

# BANGLADESH HEART JOURNAL

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# INSTRUCTION TO AUTHORS

## A. Introduction

Bangladesh Heart Journal is the official journal of Bangladesh Cardiac Society, and accepts articles for publication from home and abroad. This is a biannual, peer-reviewed journal and aims to publish work of the highest quality from all sub-specialties of cardiology and cardiovascular surgery. The aim of the publication is to promote research in Bangladesh and serve as platform for dissemination of scientific information in cardiology.

## B. Categories of Articles

The journal accepts original research, review articles, case reports, cardiovascular images and letters to the editor, for publication.

### *Original Research:*

Original, in-depth research article that represents new and significant contributions to medical science. Each manuscript should be accompanied by a structured abstract of up to 250 words using the following headings: Objective, Methods, Results, and Conclusions. Three to 5 keywords to facilitate indexing should be provided in alphabetical order below the abstract. The text should be arranged in sections on INTRODUCTION, METHODS, RESULTS and DISCUSSION. The typical text length for such contributions is up to 3000 words (including title page, abstract, tables, figures, acknowledgments and key messages). Number of references should be limited to 50.

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Only clinical photographs with or without accompanying skiagrams, pathological images, echocardiographic images, angiographic images etc. are considered for publication. Image should clearly identify the condition and have the classical characteristics of the clinical condition. Clinical photographs of condition which are very common, where diagnosis is obvious, or where diagnosis is not at all possible on images alone would not be considered. Photographs should be of high quality, usually 127 × 173 mm (5 × 7 in) but no larger than 203 × 254 mm (8 × 10 in). A short text of up to 250 words depicting the condition is needed. Figures should be placed exactly at a logical place in the manuscript. The submitted images should be of high resolution (>300 dpi). The following file types are acceptable: JPEG and TIFF. The number of authors should not exceed 3. The authors should ensure that images of similar nature have not been published earlier. Authors must obtain signed informed consent from the patient, or the legal guardian.

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All manuscripts should meet the following criteria: the material is original, study methods are appropriate, data are sound, conclusions are reasonable and supported by the data, and the information is important; the topic has general cardiology interest; and that the article is written in reasonably good English. Manuscripts which do not follow the guidelines of Bangladesh Heart Journal are likely to be sent back to authors without initiating the peer-review process. All accepted manuscripts are subject to editorial modifications to suit the language and style of Bangladesh Heart Journal and suggestions may be made to the authors by the Editorial Board to improve the scientific value of the journal.

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#### **E. Cover Letter**

The cover letter should outline the importance and uniqueness of the work. It should include the signed declaration from all authors on:

1. Category of manuscript (original research, review article, case report, cardiovascular image, letter to the Editor)
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The Introduction should address the subject of the paper. The Methods section should describe in adequate detail the laboratory or study methods followed and state the statistical procedures employed in the research. This section should also identify the ethical guidelines followed by the investigators with regard to the population, patient samples or animal specimens used. A statement should be made, where applicable, that their study conforms to widely accepted ethical principles guiding human research (such as the Declaration of Helsinki) AND also that their study has been approved by a local ethics committee. The Results section should be concise and include pertinent findings and necessary tables and figures. The Discussion should contain conclusions based on the major findings of the study, a review of the relevant literature, clinical application of the conclusions and future research implications. Following the Discussion, Acknowledgements of important contributors and funding agencies may be given.

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Collate acknowledgements in a separate section at the end of the article before the references. List here those individuals who provided help during the research (e.g., providing language help, writing assistance or proof reading the article, etc.).

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Follow internationally accepted rules and conventions: use the international system of units (SI). If other units are mentioned, please give their equivalent in SI. Generic rather than trade names of drugs should be used.

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References should follow the standards summarized in the NLM's International Committee of Medical Journal Editors (ICMJE) Recommendations for the Conduct, Reporting, Editing and Publication of Scholarly Work in Medical Journals (ICMJE recommendations), available at: <http://www.icmje.org/recommendations/>. The titles of journals should be abbreviated according to the style used for MEDLINE ([www.ncbi.nlm.nih.gov/nlmcatalog/journals](http://www.ncbi.nlm.nih.gov/nlmcatalog/journals)). Journals that are not indexed should be written in full.

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Examples of correct forms of references are given below:

**Articles in Journals** (see also *Journal article on the Internet*)

1. *Standard journal article*

List the first six authors followed by et al.

Halpern SD, Ubel PA, Caplan AL. Solid-organ transplantation in HIV-infected patients. *N Engl J Med*. 2002 Jul 25;347(4):284-7.

More than six authors:

Rose ME, Huerbin MB, Melick J, Marion DW, Palmer AM, Schiding JK, et al. Regulation of interstitial excitatory amino acid concentrations after cortical contusion injury. *Brain Res*. 2002;935(1-2):40-6.

2. *Organization as author*

Diabetes Prevention Program Research Group. Hypertension, insulin, and proinsulin in participants with impaired glucose tolerance. *Hypertension*. 2002;40(5):679-86.

3. *Both personal authors and organization as author* (List all as they appear in the byline.)

Vallancien G, Emberton M, Harving N, van Moorselaar RJ; Alf-One Study Group. Sexual dysfunction in 1,274 European men suffering from lower urinary tract symptoms. *J Urol*. 2003;169(6):2257-61.

4. *Volume with supplement*

Geraud G, Spierings EL, Keywood C. Tolerability and safety of frovatriptan with short- and long-term use for treatment of migraine and in comparison with sumatriptan. *Headache*. 2002;42Suppl 2:S93-9.

5. *Issue with supplement*

Glauser TA. Integrating clinical trial data into clinical practice. *Neurology*. 2002;58(12 Suppl 7):S6-12.

6. *Type of article indicated as needed*

Tor M, Turker H. International approaches to the prescription of long-term oxygen therapy [letter]. *Eur Respir J*. 2002;20(1):242.

Lofwall MR, Strain EC, Brooner RK, Kindbom KA, Bigelow GE. Characteristics of older methadone maintenance (MM) patients [abstract]. *Drug Alcohol Depend*. 2002;66Suppl 1:S105.

7. *Article published electronically ahead of the print version*

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**Books and Other Monographs**

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Murray PR, Rosenthal KS, Kobayashi GS, Pfaller MA. *Medical microbiology*. 4th ed. St. Louis: Mosby; 2002.

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Gilstrap LC 3rd, Cunningham FG, VanDorsten JP, editors. *Operative obstetrics*. 2nd ed. New York: McGraw-Hill; 2002.

3. *Organization(s) as author*

Advanced Life Support Group. *Acute medical emergencies: the practical approach*. London: BMJ Books; 2001. 454 p.

4. *Chapter in a book*

Meltzer PS, Kallioniemi A, Trent JM. Chromosome alterations in human solid tumors. In: Vogelstein B, Kinzler KW, editors. *The genetic basis of human cancer*. New York: McGraw-Hill; 2002. p. 93-113.

5. *Conference proceedings*

Harnden P, Joffe JK, Jones WG, editors. *Germ cell tumours V. Proceedings of the 5th Germ Cell Tumour Conference*; 2001 Sep 13-15; Leeds, UK. New York: Springer; 2002.

6. *Dissertation or thesis*

Borkowski MM. *Infant sleep and feeding: a telephone survey of Hispanic Americans [dissertation]*. Mount Pleasant (MI): Central Michigan University; 2002.

**Other Published Material**

*Newspaper article*

Tynan T. Medical improvements lower homicide rate: study sees drop in assault rate. *The Washington Post*. 2002 Aug 12;Sect. A:2 (col. 4).

## Unpublished Material

### *In press or Forthcoming*

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## Electronic Material

### 1. *Journal article on the Internet*

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Article published electronically ahead of the print version:

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### 2. *Monograph on the Internet*

Foley KM, Gelband H, editors. Improving palliative care for cancer [Internet]. Washington: National Academy Press; 2001 [cited 2002 Jul 9]. Available from: <http://www.nap.edu/books/0309074029/html/>.

### 3. *Homepage/Web site*

Cancer-Pain.org [Internet]. New York: Association of Cancer Online Resources, Inc.; c2000-01 [updated 2002 May 16; cited 2002 Jul 9]. Available from: <http://www.cancer-pain.org/>.

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2. The article is not currently being considered for publication by any other journal and will not be submitted for such review while under review by the Bangladesh Heart Journal.
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## **Heart failure – How can we face this pandemic of twenty first century?**

STM Abu Azam<sup>1</sup>, Mohammad Ullah<sup>2</sup>

(*Bangladesh Heart Journal 2016; 31(1) : 1-2*)

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Heart failure is now in the form of a pandemic across the world and a serious threat to the health and financial wellbeing of people. It affects approximately 6% of people aged between 60 and 86 years.<sup>1</sup> The incidence of heart failure increases twofold for each decade of life.<sup>2</sup> Personal, economic and health care burden of heart failure is expected to increase more in the future as life expectancy of people increases, placing further pressure on the finite health care resources.

Previously it was thought to be a problem of the people of developed world; and developing world was busy with communicable and nutritional diseases. Now along with decrease in communicable diseases in countries like Bangladesh, prevention and management of non communicable diseases have become the main concern. Almost all the non communicable diseases like, hypertension, diabetes mellitus, ischaemic heart disease, chronic kidney disease etc. end up in heart failure. Besides this, improved management of acute coronary syndrome and improved longevity of the population have also contributed to the increased magnitude of heart failure burden.

In USA 1.9% of the total population suffer from heart failure. There is no such data for Bangladesh. But Asian countries like Japan & China have got an incidence of 1% and 1.3% respectively. Overall, the prevalence of heart failure in Asia is 1.26% to 6.7%.<sup>3</sup> In Bangladesh, rheumatic heart disease still plays an important role in the incidence of heart failure, complicating the scenario. In a study conducted in National Institute of Cardiovascular Diseases and different medical colleges of Bangladesh revealed that IHD was responsible for 65% of chronic heart failure, rheumatic heart disease for 18% cases, hypertension for 12% cases and idiopathic cardiomyopathy for 5% cases.<sup>4</sup> The average age of Asian

population suffering from heart failure is 10 years lesser than that of western population. So its economic impact is also higher.<sup>5</sup>

Are we prepared to face this new monster of twenty first century? The answer is –‘No’. We have got no data regarding the magnitude of the heart failure. We must conduct study to get enough information, so that we can make out a strategy to manage the problem. The management of heart failure should be a multidisciplinary one, which will involve cardiologists, internists, respiratory physician, paramedics, nurses, nutritionists and social workers. It can be provided only by a ‘heart failure clinic’, like diabetes care centers. We should try to develop ‘heart failure clinics’ at least in all the tertiary care hospitals and regional hospitals.

To control the pandemic we must increase the awareness of the people about the risk and impact of the heart failure. We must concentrate on prevention of heart failure by providing better treatment of hypertension, diabetes mellitus, ischaemic heart disease, obesity and other risk factors which predispose to heart failure. The treatment of Stage C heart failure should be more organized and more optimized. Most of the patients of heart failure do not receive adequate follow up treatment after they are discharged from hospital. This is due to lack of adequate motivation and lack of heart failure clinics. This results in lack of understanding of a treatment plan, non adherence to medical therapy and unawareness of heart failure symptom exacerbation. But early follow up visit within one week can lead to 8% and 29% risk reduction of all cause readmission and emergency department visits.<sup>6</sup>

Many guideline directed medications are not prescribed or prescribed at a lower dose, which needs addition and optimization in follow up visits. Only a regular follow up visit by a dedicated group of people in a heart failure clinic can make it possible. Sometimes it can be titrated by trained nurses and paramedics under supervision of a heart failure specialist. Cochrane data base review

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reveals that 27 deaths can be avoided in every 1000 patients undergoing titration of the medication by nurses under medical supervision.<sup>7</sup>

Drug related problems are also a common one in heart failure patients under treatment. A probability model has been used to estimate the drug related problems in heart failure patients in USA. Drug related problems are responsible for \$76.6 billion in hospital costs, 17 million emergency department visits and 8.7 million admissions annually.<sup>8</sup> The majority of hospital admissions and emergency department visits are preventable, and an effective medication review for the successful detection of drug related problems remains an unmet clinical need, and it can only be done by patient education and regular follow up in a heart failure clinic.<sup>9</sup>

Heart failure clinics should provide services through multidisciplinary approach. They should give the pharmacological treatment as well as advice for life style modification regarding diet, exercise, smoking, substance abuse, sleep, travelling, sexual activity, adherence to drug therapy, symptoms monitoring, self care etc. Cardiac rehabilitation and palliative care may be integrated into the overall provision for patients with heart failure.

Expertise and facilities for treatment of Stage D heart failure should also be improved, though it is a costly one. But most of these treatments have already been proved to be cost effective in many countries. Different devices for the treatment of heart failure like implantable cardioverter defibrillator, cardiac resynchronization therapy, left ventricular assist device should be made available for the patients who can afford it. We must improve our facilities for implantation of these devices; and our skill and knowledge for implantation and maintenance of these devices should also be improved. Heart transplantation is another option for the patients with resistant heart failure. Like many of the developing countries, Bangladesh is far behind the goal regarding this. Our cardiologists, cardiac surgeons, policy makers, other related persons should think about it.

Heart failure will be most prevalent and important cardiovascular illness we have to face in coming decades. We are not still prepared enough to face this pandemic of twenty first century. We need a coordinated service of the related services within the healthcare system. This

needs close collaboration between heart failure practitioners and experts in allied health professions, including nurses, paramedics, dieticians, physiotherapists, psychologists, primary care providers, and social workers. 'Heart failure clinics' can bring all these services altogether to the patients.

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## Short-term outcomes associated with bilateral internal thoracic artery grafting

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### Abstract

**Background:** Bilateral internal thoracic artery (BITA) grafting is associated with improved long-term survival and graft patency compared to single internal thoracic artery (SITA) graft and saphenous vein graft in coronary artery bypass grafting (CABG). However, BITA grafting may adversely affect early in hospital mortality and morbidity due to deep sternal wound infection. Hence, we carried out this study to evaluate early outcomes of BITA grafting in different configuration in our hospital and to assess safety and applicability of BITA grafting as a routine procedure.

**Methods:** A retrospective cross sectional study was conducted in September 2014 where all 134 patients using bilateral ITA for coronary artery bypass (CABG) at United Hospital, Dhaka, Bangladesh from January 2009 to September 2014 were included. BITA grafting were used in either in-situ or Y-graft technique. We reviewed and evaluated patients' characteristics and short-term

outcomes. The short-term outcomes included in hospital mortality and major morbidities.

**Results:** Out of 134 patients, 129 (96%) patients were male. The mean age was  $48.73 \pm 8.42$  yrs ranging from 28 to 72 yrs. Hypertension and smoking were the most common cardiac risk factors. There was no mortality in both BITA in-situ and BITA Y-graft groups. Most common postoperative complications were fever (26%), and arrhythmia (7.5%). Only one female patient (0.7) had sternal wound complication. Elderly, obesity and COPD were not observed to be associated with sternal wound complication.

**Conclusion:** Short-term outcomes of BITA grafting for CABG is excellent with no significant difference between BITA in-situ and BITA Y-graft groups.

*Keywords:* BITA, CABG, short-term outcomes, Y-graft.

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### Introduction:

Despite advances in Percutaneous Coronary intervention (PCI), coronary artery bypass surgery (CABG) remains the best therapy for severe multivessel ischemic disease and the most commonly performed cardiac operations worldwide.<sup>1</sup> The gold standard for CABG is the left internal thoracic artery (LITA) to the Left Anterior Descending Artery (LAD) due to the inherent characteristics of internal mammary artery endothelium and improved run-off of the LAD territory.<sup>2</sup>

Ten years after CABG upto 95% of internal thoracic artery (ITA) grafts remain in pristine condition whereas around three quarters of vein grafts are blocked or severely diseased.<sup>3</sup> Specially, patients who received two ITA grafts

had approximately 10% risk for reintervention at 10 years, those with one ITA had 20% risk and with no ITA had 30% risk for reintervention at 10 years.<sup>4</sup> Bilateral internal thoracic artery (BITA) grafting is associated with improved survival, better event-free life and reduced reintervention relative to CABG with single ITA grafts and saphenous vein grafts.<sup>5</sup> Manipulation of the diseased aorta in elderly patients is one of the major risk factors for stroke after CABG. Composite arterial grafts including BITA graft with a non-touch aortic off pump coronary artery bypass (OPCAB) technique offers major advantages in such a population who are at highest risk of stroke.<sup>3</sup>

Although BITA grafting appears to offer superior revascularization, it is associated with increased surgical time, increased technical challenges, increase the risk of early mortality and major morbidity, in particular

increased rates of sternal wound complication.<sup>1,4</sup> In addition, patients receiving BITA grafting had higher rates of bleeding requiring postoperative mediastinal re-exploration (2.9% vs 0.6%) along with increased rates of wound complication<sup>6</sup>

Advantages of skeletonized ITA are increase conduit length, provides superior flow and greater graft diameter, reduces the incidence of deep sternal wound infection, less postoperative paresthesia and pain, improved graft patency.<sup>7</sup> Therefore, skeletonization of the ITA leaves enough of the sternal circulation intact to facilitate proper wound healing.<sup>8</sup>

A crucial point in bilateral ITA grafting is the proper use of the right ITA. When the right ITA is used in-situ, the right coronary system is the easiest to reach and next to proximal LCX branches. But the right ITA is best used as a graft to the LAD or the marginal branches (over or under the aorta).<sup>9</sup> In-situ RITA crossing midline anterior to aorta is at risk of ITA injury in redo operation. In-situ ITA graft is a better conduit as it carries its homeostatic milieu with it and so is less prone to thrombus formation.<sup>10, 11</sup> BITA Y configuration allows the larger number of arterial anastomoses and total revascularization of the whole myocardium in selected patients. However, Composite T or Y grafting with BITA brought back concern of a potential "steal phenomenon" of the LITA by the RITA.<sup>10</sup>

Considering the above, we carried out this study to review our experience of performing BITA grafting using both ITA in-situ graft and BITAY-graft technique for CABG. We also evaluated and compared short-term outcomes of BITA grafting in both configuration and assessed safety and applicability of BITA grafting as a routine procedure.

#### Patients and Methods:

A retrospective cross sectional study was conducted in which all patients undergoing bilateral internal thoracic artery grafting for coronary bypass surgery only at United Hospital, Dhaka, Bangladesh from January 2009 to September 2014 were included. During this period, 134 patients underwent BITA grafting. Out of 134 patients, 111 patients received BITA Y-graft and 23 patients BITA in-situ for CABG.

#### Surgical technique

During the study period both left and right internal thoracic artery were harvested by skeletonization technique. One ITA was used to bypass left anterior descending artery (LAD) and second ITA was used to bypass either LCX or RCA system using in-situ or Y-graft technique according to surgeon preference based on position of targeted vessels ( Figure-1 and Figure-2). All grafting were done on off pump beating heart (OPCAB).



Fig.-1: LITA and RITA in-situ

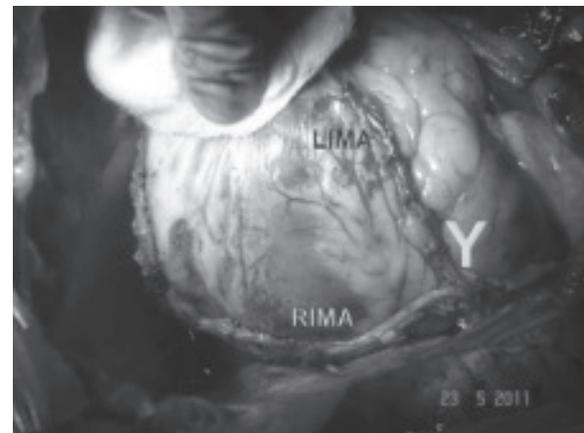


Fig.-2: LITA/LIMA and RITA/RIMA Y graft.

#### Data collection

The data were obtained by retrospective review of the patients' hospital records. The demographics, clinical profile, co-morbid factors, underlying disease pathology and severity, type of conduits used and corresponding target vessels, operation time, intensive care unit (ICU) and total hospital stay were recorded. We compared the short term outcomes as defined by the Society of Thoracic Surgeons (STS) including 30-day operative death, permanent stroke, renal dysfunction or renal failure requiring dialysis, any reoperation, prolonged ventilation (>48 hours), deep sternal wound infection, perioperative myocardial infarction, arrhythmia and fever for assessing the safety and efficacy of the procedure in both groups (insitu and Y graft group) of patients undergoing BITA grafting for CABG.

#### Statistical analysis

The data collected were entered in Epi-Info and analyzed through SPSS (version 16.00). Results are expressed as Frequencies and means, as appropriate, for the demographic data and clinical characteristics of the study

population. Statistical analysis comparing two groups with the unpaired 2-tailed t test for the means or  $\chi^2$  test for categorical variables. The frequencies with which each conduit was used for grafting were also calculated and tabulated. Significance was set at  $p < 0.05$ . The chi-square test was used to associate different post-operative complications with the pre-operative patient characteristics and to determine if any association observed was a statistically significant one.

**Results:**

134 patients were selected for the study, out of them 111 (82.84%) patients had BITA Y-grafting and rest 23 (17.16%) patients had BITA in-situ grafting. Male were 129 (96.27%) and 5 (3.73%) female patients. Patients had a wide range of age from 28 to 72 years with the mean age being  $48.73 \pm 8.42$  years. There were 13 (9.7%) patients being more than 60 yrs of age. 55 (41.02%) patients were overweight (BMI =25-30kg/m<sup>2</sup>), and three (2.27%) patients were obese (BMI >30kg/m<sup>2</sup>). Among the cardiac risk factors, Hypertension (70.1%) and Smoking (56%) were most common followed by Diabetes (41.81%). 8 (6.0%) chronic kidney disease, 5(3.7%) calcified aorta and 5 (3.7%) chronic obstructive pulmonary disease (COPD) present in these patients. Majority (64.3%) of patients had triple vessel disease (TVD) and 17.8% patients had TVD-LM. 5 (3.9%) patients had low LVEF(less than 40 %.) (Table-1).

Right internal thoracic artery (RITA) was harvested and used as an in-situ graft in 23 patients and remaining 111 patients received RITA as Y-graft with LITA. The target vessels for the in-situ LITA were mainly Left anterior descending artery (LAD), then Ramus intermedius (RI) and Obtuse marginal (OM) arteries while those for the in-situ RITA artery included Right coronary artery (RCA)

as well as LAD. Average no. of  $3.48 \pm 1.12$  and  $3.72 \pm .93$  distal anastomoses per patient done in in-situ and Y-graft group respectively and no. of arterial graft per patient was  $2.22 \pm .518$  and  $2.33 \pm .61$  in in-situ and Y-graft group respectively. Average operative time was  $5.03 \pm 1.05$  hours and  $4.87 \pm 1.14$  hours in BITA Y graft and BITA in-situ group respectively. Mean ICU stay was  $3.04 \pm 1.38$  days with range of 1 to 12 days and  $3.48 \pm 1.50$  days with range of 2 to 8 days in BITA Y graft and BITA in-situ group respectively. Average total hospital stay was relatively more in BITA in-situ group than BITA Y graft. (Table-2)

Hospital mortality and perioperative morbidity were reviewed. There was no in hospital mortality in both groups. However, 4 (3.6%) patients developed perioperative MI with raised cardiac enzymes, as type 5 MI define raised cardiac biomarker >10×99th percentile upper reference level (URL) during the first 48 h following CABG, 5 (4.5%) patients required re-opening for excessive postoperative bleeding and 1 (0.9%) patient developed respiratory complication (ARDS) that needed prolonged ventilatory support. In BITA Y-graft group, 1 (0.9%) female patient developed sternal wound complication with unstable sternum after discharge. She needed re-admission for sternal wound management. She had history of recent MI, diabetes and prolonged preoperative hospital stay (7 days) but hold normal body weight (BMI 24 kg/m<sup>2</sup>). 1 (4.3%) patient required IABP support peroperatively and postoperatively needed prolonged artificial ventilation and developed acute kidney injury (AKI) that improved with conservative treatment in BITA in-situ group. Other postoperative complications including arrhythmia (7.5%) and fever (26%) observed in both groups that increased ICU stay and total hospital stay (Table-3).

**Table-I**  
*Preoperative characteristics of patients underwent CABG with BITA grafting.*

Finding	All patients(n=134)	BITAYgraft(n=111)	BITA in-situ graft(n=23)	p-value
Male	129 (96.27)	106 (95.5)	23 (100)	.300
Female	5(3.73)	5(4.5)	0(0)	
Mean age in yrs	$48.73 \pm 8.42$	$48.29 \pm 8.57$	$50.87 \pm 7.44$	.182
>60 yrs n (%)	13(9.7)	11(9.9)	2(8.7)	
BMI>30 n (%)	3 (2.27)	2(1.9)	1( 4.36)	
Hypertension n (%)	94(70.1)	80(72.1)	14(60.9)	.285
Smoking n (%)	75(56)	60(54.1)	15(65.2)	.326
Diabetes n (%)	56(41.8)	50(45.0)	6(26.1)	.093
CKD n (%)	8(6.0)	6(5.4)	2(8.7)	.544
COPD/asthma (%)	5(3.7)	4(3.6)	1(4.3)	.864
Calcified Aorta n(%)	5(3.7)	3(2.7)	2(8.7)	
EF <40%	5(3.9)	2(1.8)	3(15.8)	
TVD	87(64.9)	71(64)	16(69.6)	

Paresenthesi (%), Mean± standard deviation

CKD-Chronic Kidney Disease, COPD-Chronic Obstructive Pulmonary Disease, EF-Ejection Fraction, TVD-Triple vessel disease,

**Table-II**  
*Distribution of patients by perioperative and postoperative findings in both groups*

Findings	All patients (n=134)	BITA Y-graft(n=111)	BITA in-situ graft(n=23)	p-value
Total Graft no.	3.68±.97	3.72±.93	3.48±1.12	.277
No. of arterial graft	2.31± 0.59	2.33±0.61	2.22±0.518	.396
No of venous graft	1.37±0.954	1.39±0.946	1.26±1.01	.752
Operative time	5±1.06 hours	5.03±1.05 hours	4.87±1.14 hours	.522
ICU stay	3.11±1.41 days	3.04±1.38 days	3.48±1.50 days	.26
Total Hospital stay	9.9±1.78 days	9.86±2.71 days	10.09±1.99 days	.43

Mean± standard deviation  
ICU-Intensive Care Unit

**Table-III**  
*Distribution of patients by major morbidity*

Morbidity	All patients (n=134)	BITA Y-graft (n=111)	BITA in-situ graft (n=23)	p-value
Perioperative MI	4(3.0)	4(3.6)	0(0.0)	.355
Re-opening for Bleeding	5(4.0)	5(4.5)	0(0.0)	.300
Prolong ventilation	2(1.5)	1(0.9)	1(4.3)	.215
Respiratory complication	1(0.7)	1(0.9)	0(0.0)	.648
Renal failure	1(0.7)	0(0.0)	1(4.3)	.027
Stroke	0(0.0)	0(0.0)	0(0.0)	
Arrhythmia	10(7.5)	9(8.1)	1(4.3)	.532
Fever	35(26.1)	29(26.1)	6(26.08)	.997
SWI	1(0.7)	1(0.9)	0(0.0)	.677
Re-admission	1(0.7)	1(0.9)	0(0.0)	.648

Paresentthesis (%)  
MI- Myocardial Infarction, SWI-Sternal wound infection

The chi-square test was used to associate different post-operative complications with the pre-operative patient characteristics. Both female sex ( $p<0.014$ ) and age greater than 60 years ( $p<0.025$ ) were associated with sternal wound infection (SWI), but diabetes ( $p=0.236$ ), obesity ( $p=0.949$ ), COPD ( $p=0.843$ ), smoking ( $p=0.258$ ), and CKD ( $p=0.800$ ) were not associated with SWI.

### Discussion

The use of the internal thoracic artery (ITA) as a conduit for CABG has changed greatly since its initial introduction in the 1950s and 1960s. Bilateral internal thoracic grafting (BITA) has been demonstrated to be associated with improved long-term survival and graft patency when compared with both single ITA grafting and with the use of venous grafts alone.<sup>12</sup> A mean 15% increase in the 20 year survival has been observed in such patients.<sup>13</sup> On the molecular level, it has been observed that there is an enhanced release of endothelial derived relaxing factor

nitric oxide (NO) from the ITA as compared to venous grafts.<sup>14</sup> NO directly regulates blood flow and inhibits platelet function and indirectly allows lesser neutrophil adhesion to the endothelium that coincides with increased short and long-term vessel patency.<sup>15-17</sup> However, some cardiothoracic surgeons have reservations about the routine use of BITA due to procedure requiring better surgical skills, takes longer time to perform, and may adversely affect early in-hospital mortality and morbidity, in particular due to deep sternal wound infections.<sup>6</sup> Most however have reported no increased risk in perioperative death or morbidity conferred.<sup>18</sup> Hence, we carried out this study to evaluate the early outcomes observed in our hospital to assess the safety and applicability of BITA grafting as a routine procedure in a tertiary care Hospital in Bangladesh.

In our study most of patients was male (96.27%) and mean age was 48.73 ±8.42 years range from 28 to 72

years. BITA grafting has traditionally been performed in younger patients having a greater life expectancy. The trend of relatively younger patients (mean age 48 years), and male (96%) having undergone BITA grafting at our hospital matches with other studies<sup>19</sup>. Thirteen (9.7%) patients were more than 60 yrs of age in this study. Elderly patients can be benefited from arterial grafts.<sup>20, 21</sup> Elderly patients have sub-optimal venous conduit due to varicosities and calcification,<sup>22</sup> prone to early occlusion. In our study, elderly patients had showed early return to normal life without complication. In our study population, most of patients (80%) had good Left ventricular ejection fraction (LVEF) and 5 (3.7%) patients had LVEF less than 40%. Most of study showed that an important criterion related to the use of arterial conduits is the functional status of the left ventricle. Patients received BITA also had a slightly better mean EF (0.55 vs. 0.52) than those who only received a LITA.<sup>2</sup> Significantly impaired left ventricular function is associated with limited life expectancy, on the other hand increase in the 20-year survival period is observed in patients with BITA grafting.<sup>23</sup>

In the study patients, 56(41.81%) patients had diabetes and 53 (39.6%) patients were dyslipidaemic. Literature suggests diabetes as a risk factor for postoperative mediastinal wound infection and hence a possible relative contraindication for doing bilateral ITA grafting in these patients.<sup>24</sup> However, in diabetic patients, increased use of skeletonized harvesting techniques when performing a BITA grafting along with other infection prevention strategies have shown a reduction in deep sternal wound infection (DSWI) comparable to that seen in LITA patients.<sup>2</sup> In our study, higher incidence of DSWI was found in diabetic patients (1 of 56; 1.8%) than in non-diabetic patients (0 of 78; 0%) but this difference was not significant ( $p=0.236$ ). In this study 75(56%) patients were smoker, 55(41%) patients overweight and 3 patients obese (BMI > 30) and five (3.7%) patients had COPD. Obesity is considered an independent risk factor for mediastinitis.<sup>25</sup> In our study, none of obese and COPD patients developed wound infection postoperatively.

BITA grafting is associated with increased early morbidity and mortality,<sup>26,27</sup> specifically the occurrence of deep sternal wound infection (DSWI) and the well-documented increased risk of death that accompanies it. DSWI has been reported to occur in as low as 0.3% and as high as 14% of BITA procedures and is thought to be a result of the decreased sternal perfusion exacerbated by bilateral versus unilateral harvest of the ITA.<sup>2</sup> Skeletonization of the ITA leaves enough of the sternal circulation intact to

facilitate proper wound healing. In our study all ITA were harvested by skeletonization technique, therefore sternal wound complication was minimum (0.7%).

The assessment of the safety of the procedure has usually been compared in literature with the results of using unilateral ITA for grafting. In our study there was no mortality But 4(3.6%) patients developed perioperative MI, 5(4.5%) patients required re-opening, 1(0.9%) patient needed prolonged ventilatory support, and 1(0.9%) female patient developed sternal wound complication in BITA Y-graft group. 1(4.3%) patient developed acute renal failure and required prolonged ventilation in BITA in-situ group. These morbidities in this study were less than other study.<sup>28</sup> In our study all ITA were harvested by Skeletonization technique, therefore sternal wound complication was minimum (0.7%). However, there is lack of association observed among diabetes, COPD, obesity and the development of wound complications.

In the study 134 patients were selected, out of them 111 (82.84%) patients had BITA Y-grafting and rest 23 (17.16%) patients had BITA in-situ grafting. In bilateral ITA grafting, the right ITA has been flexibly used as an in-situ or free graft in combination with an in situ left ITA. An in-situ right ITA can be used by anastomosing it with the left anterior descending coronary artery (LAD) and its diagonal branch along the front of the ascending aorta or to the obtuse marginal artery through the transverse sinus.<sup>9</sup> A free right ITA, on the other hand can be used as a composite graft allowing for multiple sequential anastomoses. Advocates for both the methods are found in literature.<sup>29,30</sup> However, identical patency rates have been observed in early, 1-year and the 5-year. Traditionally, inferior rates of RITA patency versus LITA patency have been documented regardless of choice of graft site<sup>31</sup> and demonstrated no difference in patients receiving an *in situ* RITA to the left or right coronary system.<sup>32</sup> A prospective study revealed that excellent patency rates were achieved using both BITA configuration with no significant difference in terms of MACCE or ITA patency.<sup>10</sup> In our study, most of the RITA grafts(82.8%) were used as a composite Y graft with in-situ LITA and only a few in-situ RITA grafts (17.16%) employed. However, there is no definitive criterion for the selection of patients suitable to undergo BITA grafting. But there was no significant different in short-term outcomes in both configurations. Therefore, excellent short-term outcomes and safety of BITA grafting found in this study and BITA bypass grafting may become a first-line option for patients receiving revascularization. Though our sample size is inadequate to comment on the outcomes, grossly both the groups

showed comparable rates of perioperative outcomes and early efficacy, with no p-value being statistically significant.

#### Conclusion:

The short-term outcomes and the safety profile of bilateral ITA grafting in both in-situ and Y graft technique for CABG seems clinically acceptable even for diabetic or obese individuals. A long-term follow-up should be done to assess the cardiac event-free survival of these individuals evaluating the long-term implication of the procedure and hence its applicability as a routine for coronary artery bypass grafting.

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## Better in-hospital outcome among hypertensive subjects developing acute myocardial infarction—study in a tertiary cardiac care center in Bangladesh

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### Abstract

**Background:**In-hospital complications and mortality in hypertensives developing acute myocardial infarction(AMI) may be different from those of normotensive counterpart.The aim of the current study was to analyze in-hospital complication and outcome of AMI in hypertensive patients and compare it with age and sex matched normotensive AMI patients. **Methods:** In-hospital complications of 112 hypertensive patients with AMI admitted over a period of 1 year (April 2014 to March 2015) were compared with the control group. Location and types of AMI were determined by ECG. Patients were considered to be hypertensive if they were taking antihypertensive treatment or were found to have a systolic blood pressure (SBP)  $\geq 140$  mmHg and/or a diastolic blood pressure (DBP)  $\geq 90$  mmHg on repeated measurements. Both groups were studied prospectively. The results were analyzed by SPSS software. **Results:**Out of total 112 patients 69 were male and 43 were female in each group. Male: female ratio was 1.6:1. Mean age of the study population was  $67.3 \pm 10.2$  yrs (range 41 to 83 yrs). Mean blood pressure values were  $145.7 \pm 11.5/88.3 \pm 8.9$  mmHg in the hypertensive and

$127.3 \pm 9.7/75.8 \pm 5.6$  mmHg in normotensive group respectively. A significantly higher prevalence of diabetes, dyslipidemia, chronic kidney disease, stroke and peripheral vascular disease were found in the hypertensive compared with the normotensive subjects. Hypertensive AMI patients had higher left ventricular ejection fraction compared with the normotensives ( $0.51 \pm 0.13$  vs  $0.47 \pm 0.15$ ). A higher frequency of paroxysmal atrial fibrillation(AF) (10.7% vs 7.1%,  $P < 0.05$ ) and a lower frequency of atrioventricular block (5.4% vs 8.0%), ventricular fibrillation (2.7% vs 4.5%), cardiogenic shock (4.5% vs 9.8%,  $P < 0.01$ ) and a lower in-hospital mortality (6.2% vs 10.7%,  $P < 0.01$ ) were found among the hypertensives compared with the normotensives. **Conclusion:** Hypertensive AMI patients had a significantly higher incidence of AF, lower incidence of cardiogenic shock and an overall better in-hospital outcome compared with the normotensives, probably owing to a better preserved left ventricular function, prior use of cardioprotective drugs and yet undefined mechanism.

*Key Words:* Hypertension, AMI, Outcome

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### Introduction:

Hypertension is a major risk factor for the development of AMI. It is considered to participate in the pathogenesis of atheromatous plaque, its ulceration and thrombosis<sup>1</sup>.

Prevalence of hypertension in AMI varies from 31% to 50% in various studies<sup>2</sup>. There is a paucity of data on hemodynamic and electrical complications of AMI in hypertensive patients. Mauri et al<sup>3</sup> reported an increased incidence of sudden death in AMI patients with a history of hypertension. On the other hand Abrignani et al<sup>4</sup> found lower incidence of shock, ventricular fibrillation, conduction disturbances, cardiac rupture and intracardiac thrombus in hypertensive patients with first MI, while atrial fibrillation was more common in these patients. More recently Rombek et al<sup>5</sup> found higher incidence of cardiogenic

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shock, pulmonary oedema, ventricular tachycardia and/or ventricular fibrillation and third degree

atrioventricular(AV) block. Other studies did not show relevant difference for in-hospital and 6 month's mortality in hypertensive and normotensive patients with MI.<sup>6</sup>

Prevalence of hypertension as well as coronary artery disease is increasing in countries of Indian subcontinent, including Bangladesh. Because of increasing number of hypertensive subjects developing acute myocardial infarction, it is an important subject to give attention. Hence, the study was done to estimate the in-hospital complications and outcome of AMI among hypertensive subjects.

### **Aim of Study**

To analyze the in-hospital complication and outcome of AMI in hypertensive subjects and compare it with age and sex matched normotensive AMI patients.

### **Material & Method**

Study design-prospective observational;case control

Inclusion criteria-first onset AMI patient with antecedent hypertension or newly diagnosed hypertension

Exclusion criteria- i.patients with previous history of myocardial infarction.ii.patients with previous baseline ECG findings of left bundle branch block, pre-excitation syndrome

The study was conducted in the coronary care unit of Chittagong Medical College Hospital.The study period was one year (April, 2014 to March, 2015).We found a total of 112 patients in each group.As control, we randomly selected age and sex matched normotensive individuals with AMI. The study protocol was approved by the ethical committee on human research of the institute. Patient's characteristics, location and types of AMI, diagnostic and therapeutic measures undertaken and various complications were recorded. AMI was diagnosed if at least two of the following criteria were met<sup>7</sup>: (i) typical chest pain lasting for at least 30 min and not relieved by nitrates (ii) ST segment and/or T-wave changes suggestive of myocardial infarction and (iii) serum troponin- i or creatine phosphokinase(CK) concentration of more than twice the upper limit of normal range. The patients were categorized as having antecedent hypertension if the diagnosis was known by the patients or his attendant to having been made by their family physician, pre-admission records showed that they were hypertension and/or they were receiving anti-hypertensive drugs. A diagnosis of new hypertension were made when BP was <140/90 mmHg on receiving the patient that persisted in that range after the patient's condition settled (in 1-2 hrs). Blood for fasting glucose and lipid profile

was drawn within 24 hrs of hospital admission. An individual was considered to be diabetic if he or she was receiving insulin or oral hypoglycemic agents or had symptoms of diabetes with fasting blood glucose >126mg/dl<sup>8</sup>. Dyslipidemia was defined when the patients had history of taking lipid lowering drugs or when any of the lipid fractions was abnormal, for example, serum cholesterol >160mg/dl or HDL<35mg/dl, LDL >100mg/dl or triglyceride >150mg/dl<sup>8</sup>. A family history was considered positive when symptomatic coronary artery disease occurred in siblings, parent's sibling's or grand parents before age 45 in male and 55 in female. Blood sample were send for estimation of serum troponin-I, CK-MB (when re-infarction suspected). The patients were treated with medications according to current recommendations. Those presenting with AMI and a very high blood pressure, a controlled reduction of BP without compromising coronary flow was undertaken with anti-hypertensive drugs to a range of goal BP of 160-170 mmHg systolic and 100-110 mmHg diastolic. Thrombolytic therapy with i.v streptokinase was administered to the eligible patients presenting within 12 hours of onset of chest pain. Those presenting with very high BP, therapy was withheld until BP was reduced to less than 160/110 mmHg with medication. Intravenous nitroglycerine (dose 0.5 to 5.0 microgm/min) was used in patients with hypertensive emergencies, and hypertensives with AMI with or without left ventricular failure. Oral beta-blocker and angiotensin converting enzyme inhibitors were used in all hypertensive AMI patients unless contraindicated. These drugs were also used in all normotensive patients with anterior wall infarction unless contraindicated. Presence of heart failure was assessed clinically according to Killip classification as follows: Class I- no evidence of heart failure Class II- bi-basilar rales±S3, Class III- pulmonary edema (with respiratory distress, diaphoresis, cold extremity), Class IV- cardiogenic shock. Hemodynamic monitoring with central venous and arterial line were done in patients with cardiogenic shock. Patients with acute left ventricular failure was managed with i.v morphine and diuretics. Those developing cardiogenic shock were managed conservatively with i.v.inotropes(dopamine and dobutamine).Arrhythmic complications were managed with appropriate anti-arrhythmic medications, e.g digitalis for atrial fibrillation, i.v lignocaine,amiodarone etc. for ventricular tachycardia, ventricular fibrillation, along with cardioversion in appropriate cases. Temporary pacemaker implantation was done for patients with AV block and hemodynamic instability, followed by implantation of permanent pacemaker in cases of

anterior wall infarction. Doppler echocardiographic study was done in all patients to assess LV function and mechanical complications post MI.

3.4. Statistical analysis—results were presented as mean ± standard deviation for continuous variables and numbers (%) for categorical variables. Student's 't' test and chi-square tests were used to determine the level of significance. P value <0.05 was considered statistically significant

**Results**

A total of 224 subjects were analysed with 112 subjects in each group, There were 69 male (61.6%) and 43 (38.4%) female in both groups. Male:female ratio was 1.6:1. Mean age of the study population was 67.3 ±10.2 yrs (range 41 to 83 years). Baseline characteristics of the patient population are shown in table I.

Concomitant risk factors of Coronary artery disease (CAD) in the two study groups are shown in table II.

DM, dyslipidaemia and a positive family history of CAD were significantly higher in hypertensives subjects compared with normotensives. Normotensive subjects had a higher prevalence of smoking compared with hypertensives (not statistically significant).

Hypertensive AMI subjects showed a higher prevalence of co-morbidities as shown in table III.

Various diagnostic and therapeutic procedures undertaken in the two study groups are shown in table IV

Anterior wall MI was the commonest in both the study groups followed by inferior MI with no significant difference between them (47% vs 46% and 35% vs 37% respectively). Seven percent of hypertensives and 10% of normotensive subjects had a combined (ant.& inf.) MI. A significantly higher percentage of hypertensive subjects had NSTEMI compared with the normotensives (19.9% vs 15.9%, P<0.05).

Majority of the hypertensive subjects (69%) presented to the CCU within 12 hrs of onset of their chest pain. Whereas, 55% of the normotensive subjects presented within 12 hrs of onset of their chest pain (p<0.05). Thus a greater number of hypertensive subjects were being thrombolysed (57.1% vs 50.9%) compared with normotensives, even though patients with BP >160/110mmHg were excluded from therapy.

Pharmacologic treatment undertaken in the two groups are shown in table V.

A higher percentage of hypertensive subjects received low molecular weight heparin (LMWH) compared with the normotensives consistent with the higher NSTEMI cases among the hypertensives, all of whom received LMWH. Other cardioprotective drugs (beta blockers, ACEI) were used significantly more in hypertensive subjects.

**Table-I**  
*Characteristics of hypertensive subjects (n=112)*

Characteristics	Frequency	Percentage (%)
Sex		
Male	69	61.6
Female	43	38.4
Antecedent hypertension	103	92.0
Poorly controlled	38	33.9
Well controlled	65	58.0
Anti-hypertensives used :		
Beta blockers + calcium channel blockers	33	29.5
ACE inhibitors / angiotensin receptor blockers	27	24.1
Beta blockers	13	11.6
Angiotensin receptor blockers +Thiazides	10	8.9
Calcium channel blockers	9	8.0
Thiazides	6	5.4
Angiotensin receptor blockers + calcium channel blockers	3	2.7
Other drugs	2	1.8
Newly diagnosed hypertension	9	8.0

\*Mean duration of raised BP: 13±7.4 yrs

**Table-II**  
*CAD risk factors in the two groups*

Risk factors	Hypertensive(n = 112)	Normotensive(n = 112)	P value
Smoking	58 (51.8)	59 (52.7)	NS
Diabetes mellitus	47 (42.0)	38 (33.9)	<0.05
Dyslipidemia	43 (38.4)	40 (35.7)	<0.05
Positive F/H of CAD	29 (25.9)	19 (17.0)	<0.01
Obesity	7 (6.2)	6 (5.4)	NS

Figures within parentheses indicate percentages

**Table-III**  
*Co-morbidities in the two groups*

Co-morbidities	Hypertensive(n = 112)	Normotensive(n = 112)	P value
Chronic obstructive pulmonary disease	13 (11.6)	9 (8.0)	<0.01
Cerebrovascular disease	11 (9.8)	5 (4.5)	<0.05
History of revascularization	8 (7.1)	5 (4.5)	NS
Chronic kidney disease	7 (6.2)	3 (2.7)	<0.05

Figures within parentheses indicate percentages

**Table-IV**  
*Diagnostic and therapeutic procedures(n=224)*

Procedures	Hypertensive(n = 112)	Normotensive(n = 112)	P value
Doppler echocardiography	112 (100.0)	112 (100.0)	–
Chest radiography	27 (24.1)	19 (17.0)	<0.01
Hemodynamic monitoring	12 (10.7)	9 (8.0)	NS
Cardio-pulmonary resuscitation	8 (7.1)	11 (9.8)	NS
Cardioversion	7 (6.2)	8 (7.1)	NS
Transvenous pacing	6 (5.4)	5 (4.5)	NS

Figures within parentheses indicate percentages

**Table-V**  
*Drug administered in CCU in two groups*

Drugs	Hypertensive(n = 112)	Normotensive(n = 112)	P value
Clopidogrel	97 (86.6)	96 (85.7)	NS
LMW heparin	70 (62.5)	63 (56.2)	NS
Thrombolytic (streptokinase)	64 (57.1)	57 (50.9)	<0.05
Oral GTN	62 (55.4)	48 (42.9)	<0.01
ACE inhibitors / angiotensin receptor blockers	47 (42.0)	39 (34.8)	<0.05
Beta blockers	37 (33.0)	26 (23.2)	<0.01
Diuretics (frusemide)	32 (28.6)	24 (21.4)	<0.05
Lignocaine	25 (22.3)	31 (27.7)	NS
GTN i.v.	23 (20.5)	17 (15.2)	<0.01
Dobutamine / Dopamine	20 (17.9)	22 (19.6)	NS
Digoxin	9 (8.0)	7 (6.2)	NS
Calcium channel blockers	8 (7.1)	3 (2.7)	NS

Figures within parentheses indicate percentages

**Evolving Complications:**

Analysis of arrhythmic complications developing in the two study populations shows that a significantly higher prevalence of paroxysmal atrial fibrillation and a lower prevalence of conduction disturbance (AV block) and ventricular arrhythmia were found in hypertensives compared with normotensives Table VII shows the various mechanical complications developing in the two study populations.

Hypertensive AMI patients had a higher prevalence of LV failure (Killip class 2-4). A significantly higher percentage of cardiogenic shock (4.5% vs 9.8%, p<0.01) was found among the normotensive subjects compared with

hypertensives. Analysis of ischemic complications (Table VIII) shows that a significantly higher reinfarction rate was present in the hypertensive group.

Mean hospital stay of the two groups were not significantly different (9±2.7 days in hypertensives vs. 9±1.8 days in normotensives). Echocardiographic examination revealed a higher left ventricular ejection fraction (LVEF) among the hypertensives compared with the normotensives (0.51±0.13 vs. 0.47±0.15). In-hospital mortality in the two groups are shown in table IX.

There was significantly lower in-hospital mortality among the hypertensive AMI subjects compared with the normotensive AMI subjects.

**Table-VI**  
*Arrhythmic complications in the two groups(n=224)*

Arrhythmic Complications	Hypertensive(n = 112)	Normotensive (n=112)	P value
Sinus tachycardia	75 (67.0)	85 (75.9)	<0.01
Paroxysmal AF	12 (10.7)	8 (7.1)	<0.05
AV block	6 (5.4)	9 (8.0)	NS
Ventricular fibrillation	3 (2.7)	5 (4.5)	NS
Ventricular tachycardia	2 (1.8)	3 (2.7)	NS

Figures within parentheses indicate percentages

**Table-VII**  
*Mechanical complications in the two groups*

Mechanical Complications	Hypertensive(n = 112)	Normotensive (n=112)	P value
LV failure	22 (19.6)	20 (17.9)	NS
Cardiogenic shock	5 (4.5)	11 (9.8)	<0.01
Mitral regurgitation	3 (2.7)	2 (1.8)	NS
LV aneurysm	3 (2.7)	5 (4.5)	NS
Ventricular septal rupture(VSR)	2 (1.8)	1 (0.9)	NS

Figures within parentheses indicate percentages

**Table-VIII**  
*Ischemic complications in the two groups*

Ischemic Complications	Hypertensive(n = 112)	Normotensive (n=112)	P value
Re-infarction	11 (9.8)	7 (6.2)	<0.05
Post MI angina	8 (7.1)	6 (5.4)	NS
Infarct expansion	3 (2.7)	2 (1.8)	NS

Figures within parentheses indicate percentages

**Table-IX**  
*In-hospital mortality in the two groups*

In-hospital mortality	Hypertensive(n = 112)	Normotensive (n=112)	P value
	7 (6.2)	12 (10.7)	<0.01

Figures within parentheses indicate percentages

### Discussion:

The role of arterial hypertension in relation to in-hospital outcome in AMI has not been definitely established. Available evidence does not clearly show an increased rate of adverse outcome after AMI including stroke, heart failure and cardiovascular death among hypertensives<sup>9</sup>. Hypertension has been found to be a weak predictor of death in AMI patients by several investigators<sup>10</sup>. Muri et al<sup>3</sup> described that the increased risk of hemodynamic complications and death is related to the extent and expansion of MI, an anterior wall involvement, an increased extent of CAD, the presence of severe ventricular arrhythmias and myocardial rupture. Mortality from CAD rises continuously with increasing levels of systolic and diastolic BP<sup>14</sup>. However the relationship between the two is complex.

Prevalence of hypertension increases with increase in age, which is itself an important predictor of poor outcome in AMI<sup>15</sup>. Despite a global reduction in mortality from AMI in general population it remains still high among elderly<sup>16</sup>. Mortality from AMI is also high among females<sup>4</sup>. In an attempt to exclude age and sex as confounding factor we matched normotensives AMI patients by these factors. Hypertension was found to be combined with increased associated co-morbidities in the form of cerebrovascular disease, peripheral arterial disease, renal failure, diabetes, dyslipidemia and chronic obstructive airway disease<sup>4</sup> and on the basis of observations of other authors, greater target organ damage, such as nephropathy, microalbuminuria etc.<sup>11</sup>. Our hypertensive AMI patients had a higher prevalence of co-morbidities (table-2) such as DM, dyslipidemia, chronic kidney disease, chronic obstructive airway disease and history of revascularization which is in agreement with the findings of Ascenzi et al<sup>17</sup>. We did not find any significant difference regarding anterior or inferior location of AMI in our patients but a significantly higher rates of non ST-elevation MI (19.9% vs 15.9%,  $P < 0.05$ ) was found in hypertensives subjects compared with normotensives. A higher prevalence of non-ST elevated MI (NSTEMI) among hypertensives were also described in epidemiologic studies, where chronic hypertension was found as the most prevalent risk factors<sup>13</sup>. This higher prevalence of NSTEMI cases among the hypertensives may have also contributed to the lower in-hospital complications and mortality which is also in agreement with the findings of Abrignin et al<sup>4</sup>. Hypertensive subjects are known to have severe coronary atherosclerosis<sup>19</sup> which allows development of more collateral circulation. It explains the more NSTEMI cases among the hypertensives.

Most of the studies regarding links between hypertension and MI consider patients with a previous history of hypertension but data are lacking regarding patients who do not have antecedent hypertension but showed elevated BP values during their hospital stay for AMI. A study on patients with AMI admitted within 6 hours of onset of pain 31.7% patients had an elevated BP ( $\geq 180/100$  mmHg) on presentation, only 6.3% of them had elevated BP after 6 hours though not treated with antihypertensive drugs<sup>20</sup>. We found such new cases of hypertension in 8% of our patients. We undertook a controlled reduction of BP in our AMI patients with hypertension in the CCU. Patients with long standing uncontrolled hypertension can not tolerate rapid great reduction of BP. The hypertensive myocardium is sensitive to BP lowering below a critical level. A rapid reduction of diastolic BP may potentially be more dangerous to the heart. Result of a recent analysis of AMI treated early with ACE inhibitors suggest caution in patients with antecedent hypertension but with low or normal BP at presentation, as these patients showed a higher first day mortality<sup>25</sup>. The recommended BP target in hypertensive AMI subjects is not completely clear. Further studies are needed to define the issue more clearly.

Analysis of arrhythmic complications showed that only paroxysmal AF was found to be more frequent in our hypertensive patients. Other arrhythmias and AV block were more in normotensive subjects. This is in contrast to the general concept that hypertensive individuals are more prone to cardiac arrhythmias<sup>21</sup>. Abrignani et al<sup>4</sup> found an almost similar findings in their hypertensive patients with first time MI. A smaller necrotic area and presence of well developed collateral may better preserve conduction pathway, explaining the lesser prevalence of conduction disturbance and ventricular arrhythmias in hypertensive subjects. A higher prevalence of paroxysmal AF among our hypertensive subjects may be related to left atrial enlargement and chronically elevated left atrial pressure<sup>22</sup>. Prevalence of cardiogenic shock was significantly lower in our hypertensive patients (4.5% vs 9.8%,  $P < 0.01$ ) compared with the normotensives. Cardiogenic shock remains the leading cause of death in patients hospitalized with AMI<sup>18</sup> but its incidence has been greatly reduced by the use of reperfusion therapy through mechanical revascularization. Because of lack of mechanical revascularization and IABP facilities we could only undertake pharmacologic reperfusion with i.v. thrombolytic and conservative management of cardiogenic shock with inotropes. A higher percentage of our hypertensive ST elevated MI (STEMI) patients received

thrombolytic therapy because of their early presentation to the CCU. Among the hypertensive STEMI subjects 69% presented to CCU within 12 hrs. Whereas only 55% of the normotensive subjects presented before 12 hrs. Higher rates of thrombolytic administration to the hypertensive subjects had possibly limited their infarct size as evident by the lower serum enzyme levels and a higher LV ejection fraction on echocardiography.

The lower incidence of cardiogenic shock and a better preserved LV function was responsible for significantly lower in-hospital mortality in the hypertensive AMI patients. Hypertensive subjects had a lower in-hospital mortality compared with the normotensives (6.2% vs. 10.7%,  $P < 0.01$ ). This is probably due to a better preserved LV function with a higher LVEF among the hypertensives ( $0.51 \pm 0.13$  vs.  $0.47 \pm 0.15$ ) compared with the normotensives. This is in accordance with the findings of Abrignani et al<sup>4</sup> who are the first to show a significantly lower in-hospital complications and morbidity in hypertensive subjects with first time AMI compared with normotensives. This lower in-hospital mortality is in contrast to the findings of Richards et al<sup>23</sup> who found the same as 8.1% in their hypertensive patients compared with 4.4% among the normotensives. Their hypertensive patients also had a higher incidence of acute LV failure (33% vs. 24%) compared with the normotensives. Literature published so far shows that this is one of the few studies that showed a significantly better in-hospital outcome in hypertensive AMI subjects. Although this better outcome may not be maintained long term because of the greater severity of CAD in hypertensives and higher co-morbidities among them<sup>25</sup> and an adverse long term outcome is not unusual. Further studies may clarify the issue.

The study has got some important limitations:

i. Besides age and sex other patient characteristics were not excluded as confounding variables. ii. 24 hours blood pressure recording of the study population were not possible. iii. primary PCI was not possible due to lack of facility. iv. only in-hospital outcome data are described here, post discharge follow up and long term complications and mortality data are lacking.

#### **Conclusion:**

Hypertensive AMI subjects had a better in-hospital outcome compared with normotensive AMI patients. These better outcomes is probably related to a better preserved LV function due to less infarct extension in hypertensive and more cases of NSTEMI among them. Early presentation of STEMI in the hypertensive group

with higher rates of thrombolytic administration, more use of cardioprotective antihypertensive drugs, presence of extended collaterals in the hypertensives and yet unidentified factors may have also contributed to the better in-hospital outcome.

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## Clinical profile of cardiac myxoma: 11 years' experience of 90 cases

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### Abstract:

**Introduction:** Cardiac myxoma is a benign neoplasm that represents the most common primary tumour of the heart. Because of nonspecific symptoms, early diagnosis may be a challenge. Although the left atrium is the most commonly involved site of origin in 75% of cases, it can arise from any of the cardiac chambers. Symptoms from a cardiac myxoma are more pronounced when the myxomas are left-sided, racemosus, and over 5 cm in diameter. Symptoms are produced by mechanical interference with cardiac function or embolization. Being intravascular and friable, myxomas account for most cases of tumor embolism. The site of embolism is dependent upon the location (left or right atrium) and the presence of an intracardiac shunt. Most atrial myxomas are benign and can be removed by surgical resection.

**Objectives:** To see clinical presentation and echocardiographic profile of cardiac myxomas.

**Methods:** 90 cardiac myxoma patients who admitted both in cardiology and cardiac surgery departments of National Institute of Cardiovascular Diseases (NICVD), Dhaka from August 2003 to July, 2014 were studied clinically and by echocardiogram. Clinical histories were reviewed, noting age, gender, and clinical presentation.

**Results:** There were 30 males and 60 females, ages ranged from 17 to 76 years. The commonest clinical feature was dyspnoea (94.44%), followed by palpitation (76.67%), chest discomfort (74.44%), constitutional symptoms (50%), pedal oedema (20%), syncope (14.44%), and embolization (7.7%). The mean duration of symptoms was 09.7 months.

**Conclusion:** The clinical presentation of cardiac myxoma is often nonspecific, so high index of clinical suspicion is important for its early and correct diagnosis. The size and appearance of the myxomas correlated with the presenting symptoms.

*Key Words:* Cardiac myxoma, Clinical presentation, Echocardiographic profile

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### Introduction:

Cardiac myxoma is a benign neoplasm that represents the most common primary tumour of the heart. Because of nonspecific symptoms, early diagnosis may be a

challenge. Although the left atrium is the most commonly involved site of origin in 75% of cases, it can arise from any of the cardiac chambers. Symptoms from a cardiac myxoma are more pronounced when the myxomas are left-sided, racemosus, and over 5 cm in diameter. Symptoms are produced by mechanical interference with cardiac function or embolization. Being intravascular and friable, myxomas account for most cases of tumor embolism. The site of embolism is dependent upon the location (left or right atrium) and the presence of an intracardiac shunt. Most atrial myxomas are benign and can be removed by surgical resection.<sup>1-5</sup>

Primary tumours of the heart are extremely rare, with an estimated incidence ranged from 0.0017% to 0.33% at autopsy<sup>1</sup>. Cardiac myxomas represent the most frequent

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benign cardiac tumors. In most surgical series, they account for almost 80% of cases.<sup>2</sup> The cells giving rise to the tumor are considered to be multipotential mesenchymal cells that persist as embryonal residues during septation of the heart.<sup>3,4</sup> They also are thought to arise from cardiomyocyte progenitor cells, subendothelial vasoformative reserve cells or primitive cells which reside in the fossa ovalis and surrounding endocardium or endocardial sensory nerve.<sup>5-8</sup> Occasionally, mucous glandular epithelium may present, which may represent rests of entrapped embryonic foregut.<sup>9,10</sup> Two types of macroscopic appearance are observed: polypoid type and papillary type.<sup>11,12</sup> The histopathological diagnosis of a cardiac myxoma depends on the identification of the myxoma cell, which has occasionally been called the lepidic cell.<sup>13</sup> The cells are arranged singly or in small clusters, or formed capillary like channels.<sup>2</sup> Some morphological and immunohistochemical features may be related to the clinical presentations. Burke<sup>4</sup> found that embolic myxomas were less often fibrotic than nonembolic myxomas and were more likely thrombosed and extensively myxoid with an irregular frond-like surface. Fibrotic and non-thrombosed tumors had a longer mean duration of clinical symptoms and were found in older persons. Recurrent, multiple, and familial myxomas were more often found in younger women and, more likely irregular surfaced and histologically myxoid. Endo's group reported that tumors associated with constitutional signs were significantly more likely to be large, multiple, or recurrent than those unassociated with constitutional signs.<sup>14</sup> Papillary surface myxomas are thought to be related to embolism, and large left atrial tumors are related to atrial fibrillation. Myxoma cells usually express IL-6, and some tumors have abnormal cellular DNA content.<sup>15</sup> A C769T PRKAR1a mutation has been observed in "familial myxomas".<sup>16</sup>

**Methods:**

90 cardiac myxoma patients who were admitted both in cardiology and cardiac surgery department in the National Institute of Cardiovascular Diseases (NICVD), Dhaka from August 2003 to July, 2014 were studied clinically and by echocardiogram. Clinical histories were reviewed, noting age, gender, and clinical presentation.

**Results:**

There were 30 males and 60 females, ages ranged from 17 to 76 years. The commonest clinical feature was dyspnoea (94.44%), followed by palpitation (76.67%), chest discomfort (74.44%), constitutional symptoms (50%), pedal oedema (20%), syncope (14.44%), and

embolization (7.7%). The mean duration of symptoms was 09.7 months.

**Table-I**  
*Presenting features of the patients. (n=90)*

Clinical features	Percentage
Dyspnoea	85 (94.44%)
Constitutional symptoms	45(50%)
embolization	07(7.77%)
palpitation	69(76.67%)
syncope	13(14.44%)
Pedal edema	18(20%)
Chest discomfort	67(74.44%)

**Table-II**  
*Sites of myxoma (n=90)*

Site	No (%)
Left atrium	80 (88.89%)
Right atrium	07(07.78%)
Biatrial	02 (02.22%)
Right ventricle	01(01.11%)

Only in 7.9% cases was the diagnosis of myxomas made clinically. 73.7% cases were initially diagnosed as having mitral valve disease, tricuspid valve disease (09%), ischemic heart disease (3.4%), cardiomyopathy (1.7%), and the remaining 4.3% were detected during family screening and follow-up. The sites of myxomas were left atrium 80 (88.89%); right atrium 07(07.78%) and biatrial 02 (02.22%). All myxomas except 5 were attached to the interatrial septum. The site, size, shape, attachment, mobility, prolapse into ventricle, and surface characteristic of myxomas were accurately assessed by 2D-echocardiography. When the morphological characteristic of myxomas were studied and correlated with clinical features, large left atrial myxomas presented more commonly with constitutional symptoms, congestive heart failure, syncope, and auscultatory findings suggestive of mitral valve disease, whereas myxomas smaller in size and with irregular surface were associated with embolization.

**Discussion:**

Myxoma is the most common primary cardiac neoplasm and accounts for approximately one-half of all primary cardiac tumors.<sup>1</sup> Because of the nonspecific clinical presentation of patients with cardiac myxomas, premortem diagnosis was not accomplished until 1952, when Goldberg et al.<sup>13</sup> demonstrated a left atrial myxoma

at cardiac angiography. The first successful surgical resection of a cardiac myxoma was performed with a cardiopulmonary bypass in 1954.<sup>14,15</sup>

In concurrence with our series, patients with cardiac myxomas are reported to have a wide age range (17–76 years), with a mean age of approximately 45 years. There appears to be a slight female predominance, with a female-to-male ratio of 5:4.<sup>1, 5, 15–17</sup> 95 % of our patients presented with at least one manifestation of a classically described clinical triad, which includes cardiac obstructive symptoms, embolic phenomena, and constitutional symptoms.<sup>5</sup>

Presenting complaints relate to tumor location, which predicts obstructive sequelae and influences embolic sites (pulmonary or systemic). Left atrial myxomas commonly cause mitral valve obstruction, and symptoms mimic those of rheumatic heart disease. Affected patients present with dyspnea and orthopnea from pulmonary venous hypertension.<sup>1, 5, 15</sup> The pedunculated and prolapsing nature of these tumors allows for positional and intermittent mitral valve obstruction. Left atrial myxomas may present with symptoms related to mitral valve obstruction. Right atrial myxomas may obstruct the tricuspid valve and cause symptoms of right-sided heart failure, peripheral edema, passive hepatic congestion, and syncope. In our series, only 10% of patients with right atrial myxoma presented with signs and symptoms of tricuspid valve obstruction. Ventricular myxoma may also cause right-sided heart obstruction.<sup>5,14</sup>

Constitutional symptoms of fever, malaise, weight loss, anemia, and elevated erythrocyte sedimentation rate (ESR) have been reported and may be related to an autoimmune reaction initiated by the tumor.<sup>1,5,8,18</sup> These symptoms were present in 50% of our patients. Cardiac arrhythmias, atrial fibrillation, and atrial flutter reportedly occur in approximately 20% of patients with cardiac myxoma.<sup>16,19</sup> In our series, 69% of patients presented with arrhythmia or palpitation. Embolic phenomena are reported in approximately 35% of left-sided and 10% of right-sided myxomas.<sup>1,15,19,20</sup> The propensity of cardiac myxomas to embolize is thought to be related to tumor morphology, with friable and gelatinous myxomas being more likely to embolize than firm and fibrous lesions.<sup>1,20,21</sup> Left-sided myxomas embolize systemically, usually to the brain, kidneys, and lower extremities, and right-sided myxomas embolize to the pulmonary circulation.<sup>1,15,19</sup> Approximately 20% of all patients with myxomas irrespective of intracardiac location are reportedly asymptomatic, and the tumors are discovered incidentally or at autopsy.<sup>22</sup>

The majority of cardiac myxomas occur sporadically. However, in approximately 7% of patients, cardiac myxomas exhibit atypical behavior, demonstrating a familial predisposition or an association with a clinical complex (Carney complex). These patients tend to be younger (mean age, 24 years) and male, and the myxomas are more likely to be multicentric (45%) or recurrent (12%–22%), whereas only 1%–3% of sporadic myxomas reportedly recur.<sup>3,4,23,24</sup> Carney complex is an autosomal dominant condition with a complex of lesions that include mammary myxoid fibroadenoma, spotty pigmented lesions of the skin, endocrine disorders, testicular tumors, and psammomatous melanotic schwannoma. Two-thirds of these patients have cardiac myxomas.<sup>23,24</sup>

Cardiac myxoma is an intracavitary, round or ovoid neoplasm that is attached to the endocardium. Although the vast majority attach at the fossa ovalis of the interatrial septum, they can arise from any endocardial surface.<sup>1</sup> The majority (75%) occur in the left atrium, with a smaller proportion in the right atrium (20%) and rare cases in right ventricular, biatrial, left ventricular, or multiple locations.<sup>1,15,25</sup> In our series, approximately two-thirds of the atrial myxomas were attached to the interatrial septum, and the tumor location differed slightly from most reports, with 88% arising from the left atrium and 7% from the right atrium.

The clinical presentation of myxomas is diverse and dependent upon tumor location, size and mobility<sup>24-27</sup>. According to a previous study, the most common symptom is dyspnea (54%), and then followed by palpitation (35%).<sup>15</sup> Dyspnea and edema of lower limbs are thought to a consequence of atrioventricular valve obstruction. Nevertheless, the intracardiac obstruction may also lead to narrowing outflow tract and atrial fibrillation, which could contribute to dyspnea and palpitation. Cough is thought to result in pulmonary venous hypertension and frank pulmonary edema. Angina may be caused by insufficient blood supply. Embolism is also a classic symptom of myxomas, which have been reported to associate with the papillary surface<sup>15</sup>. The cause of some constitutional disturbances is still unclear. Some findings suggest the cytokine interleukin-6 (IL-6) may be responsible for that. The relationship between IL-6 and constitutional syndromes is still controversial. In some cases, the right atrial myxomas may induce pulmonary hypertension because of embolism of tumor fragments. Right ventricular myxomas may mimic stenosis of pulmonary valve and cause syncope. In our series, both left and right atrial myxomas

were observed with symptoms of dyspnea, palpitation, cough, and angina. Besides, two cases presented with cerebral infarction symptoms and one case with angina and vertigo showed papillary surface, which was consistent with previous studies.<sup>4,15</sup> Other cerebral symptoms in cases of solid surface may only caused by cardiac obstruction and cerebral ischemia. About 20% of cardiac myxomas are asymptomatic; they are usually smaller than 4 cm.<sup>28, 29</sup> The maximal diameter of four asymptomatic cases of the present report was 6, 4, 3, and 3 cm, respectively. We consider it is due to the small tumor size or long growth course, which may result in adaption to the tumor.

Echocardiography is widely recognized as a sensitive preoperative diagnostic method, although it appears nonspecific to some occupying lesions. Moreover, thrombus may be misdiagnosed as myxoma in some cases. Nevertheless, echocardiography is also demonstrated as the most accurate and reliable preoperative method with which to predict the diameter, location, attachment, mobility, and morphology of cardiac myxomas.

Bone and brain metastases from glandular cardiac myxomas have been reported in the recent literature.<sup>31-38</sup> The most frequent metastatic site for cardiac myxomas is cerebrum.<sup>33</sup> Several reports have reviewed cerebral metastasis cases.<sup>34,35</sup> Since most myxomas are located in the left atrium, systemic embolism is particularly frequent. The tumor fragments metastasized to cerebral vessel walls may penetrate through the vessel wall, forming intra-arterial metastases. Some cytokines, such as CXC chemokines, may explain the metastasis potential of morphologically benign myxomas. In our series, no metastatic case was observed.

#### Conclusion:

The clinical presentation of cardiac myxoma is often nonspecific, and may be detected in asymptomatic patients, so a high index of clinical suspicion is important for its early and correct diagnosis. The size and appearance of the myxomas correlate with the presenting symptoms.

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## Outcome of off-pump coronary artery bypass graft (OPCAB) surgery: Analysis of 129 cases

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### Abstract:

Coronary artery disease (CAD) is a leading cause of morbidity and mortality worldwide, including Bangladesh. Besides medical and interventional treatment, coronary artery bypass graft (CABG) surgery in an effective modality for the management of a subset of CAD patients. Off-pump coronary artery bypass graft (OPCAB) surgery is a recent modification of conventional CABG surgery, which, like other parts of the world, is being increasingly practiced in Bangladesh. But the outcome of this relatively recent surgical approach in our setting is largely unknown. In this study, the outcomes of 129 cases off-pump CABG surgery done in

a tertiary cardiovascular centre and a private institute in Dhaka were analyzed. Majority (67, 54.2%) had triple vessel disease (TVD), while 4 (3.2%) patients had left main disease. One, two and three grafts were used in 17 (13.2%), 74 (57.4%) and 38 (29.4%) cases respectively. There was no mortality. Post-operative complications occurred in 17 (13.18%) patients; secondary wound infection in 10 (7.75%) and immediate respiratory distress in 7 (5.43%) cases. Ten (7.75%) patients needed secondary stitches.

*Key words:* Coronary artery disease, Cardiac Surgery, Coronary artery bypass graft, Off-pump.

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### Introduction:

Coronary artery bypass graft (CABG) operation has become the most completely studied surgical operation in the history of surgery and has been shown to be highly effective for the relief of severe angina.<sup>1</sup> In 1962 cardiac surgeon Dr. Sabiston conducted the first unsuccessful saphenous vein graft from the ascending aorta to the distal right coronary artery and the patient died 3 day later. The technique was then pioneered in Cleveland Clinic in USA in late 1960s. First successful CABG was done by Dr. Robest. H. Goetz and his team.<sup>2</sup> The major development was in 1970 when internal mammary artery was used as bypass conduit to the coronary artery. By mid 1970s, many centers in USA, Australia and Europe were performing CABG with low perioperative mortality and high rate of pain relief.<sup>3</sup>

Off pump coronary artery bypass graft or beating heart surgery was primarily developed in early 1990s by Dr.

Amno Atsushi and it reduces the number of complications related to cardiopulmonary bypass.<sup>4</sup>

In Bangladesh CABG and off-pump CABG started towards the end of last century. In this study short-term outcome of 129 cases of off-pump CABG was observed.

### Method and Material:

129 patients with coronary artery disease underwent off-pump CABG during July 2013 to December 2015 in the National Institute of Cardiovascular Diseases (NICVD) and a private institute in Dhaka. After proper pre anesthetic evaluation, Off-pump CABG was done in all patients maintaining standard procedures. Octopus or fixation was used in every cases to fix up the area of operation.

### Result:

Off-pump CABG was done in 129 patients with coronary artery diseases from July 2013 to December 2015. Clinical profile of the patients is shown in Table I.

Out of 129 cases undergoing OPCAB surgery, 121 were male and only 8 were female. Age ranged from 25 to 70 years and most of the patients were between the age of 40 and 70 years. (Table 2)

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**Table-I**  
*Clinical Profile of the patients undergoing off-pump CABG. (N=129)*

Total Cases	: 129
Male	: 121 (93%)
Female	: 08(07%)
Mean Age	
Male	: 51.6±8.32
Female	: 53.6±6.7
Clinical History	
Prior MI	: 58%(75)
Hypertension	: 35%(45)
Heart Failure	: 9.3%(12)
Diabetes Mellitus	: 12.5% (16)
Cigarette Smoking	
Present smoker	: 29%
Ex. smoker	: 46%
Non smoker	: 25%
Medication	
Long acting GTN	: 80(62%)
Beta Blockers	: 120(93%)
Anti-platelets	: 129(100%)
Lipid lowering drugs	: 105(81%)

**Table-II**  
*Distribution of patients by age and sex. (N= 129)*

Age range	Male	Female	Total	Percentage
21-30 years	01	00	01	0.8
31-40 years	08	01	09	6.9
41-50 years	44	02	46	35.7
51-60 years	47	02	49	37.9
61-70 years	21	03	24	18.7
Total	121	08	129	

Left main disease was present in 4 patients and 67 had triple-vessel disease. The pattern of coronary artery disease is show in Table III.

**Table-III**  
*Pattern of coronary diseases (N=129)*

Defects	Male	Female	Total	Percentage
SVD	09	03	12	9.3
DVD	41	02	43	33.3
TVD	67	03	70	54.2
Left main disease	04	0	04	3.2
Total	121	08	129	

Off pump CABG were done in all cases in standard procedures. Number of graft used are shown in Table 4.

**Table-IV**  
*Number of grafts used (N=129)*

Single graft	17	13.2%
Two grafts	74	57.4%
Three grafts	38	29.4%

There was no mortality. Post-operative complications were seen in 17 (13.18%) patients. Secondary wound infection were found in 10 (7.75%) cases and immediate respiratory distress were seen in 7 (5.43%) cases . All the patients were managed accordingly and in 10 patients secondary stitches had to be given for proper healing of the wound. Patients were discharged with advice of regular use of anti-platelets, beta blockers and statin among others.

**Discussion:**

Medical and surgical therapies for patients with coronary artery diseases have changed over last 20 years. Coronary artery bypass surgery is the treatment of choice for patients who have poorly controlled angina pain after adequate medical management. Majority of the coronary surgical procedures are performed for multiple vessels diseases and overall mortality rate is also very low i.e., around 2-3%.

In this series of 129 cases there was no mortality though majority i.e. 52.4% had triple-vessel disease and only 9.3% patients had single vessel diseases.

Now-a-days more than 25% of CABG operations are being done as off-pump CABG. It is safe as on-pump surgery and in experienced hands offers less early complications, particularly in those patients with significant comorbidity.<sup>7</sup>

This study demonstrates the feasibility and safety of OPCAB heart surgery in Bangladesh.

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# Safe Technique of Removal of Left Atrial Thrombus during Mitral Valve Replacement Surgery

Rampada Sarker<sup>1</sup>, Manoz Kumar Sarker<sup>1</sup>, AM Asif Rahim<sup>1</sup>, Abdul Khaleque Beg<sup>2</sup>

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## Abstract:

**Background:** Open mitral operation in patients with massive left atrial thrombus still with high mortality due to intra-operative embolism. To prevent this mortality due to intra-operative embolism and to prevent this danger we practiced a surgical technique which includes careful handling of heart and obliteration of left ventricular cavity by bilateral compression.

**Method:** We used this technique in patients of severe mitral stenosis with atrial thrombus during mitral valve replacement. Our technique was to obliterate the left ventricular cavity and thus keep the mitral cusps in a coapted position by placing gauge posterior to left ventricle and a compression over right ventricle by hand

of an assistant with a piece of gauze. This obliteration prevented passage of fragments of left atrial thrombus towards collapsed left ventricle.

**Result:** Before practicing this technique, 4 out of 9 patients expired due to cerebral embolism. But after implementation of this technique in 17 patients no mortality or morbidity occurred.

**Conclusion:** This technique of removal of left atrial thrombus during mitral valve replacement may be a safe procedure for preventing perioperative embolism.

*Key words:* Safe technique, Removal of LA thrombus, Mitral valve replacement.

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## Introduction:

Left atrial thrombi are common cause of embolisation<sup>1</sup>; especially in patients with mitral valve abnormalities<sup>1,2</sup> and atrial fibrillation<sup>3,4</sup>. Left atrial enlargement is common in severe rheumatic mitral stenosis<sup>1-5</sup>. Exact etiology is not known but increased left atrial pressure and weakening of left atrial wall by rheumatic pancarditis are implicated in its development<sup>1,2</sup>. The enlarged left atrium is associated with blood stasis and thrombus formation among patients with mitral stenosis with atrial fibrillation<sup>6,7</sup>. The presence of massive left atrial thrombus still clouds with uncertainty the otherwise standardised mitral operation<sup>8</sup>. Although open techniques have made the removal of the thrombus possible, the occasional massive and usually friable thrombotic mass constitutes and usually friable thrombotic mass constitutes a very real hazard of intra operative embolism<sup>8</sup>. To prevent or minimize this danger, a number of surgical maneuvers have been proposed<sup>9,10,11,12,13</sup>. Our experience in facing this problem prompted us to reconsider our technique and

introduce some modifications which may eliminate the danger of intra-operative fragment embolisation.

## Methods:

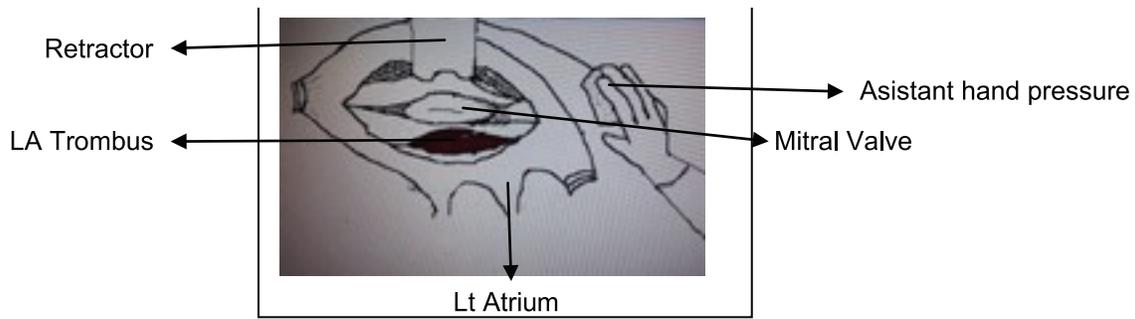
This retrospective study was carried out during the period of January 2014 to March 2016 in the Department of Cardiac Surgery in National Institute of Cardiovascular Diseases (NICVD) and Hospital, Dhaka, with permission of academic council of the institute. There were total 26 patients of severe mitral stenosis with left atrial thrombus undergoing mitral valve replacement. In 9 out of 26 patients we practiced the previous conventional technique and in 17 patients we used the new technique of thrombus removal. Data were collected retrospectively from records of operation theatre at Cardiac Surgery Department and our Cardiac Surgery Units follow-up sheet.

## Surgical technique :

Cardiopulmonary bypass was established with standard procedure of aortic and bicaval cannulation. The heart was arrested by moderate antegrade hypothermic blood-cardioplegic arrest. Two pieces of gauze were placed under the left ventricle. Another piece of gauze was placed over the right ventricle (Figure-1)

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**Fig.-1:** Safe technique of thrombus removal from left atrium by obliteration of left ventricular cavity by pressure.

A gentle pressure was exerted by an assistant on the gauge over the right ventricle with his right palm of the hand sufficient enough to collapse left ventricle. Left atriotomy was done, retractor to left atriotomy was introduced. Careful handling was ensured to avoid disrupting the thrombotic mass. A plane of cleavage was created by holding the anterior lip of the atriotomy and making a blunt dissection thereby separating the thrombus from the wall. The intracardiac sucker was placed within the left atrial cavity was washed out thrombus and valsalva maneuver were performed, there by aspirating pulmonary veins. gauges were removed after releasing compression on right ventricle. Mitral valve replacement was completed, left atrium was closed. cross-clamp was released after proper and adequate de-airation. The patients were weaned from cardiopulmonary bypass and chest was closed. Cardipulmonary by pass time aortic cross-clamp time were collected from operation theatre record book. Post-operatively all patients data were collected from operation

theatre recordbook. Post-operatively all patients were admitted into intensive care unit where record of any systemic or cerebral embolism or, any ventilation problem or mortality were searched from registered data. Oral anti coagulant warfarin was started from first post-operative day.

**Result:**

Mitral Valve Replacement were performed in both groups of patients. Mean age of the patients (table-I) were 41.35 years in our new technique group (Group-II). Seven were male and 10 were female. Mean weight was 51.59kg. All of 17 patients with new technique (group-II) were free from cerebral embolism (table-II). Mean age of patients of conventional technique group (Group-I) was 40.22 years. Six were male and three were female, mean body weight was 49.78 kg. Four patients (44.44%) out of 9 patients of old technique group(group-I) expired due to cerebral embolization in old technique group, among them 2 were male and 2 were female (table-II).

**Table-I**  
*Demographic data*

	Age(mean)years	P value	Sex		P value	Body Wt.	P value
Group-I(Old technique)	40.22	0.125	F 3(33.3%)	M 6(66.7%)	0.430	49.78	0.212
Group-II (New technique)	41.35		F 10(58.8%)	M 7(41.2%)		51.59	

**Table-II**  
*Outcome of two groups*

	Total pt.	THROMBO EMBOLISM	No THROMBO EMBOLISM	P value
Group-I (Conventional) n=9	9	4	5	0.001
Group-II (New Technique) n=17	17	0	17	

### Discussion:

Removal of an organized thrombus from left atrium can be challenging especially when it is huge in size and the presence of dense adhesion and absence of cleavage plane makes its removal difficult<sup>14</sup>. and under such circumstances residual organized material can be left<sup>14</sup>. Gallo and colleagues<sup>8</sup> practiced a technique that included occlusion of mitral valve orifice by a 24F Foley catheter to prevent entry of thrombus fragments into left ventricle. They practiced in 22 patients without any mortality or morbidity where as 7 out of 8 patients expired in their previous technique of left atrial thrombus, like their experience we lost 4 out of 9 patients with previous conventional technique and all of 17 patients with new technique of thrombus removal from left atrium were free from cerebral embolism. Our Technique is similar to them. Because the occluded mitral valve orifice and we occluded mitral valve cavity so that thrombus cannot get entry through mitral valve orifice. Excision of the small atrial thrombus does not present any surgical difficulty but a large intra-cavitary thrombotic mass creates a serious chance of intra-operative embolization<sup>8</sup>. The goal of this new technique was to avoid inadvertent handling, carefully performing atriotomy and introducing the retractors. Obliteration of left ventricular cavity avoided entry of fragments of thrombus into the left ventricle. Finally wall suction, vulsalva maneuver and thorough left atrial washing removed virtually all residual fragments. Our study was retrospective with only 17 patients. A large scale prospective study in multiple centers can judge safety of our technique.

### Conclusion:

Our technique of removal of left atrial thrombus during mitral valve replacement by occlusion of left ventricular cavity by gentle compression is safe and prevents cerebral embolization.

### Acknowledgement:

I would like to express my thankful acknowledgement to Kabita Rani Podder, ICU in-charge, and Farida Yasmin, OT in-charge, of NICVD for their cooperation during collection of data for this study.

### Conflict of interest- None.

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# Apical hypertrophic cardiomyopathy, a review of presentation, pathophysiology, diagnosis and natural course of the disease

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## Abstract

**Apical Hypertrophic Cardiomyopathy is an uncommon phenotype of hypertrophic cardiomyopathy. We present a review of best evidence on presentation, pathophysiology, diagnosis, outcomes and**

**management for patients with apical hypertrophic cardiomyopathy.**

*Key Words: Apical Hypertrophic Cardiomyopathy, Mimics, Incidence, Natural Course, Diagnosis*

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## Introduction:

Apical Hypertrophic Cardiomyopathy (AHCM) is characterized by hypertrophy of the myocardium predominantly in the left ventricular (LV) apex and has now been recognized as an uncommon phenotype of hypertrophic cardiomyopathy (HCM).<sup>1</sup> The incidence of AHCM ranges from 3% of all HCM patients in North America to up to 15% and 16% in reports from Japan and China respectively.<sup>2,3</sup> We review the latest evidence on epidemiology, pathophysiology, diagnostic modalities, management and natural course of AHCM.

## Typical Presentation

In a cohort of 208 patients from China, the most common symptom was chest discomfort that was characterized as chest pain, chest tightness and palpitations.<sup>3</sup> In another study 46% of the patients were asymptomatic.<sup>4</sup> In another cohort of 105 patients, 16% complained of angina, 14% had atypical chest pain, 10% had palpitations, 6% complained of dyspnea, and 6% had pre-syncope.<sup>5</sup>

## Diagnostic considerations and types of AHCM

The diagnosis is made on imaging studies.<sup>6</sup> On transthoracic echocardiogram (TTE) AHCM is defined as LV wall thickening confined to the most distal region of the apex, below the papillary muscle level with the ratio of

apical to basal LV thickness more than 1.3.<sup>6,7</sup> Hypertrophy at this region is best visualized in apical views.<sup>7</sup> In a cohort of 182 patients with AHCM 3 subtypes were identified based on the patterns of hypertrophy. These subtypes are pure, pure diffuse and mixed.<sup>8</sup>

AHCM is more commonly classified as “pure” when no concomitant septal hypertrophy is present, and “mixed” when evidence of septal hypertrophy is seen.<sup>3,9</sup> In two studies no significant difference was found between cardiovascular mortality and morbidity between these variants of AHCM.<sup>3,5</sup> Figure 1A and 1B show an illustration of the location of hypertrophy and differences between the two variants.

## Echocardiography-strengths and limitations

TTE is usually the first line imaging modality in patients suspected to have AHCM, because of its widespread availability and relatively low cost. Figure 2 shows typical finding of pure variant of AHCM on TTE.

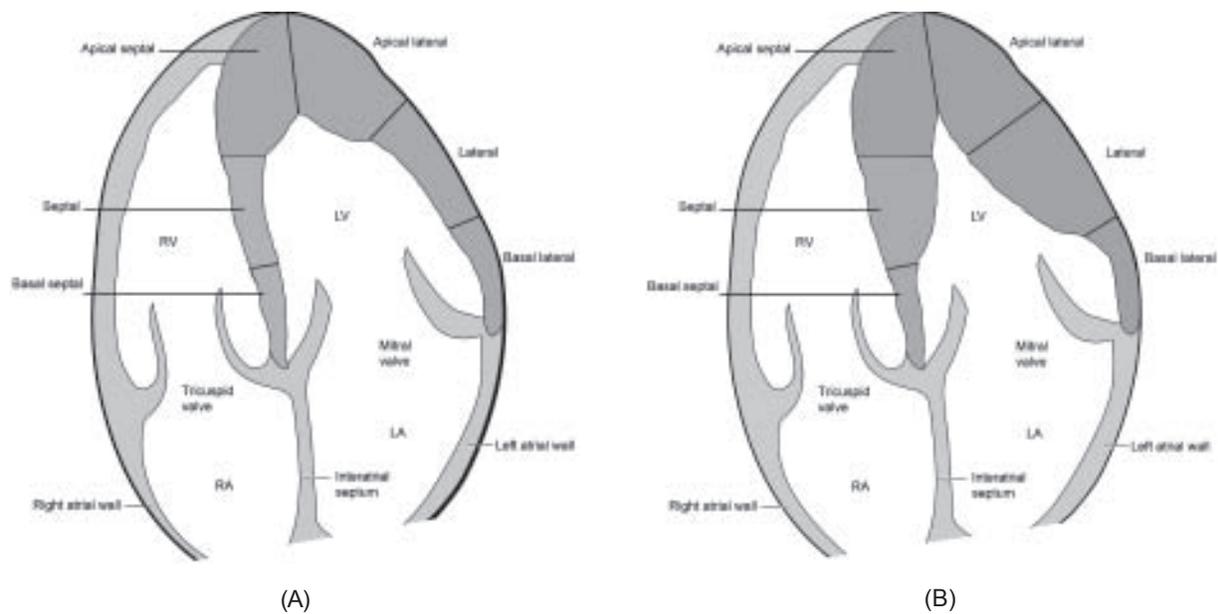
Frequent inability to visualize the apical endocardium can limit diagnosis of AHCM by TTE.<sup>10,11</sup> Furthermore the distribution of hypertrophy may be inappropriately measured on TTE and severity of wall thickening may be underestimated.<sup>12</sup> In a study TTE only detected one in four cases of AHCM related LV aneurysms.<sup>13</sup> Even in classic HCM patients TTE was only able to identify LV aneurysm in 57% of the patients.<sup>14</sup> Hence additional imaging modalities are recommended in patients with suspicion of AHCM and initial TTE is non-diagnostic or with sub optimal visualization of the apex.

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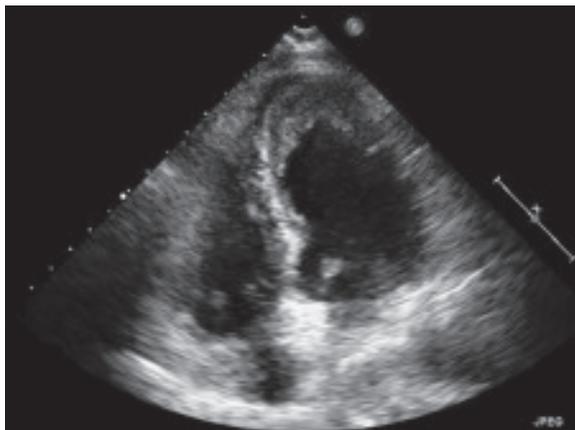
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**Fig.-1:** Pattern of hypertrophy A) pure variant of Apical Hypertrophic Cardiomyopathy (AHCM), B) mixed variant of AHCM



**Fig.-2:** Representative trans-thoracic echocardiogram image of pure variant of apical hypertrophic cardiomyopathy

#### Apical hypertrophic cardiomyopathy mimics and the role of other imaging modalities

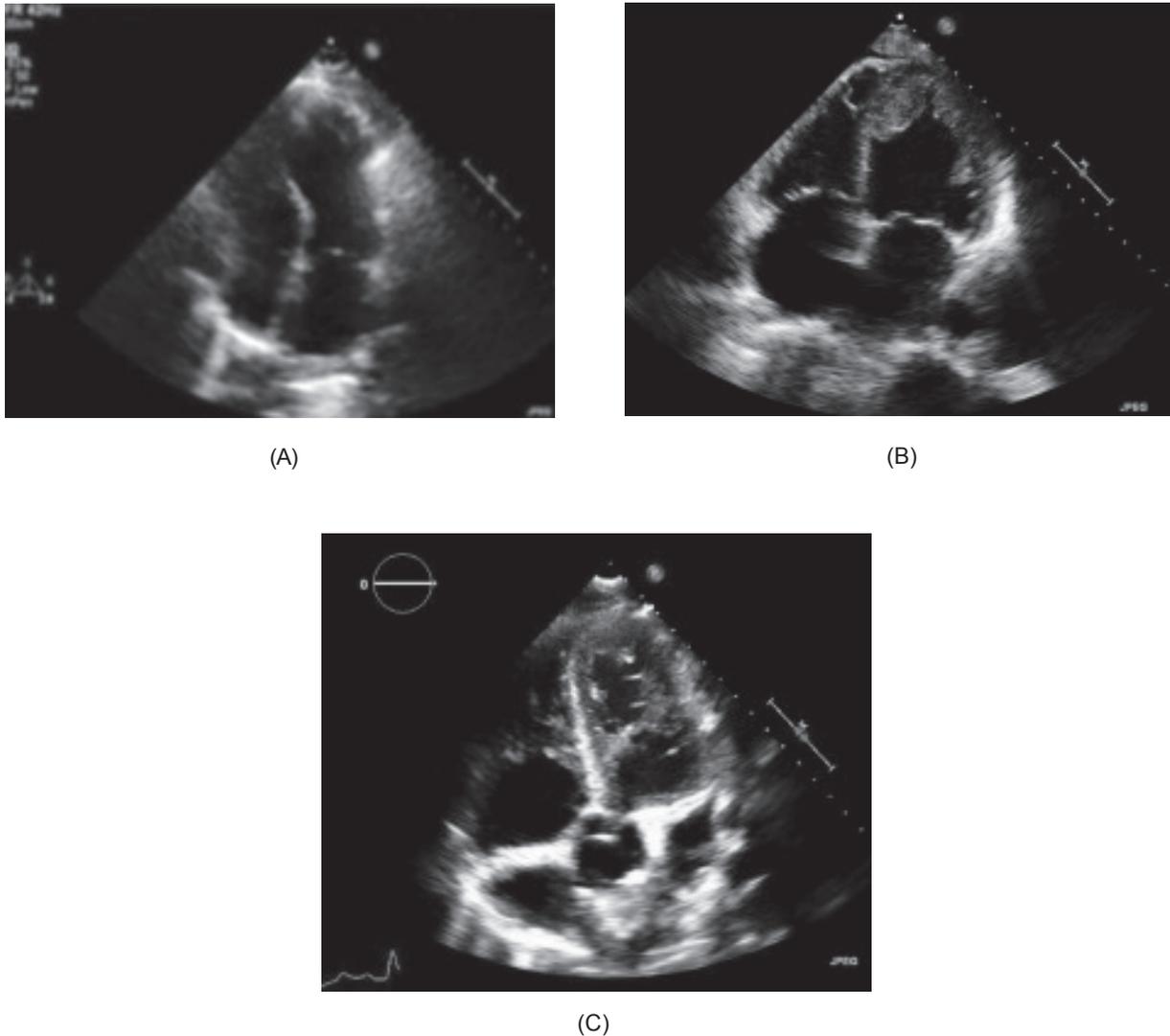
As mentioned a TTE although often the first line imaging modality can be non-diagnostic for AHCM especially if the apex is not fully visualized. Furthermore apical thrombus, Loefflers endocarditis, LV aneurysm, LV non compaction, and endomyocardial fibrosis may give a similar echocardiographic appearance on TTE to AHCM.<sup>15-17</sup> Other common imaging modality options include TTE with contrast, trans-esophageal

echocardiogram (TEE), cardiac multidetector computer tomography (MDCT) and cardiac magnetic resonance imaging (CMR).

Apical ballooning syndrome or Takotsubo cardiomyopathy can also mask underlying AHCM.<sup>18</sup> In such cases TTE with contrast can be helpful.<sup>18</sup> Figure 3A shows typical findings on TTE in Takotsubo cardiomyopathy. TTE with contrast can also help in differentiating LV thrombus and LV non-compaction from AHCM.<sup>18</sup> Figure 3B and 3C show representative images of typical findings on TTE in LV thrombus and LV non compaction respectively.

TTE with contrast can help in diagnosing AHCM in patients with high suspicion and a non-diagnostic TTE.<sup>10</sup> TEE can also accurately measure wall thickness at the apex if TTE imaging is not adequate.<sup>12</sup>

In recent years there has been a great increase in the use of CMR in the diagnosis of HCM including AHCM, because of its precise determination of myocardial anatomy and the depiction of myocardial fibrosis.<sup>15</sup> CMR has also been shown to distinguish pure from mixed variant of AHCM.<sup>19</sup> In a small cohort of ten patients with non-diagnostic TTE, AHCM was seen on CMR.<sup>20</sup> CMR has shown better performance in detection AHCM compared to TTE, and in also detecting LV aneurysms related to AHCM.<sup>13</sup>



**Fig.-3:** A) Representative 2D transthoracic echocardiogram images of Takotsubo Cardiomyopathy B) Patient with left ventricle(LV) thrombus, C) LV non compaction

Characteristic findings on single photon emission computed topography (SPECT) imaging can also aid in diagnosis. In a cohort of 20 patients with AHCM, 75% demonstrated an increased tracer uptake and characteristic spade-like deformity of the LV chamber.<sup>21</sup> Evaluation of the LV cavity on ventriculogram can also show the spade-like configuration of LV in end diastole.<sup>21</sup>

MDCT has been used with success to detect significant coronary artery stenosis and has also emerged as a novel technique to evaluate cardiac morphology. Due to its high spatial resolution MDCT enables accurate

delineation of apical endocardial border and dynamic evaluations of myocardial thickness and global and regional LV function.<sup>22</sup>

Table 1 shows the sensitivity of some of the imaging techniques from different studies.<sup>10, 12, 13, 20, 23.</sup> As only positive results from the modality being tested resulted in a confirmatory gold standard test being performed, specificities of imaging modalities could not be calculated. Although TTE has low sensitivity it has the advantage of having a low cost and widespread availability. A reasonable alternative could be TTE with contrast.

**Table-I**  
*Sensitivity of different imaging modalities in diagnosing AHCM*

Author	Imaging Modality Studied	Sample Size	Gold Standard	Sensitivity
Crowley JJ <sup>12</sup>	TEE	6	Previous Diagnosis	100%
Crowley JJ <sup>12</sup>	TTE	6	Previous Diagnosis	83%
Fatori R <sup>13</sup>	TTE	13	CMR	69%
Moon JC <sup>20</sup>	TTE	10	CMR	0
Ward RP <sup>10</sup>	TTE	26	TTE with contrast	0
Chen CC <sup>23</sup>	MDCT	14	Previous diagnosis	100%

**Electrocardiography**

The electrocardiogram (EKG) findings that have been associated with AHCM include deep inverted T waves in precordial leads, and presence of notched QRS in more than one lead.<sup>23-25</sup> T wave inversions can disappear during long term follow-up.<sup>26</sup> Figure 4A shows an EKG with characteristic T wave inversions in precordial leads associated with AHCM.

Presence of notched QRS without bundle branch block (BBB) is associated with impaired apical contraction and late gadolinium enhancement (LGE) on CMR, and is associated with higher prevalence of ventricular tachycardia.<sup>25</sup> Reports have shown the association of a notched QRS in inferior leads with an apical aneurysm.<sup>27,28</sup> Hence notched QRS without BBB may be a useful indicator of morbidity and risk of aneurysm formation in patients with AHCM. Figure 4B shows an EKG with notched QRS in inferior leads.

**Pathophysiology**

**Cellular mechanism and role of gene mutations**

Decades of intense investigation has identified more than 11 genes and 1400 mutations implicated in HCM.<sup>29</sup>

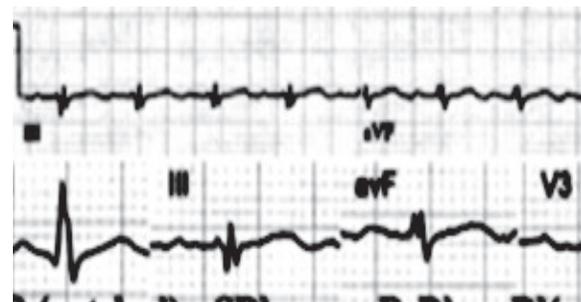
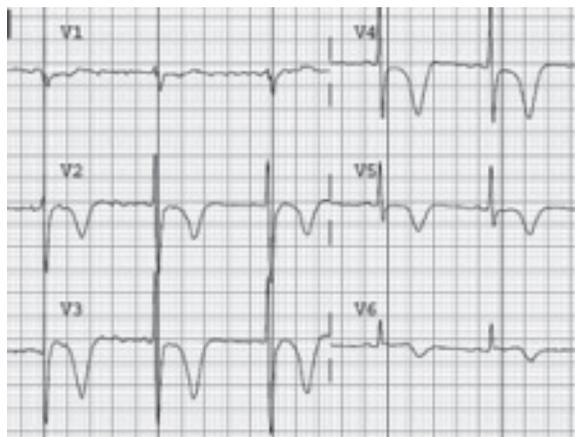
In AHCM the most common gene mutations are MYH7, MYBPC3, ACTC1 and TPM1.<sup>1,30,31</sup> The protein coded by these genes plays an important role in the contraction of the sarcomere.<sup>32</sup>

The key histological feature of HCM is myocyte and myofibrillar disarray.<sup>33</sup> Myocyte hypertrophy with nuclear enlargement and hyperchromasia is also seen.<sup>34</sup> Dysplasia of small arteries seen as medial and intimal smooth muscle proliferation with luminal narrowing can also develop.<sup>35</sup> The mechanism by which genetic mutations cause these changes is poorly understood.<sup>32</sup>

Prevalence of mutations does not correlate with phenotypic features. In the largest studied cohort of patients with AHCM only 18/71 (25%) of the patients were genotype positive with majority of mutations found in MYH7 and MYBPC3.<sup>1</sup> In another cohort of 61 patients gene mutations were found only in 8/61 (13%) of the patients.<sup>31</sup> Furthermore genotype positive patients did not have any significant differences in outcomes.

**Natural course, morbidity and mortality**

Relative to HCM, patients with AHCM have more benign outcomes. Incidence of sudden cardiac death (SCD),



**Fig.-4:** A) Electrocardiogram showing characteristic Giant T wave inversions B) notched QRS associated with Apical Hypertrophic Cardiomyopathy

cardiac dysrhythmias and heart failure is less frequent than HCM.<sup>37</sup> However, common causes of cardiovascular mortality still include myocardial infarction, congestive heart failure, dysrhythmias and left ventricular apical aneurysms.<sup>3,38,39</sup> To our knowledge only 10 studies, looking at long term outcomes in AHCM, with a sample size greater than 30, have been published. Symptoms at presentation were not specified in majority of the studies. Mortality and morbidity outcomes from these studies are summarized in Table 2 and Table 3.<sup>3, 4, 5, 16, 36, 37, 40-43</sup> Most studies did not report MI unrelated to pre-existing coronary artery disease.

AHCM patients have a relatively small burden of myocardial fibrosis and less severe diastolic dysfunction, hence have a better prognosis than HCM patients.<sup>43</sup> Some of the common causes of long term cardiovascular morbidity in patients with AHCM include atrial fibrillation, stroke, transient ischemic attack, syncope, pre-syncope and myocardial infarction. In several of the major outcome studies, atrial fibrillation was the most common, followed by stroke.<sup>4,5</sup> The incidence of atrial fibrillation is higher in mixed type.<sup>8</sup> However another report showed a higher incidence of atrial fibrillation in patients with pure variant.<sup>9</sup> The prognosis is worse in women with a higher

**Table-II**  
*Mortality rates in major studies for AHCM. \* median follow-up, \*\*not specified*

Author	Region	N	Mean age at presentation (years)	Mean Follow-up (years)	All-Cause mortality	Cardiovascular mortality
Sukamoto T et al <sup>40</sup> (1986)	China	31	47	2-13	0	0
Erickson MJ et al <sup>5</sup> (2002)	Canada	105	41.4	13.6 ± 8.3	11/105	2/105 (1.9%)
Lee CH et al <sup>37</sup> (2006)	Taiwan	40	60	6 ± 5	0	0
Chung T et al <sup>41</sup> (2010)	Australia	32	71	4 ± 3	6/32	4/32 (13%)
Chen CC et al <sup>42</sup> (2011)	Taiwan	47	60	2.95 ± 1.97	4/47	3/47 (6.4%)
Moon J et al <sup>16</sup> (2011)	Korea	453	61	3.58 ± 1.66	39/453	22/453 (4.8)
Yan L et al <sup>3</sup> (2012)	China	208	52	**	3/193	2/193 (1.0%)
Kim SH et al <sup>36</sup> (2013)	Korea	243	59	6.5*	65/243	26/243 (11%)
Klarich KW et al <sup>4</sup> (2013)	USA	193	58	**	55/187	7/187 (3.7%)
Kim EK et al <sup>43</sup> (2015)	Korea	85	56	37 months*	**	0

**Table-III**  
*Percentage of Patients in Major Studies with Symptoms at Presentation and Major Morbid Clinical Outcomes(\*not specified in study. \*\*New York Heart Association Functional Class >3)*  
*Congestive Heart Failure (CHF), Atrial Fibrillation (Afib)*  
*Ventricular Tachycardia/Ventricular Fibrillation (Vfib)*

Author	At Presentation	Giant t waves >10mm	Vfib/ Vtach	Deep inverted t waves	Afib	CHF	stroke	Syncope/ Pre-syncope
Sukamoto T et al <sup>40</sup>	* Asymptomatic	3.2 Chest Pain/ Dypnea	*	0	100	*	*	*
Erickson MJ et al <sup>5</sup>	46	30	47	3.8	93	12.4	4.8	2.9
Lee CH et al <sup>37</sup>	*	80	52.5	0	*	7.5	1	15
Chung T et al <sup>41</sup>	*	*	46.8	*	*	31.2	9.3	6.3
Chen CC et al <sup>42</sup>	*	*	59.6	4.2	*	14.8	25.5	10.6
Moon J et al <sup>16</sup>	78	7	*	*	*	16	15	6
Yan L et al <sup>3</sup>	*	91.8	28.8	2.6	91.3	9.9	7.2**	1.6
Kim SH et al <sup>36</sup>	*	*	36	*	*	*	*	*
Klarich KW et al <sup>4</sup>	*	28	11	11	90	29	8	11
Kim EK et al <sup>43</sup>	*	*	*	1.2	*	*	*	3.5

incidence of heart failure and atrial fibrillation.<sup>4</sup> The prognosis of AHCM becomes very poor with the development of apical aneurysms.<sup>38</sup> It is not clear if myocardial infarction in the apex leads to aneurysm formation in these patients.

### Management Considerations

The most recent guidelines, for management of patients with HCM, by the American College of Cardiology and American Heart Association (ACC/AHA) were published in 2011.<sup>6</sup> Risk stratification of AHCM patients, based on history of SCD, syncope, family history of SCD and ventricular tachycardia (VT) is reasonable. In such instances an intra-cardiac defibrillator (ICD) is recommended for the patient.<sup>6</sup> However the ACC/AHA guidelines do not separately address AHCM.

### Future Directions for Research

AHCM is still a poorly understood disease. There is a dearth of significant multi-center studies looking at long term follow-up in patients with AHCM. The outcome studies either have a very small sample size or short follow-up period. No significant study has been done to look at natural course specifically in the young population. The mechanisms underlying the differences in incidence and outcomes in patients from different ethnicities and gender are also unclear.

More basic science research is needed to understand the genetic and molecular interactions causing disease, at the cellular level. Little is known about the mechanism by which gene mutations result in myocyte disarray and hypertrophy. A better understanding regarding these complex mechanisms is necessary to improve treatment and outcomes.

In order to develop a more evidence based approach, there exists a need for more large scale population based studies to evaluate the most effective strategy for imaging known or suspected AHCM. More outcome based research is needed to evaluate the role of ICDs for primary prevention in patients with AHCM. Having a central registry to document all cases of AHCM will lead to a better understanding about the pathophysiology and natural course of the disease.

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## Cor triatriatum dextrum: A rare congenital cardiac abnormality

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### Abstract:

Cor triatriatum dextrum is an exceptionally rare congenital heart disease, in which the right atrium is partitioned into two chambers by a membrane to form a triatrial heart. It is caused by persistence of the right valve of sinus venosus. The aim of presenting this case is to develop awareness regarding cor triatriatum dextrum, though a rare case, can be present and may contribute to right heart failure and 2D-echocardiography

is an important tool in making early and accurate diagnosis. We are reporting a case of an elderly Bangladeshi male presented with the features of mitral stenosis with pulmonary hypertension with CCF with respiratory tract infection, where cor triatriatum dextrum with an atrial septal defect was an incidental finding on routine echocardiographic assessment.

Keywords: Cor triatriatum dextrum

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### Introduction:

Cor triatriatum was first described by Church in 1868.<sup>1</sup> It is a rare congenital anomaly that occurs when the left atrium (cor triatriatum sinistrum) or right atrium (cor triatriatum dextrum) is partitioned into two parts by a membrane, or a fibromuscular band.<sup>2</sup> On the right side of the heart, complete persistence of the right venous valve of the embryonic heart produces a septum in the right atrium separating the intercaval part of the right atrium from the atrial body. The remaining opening may vary depending on degree of partition or septation.<sup>3</sup> Typically, the right atrial partition is due to exaggerated fetal eustachian and thebesian valves, which together form an incomplete septum across the lower part of the atrium. This septum may range from a reticulum to a substantial sheet of tissue.<sup>4,5</sup>

Normally, during embryogenesis, the right horn of the sinus venosus gradually incorporates itself into the right atrium to

form the smooth posterior portion of the right atrium or the sinus venarum, whereas the original embryologic right atrium forms the trabeculated anterior portion. The right horn of the sinus venosus and the embryologic right atrium are then connected through the sinoatrial orifice, which, on either side, has 2 folds called the right and left venous valves. During this incorporation, the left valve becomes part of the septum secundum, and the right valve of the right horn of the sinus venosus divides the right atrium into 2 chambers. This right valve forms as a sheet that serves to direct oxygenated blood from the inferior vena cava across the foramen ovale to the left side of the heart during fetal life. Normally, the network regresses by 12 weeks, leaving behind the crista terminalis superiorly and the eustachian valve of the inferior vena cava and the thebesian valve of the coronary sinus inferiorly.<sup>5</sup> Complete persistence of the right sinus valve results in a separation between the smooth and trabeculated portions of the right atrium, constituting cor triatriatum dextrum.<sup>6-8</sup>

Cor triatriatum dextrum has varying clinical manifestations depending on the degree of partitioning or septation of the right atrium. When the septation is mild, the condition is often asymptomatic and is an incidental finding frequently made at postmortem examination; more severe septation can cause right-sided heart failure and elevated central venous pressures

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due to obstruction at the level of the tricuspid valve, the right ventricular outflow tract, or the inferior vena cava.<sup>9</sup> Significant sequelae is unusual with cor triatriatum dextrum, and in most instances remain undiagnosed.<sup>10</sup>

Cor triatriatum accounts for approximately 0.1% of all congenital heart diseases,<sup>11</sup> most being cor triatriatum sinistrum. In most cases, the anomaly is recorded at necropsy, either as an isolated finding in an otherwise normal heart or as an accompaniment to other congenital heart lesions.<sup>7,8</sup>

### Case Report

Our patient is a 75-year-old retired farmer who presented with acute severe breathlessness with increased intensity of cough for about a week.

Patient has cough for last six months and was productive with moderate mucoid sputum. There was no history of hemoptysis, fever, night sweat or significant weight loss. Further query revealed that he got easily fatigued and breathless, sometimes even at rest for last six months. There was associated occasional orthopnoea and paroxysmal nocturnal dyspnoea. He also gave history of smoking.

He required admission in respiratory medicine department of a tertiary care hospital about two months prior to this admission following similar episode of breathlessness. He was diagnosed clinically to have Chronic Obstructive Pulmonary Disease (COPD) with

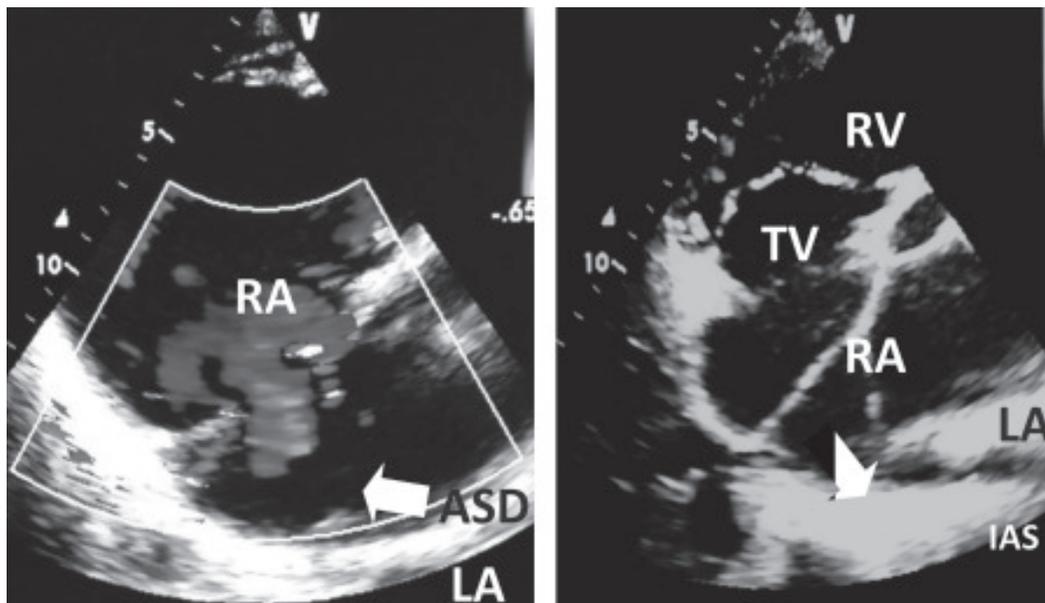
pulmonary hypertension and was treated accordingly and discharged when improved.

Physical examination revealed a chronically ill-looking elderly man, dyspnoeic at rest without cyanosis. There was no significant lymphadenopathy. We also found the patient having bilateral leg swelling up to mid-calves, raised jugular venous pressure and bibasal end-inspiratory fine crepitation and tender hepatomegaly along with left parasternal heave and loud pulmonary component of 2<sup>nd</sup> heart sound. Apex beat was normal in position and character. There was apical non-radiating mid-diastolic murmur best heard on left lateral position with breath held in expiration and there was also left lower parasternal pansystolic murmur better heard with breath held in inspiration without any radiation.

He was settled with optimal dose of diuretics and nebulized bronchodilators and anticholinergics.

An initial clinical diagnosis of mitral stenosis with pulmonary hypertension with CCF with respiratory tract infection (RTI) was made.

Electrocardiography showed sinus rhythm with right axis deviation and right atrial enlargement. X-ray chest PA view showed RV type apex, straightening of the left heart border with upper lobe diversion of pulmonary veins. Transthoracic echocardiography revealed moderate mitral stenosis and mild mitral, aortic and tricuspid insufficiency with estimated PASP 46 mm Hg. Echocardiography also showed an incomplete membranous septum diagonally dividing the right atrium into 2 parts a secundum atrial septal defect.(Figure-1, right & left panel)



**Fig.-1:** Left panel showing color flow across secundum Atrial septal defect & right panel showing a diagonal membrane (patterned arrow) across the right atrium

A final diagnosis was moderate mitral stenosis with mild mitral aortic and tricuspid regurgitation with Atrial septal defect (secundum) with mild pulmonary hypertension with Cor triatriatum dextrum with CCF with RTI. The combination of acquired Mitral stenosis & congenital ASD is known as 'Lutembacher's syndrome'.

The Patient was treated with anti-failure medications, bronchodilators and oxygen and has been counseled regarding his condition.

#### **Discussion:**

Cor triatriatum dextrum, an extremely rare form of cor triatriatum, accounting for 0.025% of all congenital heart disease.<sup>12</sup> It can occur as an isolated cardiac anomaly<sup>13</sup> or associated with other malformation of right heart structures such as pulmonary artery stenosis or atresia, pulmonary valve stenosis or atresia, hypoplastic right ventricle, tricuspid valve stenosis or atresia, atrial septal defect and Ebstein anomaly.<sup>4,14,15</sup> Unlike cor triatriatum sinistrum, which carries a higher mortality rate if not repaired, cor triatriatum dextrum has varying clinical manifestations depending on the degree of obstruction to venous flow ranging from asymptomatic to overt right-sided heart failure and elevated central venous pressures. In our patient, free flow of blood across the membrane of right atrium is probably the major element for the surprisingly good tolerance of the pathology for such a long periods of time.

Cor triatriatum dextrum may contribute to right heart failure. In our patient, heart failure was probably due to superimposition of severe respiratory tract infection upon already existing mitral stenosis, ASD (secundum) & pulmonary hypertension. There was no Cyanosis in our patient which is very rarely reported with cor triatriatum dextrum<sup>16,17</sup> and in those cases, echocardiographic assessment showed significant obstruction at the level of right ventricular inflow.<sup>16</sup> Common cardiac causes of pulmonary hypertension include diseases affecting the lung, mitral valve and left ventricle. In our patient, Cor triatriatum dextrum and ASD were incidental finding coexisting with mitral stenosis and RTI, which could not be optimally managed previously and gave rise to pulmonary hypertension and right heart failure.

There are no pathognomonic chest X-ray or electrocardiographic findings in isolated cor triatriatum dextrum. Right heart catheterization may reveal elevated pressure in the proximal right atrial chamber with a gradient across the accessory membrane. Echocardiography (transthoracic and transesophageal)<sup>14,18,19</sup> and cardiac magnetic resonance imaging (MRI)<sup>20,21</sup> are usually

diagnostic as it demonstrates the presence of a membrane within the right atrium. Since many patients are asymptomatic, transthoracic echocardiography which is more readily available and affordable has a pivotal role as a mean of ante-mortem diagnosis. In symptomatic patients, when diagnosis is established, it is amenable to relatively simple surgical resection of the dividing membrane. Percutaneous catheter disruption of the membrane has also been suggested as a preferred alternative to open heart surgery.<sup>22</sup>

#### **Conclusion:**

Careful routine echocardiographic assessment can reveal asymptomatic rare congenital anomalies of the heart which otherwise remain undiagnosed and sometimes contribute to important clue to the management of the cardiac patients.

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## Cyanotic complex congenital heart disease presenting with brain abscess at the age of 19: A case report and review of literature

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### Abstract:

**Introduction:** Brain abscess is a complication of congenital cyanotic heart disease with or without shunt anomaly. It is more common in children. Here, we delineated a case of 19-year-old young Bangladeshi - Bengali male who presenting with brain abscess, an unusual presentation of cyanotic complex congenital

heart disease. This case report focuses not only on the unusual presentation of congenital heart disease but also emphasizes the importance of early recognition of neurological complication for referral management.

*Key words:* Heart defects, Congenital, Cyanosis, Brain, Abscess.

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### Case presentation:

A 19-year-old Bangladeshi male presented with fever, cough, and breathlessness for 2 weeks, headache, confusion and focal convulsion confined to right side of face for 4 days. He had mild dehydration, congested conjunctiva, pulse- 136 bpm, blood pressure- 90/60 mm Hg, temperature-105<sup>0</sup> F, grade 4 clubbing, cyanosis and

oxygen saturation 81%, features of pulmonary stenosis, tricuspid atresia in precordium and Glasgow coma scale- 10/15, rigid neck, bilateral planter extensor, bilateral early papilloedema and crackles of right side of back of the chest. Lab reports include 18.7 gm/dl Hb%, 10,200 /mm<sup>3</sup> white cell count with 75% neutrophils. There was pneumonic consolidation of right side in chest X-ray and sinus tachycardia, left axis deviation, short PR interval and features of right atrial enlargement in electrocardiogram. Doppler echocardiogram revealed tricuspid atresia, rudimentary right ventricle, large ventricular septal defect, severe valvular pulmonary stenosis, atrial septal defect, persistent left superior vena cava with ejection fraction 63%. CT scan of brain revealed a cerebral abscess in left frontal lobe with mass effect; the lesion was operated through bar hole. Culture of pus revealed *Enterococcus faecalis*. Persistent abscess was noted on repeat CT scan after 3 weeks. Unfortunately, patient died despite giving appropriate treatment with proper antibiotic according to pus culture and antibiotic sensitivity report.

**Conclusion:** Congenital heart disease with right to left shunt should be considered if a young patient presents with brain abscess without presence of other risk factors.

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During follow-up or 1<sup>st</sup> time visit of congenital cyanotic heart disease, development of focal neurological deficits without any residual deficits or evidence of raised intracranial pressure should be monitored.

#### **Background:**

Brain abscess is a rare but well-recognized, serious life threatening complication of cyanotic congenital heart disease. It is more common in children<sup>1</sup>. Its mortality rate is high in the setting of cyanotic congenital heart disease, even in the modern era<sup>2</sup>. Here, we described a case of 19-year-old young Bangladeshi male who had undetected cyanotic complex congenital heart disease presenting with brain abscess. We emphasized on this type of presentation of congenital heart disease as it is uncommon and early detection of neurological complication in this settings gives better outcome.

#### **Case Presentation:**

A 19-year-old young Bangladeshi garments worker was admitted to Department of Cardiology, Dhaka Medical College Hospital with the complaints of fever, cough, shortness of breath for 2 weeks and headache, altered mental status, focal convulsion for 4 days. His fever came without chills and rigor, was high grade from the 1<sup>st</sup> day of symptoms, maximum recorded temperature 105<sup>0</sup> F, persisted whole day with a 2<sup>0</sup> F fluctuation even after taking anti-pyretic drugs. It was associated with cough which was dry and worsening day by day. It had no precipitating factors, not associated with chest pain, hemoptysis, wheeze, nasal symptoms, hoarseness of voice, dyspepsia, heartburn, skin rash, weight loss. He also had shortness of breath on exertion for the same duration, which came on gradually, was constant and worsening. Later he felt it even on rest. It was not associated with any precipitating or relieving factors, chest pain, pre-syncope or syncope, orthopnoea, oedema, any vasculitic symptoms, pruritus. His functional status was New York Heart Association (NYHA) class IV. For last 4 days, he had severe headache that developed gradually, persistent over whole day, more on morning and during straining. Later, He became drowsy and developed focal convulsion. Convulsion occurred several times with preceding history of forced head and eye turning to left side and was only confined to right side of face; each episode persisted for 5- 10 seconds. Neurological features were not associated with rash, limb weakness, visual disturbances, dysphasia, nausea, vomiting, recent ear and upper respiratory tract infection, head trauma. On query, patient's mother gave history of

presence of cyanosis and clubbed fingers since his childhood. Otherwise he had normal developmental milestones and no history of chest infection and family history of heart disease. He was non-smoker, non-alcoholic in personal life. He took a drug- combination of amoxicillin/ clavulanic acid for 5 days and paracetamol before admission but there was no improvement. On admission, he was mildly dehydrated, having below average body build, congested conjunctiva, regular pulse at a rate of 136bpm, 90/60 mm Hg blood pressure, 105<sup>0</sup> F surface body temperature, grade 4 clubbing, marked cyanosis in lips, tongue and nail bed and SpO<sub>2</sub> 81% in room temperature and no stigmata of infective endocarditis. He had features of pulmonary stenosis- a widely split S2, with a soft and delayed P2 with a systolic ejection murmur of grade V, more prominent in the left 3<sup>rd</sup> intercostals space that increased with inspiration and radiates diffusely along with both right and left upper sternal border and in back of chest and features of tricuspid atresia - hyperdynamic apical impulse without displacement and loud 1<sup>st</sup> heart sound. Besides this, there was also a systolic thrill in right 1<sup>st</sup> and 2<sup>nd</sup> Intercostal space along with right upper sternal border. He had 10/15 GCS (E3, M6, V1), sign of meningeal irritation (neck rigidity), features of upper motor neuron lesion- bilateral early papilloedema and bilateral extensor planter response. He also had vesicular breath sound and crackles from 7<sup>th</sup> intercostals space down to the right side of back of the chest.

Marked cyanosis, clubbing and features of valvular heart disease lead to the diagnosis of congenital cyanotic heart disease. Neurological findings in this setting were thought to be done to meningoencephalitis complicated by brain abscess. Clinical findings of chest were thought to be pneumonia.

Complete blood count revealed 18.7 gm/dl Hb%, 05 mm of Hg ESR, 10,200 /mm<sup>3</sup> white cell count with 75% neutrophils, 17.6% lymphocytes. Serum electrolytes, serum creatinine, serum urea and serum ALT were within normal limits. Chest x- ray revealed inhomogeneous opacity in right mid and lower zones, cardiac shadow was normal in transverse diameter, right ventricular type apex, reduced broncho-vascular markings.

In electrocardiogram, there were sinus tachycardia, left axis deviation, short PR interval, right atrial enlargement. Colour Doppler echocardiogram revealed tricuspid atresia, rudimentary right ventricle, large ventricular septal defect ( perimembranous and muscular), severe pulmonary stenosis (valvular type), atrial septal defect

(coronary sinus type), persistent left superior vena cava, normal atrioventricular and ventriculoarterial relationship, normal great artery relationship, good left ventricular systolic function with ejection fraction 63%.

There was also no growth in blood culture. Due to neurological manifestations, we did a CT scan of brain that revealed a large oval shaped hypodense mass lesion with perilesional oedema measuring about 7×5×4 cm<sup>3</sup> in size situated in the left frontal area and mass effect evident by compression on adjacent brain parenchyma with contralateral midline shift suggestive of cerebral abscess in left frontal lobe with mass effect. This was managed by draining through burr hole under general anaesthesia done by Department of Neurosurgery of same institute. Gram stain of pus revealed gram positive cocci and culture of pus showed enterococcus faecalis sensitive to ampicillin, gentamicin, penicillin G and vancomycin. He was treated with gentamicin and vancomycin. Three weeks later, again CT scan was done revealing persistent abscess. For that, repeat drain of brain abscess was suggested by Department of Neurosurgery. Preparation for 2<sup>nd</sup> time drain of brain abscess and cardiac catheterization after recovery from brain abscess was taken. Unfortunately, patient died before that the caregivers were not agreed to do a post-mortem evaluation.

#### Case discussion:

Brain abscess is a focal, intracerebral infection that begins as a localized area of infection and develops into a collection of pus surrounded by a well-vascularized capsule<sup>3</sup>. In 1814, Ferry described a case of brain abscess in congenital cyanotic heart disease for the first time in medical history<sup>4</sup>. It is an uncommon complication of congenital heart disease, mostly associated with untreated cyanotic congenital heart disease. Even in Bangladesh where most of the congenital heart diseases are undiagnosed and untreated, brain abscess is not common as a complication. In one study of 162 cases of brain abscess treated in different hospitals of Dhaka, Bangladesh in between 1999 to 2013, congenital heart disease is responsible in only 6.17% cases<sup>5</sup>. It is necessary to mention that cyanotic heart disease accounts for up to 69.1% of all cases of brain abscesses associated with higher incidence in children and peak incidence in between 4 to 7 years of age<sup>1, 6</sup>. Most series revealed 1.5 to 2.5 times a male preponderance over female in general population<sup>7, 8</sup>. This 19-year-old male having no significant history related to complex congenital cyanotic heart disease presented to us with brain abscess. The age and the way of presentation enriched

our knowledge as he had no significant symptoms of heart disease.

It is most commonly seen in tetralogy of fallot<sup>5, 6</sup>. However, any condition resulting in a significant right to left shunt appears to increase the risk. Though the abnormality couldn't be fully identified by colour Doppler and M Mode echocardiography in this case, it revealed coronary sinus type atrial septal defect, large perimembranous and muscular ventricular septal defect, tricuspid atresia. Why does brain abscess occur in cyanotic heart disease? It is always secondary to bacteremia. Bacteria in the bloodstream are not filtered through the pulmonary circulation due to right to left shunt of venous blood in heart, it is prone to seeding in brain, especially in the low- perfusion area “ in the junction of gray and white matter” and are often poorly encapsulated. This abscess also shows a predilection for the territory of the middle cerebral artery<sup>9</sup>.

The classic triad of presentation of brain abscess is headache, fever and a focal neurologic deficits, present only in less than 20% cases in one study. Localizing neurological signs occur relatively late in the course of the illness. It is usually solitary. However, due to hemoatogenous spread, multiple numbers of abscesses are often encountered. In presence of cyanotic heart disease, the most common organisms are *viridans streptococci*, including the *Streptococcus milleri*, *Streptococcus aureus*, *Haemophilus* and occasionally *Enterococci*<sup>9</sup>. *Atiq et al* revealed *Streptococcus milleri* was most common in patients with cyanotic heart disease<sup>11</sup>. Multiple organisms have also been reported<sup>12,13</sup>. In this case, gram stain of pus showed gram positive cocci and in culture, gram positive organism Enterococci faecalis was isolated.

In case of enterococcal disease, bacteremia and endocarditis are common presentations. It accounts for 5% - 20% of cases of endocarditis after *Streptococcus aureus*. There is a low possibility of endocarditis in this case as there was no clinical signs and evidence of endocarditis in colour Doppler and M mode echocardiography. Other than endocarditis, frequent sources of the bacteremia are usually the genitourinary and gastrointestinal tracts in patients with infections originating outside the hospital. There is a possibility of Enterococcal pneumonia supported by clinical signs and radiological features causing bacteraemia in this case though it is rare. It is also noted that it is an uncommon cause of meningitis with complication- brain abscess. Other complications of enterococcal meningitis are hydrocephalus and stroke<sup>14</sup>.

As cyanotic heart disease compromises cardiovascular system, the related patients are not a good candidate for general anesthesia. The mortality rate is very high. Following craniotomy and excision, it was 71%<sup>15</sup>. Nearly 17% of patients after aspiration develop cyanotic spells and may develop life-threatening complications<sup>13</sup>. The mortality rate was decreased over the past few decades, from around 70% to around 10% due to use of neuro-imaging in the management of brain abscess<sup>2</sup>. Early detection, availability of image guidance for aspiration and better radiological follow-up during the course of the antibiotic therapy is responsible to this achievement though the prognosis is dependent on the rapidity of progression of disease before hospitalization and patient mental status on admission<sup>2</sup>.

#### **Conclusion:**

Congenital heart disease with right to left shunt is one of risk factors for brain abscess and outcome is poor if it is diagnosed late. Development of focal neurologic abnormalities or evidence of raised intracranial pressure should be evaluated for presence of brain abscess.

#### **Consent:**

Written informed consent was obtained from the patient caregivers for the publication of this case report. A copy of the written consent is available for review by the editor of this journal.

#### **Competing Interests**

The authors declare that they have no competing interests.

#### **Authors' Contributions**

KMNS is the first author and was involved in the diagnosis of the case and writing a part of the manuscript. MSI is the communicating author and involved in drafting the manuscript. MAS, SK revised it critically and involved in the management of patient. HILRK did echocardiography and tried to reveal the riddle. AWC provided overall support. All the authors have read and approved the final manuscript.

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# Ebstein's anomaly with constrictive pericarditis

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### Abstract:

**Ebstein's anomaly is a rare congenital cardiac malformation that affects the tricuspid valve commonly, but its association with pericardial disease is even rarer. We report a case of 25 year old man presented with dyspnoea on exertion and fatigability. A diagnosis of Ebstein's anomaly with atrial septal defect (ASD) with constrictive pericarditis was confirmed using transthoracic echocardiography. Peroperatively**

**pericardiectomy was done after meticulous dissection of pericardium. Under cardiopulmonary bypass tricuspid valve was replaced with 29 mm Edward life science porcine tissue heart valve with direct closure of ASD. Patient showed excellent symptomatic improvement and was discharged on 7<sup>th</sup> POD with advice and after 3 months of follow up patient was doing well post operative day.**

*Keywords: Ebstein's anomaly, tricuspid valve, atrial septal defect, constrictive pericarditis, tricuspid valve replacement.*

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### Introduction:

In 1866, Wilhem Ebstein published a scholarly description of a tricuspid valve anomaly that bears his name. On July 7, 1864, he performed an autopsy of a 19 year old, laborer, Joseph Prescher at Breslau and his findings were accurately described and illustrated with excellent drawings by his colleague Dr Wyss<sup>1</sup>. Ebstein's anomaly is a rare congenital heart disorder occurring in approximately 1 % per 200,000 live births and accounting for <1% of all cases of congenital heart disease<sup>2</sup>. The incidence is equal in both sexes.<sup>3</sup> Familial Ebstein's anomaly has been reported rarely.<sup>4</sup> The risk of Ebstein's anomaly exists especially if the lithium drug is taken as medication during 2-6 weeks post conception.<sup>5</sup> Ebstein's anomaly may present at any stage and has a highly variable clinical course. Fetal and neonatal presentation is associated with poor outcomes whereas older children

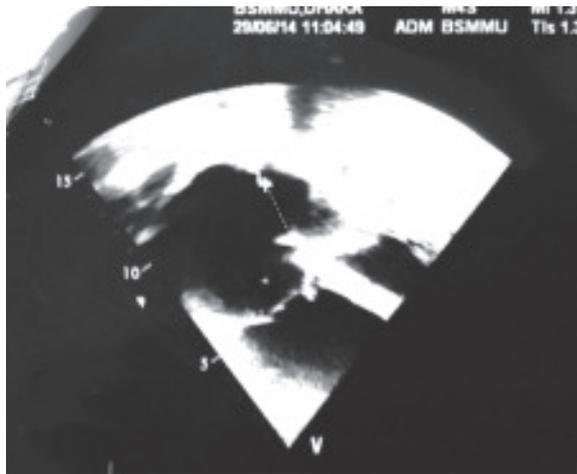
and adults long term outcome is superior.<sup>3</sup> Right ventricular abnormalities, tricuspid valve abnormalities and accessory conduction pathways (WPW syndrome) are the main primary pathophysiologic features predominating in Ebstein's anomaly.<sup>6</sup> An interarterial communication is present in 80-94 % of patients with Ebstein's anomaly. Additional anomalies include pulmonary atresia or stenosis, bicuspid or atretic aortic valves, ventricular septal defect, congenital mitral stenosis etc.<sup>7,8,9</sup> In adulthood, patients usually present with decreasing exercise tolerance, progressive cyanosis, arrhythmias or right heart failure. In the presence of interatrial communication, the risk of paradoxical embolisation, brain abscess and sudden death is increased.<sup>10</sup> Tricuspid valve repair with ASD closure is the preferred operation. However, in about 20 % to 30 % of patients, immobility or morphology of the tricuspid valve prevents repair and valve replacement is required.<sup>11,12</sup> Here, we are presenting a case of Ebstein's anomaly with constrictive pericarditis.

### Case Report:

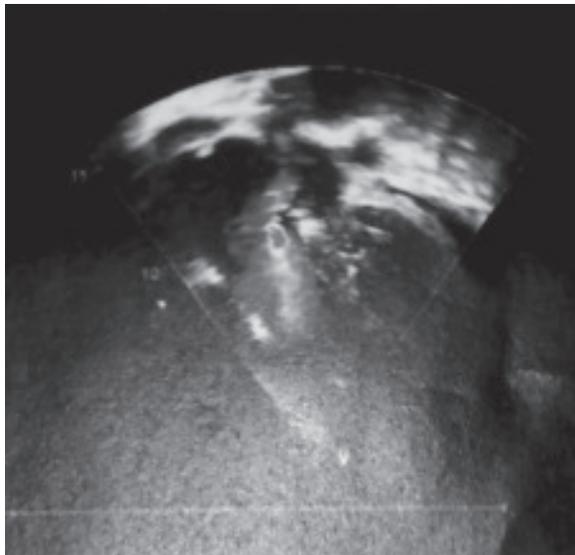
A 25 year old man was admitted in our department with chief complaints of dyspnea on exertion and fatigability for last four months. Physical examinations revealed pulse was 80 beats per minute, regular and normal in volume. Blood pressure was 110/70 mm Hg. His 1<sup>st</sup> heart sound was normal but 2<sup>nd</sup> heart sound was splitted

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at upper left parasternal border at 2<sup>nd</sup> intercostal space. There was a systolic murmur best heard at left lower parasternal border with inspiratory accentuation. Electrocardiogram revealed sinus tachycardia, right bundle branch block, right ventricular hypertrophy with inverted T waves in leads V<sub>1</sub> to V<sub>4</sub>. The chest X-ray showed marked cardiomegaly. Transthoracic colour doppler echocardiogram revealed a large ostium secundum, ASD (28 mm) with left to right shunt with PASP 53 mm Hg. Dilated RA, RV, PA with mild to moderate PAH. Ebsteinoid deformity of tricuspid valve (septal leaflet is plastered to underlying endocardium and anterior leaflet is sail like),

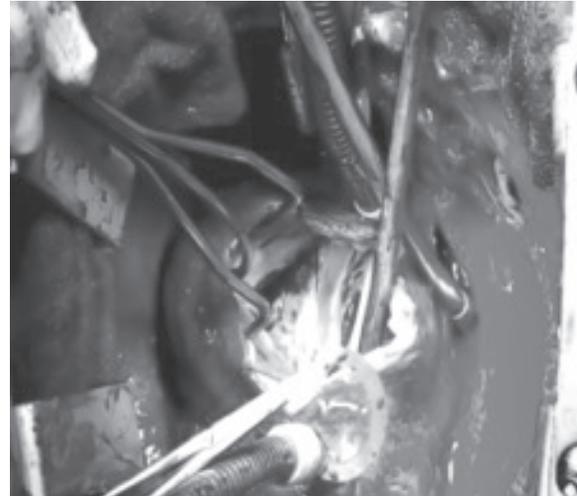


**Fig.-1:** Ebstein's anomaly of Tricuspid Valve (apical 4-chamber view).



**Fig.-2:** Color Doppler across the ASD and the Tricuspid Valve.

tricuspid valve displacement from tricuspid annulus was 16mm, mild RV systolic dysfunction with TAPSE 12mm and good LV systolic function with LVEF = 76 %. Moderate pericardial effusion (19mm), paradoxical movement of



**Fig.-3:** Anterior leaflet of Tricuspid Valve.



**Fig.-4:** Tricuspid Valve Replacement with 29 mm Porcine Bioprosthesis Valve.

IVS. IVC is 15 mm with presence of restricted respiratory variations. The patient was operated on 21<sup>st</sup> July, 2014. Under general anaesthesia, with all aseptic precaution, standard median sternotomy was done. Pericardiectomy was done with difficulty because there was gross adhesion of pericardium. Cardiopulmonary bypass was established with aortic cannulation (24 F) and bicaval cannulation with SVC (30 F) & IVC (32 F). Heart was arrested with antegrade cardioplegia under mild

hypothermia (32° C). Right atriotomy was done. A large ASD, 20 X 6 mm, secundum, was detected. Tricuspid annulus was dilated. Septal leaflet was rudimentary. Anterior leaflet was sailed approximately 1cm displaced. Tricuspid valve was beyond repairable. Tricuspid valve was replaced with 29 mm Edward life science tissue heart valve with total preservation of subvalvular structures. Direct closure of ASD was done. Right atriotomy was closed in layers. Patient was weaned from bypass without any difficulty. Total cross clamp time was 35 minutes and total bypass time was 60 minutes. After achieving proper haemostasis, chest was closed leaving two mediastinal drainage (retrosternal 24 F) connected to under water sealed drainage. The patient was shifted to ICU with minimum inotropic supports and was extubated 8 hours after arrival at ICU. The patient was discharged on 7<sup>th</sup> POD with advice of taking tablets warfarin sodium 2.5 mg everyday for 3 months followed by antiplatelet therapy. Patient remains asymptomatic in the subsequent follow up and is leading to an almost normal life.

#### Discussion :

Ebstein's anomaly is a malformation of the tricuspid valve and the right ventricle characterized by adherence of the septal and posterior leaflets to the underlying myocardium; downward displacement of the functional annulus (septal>posterior> anterior); dilatation of the "atrialized" portion of the right ventricle; with various degrees of the hypertrophy and thinning of the wall; redundancy, fenestrations and tethering of the anterior leaflet and dilatation of the right atrioventricular junction(true tricuspid annulus) .<sup>13</sup> Regarding embryology, the leaflets and tensile apparatus of the tricuspid valve are believed to be formed mostly by the process of delamination of the inner layers of the inlet zone of the right ventricle. The downward displacement of the leaflets in Ebstein's anomaly suggests that delamination from the inlet portion failed to occur.<sup>14</sup> In our case, peroperative findings were almost similar with others.<sup>13,14</sup> It is not uncommon for Ebstein's anomaly to be undiagnosed until adulthood. The mean age of diagnosis in a study of the natural history of 72 unoperated patients was 23.9±10.4 years.<sup>15</sup> As our reported case was 25 years of age which is consistent with some other authors.<sup>15</sup> The cardinal symptoms in Ebstein's anomaly are cyanosis, right-sided heart failure, arrhythmias, and sudden cardiac death.<sup>10</sup> Children more than 10 years of age and adults often present with fatigue, right-sided heart failure and arrhythmias<sup>10</sup>. Our patient had complaints of dyspnea on exertion and fatigability

for four months. A systolic murmur may be heard at lower left sternal edge due to tricuspid regurgitation.<sup>6</sup> Regarding our patient, 2nd heart sound was widely splitted and fixed at upper left parasternal border and a systolic murmur was heard at lower left parasternal border. Chest radiograph showed marked cardiomegaly with a rounded or boxlike cardiac contour with normal or oligemic lung fields.<sup>6</sup> In our patient, chest radiograph showed only cardiomegaly. The ECG is abnormal in most patients with Ebstein's anomaly. It may show tall and broad P waves with complete or incomplete right bundle-branch block patterns and bizarre morphologies of the terminal QRS pattern result from infra-Hisian conduction disturbance and abnormal activation of the atrialized right ventricle. From 6% to 36% of patients with Ebstein's anomaly have more than one accessory pathway and most accessory pathways are located around the orifice of the tri-cuspid valve. First-degree atrio-ven-tricular block occurs in 42%.<sup>16,17,18</sup> Wolff-Parkinson-White syndrome is found in 30-50%.<sup>17</sup> In our case ECG was showing sinus tachycardia with right bundle branch block with right ventricular hypertrophy and inverted T waves in leads V<sub>1</sub> to V<sub>4</sub>. Two dimensional echocardiography is the diagnostic test of choice for Ebstein's anomaly. Echocardiography allows accurate evaluation of the tricuspid valve leaflets and the size and function of the cardiac chambers. The principal feature of Ebstein's anomaly is apical displacement of the septal leaflet of the tricuspid valve from the insertion of the anterior leaflet of the mitral valve by at least 8 mm/m<sup>2</sup> body surface area.<sup>6</sup> Our patient had 16mm displacement of septal leaflet on echocardiography. Our patient have Great Ormond Street Ratio (GOSR) score 2. Echocardiographing finding of constrictive pericarditis are left ventricular free wall flattening, paradoxical motion of interventricular septum( septal bounce), premature opening of the pulmonary valve, inferior venacava and hepatic vein dilatation with restricted respiratory variation.<sup>23</sup> In our case, paradoxical motion of the IVS and IVC dilatation with restricted respiratory variation were present. Tricuspid valve repair with direct ASD closure is the preferred operation. However in 20-30% of patient, valve replacement is required.<sup>6</sup> Porcine bioprosthetic valve remains a good alternative. Most prefer bioprosthesis to mechanical valves due to relatively good durability and lack of need for anticoagulation.<sup>19</sup> A bioprosthetic valve may offer superior late survival when compared with a mechanical valve when tricuspid valve replacement is required in patients with Ebstein anomaly.<sup>20</sup> Heart transplant is rarely necessary for Ebstein's anomaly. Its indication is usually the presence of severe biventricular dysfunction (LVEF

<25 %).<sup>2</sup> In our, patient tricuspid valve apparatus were beyond repairable and tricuspid valve was replaced with 29 mm Edward life science porcine tissue heart valve. Ebstein's anomaly with pericardial diseases are rarely reported in the literature.<sup>24</sup> In our case, pericardium was densely adherent to the anterior surface of pericardium. No operative sample of the pericardium was taken for histopathological examination. The cause of pericarditis was chronic inflammation as our patient was a case of congenital heart disorder (Ebstein's anomaly with ASD). A 35 years old lady with diagnosis of Ebstein's anomaly with pericardial disease was reported in 2005.<sup>21</sup> Similarly, Ebstein's anomaly with significant pericardial effusion was reported in 1998<sup>22</sup>. Our patient had Ebstein's anomaly with constrictive pericarditis.

#### Conclusions:

Ebstein's anomaly with constrictive pericarditis has been reported in medical literature<sup>24</sup>. Untreated Ebstein's anomaly with constrictive pericarditis can be effectively managed with surgical treatment.

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