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Case report

Prenatal diagnosis of aortopulmonary window – case report and review of the literature

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Abstract

Aortopulmonary window (APW) is a rare congenital heart malformation, which is characterized by a septal defect between the aorta and the pulmonary trunk. This defect can be isolated or associated with other cardiac anomalies in 25-50% of cases. There is no possibility of spontaneous closure of the defect, so early surgical treatment is essential to prevent complications. APW is a well described malformation in paediatric cardiology and cardiac surgery literature, but it is far less described in publications for obstetricians and perinatologists. The presented patient, after in vitro fertilization, had no abnormal finding in the first trimester observed. Subsequently, cardiomegaly occurred at the 32nd week, and finally the fetus was diagnosed with APW in the tertiary centre for fetal cardiology at the 34th week of gestation. Prenatal diagnosis of this condition sped up the postnatal treatment.

Key words: aortopulmonary window, prenatal congenital heart defect, 3rd trimester.

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Introduction

Aortopulmonary window (APW) is a very uncommon congenital heart defect, which is characterized by connection between the pulmonary trunk and the ascending aorta above normally formed semilunar valves [1, 2]. This abnormality can be isolated (simple APW), but in one fourth to one half of all cases it is associated with additional cardiovascular defects (complex APW) [3, 4]. APW is a well described malformation in paediatric cardiology and cardiac surgery literature, but it is far less described in publications for obstetricians and perinatologists.

We present a case of prenatally diagnosed APW in the third trimester of pregnancy after in vitro fertilization; such an association has never been reported previously (to the best of our knowledge).

Case study

A 33-year-old woman (gravida 2 para 0) was referred to the Prenatal Cardiology Centre for targeted fetal echocardiography at 34 weeks of gestation due to suspicion of congenital heart disease (CHD).

She had a miscarriage at early gestation 2 years previously. This pregnancy was conceived after an in vitro fertilization procedure (intracytoplasmic sperm injection – ICSI). During the first trimester and mid trimester ultrasound scans, no abnormalities were detected (present nasal bone – NB, nuchal translucency – NT 1.4 mm, no tricuspid valve regurgitation, normal ductus venosus flow, normal maternal blood biochemistry in 1st trimester, normal ultrasound scan at 20 weeks of gestation). The next ultrasound exam at 32 weeks of gestation revealed cardiomegaly, and this was the reason for the referral to the tertiary fetal cardiology centre.

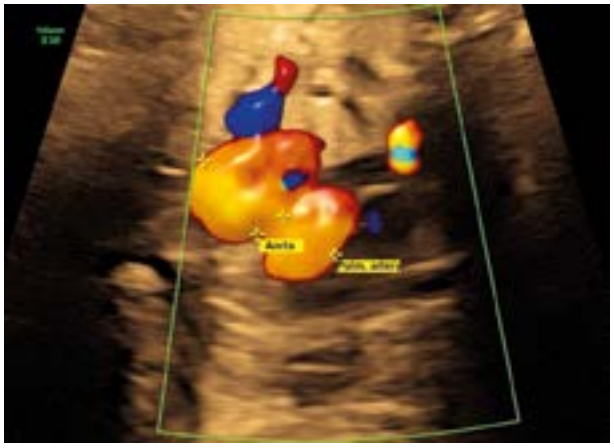

Figure 1. The 3-vessel view

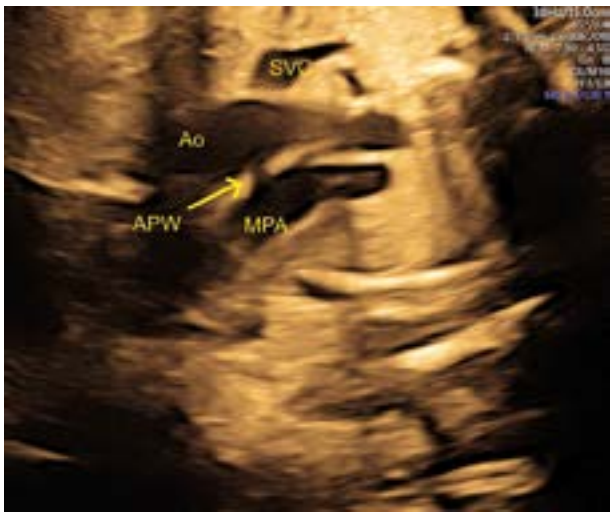

Figure 2. The defect between main pulmonary artery and the ascending aorta

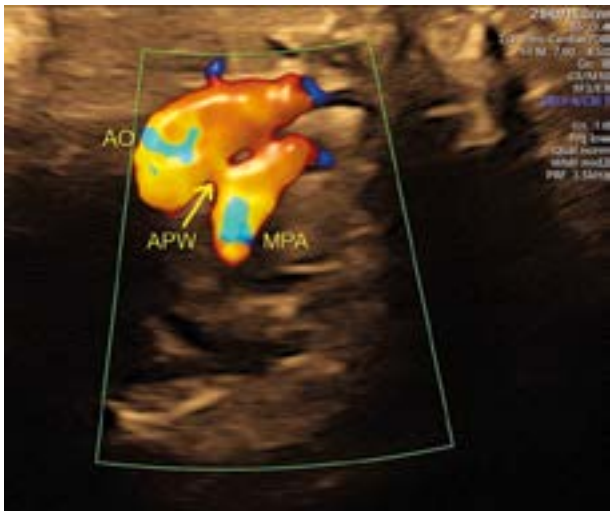

Figure 3. Colour flow mapping

Targeted fetal echocardiography at 34 weeks of gestation showed levocardia (normally positioned heart), and atrioventricular and ventriculoarterial concordant connections. Contractility of both ventricles was normal, heart area/chest area (HA/CA) ratio was 0.42, there was a 3 mm rim of pericardial effusion, and tricuspid regurgitation 2 m/s. In the 3-vessel view the aorta was 10 mm (Z-score 3.21) and the pulmonary artery was 7 mm (Z-score 0,68) (Figure 1). At the level of the 3-vessel view, a 3 mm defect was present between the main pulmonary artery and the ascending aorta above the semilunar valve (Figure 2). Colour-flow mapping confirmed unidirectional right-to-left shunting (Figure 3).

The cardiovascular profile score was assessed as 7/10 (minus 1 for HA/CA, minus 1 for pericardial effusion, and minus 1 for tricuspid valve regurgitation).

The next fetal echocardiography was performed at 38 weeks of gestation. At that time pericardial effusion was not observed and no additional abnormality was detected. The pregnant woman was admitted for delivery to our tertiary hospital (obstetrical and complex paediatric care including the Cardiac Paediatric Surgery Department).

Three days after this exam (at 38 weeks of gestation), due to abnormal CTG tracing, the male baby was delivered by caesarean section with a neonatal birth weight of 2890 g in good clinical condition (Apgar score 9/9). Postnatal echocardiography confirmed 3.8-4 mm aortopulmonary septum defect with bidirectional blood flow between the pulmonary artery and ascending aorta. Both ventricles were enlarged but with normal contractility. There was also an 8 mm foramen ovale/atrial septal defect with left-to-right blood flow.

During next couple days the condition of the neonate worsened with signs of heart failure, and multidisciplinary cardiac team decided to perform cardiosurgery on 12th day of postnatal life with a cardiovascular bypass. During the surgery, the pulmonary trunk was separated from the aorta. During this opened cardiac surgery, it was revealed that the right coronary artery arose from the pulmonary trunk, so the right coronary artery was separated from the pulmonary trunk and grafted onto the aorta.

The postoperative course was uneventful. In the control echocardiography, normal contractility of both ventricles was seen and the baby was discharged from hospital 3 weeks after the surgery.

Discussion

Aortopulmonary window, also called aortopulmonary septal defect, is a rare congenital heart lesion caused by defective development of conotruncal ridges [3]. This defect develops embryologically when there is incomplete septation of the great arteries, and it can be traced to the fifth week of intrauterine development [5]. It accounts for 0.2% to 0.6% of all CHDs and

is the rarest of congenital septal defects in the paediatric population [6]. In the Polish National Registry for Fetal Cardiac Malformations (www.orpkp.pl) in the years 2004-2018 there were 9502 fetuses with heart defects, and APW was suspected only in 3 cases (including this case). This condition has several classifications, the most widely accepted by cardiac surgeons is the classification by Richardson [2]. According to this classification, APW can be classified into 3 types: type I – proximal, the defect occurs in the proximal part of the aortopulmonary septum; type II – distal, where the defect occurs in the distal part of the aortopulmonary septum; type III – a defect of all the aortopulmonary septum [7]. The most common are proximal defects, which comprise 70-96% of all cases [8].

Aortopulmonary window is rare condition but is well known to paediatric cardiologists and cardiac surgeons, and this is well described in academic books. However, prenatal diagnosis of APW is unique and requires both good technical skills and a lot of experience even in a tertiary prenatal cardiology centre. The first report of fetal aortopulmonary window in the literature, we believe, was in 2004, although the diagnosis was made *post mortem* after autopsy. A significant pulmonary regurgitation was present and recorded during fetal echocardiography [9]. We present a table of previously published cases with prenatal diagnosis and postnatal confirmation of APW (Table 1). According to review of the previously published 39 cases, it is not easy to calculate the chance of a neonate surviving after prenatal diagnosis, partially because there are a significant number of terminations of pregnancy in the presented material and partially because current perinatal medicine is still developing, enabling better healthcare. Table 1 also shows that in the group of prenatally detected APW, there are some additional cardiac and extracardiac defects, which might influence the postnatal prognosis. The earliest presented antenatal diagnosis of APW based on Internet findings was made at 15 weeks of gestation [10].

The clinical features of APW postnatally are not specific and depend on the size of the defect. Prenatally it is not so common to detect APW, and in our case cardiomegaly, as a quite late sign of congestive heart failure, was the first sign that concerned the obstetrician, who referred the patient for further examination. In any case of fetal cardiomegaly, further echocardiographic examination is of great value. It might answer the question: what is the cause of cardiac enlargement? (as was presented in our case). Most patients with APW postnatally have manifestations of a large left-to-right shunt, which increases pulmonary blood flow and leads to pulmonary hypertension and congestive heart failure (tachypnoea, diaphoresis, failure to thrive, and recurrent respiratory difficulty). These symptoms appear rarely in the first week of life. The prognosis of untreated cases with large defects is poor, with a 40% fatality rate in the first year of life. Patients with small defects may be asymptomatic [11, 12]. The distinctiveness of fetal circulation from neonatal circulation may explain the reason for the right-to-left flow through APW in our case. In fetuses the right ventricular ejection force surpasses the left ventricular ejection force [13]. After delivery, pulmonary resistance decreases, causing a significant change in circulatory haemodynamics, but still there was bidirectional flow through APW in our case because pulmonary resistance decreased partially at the time of postnatal echocardiography. Typically left-to-right flow through APW occurs later in postnatal life.

Usually there is no possibility for APW to close spontaneously without intervention, and this defect will not decrease in size with time [5, 14]. The procedure for closure of the defect should be performed early in infancy in order to prevent complications such as pulmonary obstructive vascular disease and heart failure [14].

APW commonly is associated with other heart lesions in 25-52% of cases [2, 3], especially with aortic arch malformations (type A aortic arch interruption and severe preductal aortic coarctation). Other associated lesions are tetralogy of Fallot, transposition of great arteries, ventricular and atrial septal defects, right pulmonary artery arising from ascending aorta, and others [15]. Some of them might be detected prenatally, but some of them, e.g. atrial septal defect, might not. Rarely there is an anomalous origin of right coronary artery arising from the main pulmonary artery (ARCAPA), like in our case. It is also not feasible to detect ARCAPA during fetal echocardiography [16]. Unfortunately, ARCAPA was missed at neonatal echocardiography also, but it was detected finally during open cardiac surgery and corrected simultaneously. In a study published in 2015 among 43 APW patients, 1 had this anomaly [4].

The ARCAPA is usually an isolated CHD; only in rare cases is this anomaly associated with other cardiac malformations. When APW coexists with anomalous origin of coronary artery, increased flow in pulmonary arteries can be useful to ensure myocardial perfusion until surgery or intervention in some cases [17]. In our case, early surgery enabled us to repair the abnormal coronary origin and partially resolve the reason for heart failure and myocardial dysfunction. APW was described for the first time by a physician from London, John Elliotson, in 1830 [18], but the first successful repair of APW by ligature was made in 1948 by Gross. The patient was a 4-year-old girl. On follow-up 3 years post-surgically, the girl was in excellent condition [12, 17]. In general, surgery involves separation of the great arteries with either suture division or patch closure of the aorta and pulmonary artery, as was performed in our case, and this has low surgical mortality [12]. Since then, several techniques using the transaortic or the transpulmonary approaches have been described and used.

The prognosis depends on coexisting cardiac malformations. Complex congenital heart disease is a bad prognostic factor [12]. In our case prenatally detected cardiomegaly and ARCAPA, which was diagnosed during surgery, could worsen the prognosis, but to date the baby is in a good clinical condition. In isolated APW early repair results in an excellent long-term prognosis [2]. Studies show that fetuses conceived with IVF/ICSI methods are at increased risk of developing CHD [19]. A meta-analysis, which included eight studies with 25,856 children obtained from IVF techniques and 287,995 children conceived spontaneously, showed that there is a 44% higher

Table 1. Cases of aortopulmonary window with prenatal diagnosis [2, 13, 19-36]

Author	Gestational age at the time of diagnosis	APW (type according to Richardson)	Other cardiac abnormalities	Extracardiac abnormalities	Obstetric outcome	Genetic tests	Surgical treatment	Outcome
Our case (Poland)	34 weeks	APW type I	Atrial septal defect	None	CS at 38 weeks		At 12th day of life	Alive
Zidere et al. (2020) UK, London	32nd week	APW type 1	Ventricular septal defect, right aortic arch	–	At term	Normal CGH array	3rd week	Alive
Quintero et al. (2019) UK, London	Mid trimester	APW type I	Double aortic arch	None	VD at 38 weeks, weight	NK, 22q11 deletion excluded	At 12th day of life.	Alive
Kaya et al. (2019) Turkey, Istanbul	24 weeks	APW type I	Not found	None	CS at 27 weeks (because of HELLP syndrome)	NK, 22q11 deletion excluded	Not performed	Died at the age of 11 days
	25 weeks, (monochorionic diamniotic pregnancy)	APW type I	Hypoplastic left heart	None	CS at 28 weeks due to preterm labour	NA	Not performed	Died at the age of 9 days
Li et al. (2018) Beijing, China	27 weeks	APW type II	IAA type A, aortic origin of the right PA	None	VD at term	NK 22q11 not studied	Not performed	Died after 9 weeks
	27 weeks	APW type II	VSD	None	CS at 39 weeks	NK, 22q11 not studied	Surgical repair	Alive
	25 weeks	APW type III	Right aortic arch	None	CS at 37 weeks	NK, 22q11 not studied	Surgical repair	Alive
	27 weeks	APW type III	Not found	None	VD at term	NK, 22q11 not studied	Surgical repair	Alive
	26 weeks	APW type II	IAA type A, aortic origin of right PA	None	TOP at 26 weeks	NK, 22q11 not studied	–	–
	25 weeks	APW type III	Not found	None	VD at term	NK, 22q11 not studied	Surgical repair	Alive
	29 weeks	APW type II	Right aortic arch	None	VD at term	NK, 22q11 not studied	Surgical repair	Alive
	30 weeks	APW type II	Not found	None	VD at term	NK, 22q11not studied	Surgical repair	Alive
Yu et al. (2018) Beijing, China	24 weeks	APW type II	VSD, CoA	NA	TOP	NK, 22q11 deletion excluded	–	–
	24 weeks	APW type I	Absent arterial duct	NA	TOP	NK, 22q11 deletion excluded	–	–

Table 1. Cont.

Author	Gestational age at the time of diagnosis	APW (type according to Richardson)	Other cardiac abnormalities	Extracardiac abnormalities	Obstetric outcome	Genetic tests	Surgical treatment	Outcome
	23 weeks	APW type II	Interrupted aortic arch, aortic origin of right PA	NA	TOP	NK, 22q11 deletion excluded	–	–
	21 weeks	APW type II	Absent arterial duct	NA	TOP	Not studied	–	–
	23 weeks	APW type II	Aortic origin of right PA	NA	TOP	NK, 22q11 deletion excluded	–	–
	28 weeks	APW type II	DORV, absent arterial duct, VSD	NA	TOP	NA	–	–
Vaidyanathan et al. (2018) Kochi, Kerala, India	34 weeks	APW type II	IAA type A	NA	VD at 37 weeks	NA	On 4th day	Alive
Zhang et al. 2018) Beijing, China	24 weeks	APW type II	Berry syndrome: AORPA, intact ventricular septum, CoA	NA	TOP	11q14.2 gene deletion	–	–
	27 weeks	APW type II	Berry syndrome: AORPA, intact ventricular septum, IAA type A	NA	TOP	NA	–	–
	29 weeks	APW type II	Berry syndrome: AORPA, intact ventricular septum, IAA type A	NA	TOP	NA	–	–
	23 weeks	APW type II	Berry syndrome: AORPA, intact ventricular septum, IAA type A	NA	Lost follow-up	–	–	–
Tongprasert et al. (2017) Chiang Mai, Thailand	19 weeks	APW type I	Not found	hydrocephalus	TOP	NK, 22q11 not studied	–	–
	20 weeks	APW type I	Abnormal course of PA	None	VD at 27 weeks, 1100 g	NK, 22q11 not studied	Not performed	Died on 7th day
	21 week	APW type II	Abnormal course of great arteries	None	VD at term, 2900g	NK, 22q11 deletion excluded	Waiting for surgery	Alive
Louis-Jacques et al. (2017) USA	30 weeks	APW type III	Not found	Multiple fetal anomalies, Dandy-Walker malformation	CS at 38 weeks	NK, terminal gain in 7p and loss in 6p	Not performed	Death on 4th day
Fotaki et al. (2017) London, UK	34 weeks	APW type I	Not found	None	VD at term	NK, 22q11 deletion excluded	On day 36	Alive
	20 weeks	APW type I	IAA, ectopic position of right kidney	None	VD at term	NK, 22q11 deletion excluded	On 7th day	Alive

Table 1. Cont.

Author	Gestational age at the time of diagnosis	APW (type according to Richardson)	Other cardiac abnormalities	Extracardiac abnormalities	Obstetric outcome	Genetic tests	Surgical treatment	Outcome
García et al. (2016) Madrid, Spain	22 weeks (IVF, DC/DA twins)	APW type I	IAA type A	None	CS at 37 weeks	NK, 22q11 deletion excluded	At 5th day	Alive
Tunks et al. (2014) USA	NA	APW type II	DORV, VSD, D-malposition of the great arteries	None	CS at 38 weeks	Duplication of DNA from 5p12 and deletion from 16p12.2	At 5th day	Reoperation at 6 months. Survived
Kadohira et al. (2012) Shinjuku-ku, Japan	29 weeks	APW type I	IAA type B	None	CS at 38 weeks	NA	At 5th day	Alive
Corbacioglu et al. (2012) Istanbul, Turkey	24 weeks (MC/MA twins)	APW type I	IAA type B	None	CS at 35 weeks	NK, 22q11 deletion excluded	At 10th day	Complicated postoperative course, reoperation, died
Aslan et al. (2011) Istanbul, Turkey	33 weeks	APW type I	Not found	None	CS at 37 weeks	NA	At 1 week	Complicated postoperative course. Died.
Alvarez et al. (2011) Sevilla, Spain	26 weeks	APW type III	ASD	None	VD at term	NK, 22q11 deletion excluded	At 4 weeks	Alive
Hayashi et al. (2010) Osaka, Japan	29 weeks	APW type I	IAA type A	None	Delivered at term	NK, 22q11 deletion not studied	At 5th day	Alive
Patel et al. (2007) USA	30 weeks	APW type I	Tetralogy of Fallot	None	Delivered at term	NK, 22q11 deletion excluded	A few days after birth	Died postoperatively
Valsangiacomo et al. (2002) Canada	32 weeks	APW type I	Right aortic arch	None	Delivered at term	NA	NA	NA
Collinet et al. (2002) Lille, France	23 weeks	APW type I	VSD, ASD	None	Delivered at term	NA	At 3 weeks	Alive
Total					Total 40 (1 lost to follow-up, 11 TOP), Total 28 deliveries			19 survivors (8 deaths, 1 lost to follow-up)

AORPA – aortic origin of right pulmonary artery, ASD – atrial septal defect, CoA – coarctation of aorta, CS – caesarean section, DC/DA – dichorionic/diamniotic, DORV – double outlet right ventricle, IAA – interrupted aortic arch, NK – normal karyotype, PA – pulmonary artery, VD – vaginal delivery, VSD – ventricular septal defect.

risk of CHD in children conceived using assisted reproductive techniques [19]. According to the authors' best knowledge, this is the first case report in the literature presenting prenatally detected APW after IVF. We would recommend fetal echocardiography in every case of IVF. Genetic association with DiGeorge syndrome (22q11 deletion) has not been found, unlike in other conotruncal defects [3]. During fetal examinations the fetus was not found to be dysmorphic, and postnatal appearance occurred to be also normal.

Conclusions

Aortopulmonary window was diagnosed during pregnancy on echocardiography in the 3rd trimester in a fetal cardiology tertiary centre in pregnancy after in vitro fertilization. The main reason for referral was an increased fetus heart size in obstetrical ultrasound. Prenatal diagnosis of this anomaly provided information for an effective treatment plan: optimal perinatal care, transfer in utero, delivery in a tertiary care centre, postnatal cardiology evaluation in the same hospital, and faster treatment.

Conflict of interest

The authors declare no conflict of interest.

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