Case Report

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PERSISTENT PATENT FORAMEN OVALE IN ADULT MALE

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Abstract: In this case report, we want to present a persistent foramen ovale with length of 1.4 cm in an adult male cadaver approximately 60 years of age. The person had a heavy built up and appeared well nourished. The foramen ovale is a normal interatrial communication during fetal life. The persistence of this opening in the later life may go unnoticed if asymptomatic. But symptoms can begin in adult life due to pulmonary hypertension. It will become an added complication to any cardio-vascular disorder developing in later life. With no other anomalous findings in the heart, it appears that the foramen ovale was probe patent. There are five types of foramen ovale according to its location. The present finding belongs to type I that is patent foramen ovale of ostium secundum type. Echocardiogram, Electrocardiogram, Doppler image, Cardiac catheterization, Chest x-ray and MRI of the heart are the various imaging techniques which can detect this variance.

Keywords: Atrium, Foramen ovale (Patent oval foramen), Fossa ovalis (Oval fossa).

INTRODUCTION

An atrial septal defect (ASD) is an opening in the interatrial septum. It allows oxygen rich blood in left atrium to mix with oxygen-poor blood in right atrium. It leads to extra work for the heart to supply the body with essential oxygen. Congenital heart defects occur in about 7.5% of live births. [1] ASDs are one of the most common congenital heart defects seen in paediatric age group. The incidence of ASD among the congenital heart defects is 7-10%. Females with ASD outnumber males in the ratio 3:1. The incidence of PFO tends to decrease with advancing age. [2, 3]

An ASD is the most common heart anomaly and thankfully the least severe congenital heart disease. Ostium secundum is most frequently encountered ASD. It is often accompanied by other cardiac defects.[4] There are four types of ASDs and their location in the heart determines its type. If left untreated, the ASD may lead to significant morbidity and mortality.[4] A murmur heard during physical examination or imaging of the heart with Echocardiogram, Electrocardiogram, Doppler image, Cardiac catheterization, Chest x-ray and/ or MRI of the heart can indicate presence of such foramen ovale in absence of symptoms. [5]

MATERIAL AND METHODS

During routine undergraduate dissection, 2013-14 batch at Department of Anatomy, Sree Mookambika Institute of Medical Sciences, Kulasekharam, Tamilnadu the variation was noticed. A formalin-fixed male cadaver aged 60 years was dissected. The exact cause of death and medical history of the person is not known. As the source of cadaver is unclaimed body made available for the purpose of dissection under Anatomy Act.

Exposure of the heart after opening the thoracic cage was done following classical incision and dissection procedures. The chambers of the heart were opened to study the interior. In the right atrium, the foramen ovale was noticed. The variation was unexpected as the built of the cadaver was quite muscular and heavy. All other chambers were also opened and studied. The measurements were taken using divider and metal scale. (Fig. 1) The procedures followed were in accordance with ethical standards of handling of cadaver for learning and teaching.

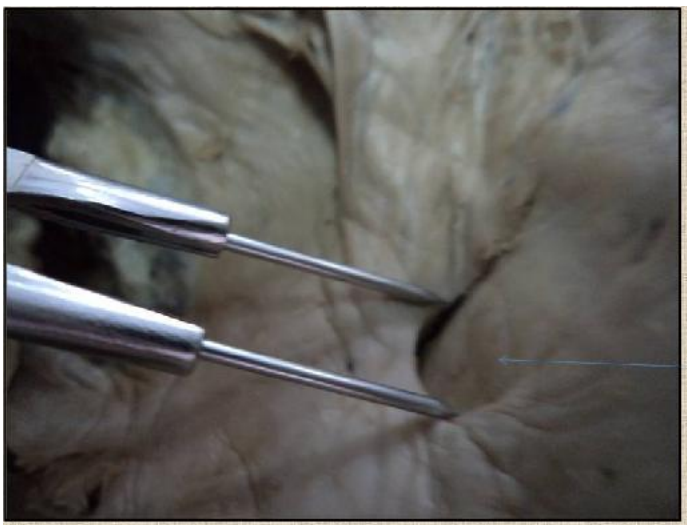

Fig 1: Measurements taken using divider and metal scale

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RESULTS

The patent foramen ovale was located at the site of fossa ovalis. It was a slit like opening of 1.4 cm along the lower rim of fossa ovalis. The dimentions of the fossa ovalis were maximum diameter of 1 cm vertically and 0.9 cm horizontally. The said foramen was 2.2 cm from tricuspid valve, 1.3 cm from coronary sinus opening, 2.8 and 2.3 cm from opening of superior and inferior vena cava openings respectively. (Fig.2) The pulmonary trunk, pulmonary arteries and aorta had normal opening. The tricuspid and the mitral valve were normal in position and structure. Coronary arteries and veins were dissected and identified. No any other anomaly was noticed during dissection of other regions.

The heart was normal in size with maximum circumference of 11.4 cm at the level of coronary sulcus. Vertically it measured 14.5 cm from the highest point of right auricle to the apex of the heart. The length of the right atrium between the openings of superior and inferior vena cava was 8 cm. the internal circumference of the right atrium was 7cm. The opening in the interatrial septum communicated right and left atrium. In the left atrium, the opening appeared like a slit and was 1.4 cm in length. It was 1.5 cm away from mitral valve. (Fig. 3)

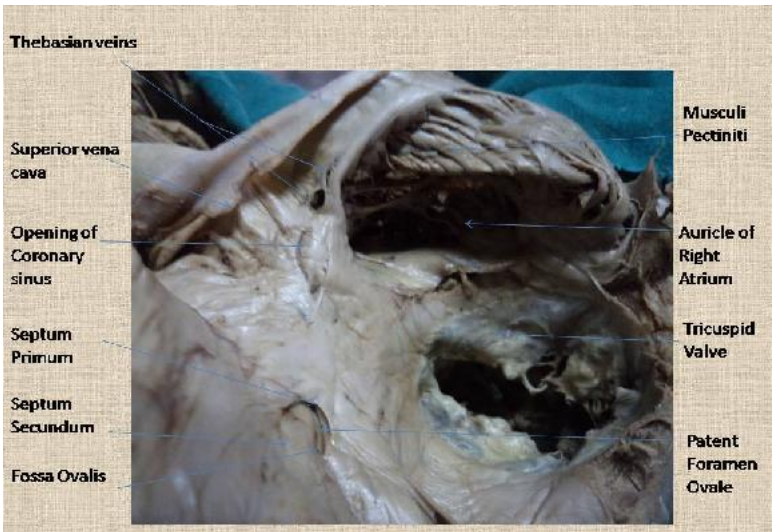

Fig 2: Interior of Right Atrium showing Patent Foramen Ovale

Fig 3: Interior of Left Atrium showing Probe passing through Patent foramen Ovale

DISCUSSION AND CONCLUSION

During fetal life, placenta acts as a site of gaseous exchange with maternal blood. So an opening between the two atrial chambers of the heart is essential to allow blood to bypass the lung's circulation. Foramen ovale is normal feature of fetal heart. Changes in the fetal circulation and occlusion of fetal vessels occur at birth. As the fetus takes first breath at birth, pulmonary respiration begins. This increases the amount of blood traversing via pulmonary trunk into pulmonary arteries. Pulmonary veins in turn carry this oxygenated blood from lungs to the left atrium.[6]

Shunting of blood to the right side of the heart, builds up pressure in the lungs. It is basis of symptoms of pulmonary hypertension that usually appear in the 30s. The defects may be multiple. In symptomatic older children defects of 2cm or more in the diameter are not unusual. A small isolated patent oval foramen is of no hemodynomic significance however, if there are other defects (e.g. pulmonary stenosis or atresia) they will present early in childhood. Blood is shunted through the foramen ovale into left atrium and produce cyanosis; a sign of deficient oxygenation of blood. A probe patent oval foramen is present in up to 25% of population. In this condition a probe can be passed from one atrium to other through the foramina. But it is functionally closed. This defect is not clinically significant. But it a potential site that may be forced open by co-existing functional pathology of the heart. [7, 8]

In the present case probe patent oval foramen seems to be formed by incomplete adhesion between the flaplike septum secundum and septum primum along the lower margin of fossa ovalis.

Symptoms of a patent foramen in the interatrial septum include shortness of breath with activity, tiring easily during activity and frequent respiratory infections in children, tachycardia and palpitations in adults, sweating, poor growth, and poor appetite. When an individual has no other congenital defect, symptoms may be absent, particularly in children. Symptoms may or may not appear during lifetime. Individuals with atrial septal defect are at an increased risk of developing a number of complications, including atrial fibrillation (in adults), heart failure, pulmonary over circulation, pulmonary hypertension, and Stroke. Severity of symptoms often depends on the size of the hole. Large ASDs may cause fatigue, pulmonary hypertension, arrhythmia, and/or an enlarged heart.[9-13]

Small defects that produce few or no symptoms may not require treatment. Many defects may even close on their own without treatment. Treatment options include open surgery for larger defects with significant symptoms. Non- invasive technique of transcatheter closure with help of device is associated with 2 to 20 % risk of air embolism, pericardial effusion, aortic laceration, residual shunt, endocarditis, device embolization or fracture, and thrombus formation on the device surface.[4,14-19] Occasionally, a murmur may be heard during physical examination, for some other ailment. Imaging of the heart done with Echocardiogram, Electrocardiogram, Doppler image, Cardiac catheterization, Chest x-ray and MRI of the heart can reveal ASD. [3, 20-22]

Different mechanisms leading to formation of ASD: [8, 9, 23] *(Fig. 4)*

A. Patent Oval foramen (POF) that results from resorption of the septum primum in abnormal locations: The septum primum is fenestrated or net like.
B. POF resulting from excessive resorption of the septum primum (short flap defect): Due to excessive resorption a short septum primum will not be able to close the foramen oval.
C. POF resulting from an abnormally large Oval foramen: Due to defective development of the septum secundum, a normal septum primum cannot close the oval foramen at birth.
D. POF resulting from an abnormally large Oval foramen and excessive resorption of the septum primum: A large ostium secundum type of ASD will occur because of combined effect.
E. Endocardial cushion defect with primum type ASD with cleft in the anterior cusp of mitral valve: It is a less common form of ASD. Several cardiac abnormalities are grouped together under this heading because they result from the same developmental defect, which is a deficiency of both endocardial cushions and AV septum. Failure of septum primum to fuse with the endocardial cushions results in ostium primum defect. Usually it is accompanied by a cleft in the anterior cusp of the mitral valve.
F. Sinus venosus ASD- A high septal defect results from abnormal absorption of the sinus venosus into the right atrium.

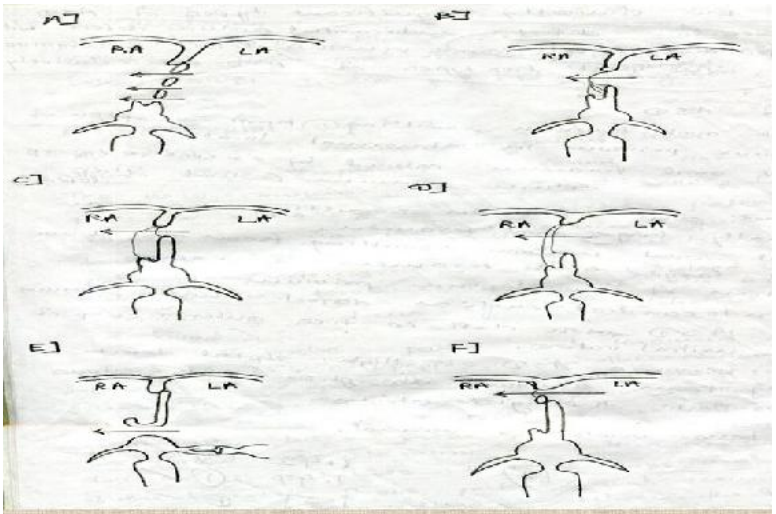

Fig 4: Different Mechanisms that lead to Atrial Septal Defect

Mechanism A and B will lead to ostium primum defect. C and D will lead to ostium secundum defect. The defect by mechanism A, B, C or D is atrial septal defect proper. Whereas in E and F Fossa ovalis is normal and ASD is accompanied by other defects.

The types of interatrial communications: [7-10, 23] ***(Fig. 5)***

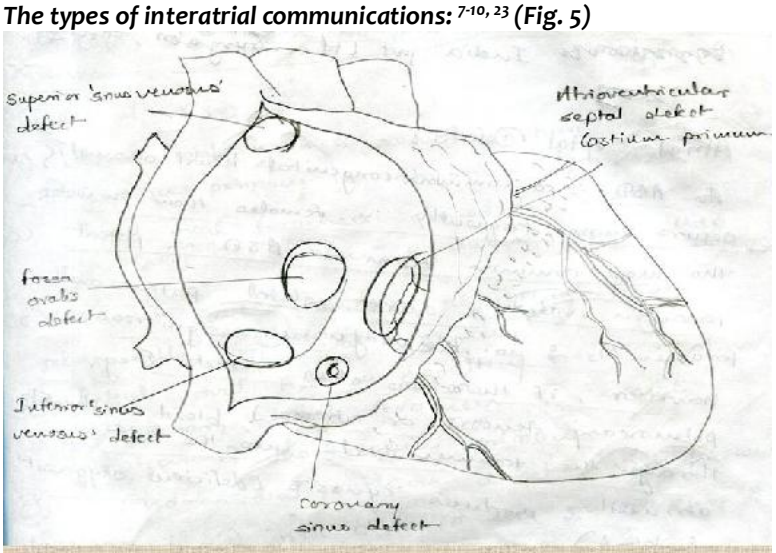

Fig 5: Types of Atrial Septal Defects depending on their location

1. ***Fossa ovalis defect:*** Only defects within the fossa ovalis are true atrial septal defects.

a. ***Ostium Primum defect:*** The patent oval foramen usually results from the abnormal resorption of the septum primum during the formation of the foramen secundum.
b. ***Ostium Secundum defect:*** It is most common type, approximately 80%.[1] Ostium secundum ASDs are in the area of the oval fossa and include both defects of the septum primum and septum secundum.

2. ***Sinus venosus defect:*** Both these are usually associated with drainage of the right pulmonary veins into the cavoatrial junction. The essential feature is the communication of either vena cava with both the atria.

a. ***Superior sinus venosus defect:*** The defect lies at site of opening of superior vena cava into right atrium. This sinus venosus defect is more frequent than its inferior counterpart.

b. ***Inferior sinus venosus defect:*** The defect in the atrial wall lies at opening of inferior vena cava into right atrium.

Most sinus venosus ASDs (high ASDs) are located in the superior part of the interatrial septum close to the entry of SVC. A sinus venosus defect is one of the rarest ASDs. They result from incomplete absorption of the sinus venosus into the right atrium and/ or abnormal development of the septum secundum. This type of ASD is commonly associated with partial anomalies of pulmonary venous connections.

3. ***Coronary sinus defect:*** The defect is located at the usual opening of coronary sinus into right atrium. The wall that generally separate coronary sinus from left atrium is deficient or absent.

In first three types the blood shunting is possible only in the atria.

1. ***Atrioventricular septal defect (ostium primum):*** During the development of the interatrial septum, the free edge of the septum primum fuses with the atrioventricular endocardial cushions. The atrio-ventricular sepum is formed subsequent to it. If this fails to occur, the entire atrioventricular junction will be malformed leading to atrioventricular septal defect. So actually it is not an ASD by defination.

A common orifice is generated at the atrio-ventricular junction. The deficiency of adjacent septal structures leads to common valve formation. The superior and inferior cusps are formed that bridge scooped out ventricular septum. These valves are tethered in both the ventricles. The left component of such valve is interpreted as a 'cleft mitral valve' though it does not resemble normal structure of the mitral valve. The shunting of blood takes place both in the atria as well as ventricle.

In the less common complete type, the endocardial cushion fails to occur. As a result, there is a large defect in the centre of the heart known as AV septal defect. Both atrioventricular valves are defective. This type of defect occurs in approximately 20% of persons with Down syndrome. Else it is relatively uncommon. This severe cardiac defect can be detected during an ultrasound examination of fetal heart.[20]

2. ***Cor triloculare biventriculare:*** Common atrium is a rare cardiac defect in which the interartrial septum is absent leading to biventricular monoatrial heart. This situation is the result of failure of the septum primum and septum secundum to develop combination of ostium secundum, ostium primum and sinus venosus defects.

3. ***Pouch formation at the interatrial septum:*** Even as the sepum primum and secundum adhere to each other leaving no foramina, the area of fossa ovalis will pouch into the left atrium. This provides a site for embolus formation.[24]

4. ***Double interatrial septum:*** It is a rare anomaly. Persistence of interatrial chamber and its communication with left atrium is a source of embolus and transient ischaemic attack.[25, 26]

5. ***Prenatal closure of foramen ovale:*** Right half of the heart is hypertrophied and left half is poorly developed. The condition is incompatible with life leading to death of neonate shortly after birth.

The condition is a congenital defect with unknown causes. Some cases may be caused by a genetic defect, Down syndrome, illness or addiction by the mother during pregnancy. Most of the time, the cause is unknown. There are no preventive measures. [27-29] Non-fatal in itself, it is a risk factor for clinical syndromes, such as paradoxical systemic embolism, embolic strokes, myocardial infarction, decompression sickness in divers, and other complications of pulmonary embolism.[25, 30-34] It is recently even linked to certain types of migraine which will respond to closure of PFO.[32] Early diagnosis and treatment can help to prevent complications.

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