Original article

# Histomorphological evaluation of myocardial infarction: autopsy study

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**Abstract:**

**Introduction:** Myocardial Infarction, is the irreversible necrosis of the heart muscle secondary to prolonged ischemia. Ischemic heart disease and stroke are the two most common cause of death worldwise.

**Aims and Objectives:** To study histomorphological changes in myocardial infarction, to determine its age-sex distribution, etiology & complications.

**Material and Methods:** The study was carried out in the autopsy section ofDepartment of Pathology, B.J. GovernmentMedical College and Sassoon General Hospital, Pune, India during January 2014 to June 2015prospectively. Total 315 hearts were studied in detail in histologically confirmed cases of myocardial infarction on Hematoxylin and eosin stains.

**Observation and results:** Commonest age group was 40-49 years with significant number of cases below 30years of age. Males are more commonly affected than females. Coronary atherosclerosis was most common etiology. Left anterior descending artery causing anterior wall MI was most common morphological finding. Ventricular aneurysm is the most common complication.

**Conclusion:** Ischemic heart disease of elderly is now shifting towards younger age group. Premenopausal women are relatively protected against myocardial infarction.

**Keywords:** Myocardial Infarction, Coronary Atherosclerosis, Autopsy

## INTRODUCTION

In addition to the high rate of Ischemic Heart Disease (IHD) mortality in the Indian Subcontinent (52%), IHD manifests almost 10 years earlier on an average in this region compared with the rest of the world (23%), [1, 2] resulting in substantial deaths occurring in working age group. Research initiatives like generation of reliable statistics on prevalence and incidence of CVD and its risk factors and CVD-related mortality through the initiation of large cohort studies and trials registries have been suggested.[3]

More than half of these deaths occurring before the stricken individual reaches hospital.The overall death rate from ischemic heart disease has fallen approximately 1/3[rd]owing to lifestyle modifications, therapeutic advances and control of diabetes.[4] Since, the changes in heart of ischemic heart disease cannot be fully revealed during life, it is universally accepted that autopsy material yields very valuable information.[5]

The present study aims at studying histomorphological changes in myocardial infarction, to correlate extent and site of coronary artery stenosis with the site of infarct in the heart. And to determine the age sex distribution, etiology and complications of myocardial infarction in the heart.

**Materials and Methods**

In the present autopsy study, hearts of all medico-legal postmortems referred to the autopsy section of B.J. Government Medical College and Sassoon General Hospital, Pune, India, for histopathological examination during January 2014 to June 2015 were studied.

Subjects of all age group were included, so were the patients dying of various external causes of death like accidents, burns, suicide etc. as well of external causes like hospital death. However, the cases in which on histopathological evaluation myocardial infarction was not found were excluded from the study.

Method for examination of heart:-

The heart was fixed in 10% formalin for at least 24 hours after washing thoroughly with water to remove the postmortem blood clots. Then the heart was weighed and examined externally for the size, shape, and obvious anomaly, signs of pericarditis, infarction or any rupture etc. Then it was opened along the direction of flow of blood as described by Virchow[6] and examined internally with a special focus on endocardium, valves, aorta and coronary ostia.

Then the epicardial coronary arteries were dissected by transverse sectioning at an interval of 3mm. The section was examined for extent and severity of atherosclerotic narrowing of the coronary artery lumen and for any occlusion by thrombus/ embolism or presence of any calcification. This site showing maximum luminal narrowing or thrombus formation or any other significant finding was sectioned for the microscopic evaluation.

Then the heart was cut into transverse slices, 1cm thick, commencing at the apex of the heart and stopping 1cm short of mitral valve. The cut surfaces were examined for variation in colour, consistency and changes if any. The routine sections were taken from the apex, anterior wall, posterior wall, lateral wall and septum. Any area showing the changes suggestive of infarction or its complication was sampled. If there was seen any atherosclerotic block in the coronary, the walls supplied by it was thoroughly examined and sampled besides the routine sampling.

The sampled sections were embedded in paraffin, cut at a thickness of 4-6μ. Sections were stained with routine heamatoxylin and eosin.

**Observations and Results**

Total 315 cases of myocardial infarction were evaluated from medico-legal cases and clinical post-mortem in an autopsy section of pathology department of B.J.G.M.C. Punefrom January 2014 to June 2015 prospectively.

Table no.1: Age and sex distribution of myocardial infarction of cases studied.

| Age (years) | Male | Female | Total | Percentage of cases |
|---|---|---|---|---|
| <20 | 01 | 0 | 01 | 0.32 |
| 20-29 | 17 | 0 | 17 | 5.43 |
| 30-39 | 55 | 01 | 56 | 17.89 |
| 40-49 | 74 | 11 | 85 | 26.84 |
| 50-59 | 73 | 03 | 76 | 23.96 |
| 60-69 | 46 | 12 | 58 | 18.53 |
| 70-79 | 08 | 06 | 14 | 4.47 |
| >/=80 | 06 | 02 | 08 | 2.56 |
| Total | 280 | 35 | 315 | 100 |

Table 2: Etiological factors for myocardial infarction.

| Etiology | No. of cases | Percentage of cases |
|---|---|---|
| Atherosclerosis | 288 | 91.43% |
| Emboli | 7 | 2.22% |
| Rheumatic Heart Disease | 4 | 1.27% |
| Aortic Dissection | 2 | 0.63% |
| Vasospasm | 14 | 4.44% |

Table 3: Distribution of atherosclerotic involvement of coronary arteries.

| Artery | No. of cases(n=288) | Percentage of cases |
|---|---|---|
| Right coronary artery | 95 | 33% |
| Left circumflex artery | 73 | 25.34% |
| Left anterior descending artery | 215 | 74.65% |

The number of vessels involved per case was more than one.

Table 4: Area wise distribution of myocardial infarcts

| Area involved | No. of cases | Percentage of cases |
|---|---|---|
| Anterior wall | 139 | 43.9% |
| Posterior wall | 110 | 34.8% |
| Lateral wall | 51 | 16.13% |
| Septal | 23 | 7.28% |
| Circumferential | 6 | 0.19% |
| Apex | 52 | 16.46% |

Anterior, antero-septal, antero-lateral were considered in anterior wall group while posterior, posteroseptal, postero-lateral in posterior group. Lateral wall alone constitutes lateral wall group. Many cases show more than one wall involvement.

Table 5: Age wise distribution of myocardial infarction in various stages

| Age group (years) | Acute MI | Healing MI | Healed MI | Acute + Healing MI | Acute + Healed MI | Healed + Healing MI | Total |
|---|---|---|---|---|---|---|---|
| 11-19 | 01 (100%) | 00 | 00 | 00 | 00 | 00 | 01 (100%) |
| 20-29 | 10 (52.63%) | 02 (10.53%) | 04 (21.05%) | 00 | 03 (15.8%) | 00 | 19 (100%) |
| 30-39 | 15 (28.85%) | 12 (23.08%) | 22 (42.31%) | 00 | 01 (1.92%) | 02 (3.85%) | 52 (100%) |
| 40-49 | 24 (27.27%) | 12 (13.63%) | 47 (53.41%) | 00 | 01 (1.14%) | 04 (4.55%) | 88 (100%) |

| 50-59 | 11 (14.10%) | 15 (19.23%) | 45 (57.7%) | 01 (1.28%) | 04 (5.13%) | 02 (2.56%) | 78 (100%) |
|---|---|---|---|---|---|---|---|
| 60-69 | 10 (18.52%) | 04 (7.41%) | 32 (59.26%) | 00 | 06 (11.11%) | 02 (3.70%) | 54 (100%) |
| 70-79 | 02 (13.33%) | 01 (6.66%) | 11 (73.33%) | 00 | 01 (6.66%) | 00 | 15 (100%) |
| >/=80 | 00 | 00 | 04 (50%) | 01 (12.5%) | 03 (37.5%) | 00 | 08 (100%) |
| Total | 73 (23.17%) | 46 (14.60%) | 165 (52.38%) | 02 (0.63%) | 19 (6.03) | 10 (3.17%) | 315 (100%) |

Healed infarcts (52.38%) were found to be most common in our study, followed by acute infarcts (23.17%)

Table :6 Complications in myocardial infarction

| Complication | No. of cases (n=315) | Percentage of cases |
|---|---|---|
| Ventricular aneurysm | 17 | 5.4% |
| Mural Thrombus | 15 | 4.76% |
| Myocardial rupture | 01 | 0.32% |
| Pericarditis | 04 | 1.27% |
| Atrial dilatation | 01 | 0.32% |
| Total | 38 | 12.06% |

**Discussion**

We have compared our results with similar studies in India and abroad. Thus the data available for comparison is from different geographic areas and over different time periods.

In the present study the youngest patient was 18 year old and the oldest was 86 year old. Maximum number of cases were seen in age group of 40-70 years with commonest age group being 40-49 years(26.84%) followed by 50-59 years(23.96%), 60-69 years(18.53%), 30-39 years(17.89%), 20-29 years (5.43%) in the present study. This is comparable with the study of Shushma Pandey et al.[7] Udnoon J et al [8] and Andrew Farb et al.[9]However, this is in contrast with that of Bardoli and Falzi et al[10], Pearl and D'Alonza[11], who found 50-59 years as commonest age group with maximum number of cases in sixth and seventh decade of life.Thus it emphasizes that ischemic heart disease was a disease of elderly but now shifting towards younger age group, which may be because of present urbanization, sedentary life style and changing dietary habits.[12]

Myocardial infarction was found to be more prevalent in males than in females. Bardoli G. et al[10]and Pearl and D'Alonza[11] also found that the incidence rate was much greater in men than in women. In the present study male to female ratio cannot be determined accurately as the sample population contains less number of females than in general population owing to males being more prone to accidents, violence, stress, smoking and alcoholism in India.[13]Hence we have received more male autopsies than female.

The difference between male to female ratio appears to decrease as age advances above 50 years, which might be due to women are remarkably protected against myocardial infarction during reproductive years. Cessation of estrogen after menopause in female probably is causing rapid development of coronary heart disease,[4,14] but this aspect could not be investigated in detail because of small sample size of females in the present study. Still we

can see the ratio decreases as the age advances and males and females are almost equally affected in seventh and eight decade of life.

Atherosclerosis was the most common etiological factor (288 cases). An analysis of etiology of myocardial infarction revealed that atherosclerosis in form of stenosing plaque causing reduction of cross-sectional area of coronary lumen by approximately 70% was observed in 91.43% cases. This is quite comparable with that of many earlier workers who have been reported from 75% to over 90% cases with narrowing of >75% of cross-sectional area of coronary lumen.[9,15-18]. Seven cases of emboli, out of which six were found in left anterior descending artery and one was found in right coronary artery. In one case prosthetic valve was seen in the region of aortic valve. Four cases of rheumatic heart disease, out of which three were aortic stenosis and one was aortic stenosis with mitral stenosis. Two cases of dissecting aneurysm of aorta were seen associated with coronary malfunction due to occlusion of coronary ostia by the torn intimal flap of aorta. Fourteen cases in which cause was not found, vasospasm was the most common suspected etiology.

In the present study left anterior descending artery (74.65%) was the commonest involved artery by the atherosclerotic process (narrowing more than 70% of cross-sectional area of the lumen) followed by right coronary artery (33%) and left circumflex artery (25.34%).

The frequency of involvement of coronaries in present study is similar to studies by Andrew Farb et al [9], Satish Chandra and Tiagi's[19] and Mc Namera et al [20]. In a study by Pandey S.et al[88] the most common wall involved was anterior wall which is comparable to the present study.Anterior wall is most predominantly involved area followed by posterior wall and lateral wall, hence it became evident that coronary lesion is always found in an artery which supplies the infarcted myocardium. This is in agreement with the observations by Crowford[21], Robertson[18]& Robert and Buja.[16]

Healed infarcts (52.38%) were found to be most common in our study, followed by acute infarcts (23.17%). Acute on healing (0.63%) are least frequency ones. Up to the age of 30 years majority of the infarcts were acute (55%). The proportion of healed infarcts increases with advancing age.

Complications of myocardial infarction were seen in 38 cases. Among them ventricular dilatation (17 cases) followed by mural thrombus (15 cases) was found as the most common complications of infarct. Becker-Anderson[22] mentioned ventricular dilatation as an important category, which can occur either as an early or late complication of acute myocardial infarction. Aneurysm developing later has a different morphology in that more scar tissue has been produced.

**Conclusion**

The present autopsy study was done on 315 cases of myocardial infarction.

Higher than expected prevalence of myocardial infarction is found at younger age groups in present study. This emphasizes that ischemic heart disease is of elderly but now shifting towards younger age group, which may be due to stress of modern fast life.Males were more commonly affected than females.Females in premenopausal age group have lower incidence of ischemic heart disease, which suggests female are protected against myocardial infarction in reproductive age years.Coronary atherosclerosis was found to be the most common cause of myocardial infarction.Left anterior descending artery was the most commonly involved artery by significant atherosclerosis, therefore anterior wall MI is most common.Up to the age of 30 years majority of the infarcts are acuteand the proportion of healed infarcts increases with advancing age.Ventricular dilatation and mural thrombus was found as the most common complications of infarct.

The knowledge about the time trends and the morphological features of ischemic disease can guide the possible preventive measures. Thus, extensive cross sectional studies depicting the histomorphological details are needed to tackle the impending epidemic of myocardial infarction.

### References

1. Gupta R. Burden of coronary heart disease in India. Indian Heart J 2005; 57: 632-8.

2. Yusuf S, Hawken S, Ounpuu S, Dans T, Avezum A, Lanas F et al. Effect of potentially modifiable risk factors associated with myocardial infarction in 52 countries (the INTERHEART study): case- control study. Lancet 2004; 346: 937-52.

3. Goyal A, Yusuf S. The burden of cardiovascular disease in the Indian subcontinent. Indian J med Res 124, September 2006:235-244

4. Schoen FJ. Blood vessels and The Heart. In. Kumar V, Abbas AK, Fausto N. Robbins and Cotran Pathological basis of disease 8th edition, Philadelphia, Saunders 2013:487-588

5. Strong JP, Mcgill Jr HC. The natural history of coronary atherosclerosis. Am J Pathol 1962;40:37

6. Rezek PR, Millard M. Heart, Thoracic Aorta and Great Vessels. In: Autopsy Pathology A Guide for Pathologists and Clinicians. Springfield, III:Charles C Thomas;1963:231-327

7. Pandey S, Jhanwar P, Jhanwar S. A prospective study of Myocardial Infarction patients admitted in a tertiary care hospital of south-eastern Rajasthan. Int J Biol Med Res.2012; 3(2):1694-1696

8. Udnoon J, Chirachariyavej T, Peonim V. Sudden unexpected deaths in different age groups at Ramathibodi Hospitals, Bangkok, Thailand: a retrospective autopsy study during 2003-2007. Southeast Asian J Trop Med Public Health.2009 Jan;40(1):162-8

9. Farb A, Jang AL, Burke AP, Sessums L, Liang Y and Virmani R. Sudden Coronary Death: Frequency of active coronary lesions, inactive coronary lesions and myocardial infarction, Circulation. 1995;1701-09.

10. Bardoli G, Falzi G, Mariani F.Sudden coronary death- a post-mortem study in 208 selected cases compared to 97 control subjects. Am.Heart J. 1979;98:20-32.

11. Pell S, D'Allonza AD. Acute myocardial infarction in a large industrial population. JAMA,1963;185:831-38.

12. WHO Scientific Group. The pathological diagnosis of acute ischemic heart disease, WHO Techn. Rep. Ser 1970; 441:5-27.

13. Garg M, Agrawal AD, Kataria SP. Coronary atherosclerosis and myocardial infarction- An autopsy study. J Indian Acad Forensic Med. 2011 Jan-Mar;33(1):39-42

14. Park K. Coronary heart disease in Epidemiology of chronic non-communicable diseases and conditions. In: Park's Textbook of Preventive and Social Medicine 16th edition. Jabalpur, M/s Banarsidas Bhanot publishers. 2000; 271-76.

15. Buja LM, Willerson JJ. Clinicopatholgic correlates of Acute Ischemic Heart Disease Syndromes. The American Journal of Cardiology. 1981;47:343-56.

16. Roberts WC, Buja ML. The frequency and significance of coronary arterial thrombi and other observations in Fatal Acute Myocardial Infarction- a study of 107 necropsy patients. Am.J.of Med.1972;52:425-43

17. Horie T, Sekiguchi M, Hirosawa K. Coronary thrombosis in pathogenesis of acute myocardial infarction- Histopathological study of coronary arteries in 108 necropsied cases using serial sections. Br. Heart J.1978;40:153-61.

18. Davies MJ, Woolf N, Robertson WB. Pathology of acute myocardial infarction. Br.Heart J. 1976;38:659-64

19. Chandra S, Tiagi. Atherosclerosis in coronary arteries, Gross study. The Antiseptic. 1965 Jul;62:75-79.

20. Mc Namera JJ, Molot MA, Stremple JF, Cutting RT. Coronary artery disease in combat casualties in Vietnam. JAMA. 1971 May 17;216(7):1185-7.

21. Crawford T. Sudden death as a manifestation of ischemic heart disease in Pathology of ischemic heart disease. London, Butterworths. 1977;77.

22. Becker AE, Anderson RH. Acquired conditions primarily affecting myocardial contractility in Cardiac Pathology. In. Becker AE, Anderson RH- An integrated text and colour atlas. London, Churchil Livingstone. 1983;3.1-3.34