Case Report

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A Case Study Showing the Actual Presentation of A Child Having Ventricular Septal Defect

Nirupam Nisha Sahu

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Abstract

Congenital Heart Disease is the major cause of death in the first year of life. Although there are more than 35 well recognised cardiac defects, the most common is ventricular septal defect. The article depicts a case study of a 3 ½ month old male child presenting with the signs and symptoms of ventricular septal defect. The importance of History taking, physical assessment, rapid response to findings with time-constrained empirical interventions, the consideration of access to health care, and a holistic approach to treatment of the patient and the family are highlighted.

Keywords: Ventricular septal defect; Failure to thrive

Introduction

Ventricular septal defects (VSD) represent the most common form of congenital heart disease. Ventricular septal defect, a hole in the heart is a common heart defect that's present at birth (congenital). The hole occurs in the wall (septum) that separates the hearts lower chamber and allows blood to pass from the left to the right side of the heart. The oxygen-rich blood then gets pumped back to the lungs instead of out to the body, causing the heart to work harder [1]. My client 2 & ½ yr old is suffering from Ventricular septal defect by birth but diagnosed at 2 month of age after he suffered from recurrent respiratory infection.

Case Report

A baby boy 2 ½ month old was admitted to some private hospital for treatment of Lower respiratory tract infection. From there he was referred to higher centre for further management. There he got cured but diagnosed as a case of ventricular septal defect after certain examination and further transferred to higher centre for management of cardiac anomaly. Baby after being received in higher centre was dull thin built, irritable, looking pale, undernourished having a weight of 3.4 kg, Heart rate 100–120 beats/ min., para systolic murmur is heard, Respiration 35 breaths/ minute also bilateral ronchi present, blood pressure 80/60 mm of Hg, SpO_2 92–93 mm of Hg and maintaining 100% @ 2 litres of oxygen, looking unhygienic. Certain investigation done as X-ray chest-reveals lower respiratory tract infection, 2 D –Echo reveals-left to right shunt, moderate size multiple, mid and apical muscular Ventricular septal defect, Hb 7.8 gm/dl, TLC4.37/mm^3 rest all parameters are within normal limit. He was on Syp. Dixin 15 drops/PO OD. Cardiac Consultants planned for surgical repair of ventricular septal defect after adequate weight is gained as the child birth weight was 3.3 kg. Also baby is now having poor appetite, oxygen saturation falls soon while feeding, low pitched voice. So, they planned for improving the appetite by slowly increasing the feeds, inserted nasogastric tubing to maintain feeds, strict intake output charting is done and hygiene maintained. Meanwhile he had aspiration pneumonia but was successfully managed by the health care team. After a month also baby's weight remained 3.6 kg, not improving

Author Affiliation: [1]Nursing Tutor, College of Nursing, All India Institute of Medical Science, Raipur, Chhattisgarh 492099, India.

Corresponding Author: Nirupam Nisha Sahu, Nursing Tutor, College of Nursing, All India Institute of Medical Science, Raipur, Chhattisgarh 492099, India.

E-mail: nitu.sahu2014@rediffmail.com

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to that level, developed congestive heart failure, still SpO_2 maintained with oxygen@ 2 litres/ min., pedal oedema present. Currently managing symptomatically.

Discussion

Ventricular septal defect (VSD) is defined as a hole in the septum (the wall) between the lower chambers of the heart (the ventricles) [2]. VSD is an acyanotic congenital heart defect, aka a left-to-right shunt, so there are no signs of cyanosis in the early stage [3]. My client was also not having cyanosis only pallor discolouration was there all over the body. Ventricular septal defect (VSD) symptoms in a baby may include: Poor eating, failure to thrive, Fast breathing or breathlessness, Easy tiring [1]. My client present with all these symptoms. Congenital VSDs are frequently associated with other congenital conditions, such as Down syndrome [4]. In my client disease is not associated with Down's Syndrome. Genetics and environmental factors may play a role. VSDs can occur alone or with other congenital heart defects [1]. My client is suffering from Ventricular septal defect alone and the cause is not known.

Pathophysiology : During ventricular contraction, or systole, some of the blood from the left ventricle leaks into the right ventricle, passes through the lungs and reenters the left ventricle via the pulmonary veins and left atrium. This has two net effects. First, the circuitous refluxing of blood causes volume overload on the left ventricle. Second, because the left ventricle normally has a much higher systolic pressure (~120 mmHg) than the right ventricle (~20 mmHg), the leakage of blood into the right ventricle therefore elevates right ventricular pressure and volume, causing pulmonary hypertension with its associated symptoms. In serious cases, the pulmonary arterial pressure can reach levels that equal the systemic pressure. This reverses the left to right shunt, so that blood then flows from the right ventricle into the left ventricle, resulting in cyanosis, as blood is by-passing the lungs for oxygenation [5]. In my client left to right shunt is seen. Diagnosis A VSD can be detected by cardiac auscultation. Classically, a VSD causes a pathognomonic holo-or pansystolic murmur. Confirmation of cardiac auscultation can be obtained by non-invasive cardiac ultrasound (echocardiography). To more accurately measure ventricular pressures, cardiac catheterization, can be performed. My client was diagnosed via cardiac auscultation and echocardiography.

Classification: Multiple, Type 1-Type 1 is sub aortic, Type 2-Type 2 also known as perimembranous, paramembranous, conoventricular, membranous septal defect, and subaortic, Type 3-Type 3 also known as inlet (or AV canal type). Type 4-Type 4 also known as muscular (trabecular), Type: Gerbode-Type: Gerbode also known as left ventricular to right atrial communication [6]. My client is having Type 4 VSD. Alberto Cresti, Raffaele Giordano etal conducted a study to evaluate the incidence and natural history of isolated VSDs and the findings are; Out of 343 newborns with an isolated VSD (incidence of 10.45/1000/births) account for 64% of all detected CHDs. VSDs location were as follows: muscular (73.8%), perimembranous (11.3%), inlet (1%), and outlet (0.8%). Of the located VSDs, 90% were small, 7.5% moderate, and 2.5% large, respectively [6].

Treatment: Most cases do not need treatment and heal during the first years of life. Treatment is either conservative or surgical. Smaller congenital VSDs often close on their own, as the heart grows, and in such cases may be treated conservatively. Some cases may necessitate surgical intervention, i.e. with the following indications: 1. Failure of congestive cardiac failure to respond to medications 2. VSD with pulmonic stenosis 3. Large VSD with pulmonary hypertension 4. VSD with aortic regurgitation. A nitinol device for closing muscular VSDs, 4 mm diameter in the centre. For my client conservative management is done [8] Epidemiology: VSDs are the most common congenital cardiac abnormalities. They are found in 30–60% of all newborns with a congenital heart defect, or about 2–6 per 1000 births [5, 7].

Conclusion

My client 3 & ½ months old baby boy was suffering from Type 4 ventricular septal defect, having frequent respiratory infections not maintaining oxygen saturation on it's own, having symptoms of failure to thrive and developing congestive heart failure was on conservative management. Lowell H Frank stated in a chapter on Ventricular septal defects that it represent the most common form of congenital heart disease. There are different anatomic subtypes with the same pathophysiology of left-to-right shunting, increased pulmonary blood flow, and possibly congestive heart failure. Echocardiography is the mainstay of diagnosis. Treatment of hemodynamically significant defects includes medical management of congestive heart failure and either catheter-based or surgical closure [8].

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