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Diagnostics of Heart Failure Development in Children with Congenital Ventricular Septal Defect After Surgery

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Abstract: Background: Congenital ventricular septal defect (VSD) is one of the most common congenital heart defects in pediatric populations. Surgical correction significantly improves outcomes; however, postoperative heart failure (HF) remains a crucial complication affecting morbidity and long-term prognosis.

Objective: To evaluate diagnostic strategies for detecting heart failure development in children following surgical repair of VSD.

Methods: We conducted a review of diagnostic modalities including clinical scores, biomarkers, echocardiographic parameters, and advanced imaging. A proposed diagnostic flowchart is presented.

Results: Early detection of HF relies on a combination of clinical assessment, laboratory biomarkers (e.g., NT-proBNP), and imaging methods such as echocardiography. Novel algorithms improve sensitivity and specificity.

Conclusion: A structured diagnostic pathway enhances early recognition and allows timely intervention to reduce adverse outcomes.

Keywords: Ventricular septal defect, pediatric cardiac surgery, heart failure, diagnostics, echocardiography, biomarkers.

1. Introduction: Congenital ventricular septal defect (VSD) accounts for 20–30% of all congenital heart anomalies. Surgical correction remains the definitive treatment for hemodynamically significant defects. Despite advances in surgical techniques, postoperative heart failure develops in a subset of children and is associated with prolonged hospitalization and increased healthcare utilization.

Early identification of HF in this population allows prompt treatment and improves overall outcomes. This article examines diagnostic modalities and proposes an evidence-based framework for clinical use.

2. Definition of the disease. Causes of the disease

A ventricular septal defect (VSD) is a congenital heart defect in which the septum between the right and left ventricles is disrupted in utero. This disruption causes them to communicate with each other, which can later lead to heart failure.

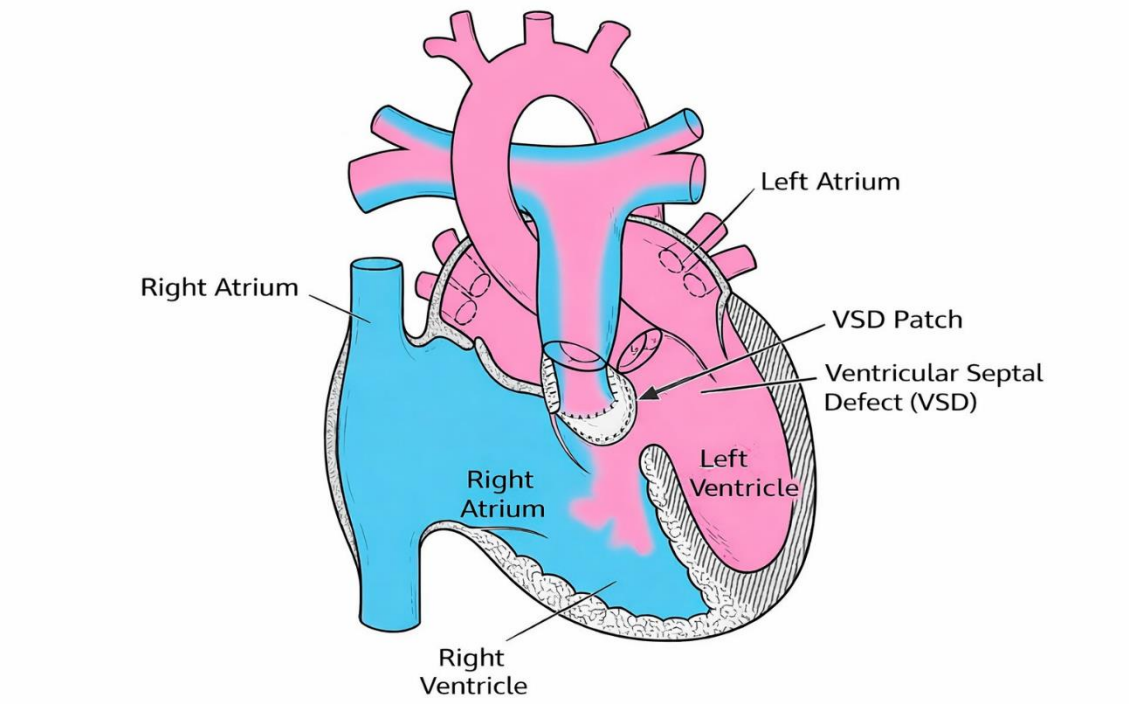

Figure 1. Ventricular septal defect

The heart's great vessels form between the 3rd and 8th weeks of pregnancy, and the actual separation of the ventricles by the septum occurs between approximately 32–33rd and 62–63rd days [3]. The countdown begins from the day of fertilization.

VSD can occur in isolation or as part of more complex heart defects, such as tetralogy of Fallot or double outlet right ventricle. Children with tetralogy of Fallot without a VSD can survive, whereas with a double outlet valve, the defect is the only opening for blood outflow from the left ventricle and is therefore essential for the child's survival.

Prevalence of VSD

Aside from bicuspid aortic valve, VSD is the most common congenital heart defect (20-30% of all defects). It occurs in boys and girls with approximately equal frequency.

Because it is a congenital heart defect, it is most often detected in childhood. In general, VSD is usually diagnosed in infancy, and the older the child, the less likely it is to be detected, as some small defects close spontaneously.

Causes of VSD

There are no clear risk factors, but VSD is more common in children whose blood relatives have congenital heart defects [20]. It has been shown that in families where close relatives have a VSD (including any complex congenital heart defect, which includes VSD), the risk of developing the defect in a child is slightly higher than in the healthy population. However, the presence of an isolated atrial septal defect or patent ductus arteriosus does not increase the risk of developing VSD in relatives.

The development of any congenital heart defect, including VSD, can also be caused by:

- ✓ bad maternal habits, such as smoking, alcohol consumption, and drug use during pregnancy;
- ✓ maternal infectious diseases during pregnancy, especially viral ones (rubella, chickenpox, etc.);
- ✓ exposure to harmful environmental factors in the first trimester.

Symptoms of a Ventricular Septal Defect

The clinical presentation of a VSD depends primarily on the size of the defect itself: the larger the defect, the more pronounced the symptoms.

Typically, with very large defects, when there is no pressure difference between the ventricles, auscultation (listening) reveals virtually no murmur.

However, parents may notice that their baby is feeding less frequently, as breathing becomes more difficult while breastfeeding, temporarily stopping to catch their breath, or sucking more slowly, and sweating more during feeding. All of these symptoms are manifestations of developing heart failure, i.e., signs of developing complications, not the VSD itself: the absence or disturbance of the murmur is the only symptom of the defect, while all other signs are caused by heart failure. If such symptoms appear, you should immediately consult a pediatrician and have an echocardiogram.

Minor defects have a different clinical picture—usually very subtle, and the only sign of a heart defect is a loud heart murmur. If the murmur is loud enough, it can be heard without a stethoscope; there may also sometimes be a systolic thrill in the chest area; both of these can be noticed by parents themselves. In any case, as soon as there is any suspicion of a congenital heart defect, it is necessary to immediately consult a doctor and have an echocardiogram performed to determine the underlying cause of the baby's heart defect.

Pathogenesis of Ventricular Septal Defect

A healthy heart has four chambers: the right atrium and ventricle and the left atrium and ventricle. The right chambers collect oxygen-poor blood, which then enters the lungs through the pulmonary artery and is enriched with oxygen. It then flows into the left ventricles, which, through the aorta—the largest and most important vessel in the human body—carry nutrient-rich and oxygen-rich blood throughout the body. This cycle repeats from birth until death.

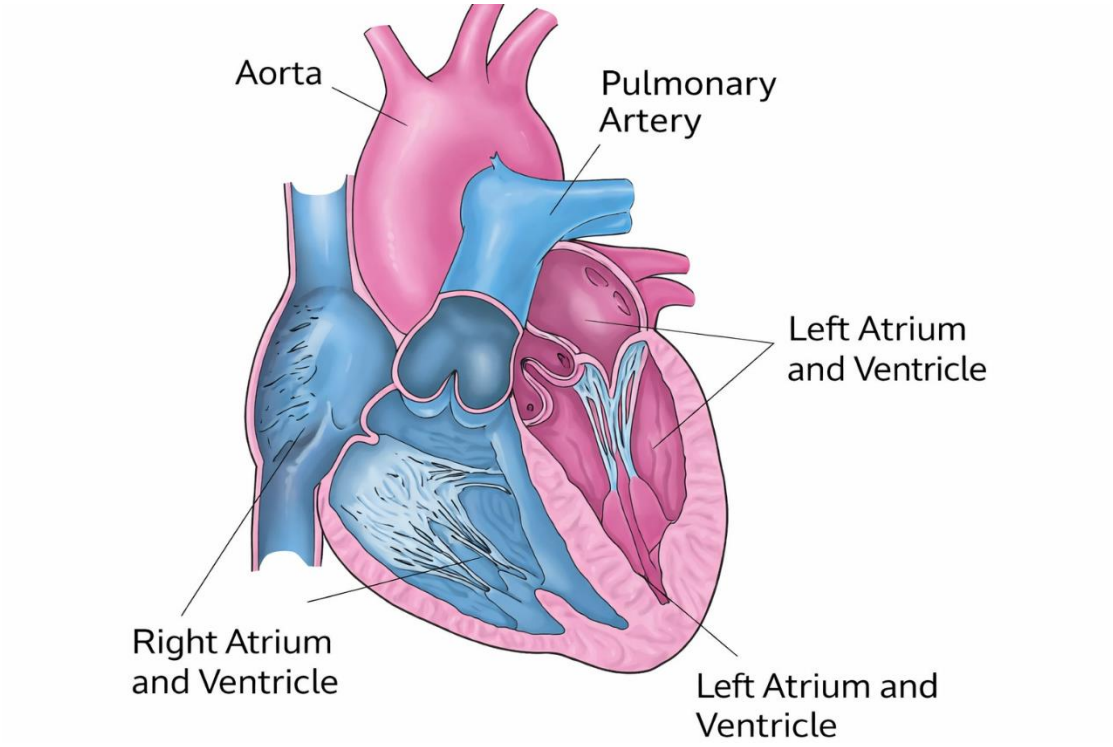

Figure 2. Structure of the heart

In the womb, the lungs play a minimal role in blood circulation and do not perform their primary function—gas exchange—at all. In this case, the mother "breathes for two": oxygen-rich blood flows from her to the fetus. Because the fetus is not breathing, the lungs are compressed, and, consequently, resistance in the pulmonary vessels is increased. Therefore, the pressure in the right ventricle is also elevated, approximately equal to the pressure in the left ventricle, preventing blood shunting through the ventricular septal defect.

When the baby is born and takes its first breath, the lungs straighten, and vascular resistance begins to decrease. After this, the right ventricle no longer needs to generate high pressure to eject blood, so it begins to drop. A pressure gradient develops between the left and right ventricles. As a result, some of the oxygen-rich blood that would normally flow into the aorta leaks into the right ventricle and back into the lungs with a

VSD. This overloads the pulmonary blood flow with additional blood volume.

As mentioned above, the larger the defect, the greater the shunt, and, consequently, the greater the blood overload in the lungs. For a time, the pulmonary vessels can accommodate the additional blood volume without any consequences. However, over time, certain changes begin to occur in their walls, which we will discuss in more detail in the "Complications" section.

Classification and stages of ventricular septal defect development

The primary classification of VSDs concerns their location within the septum itself. This classification is important for pediatric cardiologists and cardiac surgeons, as the location of the defect influences the choice of treatment and the specifics of surgical intervention.

There are four main types of defects:

1. By atrioventricular canal type;
2. Muscular defects (subdivided into posterior, middle, anterior, and apical);
3. Conoventricular defects (these include the most common membranous defects, as well as defects in the structure of more complex heart defects, such as tetralogy of Fallot or aortic arch interruption with a ventricular septal defect);
4. Conal septal defects.

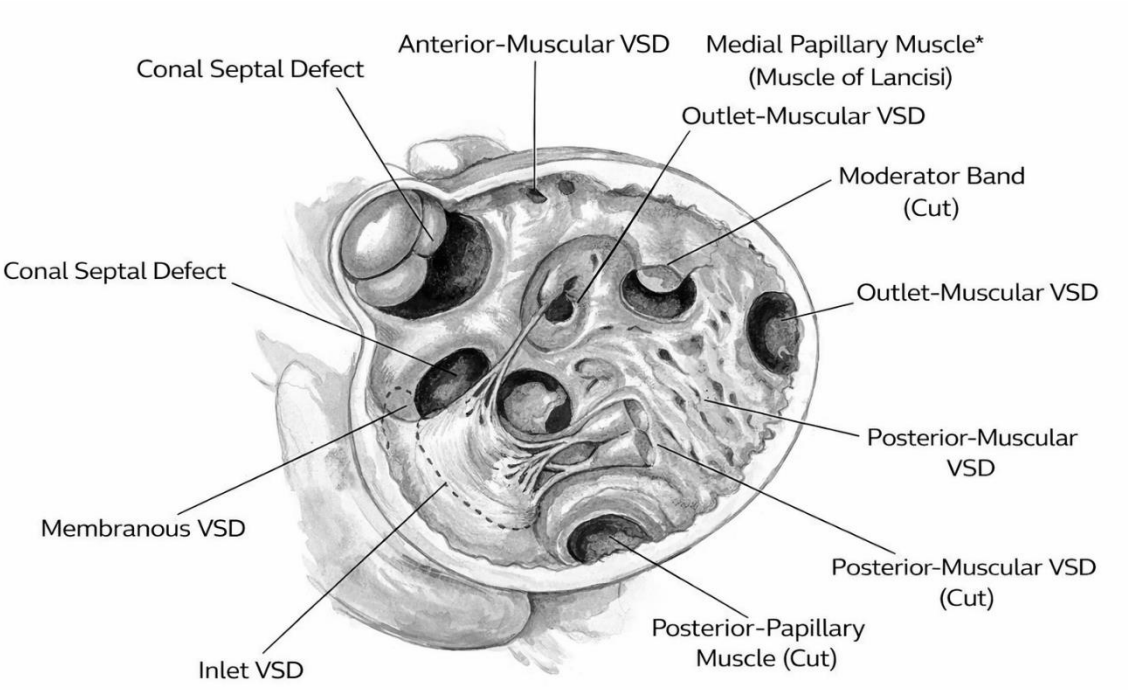

Figure 3. Types of ventricular septal defects

Conoventricular membranous defects are the most common, accounting for up to 70–80% of all VSDs. Aortic insufficiency most often develops with a conus septal defect, and slightly less frequently with conoventricular defects. AV canal-type defects may be associated with straddling of the tricuspid or mitral valve, which almost never allows surgeons to perform anatomical correction. Straddling is the attachment of papillary muscles in the "wrong ventricle," i.e., the papillary muscles of the mitral valve in the right ventricle, or the papillary muscles of the tricuspid valve in the left.

There is an additional subtype of defects called "Swiss cheese." These are multiple defects, usually muscular (or a combination of several types), that require a special approach to treatment.

Of greater interest in the course of the disease is the hemodynamic classification, which reflects the magnitude of the shunt and determines the severity of symptoms.

According to this classification, two types of defects are distinguished:

- **Small defects**—usually asymptomatic and may be discovered incidentally during a routine examination by a pediatric cardiologist at one month of age;
- **Large defects**—almost always cause significant symptoms in infants, prompting parents to consult a pediatrician.

The classification of a defect as large or small depends on the child's weight: for a 2 kg infant, a 4 mm defect can be very significant, while for a 4 kg infant, a 6 mm defect can be completely asymptomatic; for the average child (~3.2–3.5 kg), a defect larger than 6–7 mm is significant.

3. Diagnosis of Ventricular Septal Defects

Diagnosis of ventricular septal defects involves collecting a medical history, auscultation, electrocardiography, and echocardiography. Sometimes, if pulmonary vascular resistance needs to be quantified, angiography or cardiac catheterization is used.

Collection of Medical History

During the appointment, the doctor will ask about the pregnancy, whether the mother was exposed to any harmful factors (for example, living near hazardous industries, smoking, and drinking alcohol during the first eight weeks of pregnancy), and whether either parent has a history of congenital heart defects.

Auscultation

During auscultation, depending on the severity of the pressure gradient between the ventricles, a systolic murmur of varying intensity will be heard. With small defects with a high-pressure gradient, a harsh murmur (sometimes called a "machine murmur") is heard over the entire heart. With larger defects, when the pressures in the ventricles are almost equal, the murmur will be much less intense.

Electrocardiography and Echocardiography

Certain changes on the electrocardiogram may prompt the physician to consider the need for an echocardiogram. Such changes may include a left axis deviation with signs of left-sided strain.

Echocardiography is the optimal diagnostic method for most congenital heart defects, including ventricular septal defects. It allows for the precise determination of the size, location, and number of defects, as well as an assessment of the magnitude of the shunt based on the pulmonary to systemic blood flow ratio (Qp/Qs).

In most cases, these tests are sufficient to determine whether the baby is an indication for surgical treatment.

Cardiac Catheterization

Sometimes, cardiac catheterization is necessary to quantify pulmonary vascular resistance, and only then should a treatment option be selected.

Differential Diagnosis

Differential diagnosis for VSD is often performed within the diagnosis itself—the location, size, and number of defects are determined. It is also necessary to distinguish congenital ventricular septal defects from acquired ones (resulting from external trauma or myocardial infarction involving the interventricular septum). It is worth noting that such conditions are more common in adults. For example, within a week after a heart attack, a ventricular septal defect typically develops in 1–4% of patients.

4. Ventricular Septal Defect Treatment

The primary treatment for VSD is surgery. Since the vast majority of defects are conoventricular (membranous), surgery is generally performed using an open technique.

Indications for Surgery

Indications for closure of ventricular septal defects include signs of heart failure and left heart overload in infants during the first six months of life.

If there are no signs of heart failure, doctors quantify the pulmonary-to-systemic blood flow ratio (Qp/Qs). When this ratio is greater than 1.5, surgery is indicated. If the ratio is less than 1.5, the defect does not cause any significant changes in hemodynamics and is monitored. Defects (usually muscular) may close or shrink spontaneously. However, complications (aortic insufficiency or infective endocarditis) may sometimes develop during observation, which will also indicate surgical treatment. In cases of endocarditis, antibiotic or antifungal treatment may be required before surgery to reduce inflammation. If there are no indications for surgery for infective endocarditis (left-sided vegetations larger than 10 mm or critical

problems with any of the valves), antibiotic therapy is administered first. This can be empirical, i.e., broad-spectrum antibiotics pending test results, or based on cultures.

Before surgery, infants should be examined monthly until one year of age, unless an indication for surgical treatment arises sooner; after one year, the frequency of examinations is gradually reduced to once a year.

Open Surgery

Surgical interventions are typically performed through the sternum. During the main stage of the surgery, a special machine—a heart-lung machine—assists the heart and lung functions. Defects are closed with a patch, usually made of bovine pericardium, but synthetic material can also be used. Recovery after surgery depends greatly on the patient's condition at the time of surgery. On average, inpatient rehabilitation takes 10-14 days. Postoperative follow-up visits are performed first at one month, then at 3, 6, and 12 months, and then annually thereafter, unless any complaints arise.

Endovascular Surgeries

In endovascular surgeries, the defect is closed with a special device delivered through a guidewire via the femoral vein, meaning there is no need to open the chest. However, this method has some limitations: the location of the defect and the patient's weight. Such surgeries are typically performed only on muscular defects in the middle third of the septum; in some countries, similar surgeries are also performed on membranous defects, sometimes with good results, but in our country, such cases are still rare. There is also a weight limitation for the child to insert a catheter into the femoral vein without damaging it, as the delivery devices are large. The Abbott website (the manufacturer of the Amplatzer devices) states that poor results have been observed in patients weighing less than 5.2 kg, so endovascular surgery is not recommended for these children. It also states that it is contraindicated in patients with a distance from the defect edge to the valves of less than 4 mm.

In practice, this technique is not very often used, as the vast majority of defects are membranous (having edges on the aortic and tricuspid valve annulus). Few surgeries are performed on the remaining defects suitable for endovascular closure.

Surgeries for Swiss Cheese Defects

With "Swiss cheese" defects, it is quite difficult to immediately close all the defects in a child under one year of age. Surgeries for such defects are lengthy and traumatic for the heart, so children of this age have poor survival rates after primary radical repair of Swiss cheese VSDs. As the child gains weight, both the work inside the heart (finding all defects and ensuring their proper closure) and post-operative care become easier.

There is a two-stage correction technique, which first involves surgery to narrow the pulmonary artery. A special strip of synthetic fabric, called a cuff, is used for this. The goal of this surgery is to reduce the overload of the pulmonary vessels with additional blood volume and prevent the development of heart failure. As the child grows and gains weight, the cuff is removed and multiple defects are closed simultaneously.

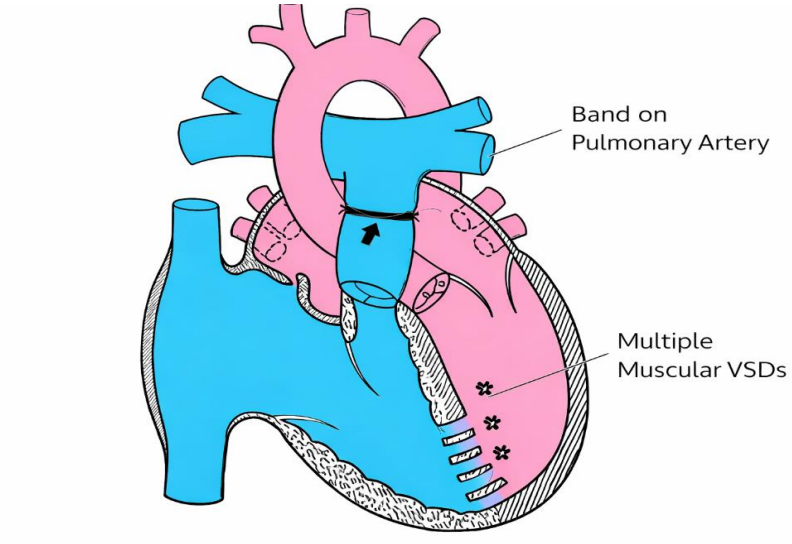

Figure 4. Heart with multiple muscular VSDs after pulmonary artery stenosis

Postoperative Complications

Potential complications are most often directly related to the procedure itself. Postoperatively, temporary or permanent atrioventricular block may develop, requiring the insertion of a pacemaker. There is also a risk of developing valve dysfunction (most commonly aortic or tricuspid) as a result of patch placement or occluder insertion.

If plastic surgery is possible, it is always preferred when valve dysfunction develops in childhood; otherwise, various types of valve replacements are performed, including mechanical/biological prostheses and autografts (Ross procedure).

Prognosis. Prevention

Small defects tend to close spontaneously in 20-30% of cases. Some relatively large defects may also spontaneously close or shrink. If the VSD does not close but does not cause serious hemodynamic disturbances, periodic monitoring is required (once a year for children over one year old who have no complaints).

The mortality rate during or after surgery is less than 1%. After surgical treatment, the life expectancy and quality of life of patients are the same as in healthy individuals [15].

There is no specific prevention for congenital heart defects. Maternal health, abstinence from bad habits, and avoidance of hazardous environmental factors and viral infections are all non-specific recommendations, and even following them cannot guarantee a 100% result.

CONCLUSION

Heart failure development after surgical repair of congenital ventricular septal defect (VSD) remains a clinically significant postoperative complication despite advances in pediatric cardiac surgery and perioperative management. Although surgical closure effectively eliminates the pathological left-to-right shunt and prevents progressive pulmonary hypertension, it does not completely eliminate the risk of postoperative myocardial dysfunction. Residual hemodynamic burden, preoperative ventricular remodeling, ischemia-reperfusion injury during cardiopulmonary bypass, and arrhythmogenic factors may contribute to transient or persistent heart failure in the postoperative period.

This study emphasizes that early and accurate diagnosis of heart failure in children following VSD repair requires a multimodal, structured diagnostic approach rather than reliance on a single parameter. Clinical evaluation alone is insufficient due to the nonspecific nature of symptoms in pediatric patients. Therefore, integration of:

- Serial biomarker monitoring (particularly NT-proBNP),
- Comprehensive echocardiographic assessment (including systolic and diastolic indices),
- Hemodynamic monitoring when indicated,
- Advanced imaging modalities in complex cases,

significantly increases diagnostic sensitivity and specificity.

Importantly, dynamic monitoring over time is more informative than isolated measurements. Trends in NT-proBNP levels, progressive ventricular dilation, or subtle reductions in ejection fraction may precede overt clinical deterioration. Early identification enables timely initiation of pharmacologic therapy (e.g., ACE inhibitors, diuretics, beta-blockers when indicated), optimization of fluid management, and prevention of long-term ventricular remodeling.

The implementation of a standardized postoperative diagnostic algorithm provides several advantages:

1. Reduction in delayed heart failure diagnosis.
2. Improved risk stratification of high-risk patients.
3. Shortened hospital stay through earlier intervention.
4. Decreased readmission rates.
5. Better long-term myocardial functional preservation.

Furthermore, incorporation of emerging diagnostic technologies such as myocardial strain imaging (speckle-tracking echocardiography) and cardiac MRI-derived functional parameters may allow detection of subclinical myocardial dysfunction before traditional parameters (e.g., LVEF) decline.

Future research should focus on:

- Identification of predictive risk factors for postoperative heart failure,
- Development of pediatric-specific biomarker cut-off values,
- Long-term follow-up studies evaluating ventricular remodeling trajectories,
- Artificial intelligence–assisted diagnostic algorithms for postoperative monitoring.

In conclusion, postoperative heart failure following VSD correction represents a multifactorial condition requiring early recognition and systematic evaluation. Adoption of evidence-based diagnostic pathways and continuous postoperative surveillance significantly improves clinical outcomes and supports favorable long-term cardiac function in pediatric patients.

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