

The Prevalence of Atrial Septal Defect in Human Adult Cadavers with its Clinical and Embryological Correlations

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Abstract

Introduction: An Atrial Septal Defect (ASD) is a persistent interatrial communication, accounts for approximately 6-10% of congenital defects. ASD types include Ostium Secundum, Ostium Primum, Sinus Venosus, and Coronary sinus defects. Small ASDs may not have significant clinical consequences, while a large defect may lead to life threatening symptoms. The objective of the study is to estimate the prevalence of ASD in the hearts of adult human cadavers.

Materials and Methods: The present study was conducted on a total of 25 Hearts obtained from cadavers allotted to the undergraduate medical students and preserved specimens from the Department of Anatomy.

Results: The presence of Ostiumsecundum ASD was seen in 1 out of 25 specimens (4%). It was an elliptical opening measuring 10×8 mm.

Conclusion: ASDs are associated with the risk of Stroke, Transient Ischaemic Attack, and Paradoxical Embolism. The Anatomical knowledge of the position of the ASD and its morphometric knowledge enables it to differentiate between various Septal Defects. ASDs are usually closed by surgical intervention or by Percutaneous Transcatheter Closure.

Keywords: Foramen ovale; Atrial septal defect; Septum secundum; Cadaveric study.

INTRODUCTION

Isolated atrial septal defects (ASD) represent 7% of all cardiac anomalies and can present at any age.¹ They usually have left to right shunt. There are 4 types of ASD. The most common one is Ostiumsecundum type, where the defect is noted in septal tissue of fossa ovalis. The Ostiumprimum type is usually large in size located in the anterior portion of the lower part of the atrial septum.

Can lead to mitral insufficiency when it involves its anterior leaflet. Other rare variants are Sinus venosus ASD, here majority of defects are situated in the posterior superior portion of the interatrial septum often very close to opening of superior vena cava. Coronary sinus ASDs are the most rare of the ASD types. Here the defects lie close to the opening of the coronary sinus. Patent foramen ovale seen in 25 to 30% of normal individuals is considered as type five and different from secundum defect. It is due to incompetence of fossa ovale valve which has right to left shunt and it may lead to paradoxical embolism.^{1,2}

The outcome of such defects are related to the size of the defect and duration of shunting. Small defects (<5mm) resolve spontaneously or may be treated with medical or expectant management. Moderate to large defect need surgical intervention.^{2,3} In the present study we made an attempt to study

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morphometric analysis and prevalence of ASD.

MATERIALS AND METHODS

The study was conducted on 25 hearts from the Department of Anatomy over a period of 3 years. The hearts were procured from the bodies given to students for dissection and also the preserved specimens were used. Hearts from both sexes were used.

The dissection was carried following the steps in Cunningham's dissection manual.⁴ After opening the rib cage vertical cut was made on pericardium anterior to phrenic nerve on either side and a horizontal cut just above the diaphragm and the heart was exposed. Heart was removed after making a cut on great vessels. Exterior of right and left atrium was defined. Cut was made just behind the sulcus terminalis extending from inferior vena cava to superior vena cava exposing the right atrium. A horizontal cut posterior to the ascending aorta will expose the left atrium. The chambers were cleaned by removing the blood clot. Interatrial septum was observed for any defect and noted. The defect when present was measured using digital verniercaliper and photographs were taken. Statistical measures like Mean, Median and Average was used.

RESULTS

SVC: Superior vena cava, IVC: Inferior vena cava, ASD: Atrial septal defect

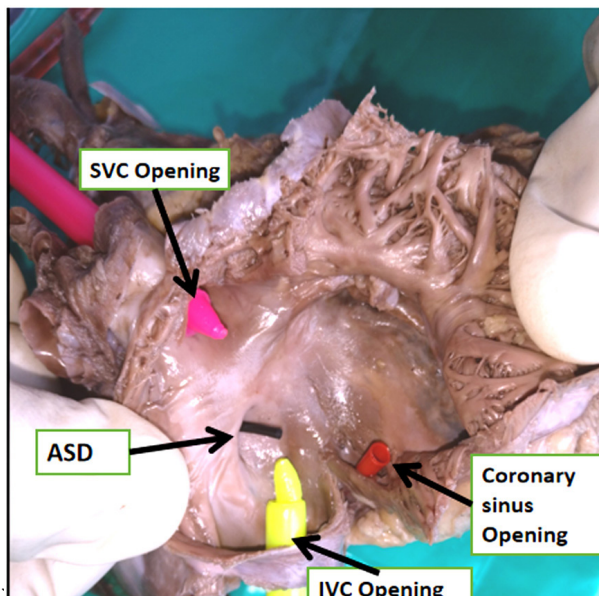


Fig. 1: Shows the elliptical septum secundum type Atrial septal defect.

Interatrial septum was observed in 25 heart specimen. In one heart, ostiumsecundum type ASD was noted (Fig. 1). It was elliptical in shape, was able to pass a thread through the defect (Fig. 2). Its mean transverse diameter was 10.37mm and antero-posterior diameter was 8.11mm (Fig. 3). The prevalence rate from the study was 4%.

DISCUSSION

Atrium develops from primitive atrial chamber. Right atrium also receives contribution from Sinus venosus which is absorbed into atrium and guarded by right and left venous valve, which fuse cranially and form marked projection septum spurium. While left atrium receive contribution from proximal part of pulmonary veins. By about 4th week atrioventricular (AV) cushion starts developing and fuse later to narrow atrioventricular orifice. At the end of 4th week a sickle shaped crest grows from roof of the common atrium called septum primum. It extends towards the endocardial AV cushion. The opening between septum primum and AV cushion is now called Ostiumprimum, which progressively narrowed. Before closure of ostiumprimum, perforations appear in the upper portion of septum primum which is called Ostiumsecundum which ensures that the blood flow from right to left atrium.

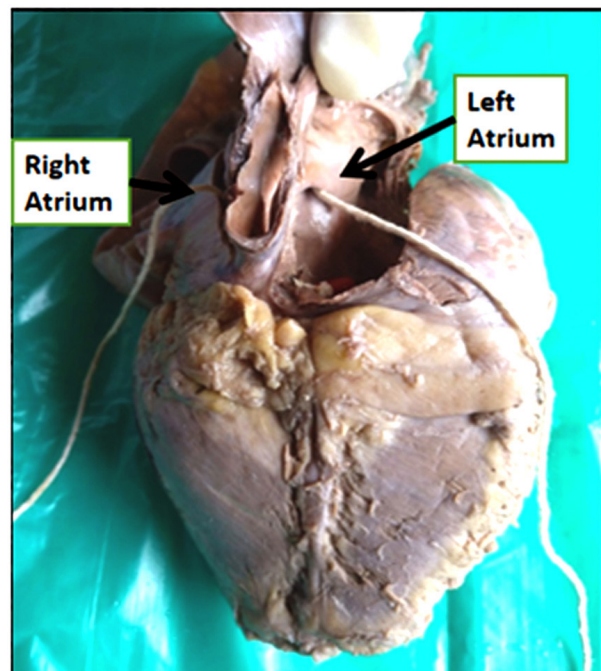


Fig. 2: Shows a thread passing through the atrial septal defect.

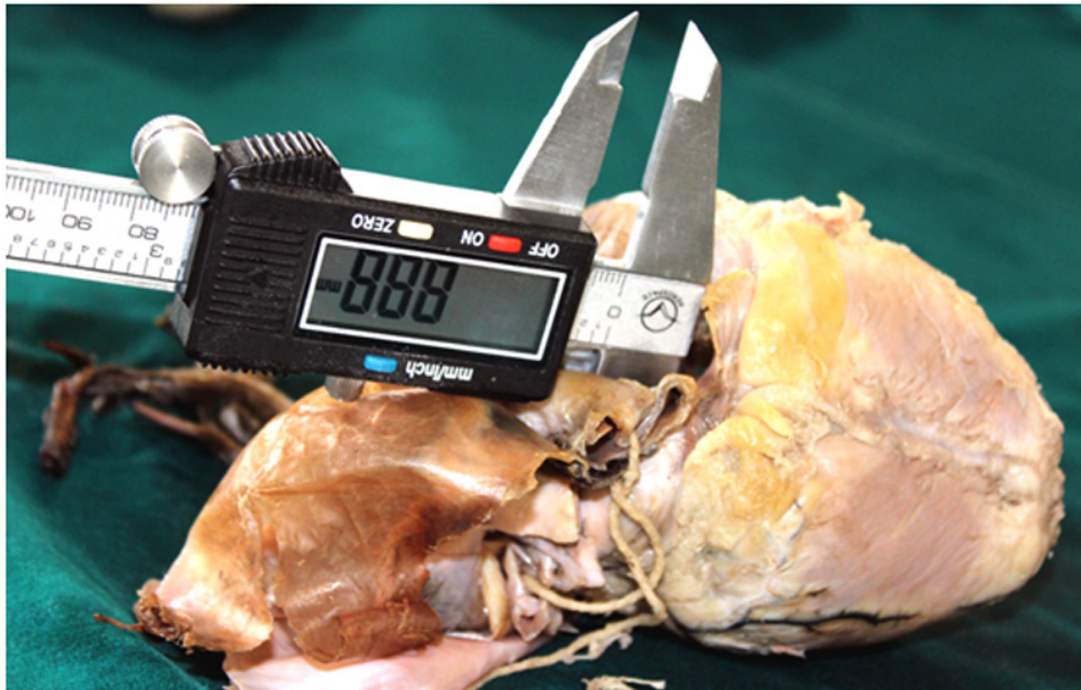


Fig. 3: Use of Vernier Calliper for measuring the width of the atrial septal defect

At about sixth to seventh week a crescent shaped thicker ridge appear in the roof called septum secundum between left venous valve and septum primum. It extends till it covers the ostium secundum. The lower crescent shape of this forms the sharp rim covering superior, anterior and posterior border of fossa ovalis in adults.

An oblique opening foramen ovale maintains the blood flow from right to left atrium in foetal life. After birth physiologically this foramen ovale will be closed by approximation of septum primum against rigid septum secundum brought about by the pressure difference in both atrial chambers. By approximately about 3 months valve of septum primum fuses with septum secundum, forming oval fossa. In 20% of cases, this fusion is incomplete forming patent foramen ovale. Excessive resorption and cell death of septum primum or by inadequate development of septum secundum will lead to ASD. Also mutations in heart specifying gene NKX 2.5 on chromosome 5q35 can produce ASD.⁵⁻⁷

In a study done on 60 cadaveric hearts probe patency of foramen ovale was found in 15% cases.⁸ Another study done on 106 cadaveric hearts shows 4.71% of patent foramen ovale where as in 10.37% has probe patency.⁹ Another study done on 50 cadaveric heart, 2 cases (4%) showed patent foramen ovale, whereas 9 cases (18%) had only probe patency.¹⁰ In another study on Japanese population, the autopsy reports and clinical backgrounds of 103

hearts were reviewed using medical records. They found prevalence of PFO was 13.6% (14/103).¹¹ Another cadaveric study done on 50 hearts, FO was patent in 3 (6.0%), whereas 24.0% had only probe patency.¹² In the present study the we found a septum secundum type ASD in 4% cases.

A study done over a period of 10 years where 117 ASD patients were reviewed. 78.4% were asymptomatic. Symptomatic usually complained of dyspnoea on exertion which increase with age.¹³ Pathophysiologic consequences of ASDs noted were arrhythmia, paradoxical embolism, cerebral abscess, pulmonary hypertension, and right ventricular failure. Early diagnosis and tratment when symptomatic will prevent the fatal complications. Two-dimensional transthoracic echocardiography with Doppler is a central aspect of the morphometric evaluation of the defect and to plan any surgical intervention.¹⁴ From an anatomoclinical point of view, ASD and PFO might be considered at the edge of a single continuum which pass throughout flat elliptical ASD and cribrosus ASD.¹

CONCLUSION

The study of atrial septal defects of prime importance, if left undiagnosed, it can lead to major complications some of which may even be lethal. Septum secundum is the most common type

of ASD on a long run can lead to patent foramen ovale. The understanding the embryological basis and early intervention in symptomatic individual can prevent fatal complications.

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