REVIEW ARTICLE

Evolution of ventricular outpouching through the fetal and postnatal periods: Unabating dilemma of serial observation or surgical correction

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Ventricular outpouching is a rare finding in prenatal sonography and the main differential diagnoses are diverticulum, aneurysm, and pseudoaneurysm in addition to congenital cysts and clefts. The various modes of fetal presentation of congenital ventricular outpouching include an abnormal four-chamber view on fetal two-dimensional echocardiogram, fetal arrhythmia, fetal hydrops, and pericardial effusion. Left ventricular aneurysm (LVA)/nonapical diverticula are usually isolated defects. Apical diverticula are always associated with midline thoracoabdominal defects (epigastric pulsating diverticulum or large omphalocele) and other structural malformations of the heart. Most patients with LVA/congenital ventricular diverticulum remain clinically asymptomatic but they can potentially give rise to complications such as ventricular tachyarrhythmias, systemic embolism, sudden death, spontaneous rupture, and severe valvular regurgitation. The treatment of asymptomatic LVA and isolated congenital ventricular diverticulum is still undefined. In this review, our aim is to outline a systematic approach to a fetus detected with ventricular outpouching. Starting with prevalence and its types, issues in fetal management, natural course and evolution postbirth, and finally the perpetual dilemma of serial observation or surgical correction is discussed.

Keywords: Cardiac magnetic resonance imaging, Congenital left ventricular diverticulum, Fetal ventricular outpouching, Left ventricular aneurysm, Transaxial helical computed tomography cardiac angiogram

Contents

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1. Introduction

Congenital ventricular diverticulum (CVD) comes up for discussion when an outpouching of the left ventricle (LVO) is detected during prenatal sonography and various perplexing terms such as left ventricular accessory chamber, left ventricular aneurysm (LVA), left ventricular diverticulum (LVD), left ventricular pseudoaneurysm (LVPA), double-chambered LV, and accessory left ventricle are used to describe the anomaly [1]. LVO can be characterized further based on (1) location: (i) anterobasal; (ii) anterolateral; (iii) apical; (iv) diaphragmal; (v) posterobasal; (vi) septal; (vii) lateral; and (viii) left ventricular outflow tract [2]; (2) myocardial layers: muscular or fibrous; and (3) contractility: synchronous or paradoxical [3,4]. Most patients with CVD remain clinically asymptomatic and no complications are observed during long-term follow-up, but it can potentially give rise to congestive cardiac failure, ventricular tachyarrhythmias, systemic embolism, sudden death, spontaneous rupture, and severe valvular regurgitation [4,5]. Management decisions are relatively streamlined in cases of symptomatic LVO (LVA/LVD) and apical LVD associated with multiple anomalies. It entails surgical correction, but the treatment of asymptomatic and isolated LVO is still undefined. The objective of this article is to review the prevalence and types of congenital LVO, approach to fetal detection of ventricular outpouching, modes of prenatal presentation of congenital LVO, issues in fetal management, its natural history postbirth, and finally the contentious subject of serial observation or surgical resection is discussed.

Abbreviations

CVD	congenital ventricular diverticulum
LVO	left ventricular outpouching
LVD	left ventricular diverticulum
CVA	congenital ventricular aneurysm
MRI	magnetic resonance imaging
FISP	fast imaging with steady state precession
FGR	fetal growth restriction
CNS	central nervous system
KUB	kidney and urinary bladder
SVT	supraventricular tachycardia

2. Methods

We searched Pub-Med, Cochrane Central Register of Controlled Trials, Google Scholar, MEDLINE, and EMBASE up to March 2016; and previous literature reviews, including cross-references and abstracts. Combining the terms Etiology/Broad[filter] AND {(''congenital''[Sub heading] OR ''congenital''[All Fields]) AND left [All Fields] AND (''heart ventricles''[MeSH Terms] OR (''heart''[All Fields] AND ''ventricles''[All Fields]) OR ''heart ventricles''[All Fields] OR ''ventricular''[All Fields]) AND (''diverticulum''[MeSH Terms] OR ''diverticulum''[All Fields])} yielded 35 results, Therapy/Broad[filter] and rest yielded eight results, Diagnosis/Broad[filter] yielded 161 articles, Prognosis/Broad[filter] yielded 56 articles, and Clinical Prediction Guides/Broad[filter] yielded 16 articles. All searches yielded only two systemic reviews on this subject. We included individual case reports with literature reviews, case series, and systemic reviews. The results of retrieved articles were reviewed for potentially relevant studies, and we focused on fetal left ventricular outpouching, diagnostic criteria, issues in the management, and patient outcome.

3. Prevalence and types

We did not find any article citing incidence of nonapical congenital LVD in the newborn population, perhaps because most cases are asymptomatic, Cantrell syndrome has prevalence of <1/1,000,000, with antenatal/neonatal onset, and prevalence of congenital LVD is suggested to be 0.5/100,000 births with equal distribution between sexes, but congenital LVD and LVA were used interchangeably in the articles [4,6].

The main differential diagnoses are diverticulum, aneurysm, and pseudoaneurysm in addition

to congenital crypts and clefts, if a ventricular outpouching is detected [1,6]. Left ventricular diverticulum (LVD) is defined as an outpouching structure that contains all three layers of the heart—endocardium, myocardium, and pericardium—and displays synchronous contraction with rest of the myocardium [5], whereas, LVA and LVPA have fibrous walls and exhibit dyskinetic or akinetic contraction. Synchronous contractility is related to the presence of muscular fibers within the diverticular wall. Furthermore, the ratio of the connection to the left ventricular cavity and the maximum diameter of the body of the anomaly is >1 in LVA, whereas, it is <1 in LVD [5]. LVD may be divided into: either congenital or acquired; apical or nonapical; muscular or fibrous. They are considered to be congenital if there is no history of conditions that have injured the myocardium and during altered embryogenesis in first 2–3 weeks, a focal defect of muscular wall have given way to an outpouching. Other potential inciting factors are *in utero* viral infection, muscle and connective-tissue defects, and excessive primordial-cell stimulation [7]. Fetal echocardiography helps to support a congenital etiology when the diagnosis is made during the prenatal period. Apical CVD is distinguished from nonapical CVD by the size of connection from the parent structure. Apical CVD is a fingerlike contractile pouch with narrow connection to the ventricle and associated with intracardiac or extracardiac malformations, whereas nonapical CVD is a large contractile pouch with wide connection to the ventricle and is an isolated anomaly [8]. Most diverticula are apical. Nonapical diverticula arise from the anterior free wall, or left ventricular outflow tract, and rarely, from both ventricles [3,8]. Apical CVD is described as a part of a syndrome of midline thoracoabdominal defect (ectopia cordis, large omphalocele, pericardial defect, sternal defect, and diaphragmatic defects) with other intracardiac malformations (tetralogy of Fallot, ventricular septal defect, atrial septal defect, tricuspid atresia, dextrocardia) that had been described by Cantrell et al. [4] whereas nonapical CVD and congenital ventricular aneurysm were shown as isolated cardiac defects. Division of diverticula into primarily muscular or fibrous is based on the amount of myocardial fibers involved. Fibrous variety is commonly located near the atrioventricular valve apparatus; its connection with the ventricular cavity is wide and usually diagnosed in older children and adults as an incidental isolated finding. Muscular diverticula are most often localized at apex, a mechanical activity synchronous with the activity of the ventricles, and contain all layers of the heart. The connection with the ventricular chamber is narrow and is usually associated with other congenital cardiac malformations or midline defects [9].

4. Approach to a fetus with ventricular outpouching

The various modes of fetal presentation of congenital LVD include an abnormal four-chamber view, fetal arrhythmia, fetal cardiomegaly, hydrops fetalis, pericardial effusion, and hydrothorax [10–12]. Once fetal outpouching is detected using fetal echocardiography four-chamber view, size of the lesion is noted and it is followed serially for its possible enlargement, mass effect on the development of lung, compromise of cardiac function, and arrhythmias (Fig. 1). This also entails look out for the presence of fetal growth restriction, hydrops, and polyhy dramnios/oligohydramnios as well as meticulous screening of fetal central nervous system and kidney and urinary bladder region.

There is a potential role of fetal cardiac evaluation with fetal magnetic resonance imaging (MRI), as it can provide additional information in cases of LVO, pericardial cyst, and pericardial effusion [13,14]. However, rapid fetal heart rate and fetal movement require appropriate techniques that allow for rapid sequence MRI imaging. Two techniques are currently being used to overcome this—half acquisition single-shot fast spin echo and steady state free precession imaging [15,16]. An electrocardiography gating signal is used to synchronize MRI acquisition with cardiac cycle. However, absence of a suitable *in utero* gating signal in the MRI environment precludes conventional fetal cardiac MRI. There are different gating alternatives that have been used to beat this obstacle: (1) real-time imaging with rapid acquisition so that no gating is required; (2) self-gating; and (3) metric optimized gating [17]. Although preliminary experiences are accumulating [18], we have not found any article addressing this issue.

Earliest gestational maturity at which LVO is reported is 13 weeks by Carles et al. [19] (intervention: termination of pregnancy) and McAuliffe et al [20] (intervention: pericardiocentesis; outcome: term live birth). The earliest time at which it can be reliably diagnosed lies in the compromise between obtaining adequate images for diagnosis and yet offering decisive diagnosis to parents for

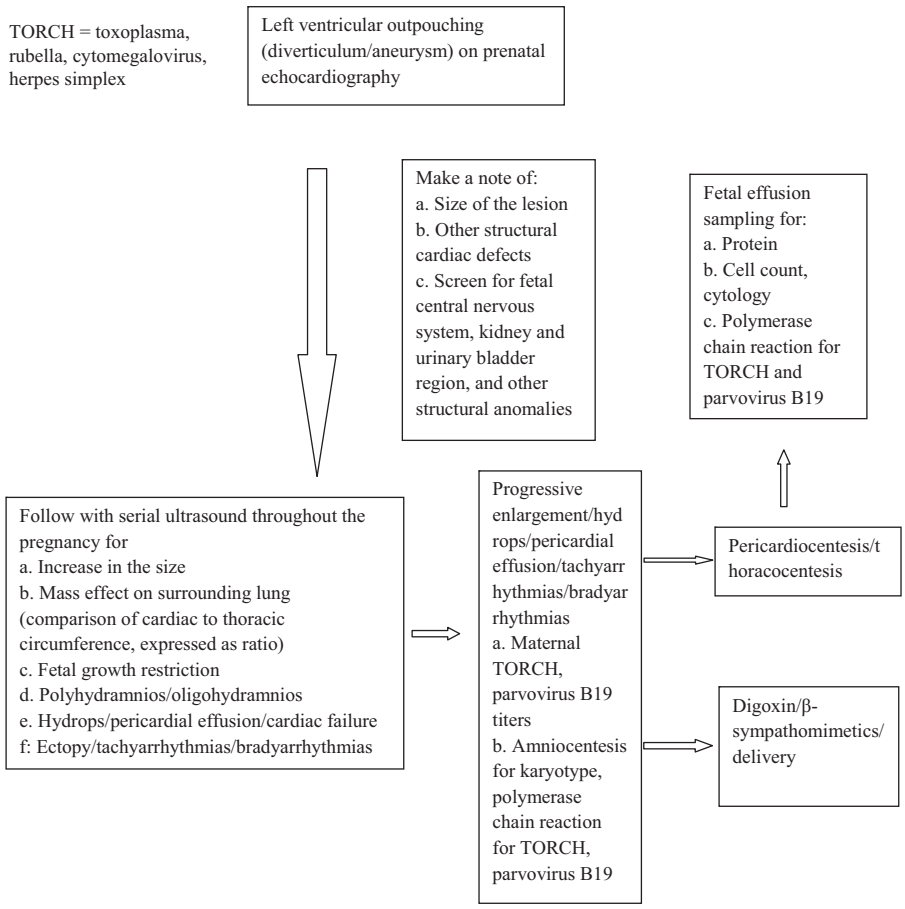

Fig. 1. Approach to a fetus with left ventricular outpouching.

considering all options ranging from conservative management to termination of pregnancy. A preliminary transabdominal or transvaginal echocardiogram at gestational age 11–15 weeks followed by a detailed examination at 18–20 weeks is the reasonable and decent approach.

Ohlow et al. [21] studied 26/42 fetuses with prenatal diagnosis of LVA and 16/42 with LVD. Mean gestational age at diagnosis was (23.8 ± 6.1)weeks. Authors found larger size of LVA as compared to LVD and apical locations in both the groups. Arrhythmias and hydrops fetalis were found more in LVA, whereas spontaneous rupture was more in LVD group. Progression in the size was found in 17% of total cases. During subsequent follow-up for a period of 29.1 ± 38.2 months, the rate of adverse events was significantly increased in patients with LVA. Cavalle-Garrido et al. [22] studied seven fetuses with diagnosis of ventricular diverticulum or aneurysm at 18–36 weeks of gestation. Out of seven fetuses, two developed hydrops and died *in utero*, and among survivors, three had decreased left ventricular function. Postnatal echocardiograms confirmed prenatal findings in all survivors. All infants remained asymptomatic, with a follow-up to 8–24 months. The authors suggested an excellent prognosis for cases diagnosed during fetal life. They further concluded feasibility of an accurate prenatal diagnosis of ventricular diverticula and aneurysms and association of outcome on the size and progression of the lesion. Pradhan et al. [23] described a case of isolated LVD associated with ventricular ectopy and mild pericardial effusion at 28 weeks of gestation and they observed restoration of sinus rhythm within 48 h. Brachlow et al. [24] reported fetal diagnosis of LVD at 32 weeks of gestation, in which infant remained asymptomatic at birth and early childhood (up to 3 years), while Bernasconi et al. [25] reported prenatal rupture of submitral fibrous diverticulum of posterior wall of left ventricle at 24 weeks of gestation and subsequent demise of fetus.

5. Management in the fetal period

Fetal management depends on: (1) persistence of irregular heart rhythm, tachy/bradyarrhythmia;

(2) hydrops; (3) compromised cardiac function; and (4) concern for high fetal loss, which consists of: (i) observation; (ii) transplacental drug therapy; (iii) thoracocentesis; (iv) pericardiocentesis; and (v) delivery of the fetus [19–23]. In general, if ectopy is infrequent (i.e., <1 beat/10 sinus beats), no further investigation is indicated but with frequent ectopy, follow-up with serial ultrasound is warranted to watch for possible progression to supraventricular tachycardia, hydrops, polyhydramnios, and a mass effect on the surrounding lung with secondary pulmonary hypoplasia and assessment of fetal wellbeing [21,26–28]. For extreme cases of hydropic fetuses with difficult to treat arrhythmia, emergency delivery should be considered.

The safest antiarrhythmic agent for the treatment of fetal tachycardia remains digoxin and flecainide is used as second-line drug in case of poor transplacental transfer of digoxin in the setting of hydrops [27].

Among fetuses presenting with bradyarrhythmias, management strategies include maternal β-sympathomimetics and delivery of the fetus once hydrops fetalis has developed or if a gestational age of 34–35 weeks has been achieved and concern for fetal loss is high [26–28].

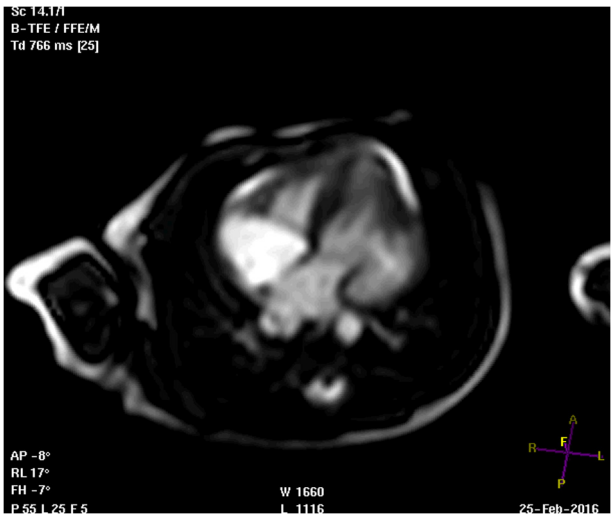

Fig. 4. Cardiac MRI showing left ventricular diverticulum.

6. Natural history and management postbirth

Postbirth, two-dimensional echocardiography (Fig. 2), multidetector computed tomography row (Fig. 3), and cardiac MRI (Fig. 4) can distinguish among muscular type LVD, fibrous LVD, LVA, and LVPA (Table 1) [29]. In particular, cardiac MRI is a very helpful tool for differentiating true LVA from LVPA [30].

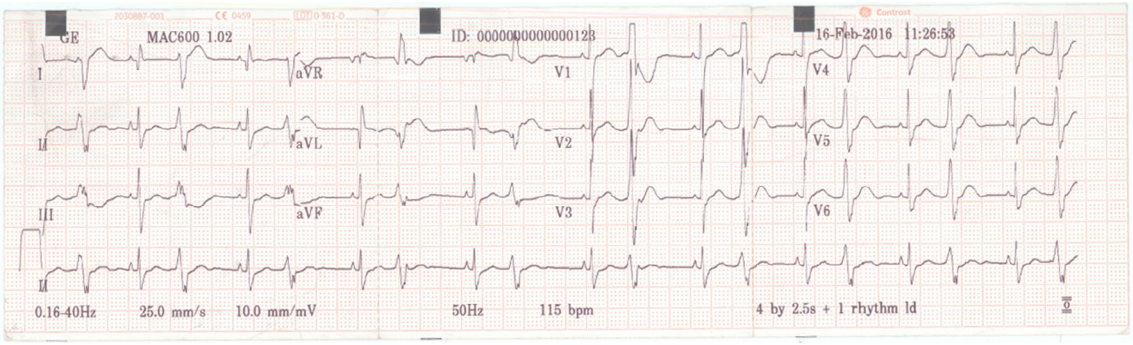

Fig. 2. LVO presenting as ventricular bigemini.

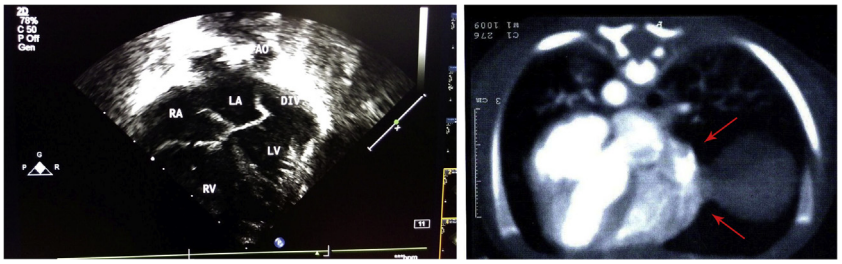

Fig. 3. 2D ECHO and CT Angio showing left ventricular diverticulum.

LVA/nonapical LVD remain clinically asymptomatic. In exceptional circumstances, it is fraught with complications such as gradual enlargement [5], thrombosis, embolism, rupture, congestive heart failure, ventricular arrhythmias [1,3], and valvular abnormalities [3]. Because of its rarity, the true incidence of these complications is not known. Mayer et al. [31] investigated 16 adult patients of arrhythmias and concluded that differential diagnosis of symptomatic ventricular tachyarrhythmias or embolic events of unknown origin should include congenital ventricular aneurysms or diverticula [31]. In newborns presenting with arrhythmias (Fig. 5), procedure of ventricular mapping of arrhythmogenic focus is difficult, but an assumption of diverticulum acting as a focus can be made, as suggested by Tsujimoto et al. [32].

Ohlow et al. [5], in their cohort of 809 cases observed more frequent cardiac death in the LVA group and caused by congestive heart failure in most of the cases, whereas spontaneous rupture was the cause of death in LVD group during follow-up. Marijon et al. [3] reported a cohort of 10 patients with LV diverticulum who were not surgically treated; two experienced spontaneous regression, and eight were alive without symptoms after a mean follow-up period of 8.4 years. Archbold et al. [33] suggested benignity of LVD after 13 years follow-up of their case. Sierra et al. [34] and Pitol et al. [35] observed LVD presenting for the first time as sustained monomorphic ventricular tachycardia (VT) in a 38-year-old Caucasian woman, and 56-year-old woman, respectively, in their case reports.

7. Serial observation/surgical correction

Treatment options are relatively straightforward in cases of symptomatic LVA and apical LVD associated with multiple anomalies and entails surgical correction, thromboembolism prevention with anticoagulants, and treatment of any associated symptoms such as arrhythmias or CHF. The same cannot be said about asymptomatic nonapical LVD/LVA, which is largely an undefined domain. The type and extension of diverticulum dictates the surgical technique applied [36]. Presence of other defects, connection of diverticulum to, and actual volume of true ventricular cavity decide the use of extracorporeal circulation [36]. Okereke et al. [36] described surgical procedure in 10 cases, which included ventricular repair with Dacron, mitral valve repair, and mitral and aortic valve replacement. Turbendian et al. [37] and

Table 1. Diagnostic tools for differential diagnoses of congenital ventricular outpouching [17].

Modality	LVD (fibrous variety)	LVD (muscular variety)	Left ventricular aneurysm	Left ventricular pseudoaneurysm
Coronary computed tomography	No volume change during the cardiac cycle, presence of fibrous tissue on the diverticulum wall, homogeneous myocardial tissue density in the ventricle chamber	Contracting in synchrony with the ventricle wall, presence of myocardial tissue on the diverticulum, homogeneous myocardial tissue density in the ventricle chamber	No volume change during the cardiac cycle, presence of fibrous tissue on the wall, hypodensity on the myocardial tissue	No volume change during the cardiac cycle; pseudoaneurysm wall is <5 mm composed by hypodense fibrous tissue (pericardium); hypodensity present
Cardiac magnetic resonance imaging	Diverticulum characterized by thinned and fibrous wall, no signal alterations of the remaining left ventricular wall with no signs of necrosis or fibrous tissue on delayed enhancement images	Diverticulum with thinned but contractile wall, no signal alterations of the left ventricular wall and no signs of necrosis or fibrous tissue on delayed enhancement images	High signal intensity on delayed enhancement images on the wall because of the presence of scar or fibrosis, lack of contraction of the myocardial tissue	Myocardial wall replaced by pericardium, significant enhancement in the surrounding myocardial tissue due to acute myocyte necrosis
Left ventricular catheterization	Rapid contrast media filling, wide neck, and no volume change during the different cardiac cycle phases	Rapid contrast media filling, narrow neck, volume reduction during systolic phase and volume increases during diastolic phase	Rapid contrast media filling, usually with a wide neck and no volume change	Differential diagnosis with aneurysm is not possible

LVD = left ventricular diverticulum.

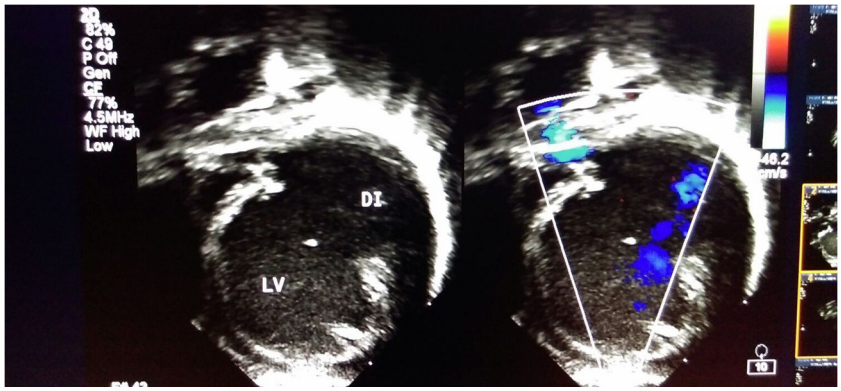

Fig. 5. 2D ECHO and colour doppler showing left ventricular diverticulum.

Vazquez-Jimanez et al. [38] described challenges in the repair of LVD in the setting of Cantrell syndrome because of its association with wide spectrum of anomalies, severity of abdominal and cardiac malformations and high mortality.

Some authors have suggested surgical resection for all the cases, keeping potential complications such as embolism, arrhythmias and spontaneous rupture in view, whereas those pitching for conservative management, are concerned with postoperative risk of resection of diverticular wall, consisting of normally contracting myocardium [39]. Possible solutions for surgical correction in asymptomatic LVA/isolated nonapical LVD are combined treatment of ventricular arrhythmias, prevention of rupture of the diverticulum and thrombus formation. Shen et al. [40] reported a case with sustained VT in association with subvalvular fibrous diverticulum refractory to medications, and surgical ablation of the arrhythmogenic focus was done after mapping but the arrhythmia recurred 1.5 years after the procedure. Similarly, Kawata et al. [41] have described successful repair of congenital LVD in an 9-day-old neonate, but it failed to make the heart quiescent and the combined ventricular bigeminy subsided only 9 months after the repair. Uchida et al. [42] advised early surgical intervention for this rare anomaly even in children without clinical symptoms after their observation of a 9-year-old child with gradual enlargement of diverticulum during 8 years out-patient follow-up.

8. Concluding remarks

When an LVO is detected during prenatal sonography, the main differential diagnoses are diverticulum, aneurysm, and pseudoaneurysm. Synchronous contractility with ventricular wall and change in volume (i.e., reduction during systole and expansion during diastole), indicate LVD (muscular variety), whereas no volume change is observed during cardiac cycle in LVA, LVD (fibrous variety), and LVPA on coronary computed tomography/LV catheterization [29] (Table 1). Similarly, presence of homogeneous myocardial density indicates LVD (muscular/fibrous) and hypodensity on the myocardial wall denotes aneurysm/pseudoaneurysm. No signal alterations on ventricular wall are observed in LVD (muscular/fibrous), whereas high signal intensity is observed in LVA and LVPA on cardiac MRI. They generally carry good fetal outcome. Presently there is lack of agreement over the best management option of asymptomatic LVA/nonapical LVDs.

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