

## **AORTIC ATHEROSCLEROSIS AS A REFLECTION OF CORONARY ATHEROSCLEROSIS – AN AUTOPSY STUDY WITH HISTORICAL REVIEW**

Kusuma Venkatesh<sup>1</sup>, C. M. Jayakeerthy<sup>2</sup>

### **HOW TO CITE THIS ARTICLE:**

Kusuma Venkatesh, C. M. Jayakeerthy. "Aortic Atherosclerosis as a Reflection of Coronary Atherosclerosis – An Autopsy Study with Historical Review". *Journal of Evidence Based Medicine and Healthcare*; Volume 1, Issue 8, October 15, 2014; Page: 937-949.

**ABSTRACT:** Atherosclerosis is a disease which is worldwide and a major cause of morbidity and mortality. In the western world half of all deaths are attributable to this vascular disease. Studies have shown that the presence of severe atherosclerosis of thoracic aorta is associated with high mortality rate and strong predictor of coronary artery disease. Autopsy study which combines morphological details with case history collected especially regarding the risk factors for atherosclerosis can be a good method in understanding the disease process. Hence this autopsy study was undertaken to study atherosclerosis of aorta and coronary arteries to assess 1) the frequency of occurrence, 2) severity at different age and sex, 3) relation to the established risk factors like hypertension, diabetes mellitus and tobacco smoking, 4) to analyze the role of socioeconomic status and diet and 5) to compare the disease process between aorta and coronary artery of the same person. The severity of coronary atherosclerosis was less than aortic atherosclerosis upto the 5<sup>th</sup> decade. There was no significant variation after 6<sup>th</sup> decade in the incidence and also the severity of atherosclerosis between Aorta and coronaries. In 88.8% persons belonging to 5<sup>th</sup> decade showed grade III and IV lesions in aorta as against 33.3% had this type lesions in coronaries. In the 6<sup>th</sup> decade all the cases had both coronary and aortic involvement. 75% cases had grade III, IV lesions in aortas with similar lesions in 66.7% of coronaries. In 7<sup>th</sup> and 8<sup>th</sup> decade, severe degree lesions were seen in both aorta and coronaries. There was clear increased frequency of atherosclerosis associated with the major risk factors, hypertension, diabetes mellitus, tobacco smoking and older age. A similar study conducted by the same author two decades later proved this fact. There is an alarming increase in the number of deaths due to coronary atherosclerosis in India and this number is expected to escalate rapidly in the next decade.

**KEYWORDS:** Aortic Atherosclerosis, Coronary Atherosclerosis, Autopsy Study.

**INTRODUCTION:** Atherosclerosis is a very common arterial disease affecting millions of people across the globe. In 1990 cardiovascular disease (CVD) accounted for 28% of all deaths and today it is known to be responsible for approximately 30% of deaths worldwide.<sup>1</sup> It is estimated that 33% of all the deaths will be the result of CVD by 2030!<sup>2</sup> India is suffering the highest loss in potentially productive years of life due to deaths from CVD affecting people between the age of 35 to 64 years (9.2 million years lost in 2000). By 2030 this loss is going to be as high as 17.9 million years, which will be 9.4 times greater than the corresponding loss in the USA!<sup>3</sup>

Atherosclerosis has been defined by the WHO as "a variable combination of changes in the intima of arteries consisting of focal accumulations of lipids, complex carbohydrates, blood

# ORIGINAL ARTICLE

---

products, fibrous tissue and calcium deposits associated with medial changes".<sup>4</sup> Especially when the deaths are increasing in the younger individuals it is indeed necessary to understand the common denominator for 'heart attack' and 'stroke' which is atherosclerosis. There are volumes written about this disease and lot of research done but still it remains mysterious. Many theories made rounds regarding the pathogenesis like the 'insudation theory', 'encrustation theory', 'monoclonal hypothesis and 'response to injury hypothesis' and now a unifying hypothesis. People are still asking which good cholesterol is and which is bad one for heart?

Study of atherosclerosis, its different stages and also in different age groups is difficult to do in the living population. Most of the studies to understand the pathogenesis have been done in experimental animals. Autopsy study which combines morphological details with case history collected especially regarding the risk factors for atherosclerosis can be a good method in understanding the disease process.

It is a disease of medium sized and large arteries known to damage aorta and its main branches along with the coronaries, cerebral arteries and renal arteries. Hence this autopsy study was undertaken with objectives to study atherosclerosis of aorta and coronary arteries to assess 1) the frequency of occurrence, 2) severity with age and sex, 3) relation to the risk factors like hypertension, diabetes mellitus and tobacco smoking, 4) to analyze the role of socioeconomic status and diet and 5) to compare the disease process between aorta and coronary artery of the same person. A similar study was conducted by the same author to find out the changing trends of coronary artery disease after two decades in 120 autopsy cases. The recent study revealed a rise of the disease that too amongst the younger people which is alarming.

**MATERIALS AND METHODS:** Totally 60 hearts with aorta up to the iliac bifurcations were studied. The material for the study was obtained from 58 medico-legal and 2 clinical autopsies conducted during the period between Feb. 1991 to Jan 1992 in Bangalore Medical College, Bangalore. Therefore this study itself is a historical review.

The hearts were collected from the department of Forensic medicine after written consent for each case. Unidentified cases severely decomposed and mutilated bodies were excluded. The socioeconomic status, dietary habits, history of past and present illness, diabetes mellitus, habits like smoking and alcoholism were obtained from the closest relative of the deceased and the autopsy protocols.

The external morphology of heart, weight and patency of coronary ostia were checked. Hearts were dissected by Virchow's method. The aorta was cut along the anterior surface after removing the excess of adventitial fat. Ventricular wall thickness was measured; any other abnormalities were recorded.

The quantitative assessment of the atherosclerotic lesions of aorta was done by using the method of Gore and Tejada.<sup>5</sup> By gross inspection, systematic observation and estimation of percentage of intimal surface involved was assessed. Quantitatively the disease involvement was categorized into five groups as follows:

# ORIGINAL ARTICLE

Group	Grade	Proportion of the intimal involvement
0	Negligible	less than 5% involved (less than 1/20 th area)
A	Minimal (+)	6 – 15% (< 1/6 th area)
B	Mild (++)	16 – 33% (< 1/3 <sup>rd</sup> area)
C	Moderate (+++)	34- 50% (<1/2 area)
D	Severe (++++)	More than 50% (> ½ area)

Each aorta was painted with Sudan III and diagrammatic representation to record the intimal involvement with different types of lesions was made and photos were taken. A minimum of two bits from the arch, thoracic, abdominal aorta and from iliac branches were processed routinely after fixation with Formaline. For both aorta and coronary artery bits, routine haematoxylin and eosin staining along with special stains like Verhoeff and Van Gieson stain to demonstrate elastic tissue, sudan III for fat, Alcian blue to demonstrate acid mucopolysaccharides, PAS for Fibrin and Alizarin red for calcium were used.

Aortic and coronary atherosclerotic lesions were classified according to the WHO study under four grades, namely 1) fatty streaks, 2) fibrous plaques, 3) atheromatous plaques and 4) complicated atheroma.<sup>5</sup> Complicated plaques had one or more additions in the form of ulceration, haemorrhage, thrombosis and calcification. 3 major coronaries, anterior descending branch of left coronary, circumflex branch and right coronary artery were studied by making serial sections along their entire course at an interval of 3 mm. Two sections were taken from the proximal and distal segment of each artery. The severity and extent of atherosclerosis of aorta were studied. The degree of stenosis of coronary arteries was graded by White, Edwards and Dry method<sup>6</sup> which depends on the amount of luminal occlusion. (Fig. 10)

**RESULTS:** 57(95%) out of 60 aortas showed atherosclerotic lesions of varying extent and severity. 68.3% was the frequency found for coronary atherosclerosis. There were 45 males and 15 females in the ratio of 3:1 (table I) and males showed more involvement by the disease by having aortic lesions in 44 out of 45 males (97.8%) and 37/45 (82.2%) having coronary atherosclerosis. On the other hand, of the 15 females studied 13 (86%) showed aortic lesions and 4 (26%) had coronary disease. The age related severity of lesions was also lesser in females with grade I lesions were seen in 86% of aortas. Table II, shows the results of grouping of the aortic lesions based on Gore and Tejada as 0, A, B, C and D as per the extent of intimal surface involvement.<sup>7</sup>

All cases in the 1<sup>st</sup> decade of life were free from aortic and coronary atherosclerosis. In the 2<sup>nd</sup> decade, all of them showed grade I (fatty streaks) aortic lesions and 25% showed coronary atherosclerosis. All the cases in 3<sup>rd</sup> decade showed aortic lesions, of which 90.9% showed grade I aortic lesions as against 45.5% of grade I coronary lesions. In the 4<sup>th</sup> decade 50% cases showed grade I lesions in aorta and in the remaining grade II, III and IV lesions were seen in equal number of cases. (Table III) The coronary disease in this decade involved in 75% of cases and grade III, IV lesions were seen in only 16%. In 88.8% persons belonging to 5<sup>th</sup> decade showed grade III and IV lesions in aorta as against 33.3% had these type of lesions in coronaries.

## ORIGINAL ARTICLE

In the 6<sup>th</sup> decade all the cases had both coronary and aortic involvement. 75% cases had grade III, IV lesions in aortas with similar lesions in 66.7% of coronaries. In 7<sup>th</sup> and 8<sup>th</sup> decade, severe degree lesions were seen in both aorta and coronaries.

TABLE 1: Analysis of Age and Sex distribution of cases.

Age Group	Males	Females	Total
New born to 1 year	1	1	2
1 – 10 years	0	1	1
11 – 20 years	3	5	8
21 – 30 years	6	5	11
31 – 40 years	10	2	12
41 – 50 years	8	1	9
51 – 60 years	12	0	12
61 – 70 years	2	0	2
71 and above	3	0	3
<b>Total</b>	<b>45</b>	<b>15</b>	<b>60</b>

Table 1

Table 2: Shows GROUPING of atherosclerotic lesions of Aorta depending on the extent of intimal surface involvement.

Age Group	Sex	Normal	0	A	B	C	D	Total
New born to 12 months	M	1	0	0	0	0	0	1
	F	1	0	0	0	0	0	1
1 – 10 yrs	M	0	0	0	0	0	0	0
	F	1	0	0	0	0	0	1
11 – 20 yrs	M	0	1	1	1	0	0	3
	F	0	1	3	1	0	0	5
21 – 30 yrs	M	0	0	6	0	0	0	6
	F	0	1	2	2	0	0	5
31 – 40 yrs	M	0	0	2	2	4	2	10
	F	0	0	0	1	1	0	2
41 – 50 yrs	M	0	0	0	0	6	2	8
	F	0	0	1	0	0	0	1
51 – 60 yrs	M	0	0	0	1	2	9	12
	F	0	0	0	0	0	0	0
61 – 70 yrs	M	0	0	0	0	0	2	2
	F	0	0	0	0	0	0	0
71 – 80 yrs	M	0	0	0	0	1	1	2
	F	0	0	0	0	0	0	0

# ORIGINAL ARTICLE

81 – 90 yrs	M	0	0	0	0	0	1	1
	F	0	0	0	0	0	0	0
<b>TOTAL</b>		<b>3</b>	<b>3</b>	<b>15</b>	<b>8</b>	<b>14</b>	<b>17</b>	<b>60</b>

Table – II

Table 3: GRADING OF THE AORTIC ATHEROSCLEROSIS.

Age Group	Sex	Normal	Grade				Total
			I	II	III	IV	
New born to 12 months	M	1	0	0	0	0	1
	F	1	0	0	0	0	1
1 – 10 yrs	M	0	0	0	0	0	0
	F	1	0	0	0	0	1
11 – 20 yrs	M	0	3	0	0	0	3
	F	0	5	0	0	0	5
21 – 30 yrs	M	0	6	0	0	0	6
	F	0	4	0	1	0	5
31 – 40 yrs	M	0	5	1	2	2	10
	F	0	1	1	0	0	2
41 – 50 yrs	M	0	0	0	6	2	8
	F	0	1	0	0	0	1
51 – 60 yrs	M	0	0	2	1	9	12
	F	0	0	0	0	0	0
61 – 70 yrs	M	0	0	0	0	2	2
	F	0	0	0	0	0	0
71 – 80 yrs	M	0	0	0	0	2	2
	F	0	0	0	0	0	0
81 – 90 yrs	M	0	0	0	0	1	1
	F	0	0	0	0	0	0
<b>TOTAL</b>		<b>3</b>	<b>25</b>	<b>4</b>	<b>10</b>	<b>18</b>	<b>60</b>

Table 3

Fatty steaks (fig. 1) were superficial, yellow or grey yellowish intimal lesions which could be selectively stained by fat stains. This type of lesion was common in 2<sup>nd</sup>, 3<sup>rd</sup> and 4<sup>th</sup> decade, present in 94% of people in that age group. These begin as minute yellow spots and then coalesce into elongated lesions 1 cm or more. Microscopically showed deposits of foamy macrophages and no extracellular ground substance. (Fig. 3, 4)

# ORIGINAL ARTICLE

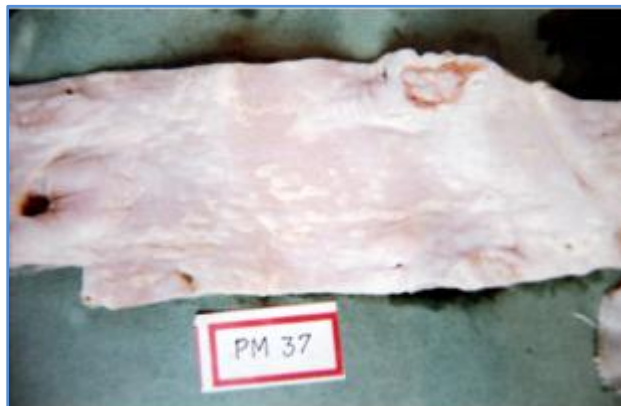
Table 4: INCIDENCE OF VARIOUS TYPE OF ATHEROSCLEROTIC LESIONS OF AORTA.

Age group	Total No. of cases	Fatty streaks	Fibrous plaques	Atheroma	Complicated plaques
New born - 10 years	3	0	0	0	0
11 – 20 yrs	8	8	1	1	0
21 – 30 yrs	11	11	6	1	0
31 – 40 yrs	12	10	5	4	2
41 – 50 yrs	9	7	5	8	2
51 – 60 yrs	12	9	6	10	9
61 – 70 yrs	2	0	1	2	2
71 – 80 yrs	2	1	1	2	2
81 – 90 yrs	1	0	0	0	1
<b>TOTAL</b>	<b>60</b>	<b>46</b>	<b>25</b>	<b>28</b>	<b>18</b>

Table 4



**Fig. 1: Aorta showing fatty streaks which are linear intimal lesions**



**Fig. 2: Aorta showing slightly elevated fatty streaks and fibrous plaques**

## ORIGINAL ARTICLE

Fibrous plaques (Fig. 2) were slightly elevated intimal thickenings, were firm and grey white and formed major type of lesion in 3<sup>rd</sup> and 4<sup>th</sup> decades. Microscopically they had less number of foam cells with more smooth muscle cells and fibroblasts.

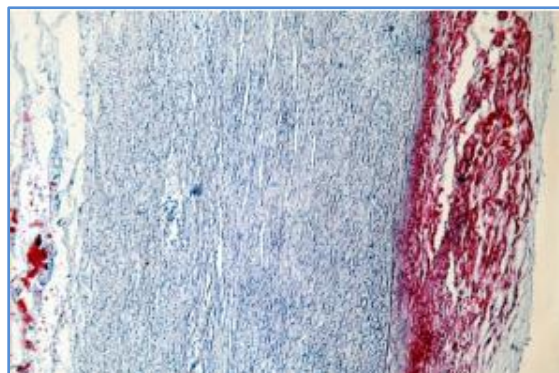
Atheromatous plaques (Fig. 5) were elevated grey white lesions with fibrous cap and soft porridge like (atheros means porridge in greek) centre. They were classically more around the orifices of the branches. Microscopically such lesions showed foam cells, lymphocytes, smooth muscle cells and cholesterol clefts representing extracellular lipid (Fig. 6). Neovascularisation was noted in a few lesions. 73.6% cases were having this type of lesion between 4<sup>th</sup> and 9<sup>th</sup> decade only 10% cases showed atheroma below the age of 30 years.

Complicated plaques (Fig. 6, 7 and 8) were seen from 4<sup>th</sup> decade onwards. Ulceration (Fig. 7, 8) was seen in 16(26.6%) cases, 8.3% showed haemorrhage, thrombosis seen in 6.6% and calcification (Fig. 7 and 8) was noted in 28.8%.

Fatty streaks were common in the arch and thoracic aorta and fibrous plaques and atheromas were more in the abdominal aorta. 7<sup>th</sup> decade onwards all were having complicated plaques. One 85 year old man had such severe calcification of the lesions (Fig. 8), the aorta on opening looked like thick bark of wood (hollow branch) and made crackling sound! This gentleman died of traffic accident not because of heart attack.



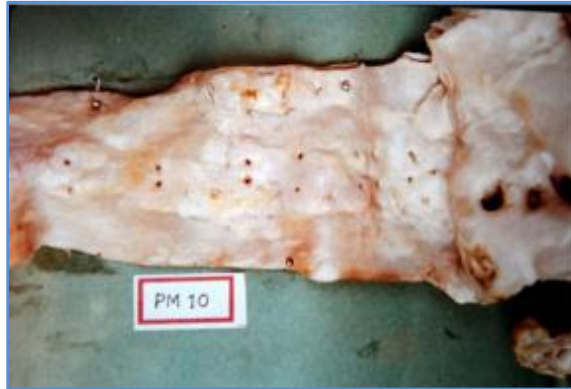
**Fig. 3: Fatty streak in aorta showing thickening of the intima and infiltration by foamy cells. Verhoeff Vangieson stain. X 400.**



**Fig. 4: Fatty streak in aorta, showing sudan positive deposits in the intima. Sudan IV stain x400**

## ORIGINAL ARTICLE

---



**Fig. 5: Aorta showing atheromas prominent around the opening of the branches**



**Fig. 6: Aorta showing haemorrhagic atheromatous plaque (arrow) and ulcerated Atheromatous plaques**



**Fig. 7: Aorta of an 85 years old man showing intima completely involved by complicated lesions which are calcified with wood bark appearance**



## ORIGINAL ARTICLE

---



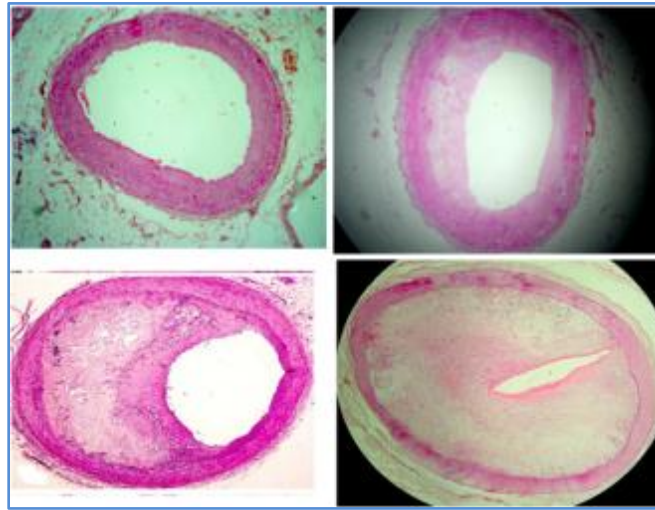
**Fig. 8: Aorta showing extensive involvement of the intimal surface by multiple complicated atheromatous plaques through out**



**Fig. 9: Heart showing atherosclerotic lesions with stenosed coronary ostia and orifices of the major arterial branches**

The coronary ostia and the orifices of arterial branches were more frequently involved by atherosclerotic plaques, causing stenosis (fig. 9). 41 out of 60 cases showed coronary atherosclerosis (68.3%) and 19 cases did not show coronary atherosclerosis (31.7%). Out of the 41 cases analysis of segmental involvement revealed single artery involvement in 14.6%, involvement of 2 arteries in 26.8% and involvement of all the 3 coronary arteries in 58.5%. Anterior descending branch of left coronary artery was the most commonly involved one. 92.7% showed atherosclerosis of the coronary artery and the severity of the lesions in vessels decreased towards the distal segments. Complicated lesions (Grade-IV) were seen mainly as calcified atheromas in 43.9%, followed by haemorrhage and thrombus formation. (Fig. 10)

Analysis of social classes (WHO) showed coronary disease in 75% of class A, 72.7% in class B and 60.9% in class C. Analysis of nutritional status revealed that 92.3% of obese persons, 64.9% of the averagely nourished persons and 50% poorly nourished had coronary atherosclerosis. Obese persons had more severe degree aortic atherosclerosis. Analysis of the diet showed 75.5% of non-vegetarians and 46.7% of vegetarians had coronary atherosclerosis.



**Fig. 10: Grade I, II, III and IV stenosis of coronary arteries Depending on the increasing luminal occlusion. H and E.stain x40**

The data presented with respect to the following risk factors includes 57 subjects after excluding two newborns and one child. Out of 57 subjects 34 were smokers and remaining 23 were non-smokers. 31 smokers (91.2%) and 10 non-smokers (43.5%) showed coronary atherosclerosis. 84% alcoholics and 62.5% non-alcoholics had coronary atherosclerosis. 91.6% of hypertensive and 62.5% of non-hypertensive had coronary atherosclerosis. 83.3% of diabetic and 66.6% of non-diabetics had coronary atherosclerosis. Hence this study revealed a higher incidence of coronary atherosclerosis in all the persons with risk factors.

The severity of coronary atherosclerosis was less than aortic atherosclerosis up to the 5<sup>th</sup> decade. There was no significant variation after 6<sup>th</sup> decade in frequency and also the severity of atherosclerosis between Aorta and coronaries. The differences in severity of aortic and coronary atherosclerosis between males and females were apparent from the 2<sup>nd</sup> decade of life and were especially marked up to the 5<sup>th</sup> decade. The extent and severity of the disease was less in females. 26.6% of the females showed coronary atherosclerosis as compared to 82.2% incidence in males.

**DISCUSSION:** Atherosclerosis does not belong to our time alone although there is no clear account of the process and its clinical manifestation before the beginning of the 18<sup>th</sup> century. Fallopius (1575) described degeneration of arteries into bone; and William Cowper noted that the passage of blood is impeded in the thickened arteries. A striking picture of aorta severely affected by atherosclerosis appears in a posthumous edition of J. J. Wepfer's observations called 'The Medico-practical deaffectibus capitis internis et externis' published in 1927. The diseased vessel which he described as containing 'bone like plugs throughout' and having its internal coat ruptured, ulcerated and rotten. Unfortunately such lesions were found in his blood vessels when the necropsy was performed on the author himself, who succumbed to the cardio vascular disease in 1895, thirty two years prior to the publication of his work.<sup>8</sup>

## ORIGINAL ARTICLE

---

The story of coronary heart disease may be said to begin with William Heber den's vivid picture of what he called 'angina pectoris' from the sensation of strangling in the chest (1768). John-Hunter (1776), examining the heart of a patient who had angina pectoris found the two coronaries which were like pieces of bone, but the connection between the attacks of pain and the condition of the coronaries was not considered. The credit for first defining a direct causal relationship between coronary artery lesions and symptoms of myocardial ischaemia is given to Edward Jenner who in 1799 correlated the original symptoms of his mentor, John Hunter to coronary artery disease. When Hunter died in 1793, his coronary arteries were found to be severely diseased and his aorta was described in the following terms." The internal membrane of this part of the vessel had lost its natural polish and was studded over with opaque white spots raised higher than the general surface". Allen Burns (Disease of the heart, 1809) produced pain in the leg by obstructing the circulation and suggested that this might also be the mechanism causing the pain in heart.<sup>9</sup>

In India, Mathur(1961)<sup>10</sup> and Bhargava(1975)<sup>11</sup> and Irwin Samuel (1961)<sup>12</sup> found that atherosclerotic lesions in the aorta in the first decade of life itself and this constituted the best evidence for the contention that atherosclerosis is a not a disease of senescence. Subramanian from Madras (1967),<sup>13</sup> noted that aortic atherosclerosis first commenced in the second decade of life, extent and the severity of the lesions, was gradual in 2<sup>nd</sup> and 3<sup>rd</sup> decades, but rapid thereafter. The peak incidence of the fatty streaks in the aorta was seen in the 3<sup>rd</sup> decade following which there was a decline. Mathur<sup>10</sup> observed that this decline could be due to their conversion into fibrous plaques and not necessarily due to their regression. Tejada and Gore (1958)<sup>14</sup> reported the occurrence of complicated lesions in the aorta in the 3<sup>rd</sup> decade in whites and in the 5<sup>th</sup> decade in Negroes for the first time.

In the present series they were seen first in the 4<sup>th</sup> decade and their incidence increased with age. All the studies show that aortic atherosclerosis is mostly concentrated on the posterior surface of the aortic intima. In this study raised lesions and complicated lesions showing calcification, ulceration and haemorrhage were found with greatest frequency in of the abdominal aorta. These observations correlate with those of Tejada et al, (1968)<sup>15</sup> and Hill (1961).<sup>16</sup> The cause for the predilection of these sites for the development of atherosclerotic lesions may be endothelial injury caused by the turbulence of blood flow, as shown by Glagov et.al.<sup>17</sup> Thus the appearance of the fatty streaks, fibrous plaques and complicated lesions in successive decades suggests a sequential evolution. With regard to social classes<sup>18</sup> clear increased frequency was found in higher class which was also true with those who were well nourished and obese and which was common to all the studies.

Clinically transesophageal echocardiography (TEE), a technique used to study and assess aortic atherosclerosis by Acarturk.et.al in suspected cases of coronary artery disease, showed a clear cut correlation between atherosclerosis of aorta and coronary arteries.<sup>19</sup> Coronary atherosclerotic lesions are presently examined and categorized according to American Heart Association classification 1995.

There is significant co-relation between the severity of coronary atherosclerosis and aortic atherosclerosis when taken with increased age, male sex and other risk factors into consideration.

# ORIGINAL ARTICLE

---

This Study showed strong positive correlation of atherosclerosis with major risk factors highlighting the importance of screening programmes.

I acknowledge with gratitude my teacher and guide, Professor Dr. C. M. Jayakeerthy who is no more and dedicate this article to him. I thank Dr. L. Thirunavukkarasu the former Professor and HOD of the Forensic Medicine Dept., Bangalore Medical College, Bangalore. I am thankful to the 60 deceased honorable people whom I did not know personally but studied their hearts after the postmortems.

## REFERENCES:

1. Gaziano T.A., Gaziano J.M., Epidimeology of cardiovascular disease Fauci A.S., Braunwald E., Kasper D.L., Hauser S.L., Longo D.L., Jameson J.L., Loscolzo J. eds, Harrisson's principle of internal medicine, 18<sup>th</sup> edition, Mc Graw Hill Medical, 2012; p.1811-1816.
2. Libby. P The pathogenesis, prevention and treatment of atherosclerosis.In: Fauci A.S., Braunwald E., Kasper D.L., Hauser S.L., Longo D.L., Jameson J.L., Loscolzo J.eds, Harrisson's principle of internal medicine, 18<sup>th</sup> edition, Mc Graw Hill Medical, 2012; p.1983 - 1991.
3. Epidimeology of Chronic Non-Communicable Diseases and Conditions, In: Park. k, Park's text book of Preventive and Social Medicine 21<sup>st</sup> edition, M/s Banarsidas Bhanot, 2011; p.336-369.
4. WHO: study group of the classification of atherosclerotic lesions.no. 143, 1958.
5. Gore, I., et al: The quantitative appraisal of atherosclerosis. Am. J. Path. 1957; 33: 875-885
6. White N.K., Edwards J E., Dry T J., the Relationship of the Degree of Coronary Atherosclerosis with Age, in Men circulation. 1950; 1, 645-654.
7. Tejada C and Gore I. Comparison of atherosclerosis in Gutemala city and New -Orleans. Am J Path. 1957; 33: 887.
8. Woolf. N. Pathology of atherosclerosis by Neville.Woolf. Butterworths and Co. Ltd. 1982.
9. Groszek and Grundy S.M.: The possible role of arterial micro circulation in the pathogenesis of atherosclerosis. J. Chron. Dis.1980; 33: 679.
10. Mathur K.S. and V. Kumar: The natural history of Atherosclerosis. The aortic lesions as seen in 500 autopsies at Agra (India).ICMR seminar on atherosclerosis and IHD. 1961; p.113-121.
11. Bhargava MK, Bhargava SV. Coronary atherosclerosis in North Karnataka. Indian J Path Micr. 1975; 18: 65-79.
12. Irwin Samuel, et al: Postmortem study on the incidence of atherosclerosis in the aorta and the coronary vessels. ICMR seminar on atherosclerosis and Ischemic Heart Disease. 1961; 105.
13. Subramaniyan. R., et al: Incidence of atherosclerotic lesions at Madras, South India. Brit. Heart J.1967; 29: 337.
14. Tejada.C., et al; Comparative severity of atherosclerosis in Costa Rica., Gautemala and New Orleans Circulation.1958; p. 92.
15. Tejada. C., et al: Distribution of aortic and coronary atherosclerosis by geographic location, race and sex. Lab. Invest.1968; 18: 509 – 526.

# ORIGINAL ARTICLE

---

16. Hill, K.R., et al: Atherosclerosis: Results of a pilot survey in the North London area. Brit. Med. J. 1961; 1: 1190.
17. Glagov, S, et al: Atherosclerosis of human aorta and its coronary and renal arteries. Arch of path.1961; 72: 558-571.
18. WHO: Chronicle: Social class and arteriosclerotic heart disease, 1969; 23: 11, p.532.
19. Acarturk E. Aortic atherosclerosis is a marker for significant coronary artery disease. Jpn heart J.Nov.1999.p.775 – 781.

## **AUTHORS:**

1. Kusuma Venkatesh
2. C. M. Jayakeerthy

## **PARTICULARS OF CONTRIBUTORS:**

1. Professor, Department of Pathology, KIMS, Bangalore.
2. Former Professor and HOD, Department of Pathology, Bangalore Medical College, Bangalore.

## **NAME ADDRESS EMAIL ID OF THE CORRESPONDING AUTHOR:**

Dr. Kusuma Venkatesh,  
Professor of Pathology,  
Kempegowda Institute of Medical Sciences,  
BSK IIInd Stage, Bangalore.  
E-mail: drkusuma\_kims@yahoo.co.in

Date of Submission: 25/09/2014.  
Date of Peer Review: 26/09/2014.  
Date of Acceptance: 29/09/2014.  
Date of Publishing: 07/10/2014.