

# Prenatal diagnosis of fetal right ventricular diverticulum with massive pericardial effusion in one of monozygotic diamniotic twins: a case report with a favorable outcome following in utero pericardiocentesis

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## Abstract

**Background:** Congenital ventricular diverticulum is a rare abnormality that may occur as an isolated malformation. Most cases are accompanied by pericardial effusion. Prenatal counseling can be difficult because the prognosis is uncertain and there is no consensus approach to prenatal management.

**Case presentation:** We describe a case of congenital cardiac diverticulum complicated by large pericardial effusion in one of monozygotic diamniotic twins. The case was diagnosed by ultrasonography at 21 weeks of gestation. Therapeutic pericardiocentesis at 22 weeks resulted in complete resolution of the effusion and led to a favorable fetal outcome. We summarize the interventions and pregnancy outcomes in cases of cardiac diverticula reported in the literature.

**Conclusions:** Better awareness of clinical features, in utero therapies, and pregnancy outcomes could help define and improve prenatal management of congenital ventricular diverticula.

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## Keywords

Ventricular diverticulum, pericardial effusion, pericardiocentesis, monochorionic diamniotic twins, fetal outcome, prenatal counseling

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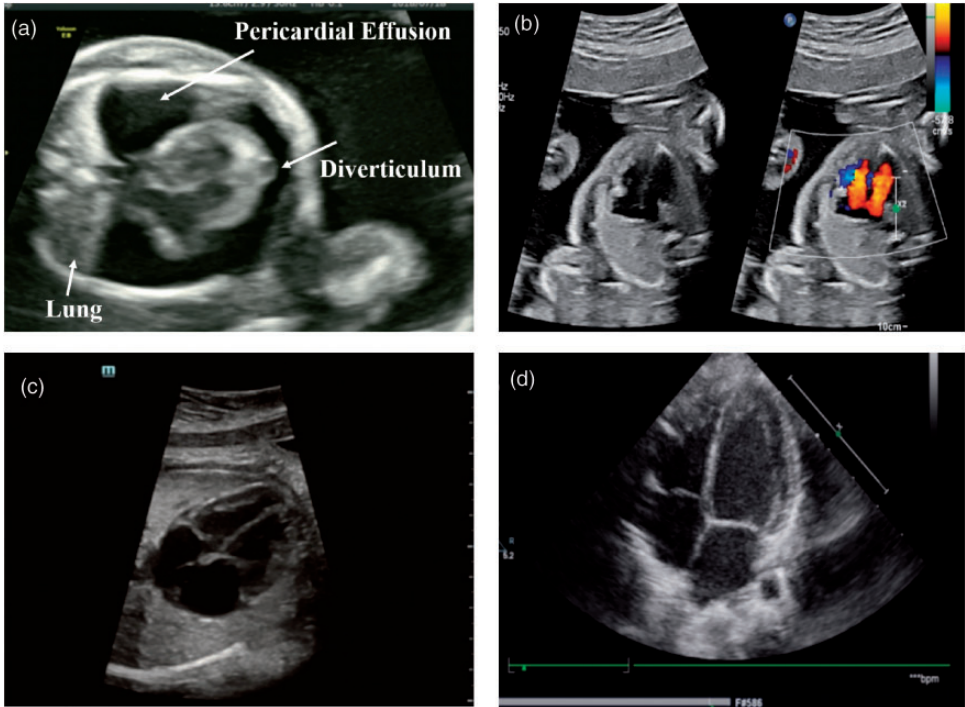
## Background

Congenital ventricular diverticulum is a rare abnormality, in which there is saccular dilatation of the cardiac ventricular wall, showing bidirectional flow on Doppler studies.<sup>1,2</sup> The cardiac diverticulum of the heart protrudes through the stenosis connection, and prenatal diagnosis may fail to detect the diverticulum. Only a few cases diagnosed in utero have been reported.<sup>3</sup> Most cases are accompanied by pericardial effusion, which can compromise the fetal circulation and lung development. Prenatal counseling can be difficult because some cases resolve spontaneously in the second trimester, whereas others progress with worsening hydrops, sometimes resulting in intrauterine fetal death.<sup>4-6</sup> Most cases are found in singleton pregnancies; only one case in the literature reports ventricular diverticulum in one fetus of monochorionic diamniotic twins. Here, we describe another monochorionic diamniotic twin fetus in whom right ventricular diverticulum associated with a massive pericardial effusion was diagnosed at 22 weeks of gestation. Therapeutic pericardiocentesis resulted in complete resolution of the effusion and led to a favorable fetal outcome.

## Case report

An 18-year-old healthy woman (gravida 2, para 1) had a spontaneous pregnancy of monochorionic diamniotic twins with unremarkable medical and family history. At the routine pregnancy check-up at 21

weeks' gestation, an abnormal fetal heart chamber was identified in one of the twins, and the patient was referred to our center with a diagnosis of bilateral hydrothorax in one fetus. Prenatal ultrasonography showed that the massive area of fluid was of pericardial, not pleural, origin. An abnormal heart chamber (4 × 5 mm) was detected in the apex of the right ventricle in the four-chamber view (Figure 1a). The presumptive diagnosis was a ventricular diverticulum or ventricular aneurysm. The connection to the ventricle was narrow, color Doppler ultrasound detected blood flow in and out of the abnormal heart chamber, and M-mode assessment indicated normal synchronous contraction of its walls with those of the ventricle; these findings were consistent with a diagnosis of ventricular diverticulum. There was an anechoic space peripherally around the heart, and both lungs were severely compressed (>50% lung compression) into a posterior position in the fetal thorax. This finding distinguished our case from that of a pleural effusion manifesting as an anechoic space located peripherally around the compressed lungs. There was no evidence of other fetal structural abnormalities or hydrops, and no structural or functional heart defects were detected. The mitral valve apparatus appeared normal, and no arrhythmias or intracardiac thrombi were seen. We detected no sign of complications of monochorionic multiple birth, such as twin-to-twin transfusion syndrome or selective intrauterine growth restriction.



**Figure 1.** Serial scans before and after intrauterine pericardiocentesis in one of monochorionic twins. (a) Transverse plane through the fetal chest showed a large pericardial effusion compressing the lungs and a  $4 \times 5$  mm, cyst-like structure at the right ventricular apex. (b) Three days after intrauterine pericardiocentesis, the fetal lungs expanded and serial scans showed an inconspicuous right ventricular cyst-like structure; the pericardial effusion did not reaccumulate. (c) A scan at 34 weeks of gestation showed no apparent right ventricular cyst-like structure, and no evidence of reaccumulation of pericardial effusion. (d) Postnatal echocardiography showed a normal heart with no hemodynamic disturbance.

Ultrasound scans of the other twin appeared normal.

The couple was counseled regarding the increased incidence of fetal chromosomal abnormalities, the possibility of pulmonary hypoplasia and progressive hydrops, and the potential effect on the other fetus caused by vascular communications in the placenta. Pericardiocentesis and amniocentesis were performed at  $22^{+2}$  weeks' gestation on the abnormal fetus only. Under ultrasound guidance, a 20-gauge needle was inserted percutaneously into the pericardial effusion, and 13 mL of straw-colored fluid was aspirated. Then, retracting the needle to the amniotic cavity, 35 mL

of amniotic fluid was taken for chromosome analysis, including G-banding karyotype and chromosome microarray analysis. The fetal lungs expanded automatically immediately after the procedure. Pericardial fluid, amniotic fluid, and maternal serum were submitted for viral studies, and all were negative for toxoplasmosis, other (syphilis, varicella-zoster), rubella, cytomegalovirus, and herpes (TORCH) infections and for parvovirus B19. Cytology of the pericardial fluid identified lymphocytes and mesothelial cells. The protein content of the pericardial fluid was 23.9 g/L, consistent with transudate. The pericardial effusion did not reaccumulate after

pericardiocentesis (Figure 1b, c). Chromosome analysis showed a normal female karyotype. Serial echocardiograms until birth showed no effusion and no cardiovascular compromise. The right ventricular diverticulum was no longer visible on ultrasound at 34 weeks' gestation. At 37 weeks' gestation, two healthy female infants were delivered, weighing 3040 and 2875 g, with Apgar scores of 9-10-10, which were appropriate for gestational age. The infants' prognoses were good and they were discharged without heart symptoms. An echocardiogram performed on the affected infant on day 10 after birth was normal (Figure 1d). The infants had regular follow-ups at our hospital and remained asymptomatic without the need for medication at 12 months of age (at the time of this writing). Our study complied with the CARE case report guidelines.<sup>7</sup> We have de-identified the case details such that the identity of the patient cannot be ascertained in any way.

## Discussion

Cardiac ventricular diverticulum is defined as a protrusion of the free ventricular wall. The main differential diagnosis is ventricular aneurysm.<sup>4</sup> The terminology is confusing and it is not always possible to distinguish by prenatal ultrasound. Sonographically, diverticula are saccular with a narrow neck communicating with the ventricle and contracting synchronously with the rest of the ventricular cavity. Aneurysms, by contrast, have a wider base and appear to be hypokinetic. Histologically, diverticula are composed of three layers: pericardium, myocardium, and endocardium, in accordance with cardiac tissue, whereas aneurysms are composed of thinned myocardium mixed with fibrous tissue.<sup>8,9</sup> Diverticula are more likely to be associated with midline defects or intracardiac abnormalities,<sup>10</sup> whereas aneurysms

are mostly isolated and have a poorer prognosis. The etiology of cardiac diverticula and aneurysms in fetal life is poorly understood. They may occur as a result of focal weakening of the ventricular wall due to an interruption during embryogenesis, such as the occurrence of congenital infection, trauma, coronary anomalies, or ischemia of the myocardium.<sup>3</sup>

Pericardial effusions associated with fetal ventricular diverticula have been reported in several cases.<sup>8,11-13</sup> The presence of pericardial effusion not only enhances the visibility of the diverticulum on ultrasound, but the effusion also acts as a cushion protecting the aneurysmal sac from rupture. The main differential diagnosis of massive fetal pericardial effusion is bilateral hydrothorax. Several cases of fetal pericardial effusion in previous reports were initially misdiagnosed as bilateral fetal hydrothorax,<sup>14</sup> as in our case. Sonographically, in our case, the lungs of the fetus diagnosed with pericardial effusion were compressed into a posterior position in the fetal thorax, a presentation distinct from a diagnosis of pleural effusion, which would present as an anechoic space located peripherally around the compressed lungs. In addition, the heart may be shifted into the contralateral hemithorax and appear smaller than normal in patients with bilateral hydrothorax.

We summarized interventions and pregnancy outcomes in cases of cardiac diverticula reported in the literature (Table 1). A total of 40 cases of prenatally diagnosed ventricular diverticula have been reported. The most frequent location is on the right ventricle (26/40, 65%), and the most frequently involved area is the heart apex (20/40, 50%). Ultrasonographic findings associated with diverticula include pericardial effusion, cardiomegaly, septal defects, arrhythmia with fetal death before delivery, and hydrops. Pericardial effusion is the most frequently associated finding (24/40,

**Table 1.** Description of prenatal cases of cardiac diverticulum reported in the literature.

Reference	Maternal age (years)	No. of fetuses	GA dx	Size	Sex	Location	Karyotype	Associated anomalies	Interventions	Prenatal progression	Neonatal	Follow-up
Kirchner et al. (1990) <sup>15</sup>	—	Singleton	33	—	Female	Apex LV (VI)	—	Cardiomegaly	No	Stable	Vaginal delivery 40 w; cardiomegaly, tachypnea, heart murmur, muscular IVC, and mild mitral regurgitation	Asymptomatic at 3.5 MOL
Hornberger et al. (1994) <sup>16</sup>	31	Singleton	31	—	—	Lateral wall below tricuspid valve (RV)	—	—	No	Stable	Vaginal delivery at term	Asymptomatic at 12 MOL
Charles et al. (1995) <sup>8</sup>	24	Singleton	13	—	Male	Apex LV	—	PE	TOP 14 w	—	—	—
Cesko et al. (1998) <sup>11</sup>	28	Singleton	17	—	Male	Apex LV	46,XY	PE	TOP 22 w	—	—	—
Cavalié-Garrido et al. (1997) <sup>6</sup>	—	Singleton	20	Large	Female	Lateral wall below mitral valve (LV)	Trisomy 18	VSD, hydrops	No	Fetal death at 26 w	—	—
Cavalié-Garrido et al. (1997) <sup>6</sup>	—	Singleton	19	Small	Female	Apex RV	—	No	No	Asymptomatic	Asymptomatic	Asymptomatic at 22 MOL
Cavalié-Garrido et al. (1997) <sup>6</sup>	—	Singleton	19	Small	—	Apex RV	—	PE	PC 20 w	Stable	Asymptomatic	Asymptomatic at 12 MOL
Cavalié-Garrido et al. (1997) <sup>6</sup>	—	Singleton	36	Small	Male	Lateral wall below tricuspid valve (RV)	—	PE	No	—	—	Asymptomatic at 18 MOL
Johnson et al. (1996) <sup>17</sup>	27	Singleton	19	3 mm	Female	Apex RV	46,XX	PE	PC 20 w	No relapse after PC, no growth	Eutocic delivery at 41 w; weight 3700 g; asymptomatic	Asymptomatic at 16 MOL
Brachlow et al. (2006) <sup>18</sup>	—	Singleton	32	3.5 cm <sup>2</sup>	—	Apex LV	—	—	No	Stable	Asymptomatic	Asymptomatic at 6 MOL
Bernasconi et al. (2004) <sup>14</sup>	29	Singleton	22	10 × 5 mm	Male	LV Lateral wall below mitral valve	46,XY	PE	PC 22 w	—	Fetal death at 26 w, probably due to diverticulum rupture	—
McAuliffe et al. (2004) <sup>19</sup>	—	Singleton	13	4 × 6 mm	Male	Apex RV	46,XY	First trimester NT 4.2 mm PE	PC 16 w	Resolution of the effusion; CD stable	Eutocic delivery at 38 w; weight 3070 g; asymptomatic	Asymptomatic at 10 MOL

(continued)

**Table 1. Continued.**

Reference	Maternal age (years)	No. of fetuses	GA dx	Size	Sex	Location	Karyotype	Associated anomalies	Interventions	Prenatal progression	Neonatal	Follow-up
McAuliffe et al. (2004) <sup>19</sup>	—	Singleton	13	4 × 3 mm	Male	Apex RV	46,XY	First trimester NT 2 mm PE	PC 14 w	Resolution of the effusion; CD stable	Eutocic delivery at 38 w; weight 3150 g; asymptomatic	Asymptomatic at 8 MOL
Prefumo et al. (2005) <sup>1</sup>	28	Singleton	14	5 × 5 mm	Male	Apex RV	46,XY	Resolution of PE and hydrops; CD stable; mild cardiomegaly	PC 16 w	Resolution of the effusion and hydrops; CD stable; mild cardiomegaly	Vaginal full-term eutocic delivery; asymptomatic	Asymptomatic at 22 MOL
Prefumo et al. (2005) <sup>1</sup>	35	Singleton	12	1 mm	—	Apex RV	—	First trimester NT 1.2 mm PE	No	Spontaneous resolution of PE with 21 w; CD stable	Full-term eutocic delivery; asymptomatic	Asymptomatic at 17 MOL
Gardiner et al. (2005) <sup>12</sup>	—	Singleton	14	2-3 mm	—	Apex RV	Normal	PE	PC 14 w	Resolution of the effusion and hydrops; CD collapsed	Asymptomatic at birth	—
Gardiner et al. (2005) <sup>12</sup>	—	Singleton	14	2-3 mm	—	Apex RV	Normal	PE	TOP	—	—	—
Del Rio et al. (2005) <sup>4</sup>	30	Singleton	13	5 × 5 mm	Female	Apex RV	46,XX	PE, AVSD	No	Spontaneous resolution at 28 w	Eutocic delivery at 40 w; weight 3400 g; asymptomatic at birth	Correction of AVSD at 3 MOL, resection of diverticulum; asymptomatic at 8 months of life
Wax et al. (2007) <sup>20</sup>	23	Singleton	20	6 × 9 mm	Male	Junction base RV infundibulum	—	No	No	Stable	Full-term eutocic delivery; weight 3689 g; asymptomatic; small permeable FO	Asymptomatic at 18 MOL

(continued)

**Table 1. Continued.**

Reference	Maternal age (years)	No. of fetuses	GA dx	Size	Sex	Location	Karyotype	Associated anomalies	Interventions	Prenatal progression	Neonatal	Follow-up
Koshiishi et al. (2007) <sup>21</sup>	25	MC pregnancy with laser intervention for TTTS	24	7 × 10 mm	–	Lateral wall below tricuspid valve (RV)	–	Mild PE; MC pregnancy with laser intervention for TTTS at week 20 when donor fetus died	No	Stable	Prenatal fetal death at 29 w	–
Pradhan et al. (2008) <sup>3</sup>	30	Singleton	28	–	–	Apex LV	–	Fetal arrhythmia, hydrops fetalis	Medical treatment (digoxin) PC 20 w	–	Vaginal delivery at 40 w	Asymptomatic at 12 MOL
Barberato et al. (2009) <sup>9</sup>	34	Singleton	16	5 × 5.7 mm	–	Apex LV	–	Mild PE	–	Discrete enlargement of PE with normal heart function	Prenatal fetal death at 37 w	–
Barberato et al. (2009) <sup>9</sup>	25	Singleton	30	12 × 13 mm	–	Mitral subvalvular	–	LV dilatation and reduced systolic function	No	Stable	–	Asymptomatic at 6 MOL
Davidson et al. (2006) <sup>5</sup>	–	Singleton	20	–	–	Apex RV	–	PE	No	Spontaneous resolution	–	Surgical treatment
Abi-Nader et al. (2009) <sup>2</sup>	38	Singleton	21	5 × 5.5 mm	Male	RV	–	PE	PC 24 w	Mild tricuspid regurgitation at 31 w; CD stable	Full-term delivery	Asymptomatic at 12 MOL
Perlitz et al. (2009) <sup>22</sup>	23	Singleton	22	7 × 4 mm	Male	RV lateral wall	–	No	No	Stable, CD growth up to 9 × 9 mm	Eutocic delivery at 40 w; weight 4010 g; asymptomatic at birth	Asymptomatic at 12 MOL
Menahem (2010) <sup>23</sup>	–	Singleton	19	–	–	Apex LV	–	PE	No	No controls performed	Full-term live birth	Asymptomatic at 10 MOL
Carrard et al. (2010) <sup>24</sup>	27	Singleton	13	2.6 × 2.9 mm	Male	RV lateral wall	46,XY	First trimester NT 2.2 mm PE	PC 17 w	Resolution after PC; CD collapsed at 26 w	Eutocic delivery at 40 w; weight 2780 g	Asymptomatic at 11 MOL

(continued)

**Table 1. Continued.**

Reference	Maternal age (years)	No. of fetuses	GA dx	Size	Sex	Location	Karyotype	Associated anomalies	Interventions	Prenatal progression	Neonatal	Follow-up
Williams et al. (2009) <sup>13</sup>	43	Singleton	22	3–4 mm	Male	RV	46,XY	PE	No	Resolution at 32–33 w	PROM 34 w; intubation due to prematurity; caesarean section; weight 2460 g; 2 muscle IVCs	Asymptomatic at 14 MOL
Williams et al. (2009) <sup>13</sup>	19	Singleton	21	11 × 15 mm	Male	RV lateral wall below tricuspid valve	–	Isolated	–	–	Eutocic delivery; weight 2780 g; asymptomatic at birth	Asymptomatic at 16 MOL
Williams et al. (2009) <sup>13</sup>	18	Singleton	25	26 × 16 mm (37 s)	Male	RV	–	Arrhythmia and reduced systolic function	Induced delivery	–	Caesarean section 38 + 5 w; weight 3270 g; mild reduction of systolic function and premature ventricular contractions at birth	Asymptomatic at 36 MOL, on prophylactic treatment with acetylsalicylic acid
Paoletti and Robertson (2012) <sup>25</sup>	40	Singleton	17	–	–	Apex LV	Normal	Mesocardia, perimembranous IVC	No	Stable	Full-term live birth	Asymptomatic at 24 MOL
Nam et al. (2010) <sup>10</sup>	30	Singleton	21	–	–	Apex LV	Normal	Defect on thoracoabdominal midline	TOP	–	–	–
Olorón et al. (2011) <sup>26</sup>	–	Singleton	31	12 (postnatal)	Male	RV lateral wall below tricuspid valve	–	VSD	No	VSD	Full-term live birth; asymptomatic at birth; symptoms at 45 days of life; closure of VSD at 3 MOL	Asymptomatic at 10 MOL
García et al. (2015) <sup>27</sup>	22	Singleton	14	2	Male	Apex RV	46,XY	PE	PC 17 w	PE resolved after treatment; CD stable; moderate cardiomegaly; normal heart function	Full-term live birth; spontaneous eutocic delivery at 40 + 1 w; weight 3150 g	Asymptomatic at 48 MOL

(continued)



**Table 1. Continued.**

Reference	Maternal age (years)	No. of fetuses	GA dx	Size	Sex	Location	Karyotype	Associated anomalies	Interventions	Prenatal progression	Neonatal	Follow-up
Erek et al. (2013) <sup>28</sup>	–	Singleton	24	5 × 10 mm	–	Connection with RV	Normal	Fetal VSD, pericardial cyst, and PE	No	–	On DOL 3, the patient underwent surgical ASD and VSD closure and excision of diverticulum with cyst spontaneously	–
Demir et al. (2015) <sup>29</sup>	37	Singleton	28	30 × 14 mm	–	LV free wall	–	Very small PE	No	PE resolved	spontaneously	Full-term live birth; mild tachypnea; Dor procedure was performed at 17 DOL
Asymptomatic at 4 MOL												
Katsura et al. (2016) <sup>30</sup>	35	Singleton	32	22 × 10 mm	–	Outside the RV	–	No	No	Stable	Full-term live birth	Asymptomatic at 6 MOL
Hunter et al. (2016) <sup>31</sup>	26	Singleton	18	6 × 5 mm	–	LV posterior wall	–	No	No	At 29 w, the diverticulum had enlarged to 12.6 × 9.5 mm; by 35 w, diverticulum measured 20 × 15 mm and obscured the left atrium cavity	Full-term live birth; LV outflow tract diverticulum was resected successfully and extended aortic arch repair undertaken	–
Our case	18	MCDA	22	4 × 4 mm	Male	Apex RV	46,XY	PE	PC 22 w	Stable	Full-term live birth	Asymptomatic at 12 MOL

GA dx, gestational age at diagnosis; RV, right ventricle, LV, left ventricle; w, weeks of pregnancy; TOP, termination of pregnancy; PC, pericardocentesis; CD, cardiac diverticulum; IVC, interventricular communication; PE, pericardial effusion; NT, nuchal translucency; FO, foramen ovale; VSD, ventricular septal defect; ASD, atrial septal defect; AVSD, atrioventricular septal defect; MC, monochorionic; MCDA, monochorionic diamniotic; TTTS, twin-to-twin transfusion syndrome; PROM, premature rupture of membranes; w, weeks; DOL, days of life; MOL, months of life.

60%) and should be considered an indirect sign of the presence of cardiac diverticula. Of these 40 cases, 32 pregnancies resulted in liveborn neonates, 4 patients opted for pregnancy termination, and fetal death occurred in 4 cases, one of which was a result of trisomy 18.

In reported cases of ventricular diverticula associated with massive pericardial effusion, the most common outcome was termination of pregnancy before a prenatal intervention was implemented. Intrauterine pericardiocentesis allows compressed lungs to re-expand, reduces systemic venous pressure, and prevents potential fetal edema and intrauterine death. Of the 40 cases of fetal ventricular diverticula reported in the literature plus our case, 24 were associated with pericardial effusion. Of these 24, 3 patients opted for pregnancy termination because of the perceived risk of pulmonary hypoplasia, and 9 patients opted for expectant management. Of these 9 patients, fetal death occurred in 2, spontaneous resolution occurred in 5, and, of the remaining 2, 1 was asymptomatic at 18 months of life and 1 underwent surgery postnatally. Of the 24 cases in the literature associated with pericardial effusion, 12 underwent intrauterine pericardiocentesis (1 fetal death occurred, likely because of rupture of the diverticulum, and 11 cases had a favorable prognosis). In our case, the effusion completely absorbed and the lungs re-expanded rapidly after intrauterine pericardiocentesis. Although the natural evolution of this condition remains unknown, some researchers speculate that without proper intervention, persistent lung compression may result in fatal pulmonary hypoplasia. Nevertheless, spontaneous resolution has been reported several times with good prognosis. Concerns about lung development and heart failure have led some researchers to recommend intrauterine pericardiocentesis when a large pericardial effusion is diagnosed. Further studies

are needed to determine whether intrauterine pericardiocentesis throughout pregnancy could be of benefit to the fetus.

## **Conclusion**

We report a case of right ventricular diverticulum associated with a massive pericardial effusion in one (monochorionic diamniotic) twin diagnosed at 22 weeks that resulted in a favorable fetal outcome following intrauterine pericardiocentesis. In addition, we summarized interventions and pregnancy outcomes of 40 cases of cardiac diverticula reported in the literature. Better awareness of the clinical features, intrauterine therapies, and pregnancy outcomes will help in defining appropriate prenatal management of congenital ventricular diverticula.

## **Ethics statement**

The study was approved by the Institutional Review Board/Medical Ethics Committee of Guangdong Women and Children Hospital (IRB reference number: 201612089). Written informed consent was obtained from the participating family for publication of the case report.


## **Declaration of conflicting interest**


The authors declare that there is no conflict of interest.

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