome was entirely attributable to the bilateral pulmonary artery agenesis and consequent pulmonary hypoplasia.

Clinical Implications and Prenatal Diagnosis:

This case highlights several important clinical considerations. First, it emphasizes the critical importance of comprehensive prenatal cardiovascular screening[15]. While routine second-trimester ultrasound examination includes basic cardiac views, detailed evaluation of the great vessels and pulmonary arteries requires specialized fetal echocardiography performed by experienced operators. Improved prenatal diagnosis of severe cardiovascular malformations allows for appropriate counseling of families, opportunity for pregnancy termination where legally and ethically acceptable, and planning for optimal perinatal management.

Second, this case contributes to our understanding of the embryological relationships between different components of the aortic arch system. The simultaneous occurrence of fourth arch abnormality (double aortic arch) and sixth arch abnormality (bilateral pulmonary artery agenesis) suggests potential common pathogenic mechanisms that warrant further investigation[21]. Future research utilizing molecular genetic techniques in similar cases may elucidate specific genes or developmental pathways whose disruption leads to these combined malformations.

Finally, this case serves as an important addition to the limited literature on bilateral pulmonary artery agenesis, a malformation so rare that systematic study is challenging. Documentation and publication of such cases contributes to the medical literature and enhances our collective understanding of rare congenital cardiovascular anomalies.

Conclusion

We describe a unique and invariably fatal case of double aortic arch associated with complete bilateral pulmonary artery agenesis in a term neonate. This rare combination of malformations results from catastrophic failure of normal aortic arch development, affecting both the fourth and sixth aortic arch systems during critical periods of embryogenesis. The bilateral pulmonary artery agenesis prevented any pulmonary perfusion, leading to severe bilateral pulmonary

hypoplasia that rendered survival impossible despite immediate resuscitative efforts.

This case contrasts sharply with unilateral pulmonary artery agenesis, which, while serious, demonstrates variable clinical presentations ranging from asymptomatic incidental findings to significant morbidity requiring intervention, but is compatible with survival and often favorable long-term outcomes with appropriate management. Recognition and accurate diagnosis of these cardiovascular anomalies require thorough imaging evaluation and comprehensive understanding of cardiovascular embryology, which remain essential for appropriate clinical management and family counseling.

The extreme rarity of this combination of malformations underscores the need for improved prenatal cardiovascular screening capabilities and continued documentation of unusual cases to enhance our understanding of congenital heart disease. Future research should focus on elucidating the molecular and genetic mechanisms underlying aortic arch development to potentially identify at-risk pregnancies and develop preventive strategies.

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Ethics Statement

The authors state that every effort was made to follow all local and international ethical guidelines and laws that pertain to the use of human cadaveric donors in anatomical research (Iwanaga *et al*., 2022)[26].

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Mini Curriculum and Author's Contribution

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