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Prenatal diagnosis and postnatal outcomes of absent pulmonary valve syndrome: A case series with genetic and hemodynamic insights

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ABSTRACT

Objectives: This study therefore aims to determine the perinatal prognosis and delineate the key risk factors associated with outcomes in fetuses with a prenatal diagnosis of absence of pulmonary valve syndrome (APVS), with particular emphasis on Doppler ultrasound parameters, the presence of extracardiac anomalies, and comprehensive genetic findings - including rare monogenic mutations - as significant contributors to the observed perinatal course.

Methods: This retrospective study included eight fetuses diagnosed with absent pulmonary valve syndrome (APVS) between 2020 and 2024 at a tertiary perinatology referral center. One patient with major extracardiac anomalies was electively terminated and excluded from the outcome analysis. For the remaining seven fetuses, detailed fetal echocardiographic assessments—including cardiac anatomy and Doppler hemodynamic parameters - were evaluated alongside genetic testing results (prenatal and/or postnatal), associated extracardiac anomalies, and postnatal clinical and surgical outcomes.

Results: Among eight fetuses prenatally diagnosed with APVS, one case was electively terminated due to major extracardiac anomalies and excluded from further analysis. All of the remaining seven cases resulted in live births. Four neonates underwent surgical intervention, three of whom survived postoperatively, yielding a surgical survival rate of 75%. Two fetuses that developed hydrops fetalis died in the early postnatal period before surgery could be performed. The overall perinatal mortality rate was 57.1%. Clinically significant genetic anomalies, including trisomy 21, 22q11.2 deletion, and a novel ABAT gene mutation detected via prenatal whole-exome sequencing, were identified in three patients (42.9%). Nonsurvivors were more likely to present with an absent ductus arteriosus and severely dilated pulmonary arteries.

Conclusions: Our study highlights that prognosis is more strongly influenced by prenatal hemodynamic markers - such as pulmonary artery velocities, ductus arteriosus status, and hydrops - than by anatomic subtype. The identification of both common chromosomal anomalies and novel ABAT gene mutations underscores the value of comprehensive genetic evaluation.

Keywords: Absent pulmonary valve syndrome, Fallot tetralogy, fetal echocardiography, ABAT gene mutation, hydrops fetalis

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Absent pulmonary valve syndrome (APVS) is a rare anomaly constituting approximately 0.2–0.4% of congenital heart diseases, and its incidence in live births is reported to be 1 in 20,000–33,000 [1]. Although it is most commonly associated with tetralogy of Fallot (TOF), it may also occur with other complex cardiac anomalies or rarely in isolation [2]. The distinctive features of the disease are the rudimentary or complete absence of pulmonary valve leaflets. This leads to free pulmonary regurgitation and various degrees of pulmonary stenosis. As a result, marked pulmonary artery dilatation may cause airway obstruction, polyhydramnios, hydrops fetalis and high perinatal mortality by creating pressure on the tracheobronchial system [2, 3].

Prenatal diagnosis of APVS is critically important in terms of family counseling, risk stratification and postnatal management planning. Although the diagnosis is usually made in the second trimester, earlier diagnosis has become possible with the development of high-resolution ultrasound and fetal echocardiography technologies [3]. Although surgical and intensive care practices have improved in the postnatal period, the prognosis of this syndrome is still poor, and high mortality rates have been reported [4].

A strong association has been found between APVS and genetic disorders, including 22q11.2 deletion (DiGeorge syndrome) and trisomy 21. Therefore, recommending genetic testing in the prenatal period is important both diagnostically and prognostically. Detailed fetal evaluation is not only a diagnostic tool but also a prognostic tool [5].

This study aimed to evaluate the clinical features, associated cardiac and extracardiac anomalies, genetic findings and postnatal outcomes of fetuses prenatally diagnosed with APVS in a tertiary referral center. Absent pulmonary valve syndrome is a very rare disease and has been reported in the literature, usually as small series or case reports [6]. In this context, with this retrospective analysis, we aimed to contribute to the knowledge on APVS and to draw attention to the effect of systematic prenatal evaluation on perinatal prognosis.

METHODS

This retrospective case series study was conducted in a tertiary referral hospital in Istanbul, Turkey, using the joint databases of the Perinatology and Pediatric Cardiology clinics. The study included fetuses prenatally diagnosed with absent pulmonary valve syndrome (APVS) between January 2020 and June 2024.

The patients included in the study were selected from those diagnosed prenatally with APVS by an experienced perinatologist via fetal echocardiography and confirmed postnatally by a pediatric cardiologist. The inclusion criteria were as follows: (i) complete ultrasonography and fetal echocardiography data and (ii) postnatal follow-up results on file. Patients with uncertain diagnoses or missing data were excluded. All the data from the hospital information management system were retrospectively reviewed. Demographic information, including maternal age, gravidity, parity, body mass index, gestational week at diagnosis, amniotic fluid volume, Doppler parameters, associated anomalies, and postnatal outcomes, was recorded. Echocardiographic examinations included measurements of the main pulmonary artery (MPA), right pulmonary artery (RPA), and left pulmonary artery (LPA) diameters, peak systolic velocity (PSV), pulmonary regurgitation, and stenosis findings. Associated cardiac anomalies (e.g., TOF, double outlet right ventricle [DORV], and arch anomalies) were recorded. Fetal biometric measurements and Doppler examinations were also performed.

In the postnatal period, birth weight, 5-minute Apgar scores, the need for intensive care, the need for mechanical ventilation, and surgical interventions were analyzed; postoperative survival and mortality outcomes were followed from birth to discharge or death.

All ultrasonographic and Doppler evaluations were performed jointly by the same experienced perinatologist and pediatric cardiologist. A Fujifilm Arietta 850 (Fujifilm Healthcare, Tokyo, Japan) ultrasonography system and a convex abdominal transducer (LISENDO™ C251) were used for imaging. Fetal biometry (biparietal diameter [BPD], head circumference [HC], abdominal circumference [AC], femur length [FL]) was performed as part of routine evaluation, and estimated fetal weight (EFW) was calculated via the Hadlock formula. The amniotic fluid index (AFI) was measured, and a detailed fetal anatomical scan was performed according to the International Society of Ultrasound in Obstetrics and Gynecology (ISUOG) guidelines.

All cardiac assessments were performed jointly by two separate experts experienced in perinatology and pediatric cardiology. The following basic cardiac sections were systematically obtained and analyzed:

(1) Four-chamber view: Right ventricular hypertrophy and ventricular septal defects (VSDs) were investigated.

(2) Right ventricular outflow tract (RVOT): Failure to visualize the pulmonary valve and aneurysmatic dilatation of the pulmonary artery were evaluated.

(3) Three-vessel trachea (3VT) section: Posterior compression of the trachea by dilated pulmonary arteries was investigated.

(4) Color Doppler: A "to-and-fro" flow pattern was observed at the pulmonary artery level.

The main, right and left pulmonary artery diameters were measured, and z scores were calculated according to gestational week. Pulmonary artery PSV was recorded, and pulmonary flow hemodynamics were evaluated.

Invasive prenatal genetic diagnosis was recommended for all patients. The methods used included conventional karyotyping after amniocentesis or cordocentesis, Fluorescence in situ hybridization (FISH) analysis for 22q11.2 deletion and chromosomal microARRAY (CMA). Next-generation sequencing (NGS)-based analysis of a large panel of genes, including the 4-aminobutyrate aminotransferase (ABAT) gene, was performed in selected cases.

The study was conducted in accordance with the principles of the Declaration of Helsinki, and ethical approval was obtained from the Clinical Research Ethics Committee of the Başakşehir Çam and Sakura City Hospital 1 (Decision No: 2024-63). Written informed consent was obtained from the parents of all patients to use the data for scientific purposes.

Statistical Analysis

Statistical analyses were performed via IBM SPSS Statistics v26.0 (IBM Corp, Armonk, NY, USA). Continuous variables are presented as the means ± standard deviations or medians (min–max); categorical variables are presented as frequencies and percentages (%). Owing to the limited sample size, descriptive analyses were performed, and comparative statistical tests (e.g., P values) were not included.

RESULTS

In this retrospective series, a total of 8 pregnant women with a prenatal diagnosis of APVS were evaluated. One

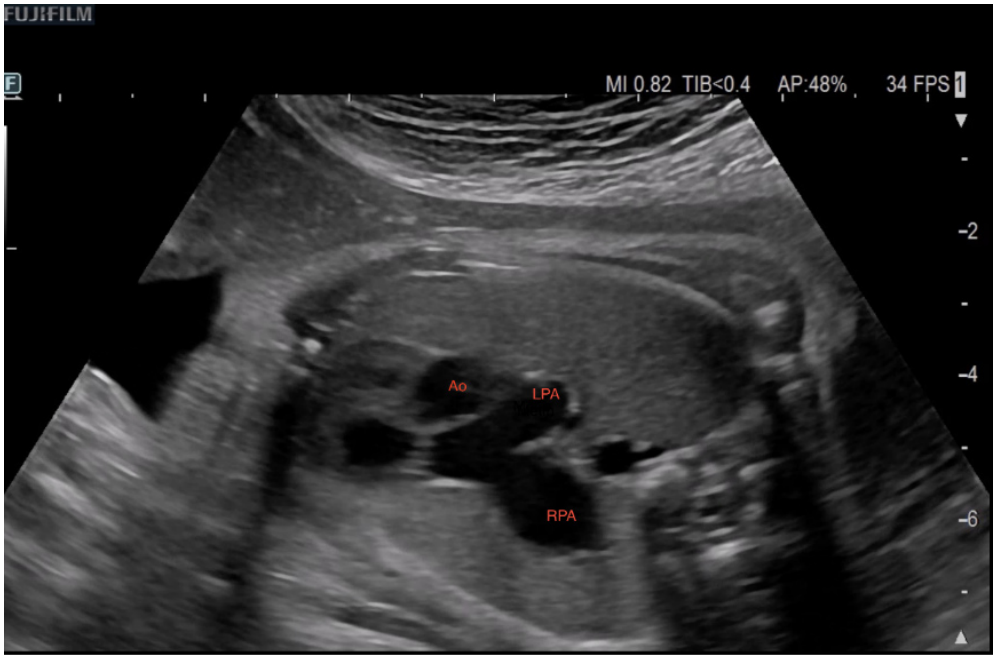

Fig. 1. Dilated right and left pulmonary arteries (RPA-LPA) and the aorta (Ao) in a fetus with pulmonary valve absence (Ao=Aorta, RPA=Right Pulmonary Artery, LPA=Left Pulmonary Artery).

of these cases was terminated in the second trimester because of associated extracardiac anomalies (acrania and omphalocele). Therefore, the clinical findings and outcome analyses were based on 7 live births.

The mean age of the pregnant women was 28.25±6.64 years, and the age range was 21-39 years. The mean body mass index (BMI) was 30.11±4.75 kg/m², and the values varied between 22.7 and 36.3. The mean gravida was 2.87±1.88, the mean parity was 1.12±0.99 (range: 0-2), and the mean abortion rate was 0.75±1.38 (range: 0-4).

With respect to the mode of delivery, 6 (85.7%) patients had cesarean section, and 1 (14.3%) patient had normal delivery. When the fetal sex distribution was analyzed, 5 (71.4%) patients were male, and 2 (28.6%) patients were female. Fetal birth weights ranged between 1200 and 3150 grams, with a mean of 2178.33±780.13 grams. When the gestational weeks of the prenatally diagnosed cases were analyzed, the earliest gestational week at the time of diagnosis was recorded as 18+2 (128 days), and the latest was recorded as 28+5 (201 days). The mean gestational age at presentation was 24+5 weeks (173 days) ±3.5 weeks.

When the postnatal Apgar scores were examined, the 1-minute Apgar values ranged between a minimum of 1 and a maximum of 8, with a mean of 4.00±2.37. The 5-minute Apgar scores ranged from a minimum of 5 to a maximum of 8, with a mean of 5.71±1.38. Postnatal oxygen saturation values ranged from a minimum of 70% to a maximum of 88%, with a median of 83%. In two (28.6%) patients, postnatal respiratory distress and oxygen desaturation necessitated endotracheal intubation. Amniotic fluid assessment revealed polyhydramnios in four (57.1%) patients, normal fluid volume in two (28.6%) patients, and oligohydramnios in one (14.3%) patient.

When the gestational weeks were analyzed, the earliest gestational week was 30+3 (day 213), and the latest gestational week was 38+5 (day 271), after one patient who was excluded from the study because of termination. The mean number of gestational weeks was 31+2 (219th day), and the median number of gestational weeks was 32+1 (225th day). The standard deviation was ±6.45 weeks (~45.2 days).

In one patient with prenatal acrania and omphalocele, the pregnancy was terminated in the second trimester, and this patient was excluded from the study. In the evaluation of the remaining seven live fetuses, extracardiac structural anomalies were detected in two (28.6%) cases. One of these patients had esophageal atresia and skeletal dysplasia, and the other had bilateral pyelectasis and agenesis of the corpus callosum. The other five (71.4%) fetuses had no extracardiac structural abnormalities.

In terms of cardiac anatomy, 6 (85.7%) patients lacked a pulmonary valve with TOF, and 1 patient (14.3%) lacked a pulmonary valve with a DORV. A right aortic arch anomaly was found in two patients (33.3%) with TOF and left pulmonary artery hypoplasia, and direct origin of the left pulmonary artery from the aortic arch (AOLPA) was observed in one patient (16.7%).

Pulmonary artery diameters were analyzed as follows:

(1) MPA diameter ranged from 6.5-10.3 mm, with a mean of 8.53±1.38 mm.

(2) LPA diameter ranged from 1.5-10.6 mm, with a mean of 7.65±3.24 mm. AOLPA was present in one patient with an LPA diameter of 1.5 mm.

(3) RPA diameter ranged from 6.8-12.7 mm, with a mean of 8.6±2.08 mm.

Fig. 1 shows the markedly dilated right and left pulmonary arteries along the aorta in a thoracic axial three-vessel view of a fetus diagnosed with absent pulmonary valve syndrome. This image supports the typical prenatal echocardiographic findings associated with the condition.

In fetuses with pulmonary regurgitation, PSV values ranged between 127 and 210 cm/sec. The mean PSV was 178.8±36.1 cm/sec, and the median value was 200.0 cm/sec. In patients with pulmonary stenosis, the PSV ranged between 120.0 and 260.0 cm/sec, with a mean of 184.0±50.8 cm/sec and a median of 187.0 cm/sec.

Postnatal cardiac evaluation revealed TOF in 6 of the 7 patients (85.7%), and 1 patient (14.3%) had a DORV associated with this condition. Pulmonary regurgitation was observed in all patients (100%); 3 (42.9%) had free regurgitation, and 4 (57.1%) had moderate to significant regurgitation. Pulmonary stenosis was present in 6 patients (85.7%), of whom 5 (71.4%) had valvular stenosis of moderate to severe severity.

Among the postnatal echocardiography controls, patency at the atrial level was found in 5 cases (71.4%); 3 (42.9%) of these cases were defined as PFO, and 2 (28.6%) were defined as wide secondum

type ASDs. Patent ductus arteriosus (PDA) was observed in 1 patient (14.3%). The right aortic arch was confirmed postnatally in 2 patients (28.6%).

When the clinical outcomes of 7 live-born APVS patients were evaluated at the postnatal follow-up, 4 patients (57.1%) underwent surgery. Three of these patients (42.9%) survived postoperatively- one followed up to 19 months, one to 6 months, and one in the early postoperative period. One (14.3%) patient died in the second postoperative month during intensive care unit follow-up. Two (28.6%) patients with severe cardiac failure and hydrops fetalis died in the early neonatal period before surgery. Another patient (14.3%) died on postnatal day 40 before reaching surgery.

Fig. 2 provides a visual summary of the perinatal course and postnatal outcomes of the seven live-born cases, including surgical interventions, survival status, and causes of mortality. This diagram enhances the understanding of the clinical prognosis of APVS patients.

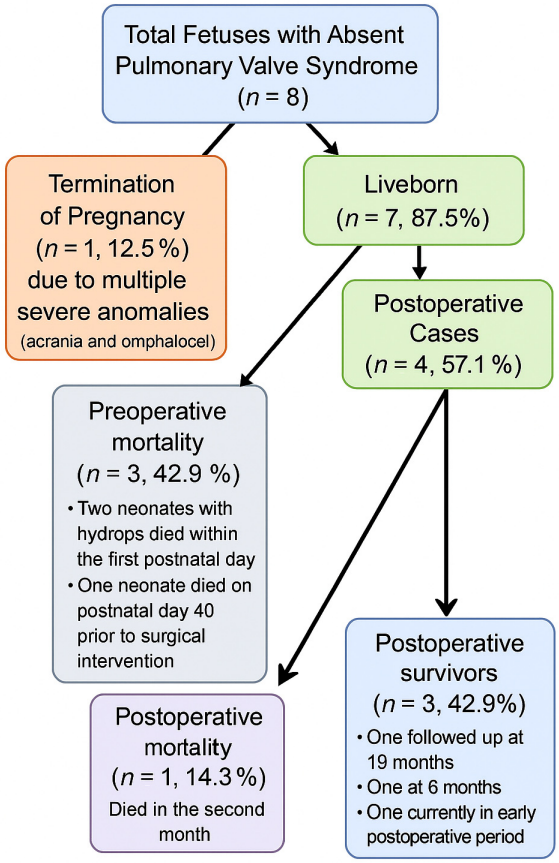

Fig. 2. Clinical outcomes of fetuses with APVS: From rermination to postoperative survival (APVS=Absent Pulmonary Valve Syndrome).

In the series of 7 cases, genetic analysis was performed prenatally in three cases and postnatally in two cases. One of the three patients tested prenatally was found to have ABAT gene mutations, and the other two were reported to be normal. Among the two postnatal cases, one was compatible with trisomy 21 (Down syndrome), and the other had a 22q11.2 deletion (DiGeorge syndrome). Accordingly, genetic anomalies were found in a total of three cases (42.8%) in the whole series: (1) One case (14.3%) of ABAT gene mutation (by prenatal WES); (2) 1 patient (14.3%) with trisomy 21; and (3) One patient (14.3%) had a 22q11.2 deletion.

In two of the remaining four cases, prenatal tests were reported as normal, and genetic testing was not performed in two cases.

DISCUSSION

Absent pulmonary valve syndrome (APVS) is a rare congenital heart defect characterized by high perinatal mortality and complex structural anomalies that pose significant challenges for surgical repair, necessitating accurate prenatal diagnosis, detailed genetic evaluation, and coordinated postnatal multidisciplinary care [6]. Despite its clinical importance, large-scale case series on APVS remain scarce in the literature, with most information derived from isolated case reports and limited retrospective analyses [7]. In this context, our study provides a comprehensive evaluation of seven live-born fetuses among eight prenatally diagnosed APVS cases, focusing on the timing of diagnosis, associated cardiac and extracardiac anomalies, Doppler findings, genetic results, and postnatal surgical outcomes.

In our series, 87.5% of fetuses had tetralogy of Fallot, 28.6% had extracardiac anomalies, 57.1% experienced perinatal mortality, and 42.9% presented with genetic abnormalities. The presence of the ductus arteriosus and hydrops fetalis are key predictors of postnatal survival. These findings highlight the prognostic value of early diagnosis and stratification, and our comparative analysis aims to deepen the understanding of this rare and complex condition.

One of the key prenatal indicators of APVS is the "to-and-fro" flow pattern observed via color Doppler ultrasonography, which is characterized by antegrade

systolic flow into the pulmonary arteries and diastolic backflow into the right ventricle due to the absence or rudimentary pulmonary valve leaflets. This bidirectional mosaic flow at pulmonary artery outflow supports the diagnosis and aids in distinguishing APVS from other congenital heart defects, such as TOF and pulmonary atresia [8, 9]. In our series, this pattern was observed in all patients and served as the principal diagnostic marker. It was also associated with severe valvular regurgitation and pulmonary artery dilatation. Notably, these Doppler findings may predict postnatal complications, including hemodynamic overload and tracheobronchial compression. Thus, careful evaluation during fetal echocardiography is essential for accurate differential diagnosis of outflow tract anomalies.

Prenatal diagnosis of APVS typically occurs between 18-24 weeks of gestation, and the mean gestational age at diagnosis in our series was 24+5 weeks, which is consistent with the 23-week average reported in the literature [10]. First-trimester diagnoses are rare and generally associated with poor prognosis [11]. The absence of such early cases in our series may reflect both the rarity of early-onset forms and the imaging limitations during the first trimester.

Hydrops fetalis was identified antenatally in two fetuses, both of which were born alive but died in the early postnatal period, resulting in a hydrops-related perinatal mortality rate of 100%, which is in line with previous reports [8].

In one of these cases, postnatal evaluation revealed a patent ductus arteriosus (PDA), suggesting its contribution to perinatal decompensation via increased right ventricular volume load and worsened pulmonary regurgitation. These findings support the notion that PDA may exacerbate the hemodynamic burden in select cases. The interplay between PDA-related volume overload, pulmonary regurgitation, and right ventricular dilatation may lead to pulmonary vascular congestion and hydrops, increasing the risk of mortality. Therefore, the presence of hydrops and their hemodynamic contributors, including PDA, should be carefully assessed in the prenatal evaluation of APVS.

Careful assessment of the ductus arteriosus via fetal echocardiography is essential, as its presence has been associated with increased perinatal mortality, warranting appropriate genetic and prognostic counseling. Conversely, some studies suggest that the absence of PDA may contribute to hemodynamic stability and a more favorable prognosis in APVS patients [10, 11]. Thus, PDA should be regarded not only as an anatomical variant but also as a dynamic factor influencing clinical outcomes.

Pulmonary artery dilatation has been linked to polyhydramnios, hydrops fetalis, and postnatal respiratory failure due to tracheobronchial compression [12, 13]. In our series, the APA (8.53±1.38 mm), LPA (7.65±3.24 mm), and RPA (8.6±2.08 mm) diameters exceeded the gestational age-specific reference values, indicating a risk for airway obstruction. These findings were corroborated by indirect signs such as polyhydramnios and mediastinal shift during the prenatal period.

Therefore, pulmonary artery diameters in APVSs should be evaluated not only for diagnostic purposes but also for their prognostic implications in predicting postnatal respiratory morbidity. Quantitative assessment during fetal echocardiography provides valuable guidance for prenatal counseling and postnatal management [14].

The extracardiac anomalies observed in our series involved multiple systems, including fetal growth restriction (FGR), esophageal atresia, skeletal dysplasia, acrania, omphalocele, bilateral pyelectasis, and agenesis of the corpus callosum. These findings suggest that APVS may often present as part of syndromic or monogenic conditions rather than as an isolated cardiac defect. The most commonly affected systems in the literature are the central nervous, gastrointestinal, and genitourinary systems [14, 15].

The incidence of extracardiac anomalies in our study was 28.6%, which aligns with the reported range of 21.6–33.8% in previous studies [16]. This rate may reflect both the extent of systematic prenatal screening and the thoroughness of individual case evaluations.

Among the seven cases in our series, genetic analysis was performed in five—three prenatally and two postnatally. Clinically significant genetic anomalies were identified in three patients (42.9%): an ABAT gene mutation was detected prenatally, whereas postnatal analyses revealed trisomy 21 (Down syndrome) and 22q11.2 microdeletion (DiGeorge syndrome). Trisomy 21 and 22q11.2 deletions are well-documented syndromic associations with APVS, whereas the ABAT mutation represents a novel finding, potentially implicating a new genetic etiology.

The genetic diagnosis rate in our cohort (42.9%)

exceeded the 39.3% reported in the meta-analysis by Recker *et al*. [1, 7], possibly due to our small sample size and the application of advanced, phenotype-guided analyses such as prenatal whole exome sequencing (WES). Notably, the ABAT mutation was identified via prenatal WES in a fetus presenting with FGR, esophageal atresia, and limb shortness, highlighting the diagnostic value of phenotype-driven testing. Therefore, in addition to conventional chromosomal anomalies, specific monogenic syndromes should be considered in all fetuses with APVS, as genetic abnormalities may impact both cardiac and extracardiac development as well as perinatal prognosis [15, 16]. Stratified, phenotype-oriented screening approaches may thus enhance prenatal counseling and postnatal management.

Over the past 10-15 years, advancements in cardiac surgery, neonatal intensive care, and multidisciplinary management have significantly improved APVS outcomes. Reports from high-volume centers demonstrate high survival among patients undergoing surgery [16-18]. For example, the Toronto SickKids group reported 5- and 10-year survival rates of 93% and 87%, respectively, in 62 surgical patients - most of whom required prolonged neonatal ventilation (mean: 36 days) [19]. Similarly, data from CHOP indicate notable improvements in postnatal survival compared with earlier decades [20].

In our cohort, one of the eight cases was excluded due to pregnancy termination for severe extracardiac anomalies. Among the remaining seven live-born individuals, four underwent surgery; three survived, and one died in the second postnatal month, yielding a surgical survival rate of 75% and a mortality rate of 25%. The overall perinatal mortality rate among live births was 57.1% (4/7). These outcomes approach the upper limit of the 50-64% survival range reported in the literature and may reflect the benefits of early diagnosis, proactive postnatal evaluation, and multidisciplinary care at our center.

The pulmonary artery PSV is a key Doppler parameter for evaluating pulmonary outflow in APVSs. In healthy fetuses, second- and third-trimester values typically range from 120-160 cm/sec; values >200 cm/sec may indicate severe regurgitation and increased volume load [15-17]. In our series, the mean PSV was 178.7 cm/sec (range: 140-260). The highest PSV (260 cm/sec) occurred in a patient who died postnatally due to respiratory failure and tracheobronchial compression. Survivors had a PSV <200 cm/sec and underwent timely surgery, suggesting that a higher PSV may predict adverse outcomes.

These findings highlight the prognostic value of Doppler-based hemodynamic assessment. Although fetal MRI was not performed, indirect echocardiographic signs - such as marked pulmonary artery dilatation, polyhydramnios, and narrowed tracheal diameter on the 3VT view - supported the presence of airway compromise. Fetal MRI has been reported as a complementary tool for assessing tracheal compression and predicting postnatal respiratory morbidity in APVSs [12]. However, current evidence does not support its routine use. Instead, MRI should be selectively considered in patients with severe pulmonary artery dilatation, mediastinal shift, or marked polyhydramnios.

Most APVS cases exhibit TOF morphology, with reported rates of 84-93% [9, 10, 15]. Our series aligns with this trend but also includes a rare variant involving the DORV, characterized by both great arteries originating from the right ventricle, a large perimembranous VSD, and complete pulmonary valve aplasia. This highlights that APVSs may present with a broader morphologic spectrum than previously recognized, underscoring the need for careful differential diagnosis. By reporting both typical and rare anatomical variants, our study contributes novel insights to the literature.

CONCLUSION

This study represents one of the few prenatally diagnosed case series of APVS, highlighting its morphological and genetic heterogeneity and its association with high perinatal mortality. Among the seven live-born patients, genetic anomalies - including Down syndrome, 22q11.2 deletion, and a novel ABAT gene mutation - were identified in 42.9% of the patients, emphasizing the syndromic nature of APVS. A surgical survival rate of 75% was achieved among operated patients, while all patients with hydrops fetalis died before surgery, underscoring the prognostic severity of this complication. These findings confirm the importance of detailed fetal echocardiography, genetic evaluation, and hemodynamic monitoring in prenatal

care. Individualized, multidisciplinary management plans based on early risk stratification may improve both postnatal outcomes and the quality of prenatal counseling. Larger, prospective studies are needed to refine the diagnostic pathways and therapeutic strategies for APVS.

Ethics Approval and Consent to Participate

This study was approved by the Başakşehir Çam and Sakura City Hospital Clinical Research Ethics Committee (Decision No: 2024-63; date: 06.05.2024). All procedures were conducted in accordance with the ethical standards of the institutional and national research committee and with the 1964 Helsinki Declaration and its later amendments. Written informed consent was obtained from the parents of all patients to use the data for scientific purposes.

Data Availability

All data generated or analyzed during this study are included in this published article. Additional information and supporting data related to the findings of this study are available from the corresponding author upon reasonable request.

Authors' Contribution

Study Conception: TA; Study Design: VAT; Supervision: TA, VA; Funding: N/A; Materials: TA, İÖ; Data Collection and/or Processing: TA; Statistical Analysis and/or Data Interpretation: TA, İÖ; Literature Review: TA; Manuscript Preparation: TA; and Critical Review: TA.

Conflict of Interest

The author(s) disclosed no conflict of interest during the preparation or publication of this manuscript.

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Generative Artificial Intelligence Statement

The author(s) declare that no artificial intelligence-based tools or applications were used during the preparation process of this manuscript. The all content of the study was produced by the author(s) in accordance with scientific research methods and academic ethical principles.

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