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Occurrence of Intimal Thickening in Aging Ascending Aorta as a Most Initial Marker of Atherosclerotic Changes in Cardiovascular Diseases

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ABSTRACT

Introduction: Atherosclerotic Changes are the most common causes of cardiovascular diseases. So it's always been a matter of discussion that whether Intimal Thickening (ITN) has its relation to age or preexisting lipid mass or it's biased to any specific part of ascending aorta.

Methods: We used 120 autopsied Ascending Aorta and instrumented histological procedure followed by H&E staining.

Results & Discussion: Accumulation of SMCs in Intima of Ascending Aorta (AA) increase with advancing Age and it happens equally in all segments of AA. This is in coinciding with previous workers.

Conclusion: Though ITN is the most initial stage of Atherosclerotic change but it's a very important indicator for the same. It increases uniformly with Age.

Keywords: Atherosclerotic Changes, Intimal Thickening, Ascending Aorta, Smooth Muscle Cells, Aging.

INTRODUCTION

Cardiovascular diseases are the main causes of morbidity, mortality, and disability in today's world. This cardiovascular disease infestation is occurring despite unequalled advances in the diagnosis and treatment of these conditions. This situation is only expected to worsen because the world population is aging. Intimal thickening meant for the accumulation of Smooth muscle Cells (SMC) in tunica Intima of Aorta; though is not considered as a form of atherosclerosis but it may be present as initial evidence of the same. Most of the aortic adult human lesions arise as preexisting intimal masses. for this statement comes in part from the studies by Kim et al, who showed that atherosclerotic lesions produced in coronary arteries of hypercholesterolemic swine arise almost. Moreover, the distribution of these normal, developmental intimal masses in children can be correlated with the distribution of characteristic lesions seen in adult humans. (Velican D et al). The cause of fat collection subjacent to early little, exceptionally central, prior intimal masses may clarify the following conundrum. Unfortunately, there are not many articles on the advancement of right on time intimal cell masses in people, and none of these explain their exact obsessive systems of advancement. Another explanation behind considering the job of the intima in offering ascend to clinically huge sores originates from our perception that most of disintegrations happen over regions of intimal thickening, with negligible or no proof of a lipid center. The main objective of the study was to see the occurrence of Intimal Thickening in Ascending Aorta with aging and whether any specific

segment of Ascending Aorta was more prone for the Intimal Thickening.

METHODS

We procured 120 Ascending Aorta samples of autopsied and cadaveric bodies from the Department of Forensic Medicine and Department of Anatomy of SMIMER Medical College, Surat. Cases without any visible diseases like cardiovascular, diabetes, epilepsy or hypertensive were only included for the study. We divided them into three groups namely Group I, Group II and Group III which consist of age between 19 to 44, 45 to 64 and 65 years and above respectively. Normal histological procedure was followed and tissues were stained with H&E stains. Slides were then microphotographed. Data were presented as absolute numbers and relative percentages. Cross tabulations, Frequencies and Descriptive analysis were done using Descriptive statistics using SPSS software.

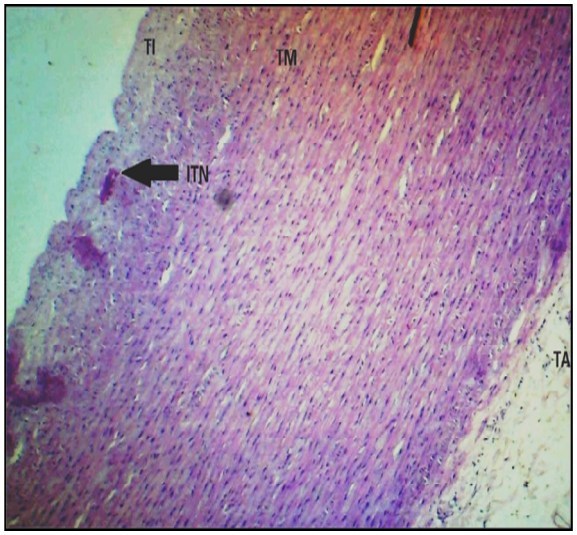

Figure1. Tunica Intima of Ascending Aorta showing Intimal Thickening (ITN) (Slide AA 16 D2)

RESULTS

There are 10 out of 50 cases (20%) shows intimal thickening in proximal Segment of Ascending Aorta of Age Group I. There are 18 cases out of 45 (40%) who show intimal thickening in Age Group II. There are 17 out of 25 cases (68.00%) of Age Group III shows intimal thickening in their proximal segments. Age Group III shows high incidence of intimal thickening. There is significant difference (P< 0.0001) with presence of intimal thickening within groups. **(Table: 1)**

There are 14 out of 50 (28%) cases shows intimal thickening of middle segment of Ascending Aorta of Age Group I. 17 out of 45 cases (37.8%) shows intimal thickening of Age Group II . 19 out of 25 cases (76%) shows intimal thickening of Age Group III in their middle segment. Age Group III that comprise the cases from 65 years and above shows the highest incidence of intimal thickening in their middle segment. The presence is statically significant as the chi square test show the p value is very much less than 0.0001. As shown in (Table 2).

Table 1: Intimal Thickening of Proximal Segment of Ascending Aorta with Age Groups

Age Group	Intimal Thickening of Proximal Segment of Ascending Aorta		
	No (%)	Yes (%)	Total (%)
19-44 yrs	40 (80)	10 (20)	50 (100)
45-64 yrs	27 (60)	18 (40)	45 (100)
65 and Above	8 (32)	17 (68)	25 (100)
Total	75 (62.5)	45 (37.5)	120 (100)

P value = <0.001

Table.2: Intimal Thickening of Middle Segment of Ascending Aorta with Age Groups

Age Group	Intimal Thickening of Middle Segment of Ascending Aorta		
	No (%)	Yes (%)	Total (%)
19-44 yrs	36 (72)	14 (28)	50 (100)
45-64 yrs	28 (62.2)	17 (37.8)	45 (100)
65 and Above	6 (24)	19 (76)	25 (100)
Total	70 (58.3)	50 (41.7)	120 (100)

P value = <0.001

Table 3: Intimal Thickening of Distal Segment of Ascending Aorta with Age Groups

Age Group	Intimal Thickening of Distal Segment of Ascending Aorta		
	No (%)	Yes (%)	Total (%)
19-44 yrs	42 (84)	8 (16)	50 (100)
45-64 yrs	28 (62.2)	17 (37.8)	45 (100)
65 and Above	6 (24)	19 (76)	25 (100)
Total	76 (63.3)	44 (36.7)	120 (100)

P value = <0.001

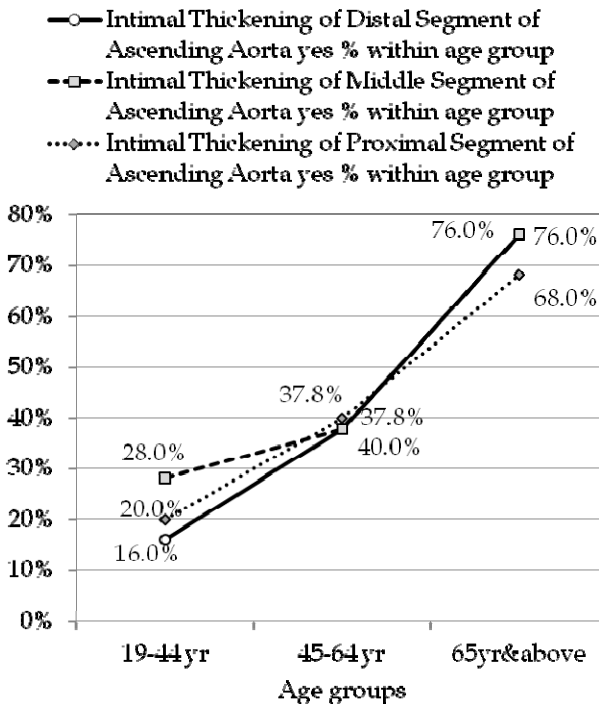

Figure 1: Occurrence of Intimal Thickening of each Age Group with Each Segment of the Ascending Aorta

There are 8 out of 50 (16%) cases shows intimal thickening of Distal segment of Ascending Aorta of Age Group I. 17 out of 45 cases (37.8%) shows intimal thickening of Age Group II . 19 out of 25 cases (76%) shows intimal thickening of Age Group III in their Distal segment. Age Group III that comprise the cases from 65 years and above shows the highest incidence of intimal thickening in their distal segment. Intimal thickening shows significant relation with Age Groups ($p<0.0001$) and increases with Age as shown in (**Table 3)**

So there is increase in occurrence of ITN in all Segments i.e. Proximal, Middle and Distal as age advances. If we compare all segments of AA in same age group we will find that there is no any significant difference in occurrence of ITN. Intimal Thickening occurs unanimously in all segments.

DISCUSSION

The normal accumulations of Smooth Muscle Cells (SMC) in intima in the absence of lipid or macrophage foam cells. In humans, ITN develops in specific arteries, such as coronary arteries and abdominal aorta, and is closely related to susceptibility to developing atherosclerosis. ITN, per se, is not a part of atherosclerosis, but can act as a depot for extracellular lipids in the earliest initial stages of atherosclerosis. In our study ITN increases in all segments (Proximal, Middle and Distal) of Ascending Aorta unexceptionally with age. It shows that SMCs from Tunica media constantly migrate to tunica intima as age advances. Our study confirms the study done by Movat HZ et al in 1958. As they suggested the structure of the diffuse intimal thickening increases with age. The focal arteriosclerotic lesions are superimposed on the diffuse intimal thickening. We are also agree with Ainsworth RW et al (1961) who reported young adults (aged between 24 and 36 years) showed a minor degree of intimal thickening in some of the their medium-sized arteries (main stem and large branches). It is showed by other large arteries in same pattern of occurrence as it shows in Ascending aorta and pulmonary trunk in our work. Like Jarvinen O et al (1996) reported that there was a correlation between age and thickness of the intima in the Superior Mesenteric Artery (r=0.33 mm), Coeliac Artery (r=0.25 mm), and Inferior mesenteric Artery (r= 0.27mm). When all three mesenteric Arteries included, the occurrence of intimal thickening of class II and III was only 6% in the group aged less than 50 years, while it was 23% among those who were above 50. There was no significant correlation between sex and intimal thickening. The diffuse thickening of the aorta is considered to be a normal developmental and aging process and not a part of arteriosclerosis although it undoubtedly represents one of the local factors which predisposes to the inception of arteriosclerosis. The focal arteriosclerotic lesions are superimposed on the diffuse intimal thickening. There was either duplication of the internal elastic lamina and an increase in collagen together with some smooth muscle in the intima or thickening by numerous thin elastic laminae, collagen fibres, and some smooth muscle cells.

CONCLUSION

An analysis for the occurrence of ITN in the wall of Ascending Aorta was done to ascertain the progress of Intimal Thickening with aging in autopsied 120 samples in three segments of Ascending Aorta. Normal histological process was followed and slides were stained in H&E stains. Results were found that Intimal Thickening increases as age advances. Moreover all the segments of aorta i.e. proximal, middle and distal were equally involved in accumulation of SMCs in their Intima. This statement is in coincides with previous works.

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