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Ebstein Anomaly

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ABSTRACT: The rare congenital heart condition known as Ebstein anomaly (EA) can manifest in a variety of ways. Ebstein anomaly is a rare congenital cardiac defect, accounting for approximately 1% of all congenital heart disease. It is characterized by malformations in the tricuspid valve especially failure of valve leaflet delamination and variable degrees of apical displacement of the septal and posterior leaflets, which lead to arterialization of part of the right ventricle and significant tricuspid regurgitation. The anatomical and functional heterogeneity in right ventricular morphology contributes to a broad clinical spectrum, from severely symptomatic neonates to asymptomatic adults. Prognostic factors associated with worse outcomes include severe tricuspid regurgitation, marked right ventricle dilation, reduced right ventricular function, presence of associated defects, and late age at intervention. For asymptomatic patients, medical therapy and observation are frequently advised, and they may be effective for many years. Emerging areas in research include optimal timing of intervention, refinement in imaging-based prognostication, regenerative therapies, and strategies to mitigate right ventricular myopathy. This review synthesizes current knowledge of Ebstein anomaly—from pathophysiology, diagnosis, and imaging, to surgical and medical management—and outlines future directions to improve outcomes in this heterogeneous patient population.

KEYWORDS: Ebstein abnormally, Congenital, Arterialization, heterogeneous, Right ventricle dilation

I. INTRODUCTION

About 1 in 200,000 live newborns have Ebstein anomaly (EA), a rare congenital heart condition that was discovered in 1866. Depending on the degree of the disease, this ailment, which is present from birth, exhibits a range of symptoms throughout life. The tricuspid valve (TV) in a healthy heart has three leaflets: the anterior, posterior, and septal. [1, 2, 3] The entire right ventricle is afflicted by Ebstein's abnormality. The apical displacement of the valve, the aberrant distal attachment of the leaflets, the size of the functional right ventricle, the degree of tricuspid regurgitation, and changes in the left ventricle are some of the defects that make up this spectrum. Approximately 0.3 to 0.7% of patients with congenital cardiac disease have it.[4] Ebstein anomaly (EA), a rare congenital heart disorder identified in 1866, affects around 1 in 200,000 live births. This condition is present from birth and manifests a variety of symptoms during the course of life, depending on the severity of the sickness. The anterior, posterior, and septal leaflets make up the tricuspid valve (TV) of a healthy heart. Ebstein's anomaly affects the entire right ventricle. Defects that comprise this spectrum include the size of the functional right ventricle, the degree of tricuspid regurgitation, the abnormal distal attachment of the leaflets, the apical displacement of the valve, and alterations in the left ventricle. It is seen in between 0.3 and 0.7% of congenital heart disease patients [5].

II. HISTORY

Tricuspid regurgitation, apical displacement of the tricuspid valve, and occasionally an elevated cardiothoracic ratio are indicators of Ebstein malformation that can be identified in utero. [6]

At 20% and 45%, respectively, the rates of fetal death and perinatal death are high. Fetal echocardiographic parameters linked to increased perinatal mortality were found to be a gestational age of less than 32 weeks at diagnosis, a cardiothoracic area ratio of greater than 0.47 ± 0.12, a larger tricuspid annulus diameter by z-score, the presence of pulmonary regurgitation, and pericardial effusion, according to a large multicenter study. [7] Early childhood heart failure symptoms may include a noticeable v-wave in the jugular venous pulse from severe tricuspid regurgitation; torrential tricuspid regurgitation may not exhibit this finding because of the quick equalization of pressures across the tricuspid valve. [8] Exertional dyspnea symptoms are frequently seen in elderly people. Hypoxemia resulting from right-to-left

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shunting can cause dyspnea. Other clinical signs of heart failure include lower extremities edema and chronic tiredness. On rare occasions, paradoxical emboli over an intracardiac shunt might cause patients to present with myocardial infarction, stroke, or brain abscess. [9.10]

III. PATHOPHYSIOLOGY

Anatomic Abnormalities

Another way to define this apical displacement is as a rotating displacement of the tricuspid leaflets in the direction of the RV outflow tract. The septal leaflet's displacement from the anatomic tricuspid annulus is often characterized as greater than 8 mm/m2. The tricuspid valve's anterior leaflet frequently has aberrant chordal attachments and becomes hypermobile, or "sail-like." Alternatively, the anterior leaflet may be tethered, resulting in limited mobility. [10,11,12] This apical displacement can also be described as the tricuspid leaflets turning in the direction of the RV outflow tract. The displacement of the septal leaflet from the anatomic tricuspid annulus is frequently described as being larger than 8 mm/m2. The anterior leaflet of the tricuspid valve often has abnormal chordal attachments and becomes hypermobile, or "sail-like." As an alternative, the anterior leaflet might be tied, which would limit its range of motion. [13-14] While a tiny percentage of people with less severe abnormalities present in maturity, the majority are diagnosed in infancy or childhood. As a result of TR, neonates typically exhibit cyanosis and cardiac failure. Exertional dyspnea, exhaustion, cyanosis, and palpitations are common symptoms in children, adolescents, and adults, though many may not notice any of these. Interatrial communication may be the secondary cause of paradoxical embolization. [15]

Classification

The results of echocardiographic measurements that calculated the ratio of the combined area of the right atrium and arterialized RV to that of the functional RV and left heart in a four-chamber view at end diastole, the standard classification of Ebstein's anomaly was as follows: Grade 1, ratio < 0.5; Grade 2, ratio 0.5–0.99; Grade 3, ratio 1–1.49; and Grade 4, ratio > 1.5. A greater grade, or increasing severity, was linked to a higher death rate. The following categories were determined using the Carpentier classification: Type A, which had a small arterialized RV and minimal septal leaflet attachment displacement; Type B, which had a large arterialized RV and moderate septal leaflet attachment displacement; Type D: tricuspid sack; Type C: significant displacement of septal and posteroinferior leaflet attachment with short muscles, nonatrialized or dyskinetic atrialized RV, and reduced anterior leaflet motion. [16]

Clinical features

Cyanosis, right-sided heart failure, arrhythmias, and sudden cardiac death are the hallmarks of Ebstein's heart defect. The degree of right-to-left interatrial shunting, histology, anatomic severity, and age at presentation all affect the hemodynamic changes and clinical presentation. [22] Despite significant regurgitation of the tricuspid valve, the jugular venous pulse rarely exhibits a large V wave upon examination because the additional volume is taken in by the massive right atrium. Typically, there are multiple other noises in along with a heart sound that is broadly constant for every split second. It is possible to hear a systolic murmur. Clubbing digitally depends on the level of cyanosis. [17]

Diagnostic evaluations:

Echocardiography

Heart catheterization has mostly been spared thanks to echocardiography, the preferred diagnostic procedure for Ebstein's abnormality. Accurate assessment of the tricuspid valve leaflets as well as the shape and function of the heart chambers is made possible by ultrasound. [18,19]. When the combined area of the right atrium and atrialized right ventricle is bigger than the combined area of the left atrium, left ventricle, and functional right ventricle as measured in the apical 4-chamber view at end diastole, there is a visible enlargement of the right atrium and atrialized right ventricle [20]. (Figure -3)

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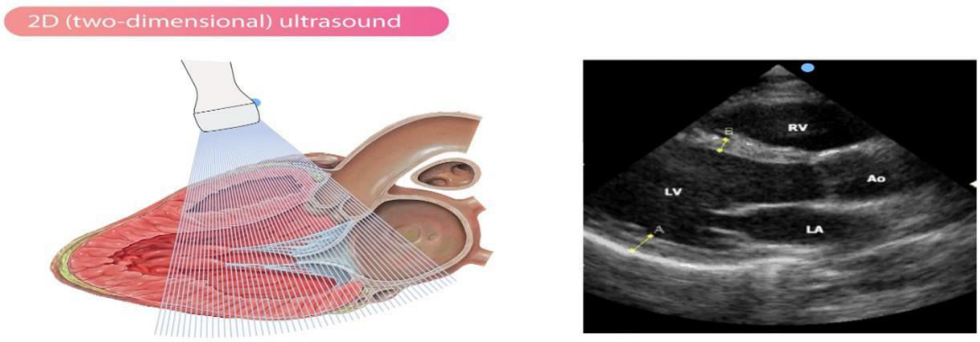

Figure -3:A picture of an echocardiography (4-chamber view, apex down) displaying a severely incorrect septal leaflet (arrow) in a patient with severe Ebstein's abnormality. The anterior leaflet is almost immobile and heavily tethered. The right ventricle (RV) is small while it is operating. LA means left atrium; LV for left ventricle; RA for right atrium; and ARV for cardiac right ventricle.

IV. THE PRACTICE OF ELECTROCARDIOGRAPHY

Most individuals with Ebstein's anomaly have an abnormal ECG. In addition to total or partial right bundle-branch block, it can show tall and broad P waves due to right atrial growth. [21] Leads V1 and V2 have tiny R waves. Abnormal activation of the atrialized right ventricle and change of infra-Hisian conduction result in strange morphologies of the terminal QRS pattern. [22] Figure 4 exhibits a typical ECG.

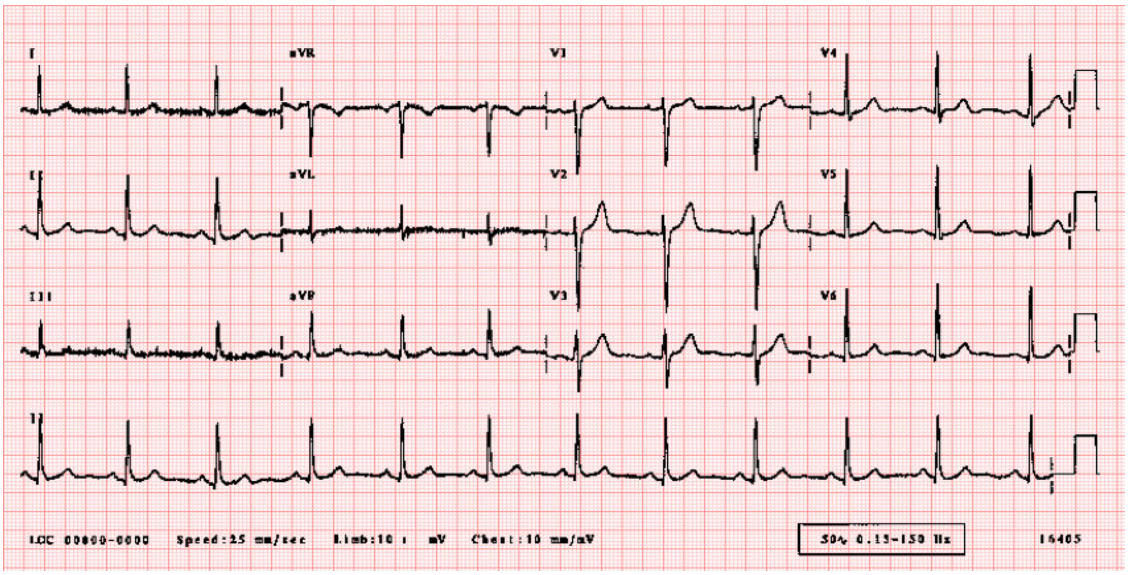

Figure 4: The ECG of a patient with severe Ebstein's anomaly shows the usual defects, namely a right bundle-branch block, an extended PR interval (226 ms), and a somewhat abnormal QRS complex shape.

Management

Medical treatment: Ebstein's anomaly patients who do not exhibit symptoms can be closely watched. Moderate therapy is an option for patients in New York cardiac Association cardiac function Classes I and II. Exercise restriction and changes to heart rate and preload are part of the treatment for right heart failure. Digoxin, oral diuretics, low-dose angiotensin-converting enzyme inhibitors like enalapril, and a low-sodium diet are a few among these. Diuretics, vasodilators, diuretics, low-sodium diets, and abdominal paracentesis were the mainstays of treatment for acute cases of right heart failure. These treatments can be useful in reducing congestive heart failure, but they are not highly effective at preventing effusions. [23]

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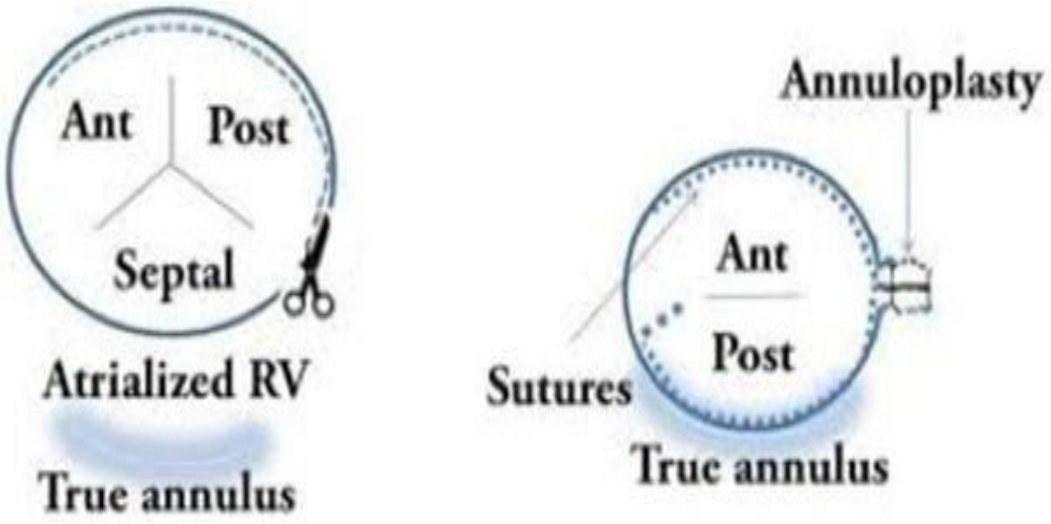
. Supporting the FRV is the aim of medical care in order to aid in the antegrade pulmonary flow. As the PVR steadily declines, this usually gets better. In assessing antegrade pulmonary blood flow, serial echocardiograms are helpful. Maintaining ductal patency requires prostaglandin, especially in newborns with pulmonary atresia. Conversely, in cases of functional pulmonary atresia, a prostaglandin withdrawal trial might be needed. Likewise, an early withdrawal might be required in a circular shunt. One study found that if anatomic blockage was ruled out, early prostaglandin withdrawal reduced mortality (24)

Surgical interventions: The majority of patients may profit from a biventricular repair that involves reconstruction of a competent monocuspid tricuspid valve, right ventriculorrhaphy, subtotal ASD closure, and aggressive reduction atrioplasty, as advised by Knott-Craig et al. Patients with poor RV function should consider a 1.5-ventricular repair (bidirectional Glenn shunt), and those with severe left ventricular dysfunction need to look at heart transplantation. The surgical techniques that Danielson's team reported. [25,26]

. It comprised right atrial reduction plasty, posterior abdominal annuloplasty, and transverse plication of the atrialized RV. By releasing the anterior tricuspid leaflet from its in situ position, an active monocusp valve was created as the basis for the tricuspid valve annuloplasty. It is known how to remove the anterior and posterior tricuspid valve leaflets from their abnormal attachments in the RV, move the free edge clockwise, and then surgically suture it to the anterior leaflet's septal border as the “cone” operation figure-1. [27]



In addition to correcting Ebstein's anomaly, the idea of a 1.5-ventricle repair has been expanded to include other abnormalities such straddling AV valves and abnormal AV septal defects. Hemi-Fontan or bidirectional Glenn shunt operations can be done with the goal of decreasing RV volume load and supplying or increasing pulmonary artery flow. RV preload and workload can be reduced by moving blood flow from the superior vena cava to the pulmonary arteries. In people with compromised RV function and Ebstein's abnormality,

For patients with severe Ebstein's anomaly and an impaired RV who are at high risk for standard surgical treatment, the 1.5-ventricle repair may be an alternative. The dual Glenn shunt may facilitate the surgical treatment by unloading the RV and providing preload to the left ventricle. [28,29] In the example of a neonate with severe Ebstein's anomaly, a surgical procedure requiring tricuspid valve and pulmonary artery closures, residual AV communication that is minimal for decompressing the RV when closing this valve, and construction of cardiovascular artery shunt, then hemi-Fontan and closed Fontan treatments in their terminal stages. A modified method for bidirectional cavopulmonary shunts involves creating a blind connection between the right pulmonary artery and the opening roof of the right atrium (figure 2). [30, 31]

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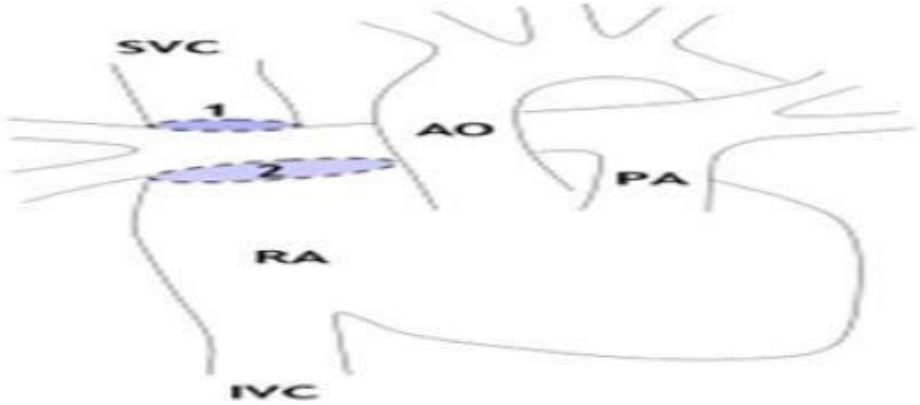

V. DISCUSSION

Ebstein's anomalous follow-up and clinical diagnostic results have been thoroughly documented in numerous communications. [32] A precise diagnosis using a noninvasive method would be helpful due to the previously documented high frequency of cardiac arrhythmias during catheterization and angiography. The early systolic sound, a characteristic of Ebstein's abnormality that was previously ascribed to either tricuspid closure [33] or pulmonary ejection [34], must be precisely timed.

Catheterization data were compared with echocardiographic and phonocardiographic results. The early systolic sound and the point of pressure development shift in the right ventricular pulse pressure were both correlated with the posterior position of the tricuspid valve echo. The synchronicity of the atrial and ventricular pulse pressure waves indicated that the tricuspid valve had closed by the time the early systolic sound occurred. The tricuspid valve leaflets are forced posteriorly toward the atrium when the right ventricle begins to contract and its pressure rises. The systolic sound is produced at this posterior limit of excursion. At this point, the right ventricular pressure exhibits a sudden increase in pressure development. By looking at the shape of the pulmonary artery pulse pressure, one can also observe these two stages of pressure development. [35]

VI. CONCLUSION

Rare is Ebstein's oddity. MYH7 and NKX2.5 mutations, among others, may be linked to the genetic basis of this congenital cardiac defect. When there is evidence of right heart dilatation and increasing impairment of ventricular systolic function, operation is advised; otherwise, asymptomatic patients with Ebstein's condition can be conservatively treated and closely followed. Even though 1.5-ventricular repair (bidirectional Glenn shunt) is an option for patients with poor RV function, heart transplantation is used for patients with severe left ventricular dysfunction. Most patients benefit from a biventricular repair consisting of reconstruction of a competent monocuspid tricuspid valve, right ventriculorrhaphy, subtotal ASD closure, and aggressive reduction ventricular remodeling. The outcome for people with Ebstein's abnormality may be improved by closely following the surgical indications.

In sum, while progress in imaging, surgical methods (notably the cone repair), and understanding of prognostic markers have significantly improved the care of patients with Ebstein’s Anomaly, there is still a substantial burden of disease, particularly in severe neonatal cases. Future research should aim to refine genotype-phenotype correlations, improve long-term surgical outcomes, and develop individualized management strategies to maximize both survival and quality of life.

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