

Congenital Ventricular Septal Defects: The Experience of Marrakech's University Hospital and the Epidemiological Outcomes

Ch.Chehbouni¹, Hassouni A¹, Safi J¹, Oualim S¹, Chniber R¹, Aboutaleb F², Zouizra Z Boumzebra D² Elhattaoui M¹, Elkarimi S^{1*}

¹Department of Cardiology, UHC Med VI, Marrakech, Morocco

²Department of cardiovascular surgery, UHC Med VI, Marrakech, Morocco

*Corresponding author: Elkarimi S

| Received: 12.03.2019 | Accepted: 19.03.2019 | Published: 30.03.2019

DOI: [10.36347/sjams.2019.v07i03.050](https://doi.org/10.36347/sjams.2019.v07i03.050)

Abstract

Original Research Article

Inter-ventricular septal defects (VSD) are dehiscences of the interventricular septum. It is the most common congenital cardiac anomaly in children, characterized by its anatomical diversity, which reflects the wide variety of its clinical expression and evolutionary profile. Colored Doppler transthoracic echocardiography (TTE) made the diagnosis easier. The purpose was to determine the frequency, epidemiological, clinical, and echocardiographic aspects of VSD in our population. We conducted a descriptive mono-centric study between January 2013 to December 2017 at the department of echocardiography of Marrakech's university hospital. We had integrated all cases in which a single VSD was found. We had excluded all VSD that were part of complex congenital cardiopathy. VSD was the first cardiac malformation representing 18.51% of all congenital cardiac malformations. It was diagnosed in 45 % of cases in the first two years, while 11 cases over the age of 18 years were identified. Seventy percent were from rural area. The peri-membranous topography was the most common form, accounted for 43.6%. Most of our cases have been discovered with fixed pulmonary hypertension. Surgery was proposed in 78% of cases based on clinical presentation and ultrasound data. The evolution of VSD may spontaneously lead to its closure if they do not, defect can lead to detrimental complications such as pulmonary hypertension (PHT), ventricular dysfunction and an increased risk of arrhythmias. Early and appropriate management is the only guarantee of a good evolution. But a main question remains: why the diagnosis of VSD is made at the stage of late complication?

Keyword: Inter-Ventricular septal defects; congenital malformation; transthoracic echocardiography; pulmonary hypertension; Surgery.

Copyright © 2019: This is an open-access article distributed under the terms of the Creative Commons Attribution license which permits unrestricted use, distribution, and reproduction in any medium for non-commercial use (NonCommercial, or CC-BY-NC) provided the original author and source are credited.

INTRODUCTION

Ventricular septal defects (VSD) are the most common congenital cardiovascular malformations [1] reaching up to 5 per 1000 births in the Moroccan context [1]. They may be part of complex malformations or isolated entities, which account for more than 20% of all congenital heart diseases [1]. VSD can be classified according to several criteria's, including the size, location or hemodynamic impact of the communication.

Doppler echocardiography remains the method of choice for the diagnosis of these abnormalities. Angiography is limited to certain situations in order to clarify echoardiographic data and to guide therapeutic choices.

MATERIALS AND METHODS

We conducted a mono-centric descriptive and transversal study between January 1st; 2013 to

December 31, 2017 at the echocardiographic laboratory in the cardiology and vascular diseases department of university hospital Mohamed VI in Marrakech. We had integrated all the cases in which a single VSD was found. We had therefore excluded all VSD that were part of complex congenital cardiopathy such as tetralogy of Fallot and transposition of greatarteries.

RESULTS

In our study, 87 patients had a ventricular septal defect from a total of 470 patients followed for congenital heart disease during this period representing the most common congenital heart disease (18, 51%). Our patient's average age was 12 and a half years, 45% of patients were under 2 years old and in 11 patients (12, 6%), VSD was discovered after 18 years old. A female predominance was noticed with a Sex ratio of 0.72. Seventy percent were from rural area, and 60, 9% were from southern regions.

The complaints were varied. For young children, the main symptoms were dyspnea, recurrent pulmonary infections and cyanosis. In 10% of cases, the VSD was revealed by the discovery of a cardiac murmur during a routine medical checkup.

The echodopplercardiograph allowed the diagnosis of VSD in all patients. The average size of VSD was 7.5 mm (3 to 27 mm). Peri-membranous VSD were the most common type (38 cases 43.7 %) followed by conal VSD (27 cases or 31 %) (Figure 1, 2). Our study showed that 67% of VSD cases were at pulmonary arterial hypertension (PHT) stage of which 62% were already severe with an average of 65 mmhg and 5 cases of Eisenmenger stadium. Beside the PHT,

there was also the left ventricle dilatation. It was found in 21 % of our cases, table 1 shows the different complications (table 1). Shunt was left to right in 89% of cases. In 48.2% of patients VSD co-existed with other minor abnormalities including atrial septal defect in 34 % of cases (Table 2).

In all patients there was a use of medical treatment based on diuretic (60%), vasodilators (35%) iron supplementation (25%).80% of patients were proposed for a surgical treatment. waiting for surgical procedure, a pulmonary cerclage was indicated in 21 (24.1%) cases as an palliative method . This gesture was complicated in a single case.

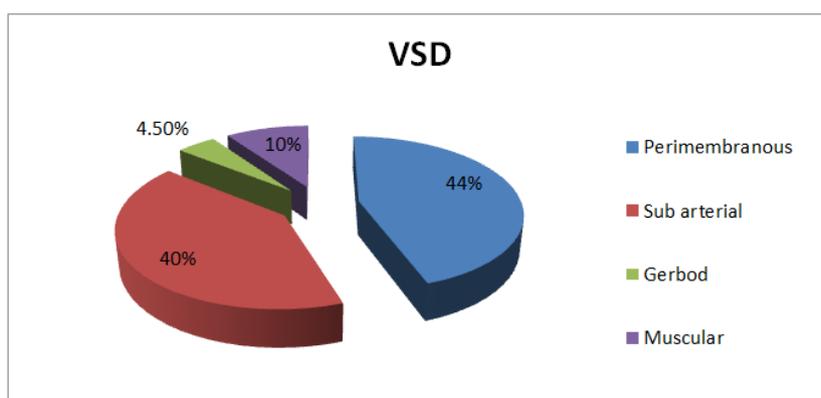
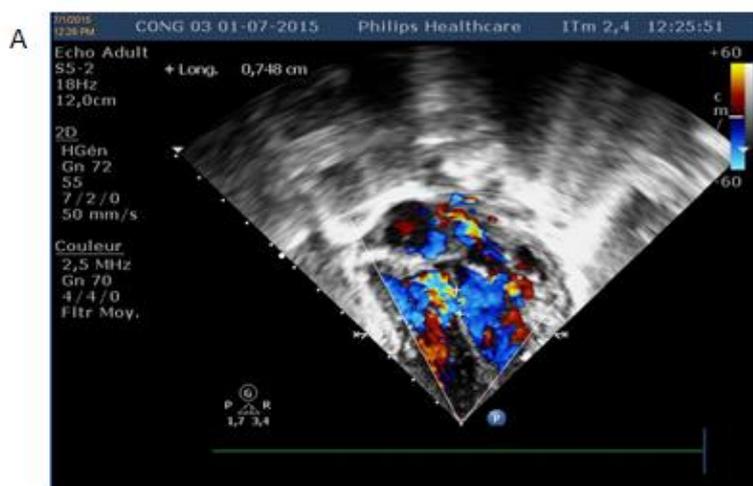


Fig-1: types of VSD



A) Muscular VSD



B)Sub arterial VSD



C)Perimembranus VSD



D)Gerbod VSD



VSD+ aortic insufficiency (laubry pezzi syndrome)



F: VSD+ aortic insufficiency (laubry pezzi syndrome)
Fig-2: Echocardiography images of different types of VSD

Table-1: Complications of VSD in our study

VSD without repercussions	20,7% (18 cases)	20,7% (18 cases)	
VSD with repercussion	79.3% (69 cases)	Pulmonary artery hypertension	84 % (58 cases)
		Left ventricular defect	27.5% (19 cases)
		Eisenmenger stadium	7.2% (5 cases)
		VSD+ aortic insufficiency (laubry pezzi syndrome)	4.6% (4 cases)

Table-2: VSD co-existed with other minor abnormalities

VSD+ arterial channel persistence	10.3%(9 cases)
VSD+atrial communication	34%(29 cases)
Multiple VSD	9.2%(8 cases)
VSD+ under aortic membrane	4.6%(4 cases)

Table-3: Among all congenital cardiopathies, the prevalence of VSD in our study is similar with most published studies

Study	Country	Study duration	Number of cases in the study	Incidence of VSD
Chauvet5	Abidjan	10	80	26.6 %
Malek chaabouniet coll.6	Tunisia	5	123	28,45 %
Bendriss L 7	Morocco	3	44	10,6 %
Nada hmamouche 8	Morroco	4	467	26 %
Our study	Morroco	4	80	20 %

DISCUSSION

Isolated VSD is the most common congenital heart malformation [2, 3]. The actual prevalence of VSD is however difficult to establish because it is variable according to patient age and method of diagnosis. It is not noted in the literature any predominance of sex despite the female predominance note in our serie.

Before the appearance of echocardiography, the estimated prevalence of VSD based on clinical data ranged from 1.35 to 2.94 per 1,000 births [4]. While the doppler colour was not yet available, the prevalence of CIV at birth was 2.01 to 3.94 per 1,000 births in well-defined populations [5, 6]. With the advance of the doppler making it easy to identify small trabeculated VSD's, the prevalence of VSD's has become significantly higher up to 17.9 % [4]. Among all congenital cardiopathies, the prevalence of VSD in our study is similar with most published studies [7, 8] (Table 3).

The etiology of VSD is unknown in the majority of cases; a Finnish study [9] highlighted the possible effects of genetic and environmental factors. The study found that maternal alcohol consumption was the first factor that could induce a VSD, followed by exposure to organic solvents. According to Hernández-Díaz [10], folic acid antagonists, including Trimethoprim, Triamterene, Carbamazepine, Phenytoin, phenobarbital, and primidone, can increase the risk of VSD, while folic acid can reduce this risk.

Also recent studies conclude that exposure to ACEs during the first trimester of pregnancy cannot be considered safe and should be avoided [9-11] while others study the influence of gene factors on VSD.

Ventricular septal defects are rarely detected in new born children. The literature describes that discovering a VSD occurs almost incidentally during a systematic check up by the finding of a cardiac murmur [12]. In some cases, the ventricular septal defect is diagnosed in second childhood or in exceptional cases, in adolescence. The main symptoms are dyspnea and cyanosis [12]. These constants are the same in our study with 12 % of cases was discovered after 18 years. More than that, our study showed that 79 % of VSD cases were with repercussion. This finding can be explained by several factors:

These results can be explained by several factors. The main reason for the delay in taking over would be, first and foremost, the Moroccan socio-cultural framework. Indeed, the Moroccan population, mainly mothers, would not be familiar with the detection of symptoms, especially those who are alleviated, and would be inclined, as they did for them, to inculcate the notion of patience and resistance to illness, thus delaying their expression at the level of the patient as well as the people in charge of them. Once parents have detected these anomalies, access to care structures remains very difficult because of the isolation of the rural environment, and also the presence of an incomplete technical-human platform that most often generates references to hospitals with more resources, but more distant and overloaded by the flow giving rise to later consultations. In addition, antenatal diagnosis is also compromised by the persistence of home delivery and the absence of health professionals trained in the detection of congenital anomalies in maternity homes. Absence of the doctor. All this, not taking into account the innumerable abundant during the course.

Hemodynamically class II was the most found in our study as in other series reported in literature. Explanation could be that VSD's become symptomatic at this stage leading patients to consultation [13]. The clinical symptoms especially breathlessness and failure to thrive are significantly correlated with the size of the VSD and lung congestion, testifying about the importance of the shunt, these symptoms are similar reported in our study [14].

Doppler echocardiography is the best method of diagnosis and follow-up of VSD, allowing both to affirm the diagnosis with specifying the site of the defect whether if it is a small one when using the color Doppler and to evaluate the hemodynamic consequences [15]. It allows also to indentify the presence of associated anomalies and to detect potential complications. Doppler echocardiography has proved its usefulness of providing essential information to assess the potential for spontaneous closure and to propose, when appropriate, surgical treatment without the use of cardiac catheterization [16].

Therapeutic management includes medical treatment, transcatheter closure and surgical repair. Medical therapy is indicated specially for patients with large VSD, responsible for congestive heart failure while waiting for surgery or spontaneous improvement

[17]. Transcatheter VSD closure has evolved significantly over the last 20 years as the tools and techniques have improved despite these improvements, the risk of adverse events, particularly serious conduction abnormalities, remains. That is why careful patient selection is important [18]. The development of devices designed for transcatheter VSD closure continue to advance the success of this procedure going forward [18]. Surgical treatment under extracorporeal circulation (ECC) consisting, closure of sub-valvular VSD's and some muscular VSD's became common practice in the first months of life [18].

Pulmonary cerclage is a palliative method that has had an important role in improving functional symptoms at a time when the repair at the outset was responsible for high mortality and morbidity. Nowadays, pulmonary cerclage has limited indications of multiple VSD's in low-weight children in a case of VSD associated with aortic coarctation, absorbable strapping may be proposed [18]. In our context; despite being abandoned; this method always keeps its place in our therapeutic protocol, especially in young children with a small weight. It helps protect the lungs waiting for the total cure, with good results.

The VSDs are one of the most frequent congenital malformations. Their prognosis depends essentially on the delay in therapeutic management when they require closure. our study found that most of the VSD, since the establishment of their diagnosis, were at the HTP stage. Whereas in comparison to other studies, most of the VSDs were without HTP. Why this observation? Are there any specificities related to the Moroccan context, particularly at the Marrakech region, which would explain the late diagnosis of these malformations. This can be explained by the profile of patients in our context cited above (rural, low socio-economic level).

Despite the late diagnosis of our patients at the HTP stage, most of them are still relevant for surgical treatment except for our 4 cases at the Esseimeger stadium.

CONCLUSION

Ventricular septal defect (VSD) is the most common congenital cardiac malformation of all newborn's congenital heart disease. With echocardiography and colour Doppler, the diagnosis is easier and more accurate than previously. Medical treatment may be indicated before surgical treatment or spontaneous improvement, but still be a palliative issue. The surgical treatment is a curative one, and consists in a defect surgical closure under bypass, since first months of life. Interventional catheterization is actually in improvement but still rarely used in our context. This descriptive study underscores the value of antenatal diagnosis, and the importance of patient awareness for an early diagnosis in front of the slightest symptoms

REFERENCES

1. El Bouchikhi I, Ouldim K, Moufid FZ, Houssaini MI, Atmani S. Caractérisation épidémiologique de la communication interventriculaire (CIV) dans la population marocaine. *Revue d'Épidémiologie et de Santé Publique*. 2015 May 1;63:S64.
2. Mehta AV, Chidambaram B. Ventricular septal defect in the first year of life. *The American journal of cardiology*. 1992 Aug 1;70(3):364-6.
3. Roguin N, Du ZD, Barak M, Nasser N, Hershkowitz S, Milgram E. High prevalence of muscular ventricular septal defect in neonates. *Journal of the American College of Cardiology*. 1995 Nov 15;26(6):1545-8.
4. Du ZD, Roguin N, Barak M, Bihari SG, Ben-Elisha M. High prevalence of muscular ventricular septal defect in preterm neonates. *The American journal of cardiology*. 1996 Nov 15;78(10):1183-5.
5. Chauvet J, Fioklou M, Seka R, Ekra A, Coulibaly A, Ouattara K, Metras D, Bertrand E. Les formes hémodynamiques du canal artériel déterminent les formes cliniques: a propos de 52 cas explorés et opérés à Abidjan. *Cardiologie tropicale*. 1986;12(47):137-45.
6. Chaabouni M, Daoud M. Aspects épidémiologiques et évolutifs des cardiopathies congénitales dans le service de pédiatrie de Sfax: A propos de 123 cas. *Tunisie médicale*. 1999;77(5):264-71.
7. Bendriss L, Sedrati M, Haddour L, Arharbi M. Communications interventriculaires: aspects anatomocliniques, thérapeutiques et évolutifs: À propos de 44 observations. *La Presse Médicale*. 2006 Apr 1;35(4):593-7.
8. CHERRABI FZ. *Les communications interventriculaires (à propos de 148 cas)* (Doctoral dissertation, Thèse de doctorat en Médecine. Université sidi Mohammed ben Abdellah, Faculté de médecine et de pharmacie-Fes).
9. Tikkanen J, Heinonen OP. Risk factors for ventricular septal defect in Finland. *Public health*. 1991 Mar 1;105(2):99-112.
10. Hernández-Díaz S, Werler MM, Walker AM, Mitchell AA. Folic acid antagonists during pregnancy and the risk of birth defects. *New England journal of medicine*. 2000 Nov 30;343(22):1608-14.
11. Cooper WO, Hernandez-Diaz S, Arbogast PG, Dudley JA, Dyer S, Gideon PS, Hall K, Ray WA. Major congenital malformations after first-trimester exposure to ACE inhibitors. *New England Journal of Medicine*. 2006 Jun 8;354(23):2443-51.
12. Chantepie A. *Communications interventriculaires*. *EMC-Cardiologie-Angéiologie*. 2005 May 1;2(2):202-30.
13. Menta I, Bâ HO, Dimzoré S, Kéita A, Thiam C, Traoré D, Daffe S, Touré M, Diarra MB. A descriptive study of ventricular septal defect (VSD) in the Luxembourg mother-child Hospital in Bamako. *Le Mali medical*. 2015;30(1):34-7.

14. Fogel MA, editor. Ventricular function and blood flow in congenital heart disease. John Wiley & Sons; 2008 Apr 15.
15. Penny DJ, Vick GW 3rd. Ventricular septal defect. *Lancet*. 2011; 377:1103.
16. Cossor W, Cui VW, Roberson DA. Three-dimensional echocardiographic en face views of ventricular septal defects: feasibility, accuracy, imaging protocols and reference image collection. *Journal of the American Society of Echocardiography*. 2015 Sep 1;28(9):1020-9.
17. Berman Jr W, Yabek SM, Dillon T, Niland C, Corlew S, Christensen D. Effects of digoxin in infants with a congested circulatory state due to a ventricular septal defect. *New England Journal of Medicine*. 1983 Feb 17;308(7):363-6.
18. You T, Yi K, Ding ZH, Hou XD, Liu XG, Wang XK, Ge L, Tian JH. Transcatheter closure, mini-invasive closure and open-heart surgical repair for treatment of perimembranous ventricular septal defects in children: a protocol for a network meta-analysis. *BMJ open*. 2017 Jun 1;7(6):e015642.