



RESEARCH ARTICLE

A STUDY OF SEPTAL BRANCHES OF HUMAN CORONARY ARTERIES USING LUMINAL CAST

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ABSTRACT

Introduction: Coronary artery disease is one of the major causes of death in young athletes all over the world, leading to sudden cardiac arrest. Knowledge of the coronary arteries along with its septal branches is very important. Interventional cardiologists treat these conditions by approaching the specific interventricular septal branches of the coronary arteries.

Objectives: To study the septal branches of the interventricular septum arising from both the coronary arteries and their variations.

Materials and method: This study was conducted on 50 specimens. The lumen of the coronary arteries were injected with cast material and made like thick scaffoldings then dissected under water and the variations were studied.

Results: 64% were right dominant, 16% left dominant and 20% co-dominant. The branches to the interventricular septum were maximum in case of co-dominance.

Discussion: In Hypertrophic cardiomyopathy, the volume of the left ventricle reduces gradually and decreases cardiac output. This condition can be treated by approaching the interventricular septum through the septal branches and causing local necrosis of the septum using alcohol.

Conclusion: This study helps the interventional cardiologists to plan the line of treatment in Hypertrophic cardiomyopathy and ablate the septum using alcohol.

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INTRODUCTION

In the recent years, incidence of coronary arterial disease is on the rise at an alarmingly fast pace, resulting in sudden death. Middle-aged people in the developing countries are becoming the victims.⁽¹⁾ Modifiable risk factors like Stressful, competitive life, change in food habits and lifestyle, are some of the contributing factors.⁽²⁾ Availability of micro vascular surgical procedures, bypass surgeries, hi-tech non-invasive curative procedures helps the patients to recoup. Second in line in the causation of sudden cardiac death in young athletes is due to Hypertrophic obstructive cardiomyopathy (HOCM), which accounts for one third of such cases. HOCM, because of the interventricular septal (IVS) hypertrophy reduces left ventricular ejection fraction due to left ventricular out flow obstruction.⁽³⁾ It is one of the commonest causes of sudden death in young and competitive athletes.⁽⁴⁾ Hypertrophy may develop or progress spontaneously from child hood.⁽⁵⁾ Screening for HOCM in competitive athletes prevented significant number of deaths during 1979 – 1996 in Italy.⁽⁶⁾ Interventional cardiologists treat this by approaching the IVS branches and ablate the septum by injecting ethanol.⁽⁷⁾ In the present scenario, detailed study of the blood supply of the heart is under taken. Normally the septum is supplied by the branches of anterior and posterior interventricular arteries. The ratio of supply being variable. Obstructions of these arteries invariably jeopardize the septal nutrition and may result in infarction. Hence, in the present study arterial supply of the interventricular septum was studied in detail. Knowledge of the septal vascular supply and variations are very essential to the cardiovascular surgeon and it helps

to plan vascular surgery to keep up vitality of the septum to prevent septal rupture.

Aims and objectives

To study the interventricular septal branches arising from both the coronary arteries and their variations.

MATERIALS AND METHODS

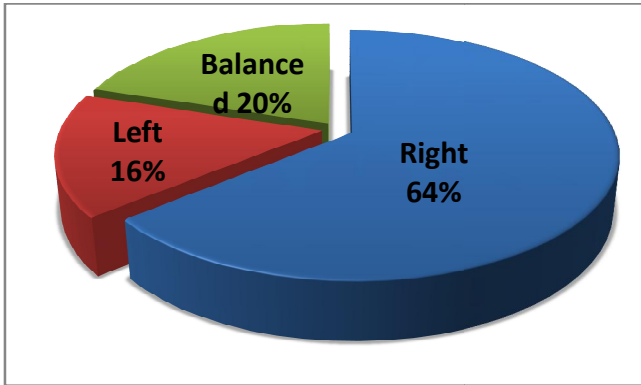
In the present study, 50 human heart specimens were studied by dissection method from a private medical college in Karnataka, South India. About 10% formalin was used preservative for the specimens. In addition, dissection instruments, butyl butyrate granules, acetone, glass syringe, 16 gauge needle and oil paints were also used. Using a 50ml syringe and a canula, plenty of water was injected into the coronary arteries to wash the blood clots. After complete wash, 10% solution of formalin was injected. butyl butyrate granules dissolved completely in acetone was taken in a 5ml glass syringe using a 16 gauge needle, injected into the coronary ostia with constant pressure. Proximal ends were blocked using artery forceps. Immediately these specimens were dipped in cold water. The butyl butyrate within the septals formed a scaffold. Ventricles were opened and septal branches were traced. Those were painted with red oil paint and documented by taking photographs.

RESULTS

The present study revealed normal pattern in 32 specimens. Septal blood supply was normal (Right dominance), Left coronary artery arising from anterior aortic sinus, and descending in anterior interventricular groove and terminating in the apex of the heart.

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Posterior interventricular artery was arising from right coronary artery, at the crux of the heart. (Graph 1). Left dominance was observed in 8 specimens, wherein posterior interventricular branch was originating from circumflex branch of Left coronary artery, and supplying the septum. Terminal part of Right coronary artery was very small and ended in supplying the myocardium before reaching the crux of the heart. (Graph 1). Balanced type of supply was seen in 10 specimens. In these cases, the posterior interventricular branch was arising from both right coronary artery and the circumflex branch of the left coronary artery. The number of septal branches depends on the level of termination of the Coronary arteries and coronary dominance.



Graph - 1. Coronary artery dominance

Septal arterial branches from the anterior and posterior interventricular septum were traced and counted. The septal branches were perpendicular to the main arteries and immediately re-branch after a short distance in the septum. The first perforator branch was prominent and arose at a distance of about 12- 17mm from the origin of the anterior interventricular artery. In case of right dominance, anterior and posterior interventricular artery branches range from 6-16 and 4-10 respectively. In case of left dominance, it was 9-12 and 3-10 branches. In case of co-dominance, these branches range from 8-14 and 5-12. The average number of branches given by the anterior interventricular artery was 11 and by the posterior interventricular artery were 6. (Table 1, Fig 1-4).

Table 1. Range of branches from the AIV and PIV in case of coronary dominance

Right dominance (32 specimens)		Left dominance (8 specimens)		Balanced type (10 specimens)	
AIV	PIV	AIV	PIV	AIV	PIV
6-16	4-10	9-12	3-10	8-14	5-12

AIV – Anterior interventricular artery, PIV –Posterior interventricular artery

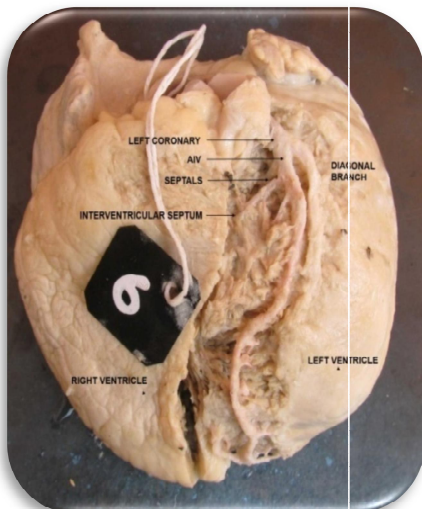


Fig. 1. AIV artery with septal branches

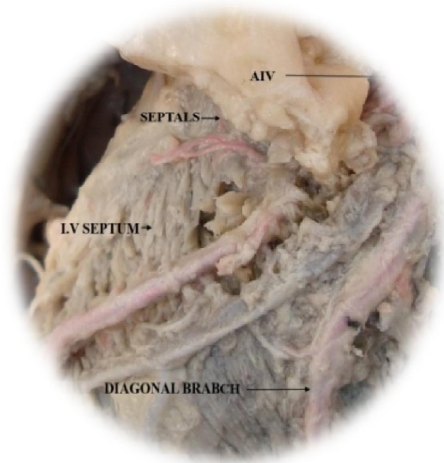


Fig 2. Enlarged view - Septal branches

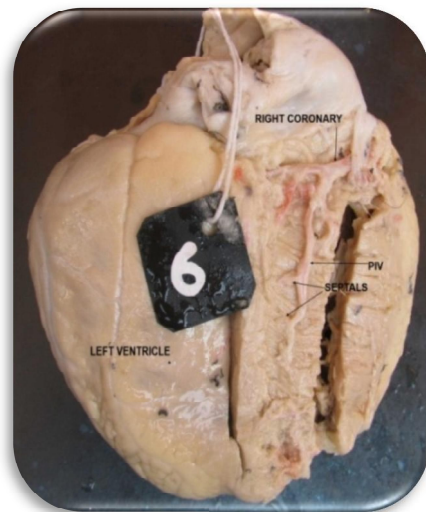


Fig 3. PIV artery arising form RCA

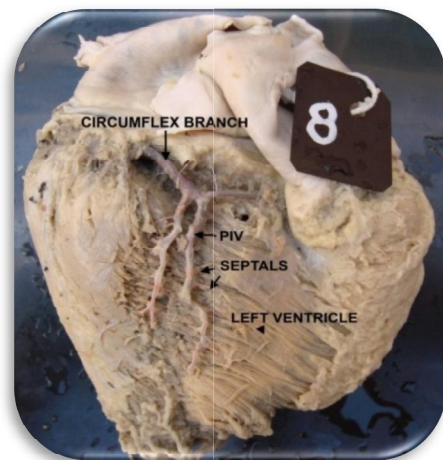


Fig 4. PIV artery arising from Cx branch of LCA

(AIV – Anterior interventricular artery, PIV – Posterior interventricular artery, RCA – Right coronary artery, LCA - Left coronary artery, Cx - Circumflex branch of left coronary artery)

DISCUSSION

Coronary artery disease takes top priority among the deaths due to cardiac diseases.⁽⁸⁾ Post infarction interventricular septal rupture is seen as sequelae to the extensive coronary block and Myocardial infarction. Septal rupture may be due to cardiogenic shock.^(9, 10) In such cases multiple grafts may help to keep vitality of septum.⁽¹¹⁾ Conduction system defects and HOCM are next in the order. It is a type of genetically inherited cardiomyopathy characterised by hypertrophy of the left ventricle including the interventricular septum. Symptoms may appear at any age. Most of the cases have autosomal dominant inheritance pattern of occurrence.⁽¹²⁾ The symptoms may range from simple cardiovascular symptoms to recurrent arrhythmias. The fatal complications like sudden cardiac death and the end stage heart failure depends on the risk factors like age, myocardial pathology, and impaired septal blood supply.⁽¹³⁾ Cross sectional slice of heart studied by hemodynamic, angiographic, echocardiographic techniques helps to identify the hypertrophy of interventricular septum. Hypertrophy of IVS may be total or localized, basal or intermediate or apical regions. The septum is more involved than the free left ventricular wall.⁽¹⁴⁾ One of the important remedial measures taken these days, with the advancement of interventional cardiology is approach of the IVS arteries to inject ethanol, thereby inducing septal infarction. This technique known as septal ablation needs a thorough knowledge of the arterial supply of IVS. The particular focal branches have to be approached in cases of localized IVS hypertrophy. Other modes of treatment include septal myectomy, and implantable cardioverter defibrillators.⁽¹⁵⁾ Echocardiography has helped to diagnose septal hypertrophy at different levels, which may be basal, apical or intermediate. Knowledge of these septal branching patterns helps to induce septal ablation in the focal point where ever it is needed.

Conclusion

The knowledge of the septal arterial supply and variations can guide the interventional cardiologists to carry out successfully the septal ablation by alcohol injection. Approach of the septal branches for septal ablation can be planned to induce septal infarction in the selected region. If IVS hypertrophy is diagnosed, preventive measures should be taken to prevent sudden death. It is advisable to thoroughly check the structural status of the IVS in active young athletes and those to be selected for defence services. It should be made mandatory for all the candidates to undergo echocardiographic evaluation during routine medical check up.

REFERENCES

1. Michales L. 1996. Aetiology of coronary artery disease, an historic approach. *Brit. Heart. J.*, 28: 258.
2. Stampfer MJ, Frank BH, Manson JAE. *et al.* 2000. Primary Prevention of Coronary Heart Disease in Women through Diet and Lifestyle. *N Engl J Med.*, 16(22): 343.

3. Kofflard MJM, Cate FJT, Lee CVD, *et al.* 2003. Hypertrophic cardiomyopathy in a large community-based population: clinical outcome and identification of risk factors for sudden cardiac death and clinical deterioration. *J Am Coll Cardiol.*, 41(6):987-993.
4. Maron BJ. 2003. Sudden Death in Young Athletes. *N Engl J Med.*, 349:1064-1075.
5. Maren BJ, Spirit P, Wesley Y, Arcej. 1986. Development and Progression of Left Ventricular Hypertrophy In Children With Hypertrophic Cardiomyopathy. *N Engl J Med.*, 315(10): 610-614.
6. Corrado D, Basso C, Schiavon M, *et al.* 1998. Screening for Hypertrophic Cardiomyopathy in Young Athletes. *N Engl J Med.*, 339: 364-369.
7. Alam M, Dokainish H, Lakkis N. 2006. Alcohol Septal Ablation for Hypertrophic Obstructive Cardiomyopathy: A Systematic Review of Published Studies. *Journal of Interventional Cardiology*, 19 (4): 319-327.
8. Gaziano TA, Bitton A, Anand S, *et al.* 2010. Growing Epidemic of Coronary Heart Disease in Low- and Middle-Income Countries. *Current Problems in Cardiology.*, 35(2): 72-115.
9. Menon V, Webb JG, Hillis LD, *et al.* 2000. Outcome and profile of ventricular septal rupture with cardiogenic shock after myocardial infarction. *J Am Coll Cardiol.*, 36(3s1): 1110-1116.
10. Moore CA, Nygaard TW, Kaiser DL, *et al.* 1998. Post infarction ventricular septal rupture: the importance of location of infarction and right ventricular function in determining survival. *Circulation.*, 74: 45-55.
11. Jansen EWL, Borst C, Lahpor JR, *et al.* 1998. Coronary artery bypass grafting without cardiopulmonary bypass using the octopus method: results in the first one hundred patients. *The Journal of Thoracic and Cardiovascular Surgery*, 116 (1): 60-67.
12. Greaves SC, Roch AH, Neutze JM, *e et al.* 1998. Inheritance of hypertrophic cardiomyopathy: a cross sectional and M mode echocardiographic study of 50 families. *Br Heart J.*, 58: 259-266
13. Elliot P, Kenna MWJ. 2004. Hypertrophic cardiomyopathy. *Lancet*, 363 (9424): 1881-91.
14. Maron BJ, Bonow RO, Seshagiri TNR, *et al.* 1982. Hypertrophic cardiomyopathy with ventricular septal hypertrophy localized to the apical region of the left ventricle (apical hypertrophic cardiomyopathy). *The American Journal of Cardiology*, 49(8):1838-1848.
15. Maron BJ, Spirito P, Shen WK. 2007. Implantable Cardioverter-Defibrillators and Prevention of Sudden Cardiac Death in Hypertrophic Cardiomyopathy. *JAMA.*, 298(4): 405-412.
16. Chang SA, Kim HK, Kim DH. *et al.* 2010. Left ventricular twist mechanics in patients with apical hypertrophic cardiomyopathy: assessment with 2D speckle tracking echocardiography. *Heart*, 96: 49-55.
