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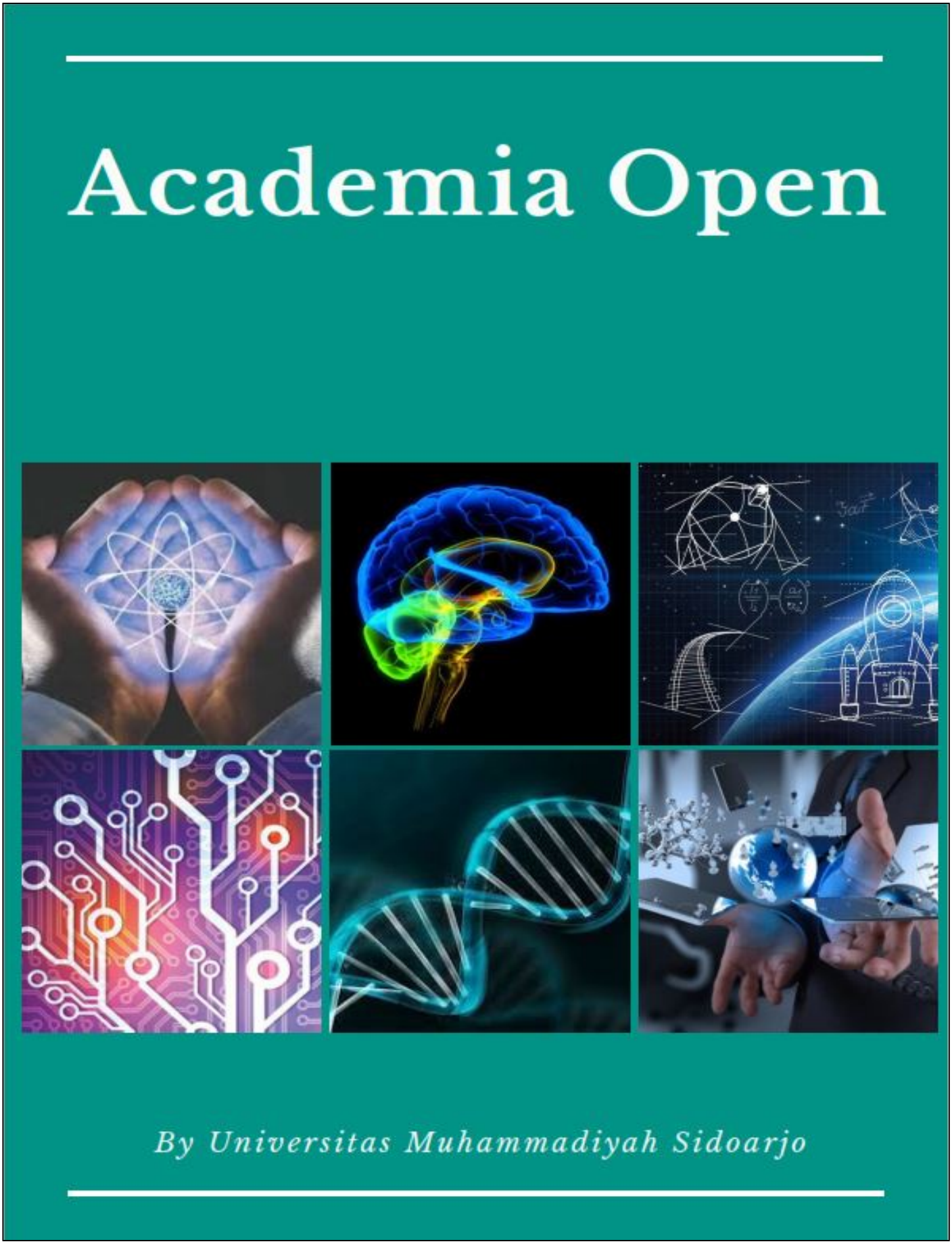

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Ventricular Septal Defect: A Review Article

Cacat Septum Ventrikel: Artikel Ulasan

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Abstract

Of all congenital heart abnormalities, up to 40% are caused by anomalies in the ventricle septum. A wide variety of abnormalities are included in the diagnosis, including those linked to other congenital cardiac deformities and solitary problems. The age of the patient, the size and anatomical correlates of the defect, and the degree of diagnostic and interventional expertise in the field all influence the presentation, signs, course, and treatment of abnormalities in the ventricular septum.

Highlights:

1. Cause & Diagnosis: Ventricular septal anomalies cause 40% of congenital heart defects.
2. Influencing Factors: Age, defect size, anatomy, and medical expertise affect symptoms.
3. Treatment & Course: Diagnosis and intervention determine management and patient outcomes.

Keywords: Ventricular Septal Defect, Review

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Introduction

The development of transcatheter procedures to treat perimembranous lesions has generated a lot of attention. Due to the intolerable risk of post-procedure heart block linked to readily available devices, most units no longer perform this technique. Given the prevalence of late-onset heart block, it is particularly troubling that this risk does not diminish or disappear with time [1]. Since many minor birth defects go away quickly, the frequency of this defect varies with age upon evaluation. It also depends on how sensitive the examination method is. Using highly sensitive color Doppler echocardiography for screening has revealed a prevalence of up to 5% in newborns. The majority are minor muscle abnormalities that go away in the first year of life [2].

Depending on the method of diagnosis and population age, the exact prevalence of ventricular septal defect differs throughout research because many patients may not exhibit any symptoms, and many abnormalities resolve over time. Compared to earlier research that depended on either clinical examination or post-mortem examinations, papers that employed echocardiography in the diagnosis algorithm have reported a prevalence of up to 3·94 per 1000 patients [3, 4].

Genetics and Cause

Our comprehension of the processes that result in normal cardiac septation limits our ability to comprehend the causes of ventricular septal defects. According to available data, the septum contains both muscular and mesenchymal components [5]. The conotruncal and atrioventricular endocardial cushions fuse to form the mesenchymal element. Less is known about the mechanisms that start the muscular septum's development, it develops passively inwards as the ventricular cavities deepen [6]. Another idea states that the primordial interventricular septum, a collection of cells, actively extends outward toward the atrioventricular canal cushions to form the muscular septum [7].

Ventricular septal defects and the majority of other congenital heart diseases have several underlying causes [8, 9]. In certain instances, monogenic flaws are causal. The molecular characterization of these anomalies has sparked a lot of interest since it has made it easier to identify key components of the signaling networks that control heart development [10-12]. Special emphasis has been paid to mutations in the transcription factors GATA4 and TBX5. The heart coexpresses these factors, and their interplay is essential for healthy cardiac septation [13]. The autosomal dominant Holt-Oram syndrome is linked to the most often reported mutation in this transcription factor [14, 15] characterized by several heart defects, including ventricular septal defect, and anomalies of the forelimbs. In the Chinese Han population [16]. GATA4 sequence variations have been found in some patients with random ventricular septal defects and familial forms of septal abnormalities, especially atrial [17-20].

Ventricular septal defects have been linked to environmental variables such as untreated metabolic disorders, maternal infections, and teratogens in mothers (such as pregestational diabetes and phenylketonuria) [21]. Purely stochastic events may also play a significant part. Because cardiac development is so complex, it must be completed with precision [22, 23].

Anatomy

Among the less complicated types of congenital cardiac abnormality. But there isn't a consensus on how to categorize it [24-27]. Defects can often be categorized based on where they are found: along the edges of the muscular septum or inside it (muscular defects). Atrioventricular valve (perimembranous) or arterial valve (juxta-arterial or subarterial) leaflet hinge points may be connected to ventricular septal defects at the edges. In a healthy heart, doubly committed and juxta-arterial anomalies are found in the muscular infundibulum. If this region is abnormal, the aortic and pulmonary valves will show recognizable continuity [28].

Pathophysiology

The systemic and pulmonary vascular beds' relative resistances are important factors that determine the resulting interventricular flow and symptoms when the lesion is non-restrictive. Crucially, this relationship can be highly erratic and contingent, especially on the patient's age. Due to the high pulmonary vascular resistance that characterizes the early newborn period, neonates may initially have relatively big abnormalities with minimal left-to-right shunting [29]. Due to ventricular septal defects, the normal postnatal decline in pulmonary vascular resistance may be caused or delayed [30, 31].

Eisenmenger's syndrome is linked to structural and functional changes in the pulmonary vasculature and is caused by persistent increases in pressure and flow [32, 33]. Important functional alterations include elevated pulmonary vasoreactivity and resistance as well as structural microvascular alterations, such as medial enlargement, and smooth muscle migration distally into normally un-muscularized microvessels [34, 35]. Every stage of this process

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is influenced by abnormalities within the endothelium. Both physically and functionally [35, 36] and in clinical conditions [37].

Patients with VSD may experience significant clinical changes as a result of secondary structural heart abnormalities. Since these anomalies may have an impact on clinical management, ongoing monitoring of all impacted individuals is necessary to track their development. Aortic valve prolapse and regurgitation can exacerbate malformations surrounding the aortic valve (muscular, perimembranous, or doubly committed). These circumstances result from the development of Venturi forces, wherein the aortic valve leaflet is drawn into the restrictive defect by the high-velocity jet [38].

Muscular band expansion leads the ventricle to become blocked in the middle, leading to the condition known as double-chambered right ventricle [39]. In some situations, the outflow septum may exhibit a small anterior deviation. According to published studies [40].

Diagnosis

Patients with modest ventricular septal abnormalities may have normal ECG readings. The left ventricle's volume loading may result in left ventricular hypertrophy. Ventricular septal problems can now be accurately detected with cross-sectional echocardiography [41-43]. The use of spectral and color Doppler imaging in conjunction with two-dimensional (2D) echocardiography greatly facilitates the identification and characterization of ventricular septal abnormalities [44-46]. Accurate measurements of the pressures in the left and right ventricles, pulmonary arteries, and right ventricle can frequently be obtained using 2D-directed [47].

Since there may be clinically significant abnormalities of the aorta, particularly coarctation, as well as pulmonary arteries, pulmonary veins, and systemic veins, the echocardiographer should additionally evaluate extracardiac vascular structures. Transoesophageal echocardiography makes it much easier to confirm repair and identify and correct any residual lesion early on, it has become a crucial tool in the intraoperative evaluation of ventricular septal defects [48, 49]. With its increasing accessibility, three-dimensional echocardiography may offer crucial diagnostic support for evaluating ventricular septal defects in odd locations as well as those linked to intricate congenital cardiac abnormalities [50].

Before and after surgery, MRI is being utilized more and more to evaluate patients with various types of congenital cardiac disease. MRI may be helpful, especially in patients with subpar echocardiographic pictures, even if clinical examination and echocardiogram can provide sufficient diagnostic information for the majority of people with ventricular heart abnormalities [51].

Ventricular Septal Defect Closure

Surgery

VSD has been repaired via sternotomy and cardiopulmonary bypass for nearly 50 years. A low rate of operational mortality and an uncommon occurrence of significant postoperative morbidity are the results of improved patient selection, early surgery, and advancements in perioperative care [52]. The semilunar or atrioventricular valves are usually used to gain access to the defect to avoid ventriculotomy. If the tricuspid valve's anterior and septal leaflets are momentarily separated during surgery, some patients may be more susceptible to the problem [53]. Although there may be behavioral and intellectual problems in early childhood [54].

As heart surgery becomes more common in developing nations [55], More focus has been placed on methods to correct anomalies in patients who have elevated pulmonary vascular resistance after not having access to surgery during infancy. In these high-risk patients, surgical closure can dramatically lower morbidity and death because right-ventricular failure is brought on by rises in both right-ventricular and pulmonary-arterial pressure [56]. Even if some have questioned the advantages of this method over traditional closure [57].

Transcatheter Closure

Transcatheter methods for sealing ventricular septal defects have been developed throughout the last ten or so years [58]. These methods have been especially beneficial for anomalies of the muscles, which might be the most difficult to evaluate medically [59]. There has been a lot of interest in the development of transcatheter techniques to treat perimembranous lesions. The majority of units no longer employ this technique. Given the prevalence of late-onset heart block, it is particularly troubling that this risk does not diminish or disappear with time [60].

Hybrid Techniques

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A hybrid closure procedure that combines surgery and interventional modalities has been used for babies with muscular ventricular septal abnormalities, for whom traditional surgery and transcatheterization present difficulties. Using this method, a sternotomy is performed as usual [61].

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