ORIGINAL RESEARCH

Analysis of age-related changes in the microanatomy of the ascending aorta and the pulmonary trunk

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ABSTRACT

Aim: Analysis of age-related changes in the microanatomy of the ascending aorta and the pulmonary trunk. **Materials and Methods:** Throughout the course of the autopsies that were performed in the anatomy department, the ascending aorta and the pulmonary trunks were removed. There were a total of 50 samples taken, four of which belonged to the fetal group (one of 32 weeks and another of 34 weeks). All of the samples were taken during the first six hours after the subject's death to prevent any autolytic alterations. **Results:** There was a lack of subendothelial connective tissue in group I (the fetal group) in both the AA and PT studies. It was found between the internal elastic lamina (IEL) and the endothelium beginning in group II and continuing forward. With increasing age, there was a continuous rise in the thickness of tunica intima of both the vessels, and statistically speaking, this was a significant finding (p 0.001). The average intimal thickness for AA ranged from 5.77 to 271.52 microns, with group I having 5.77 microns and group IV having 271.52 microns. When it came to PT, the average thickness grew from 2.11 to 150.25 microns from group I to group V. In the case of PT, the 20-40 range had the largest average complete wall thickness. It was 839µ. While there were variations in complete wall thickness that were statistically significant across various age groups (p 0.001), the comparison of people with ages less than or equal to 60 years and those older than 60 years did not indicate any statistical significance (p = 0.36). **Conclusion:** Both of the large vessels exhibited signs of degeneration as they became older; however, during the first decade of life, the alterations were far less severe in the PT than they were in the AA.

Keywords: Ascending aorta, Pulmonary trunk, Elastic fragmentation

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INTRODUCTION

The process of ageing is a complex one that has an effect on both the general functions and the structure of an organ. As a consequence, there are gradual changes in the dysfunction of the organ. [1,2] It is common knowledge that old age is a significant risk factor for a variety of ailments, particularly cardiovascular conditions (CVD). The prevalence of CVD is rising all across the globe. [1,3] The incidence of cardiovascular disease (CVD) grows with age and, as a rule, strikes those in their latter years. [4] As a consequence, this finding suggests that the changes associated with ageing contribute to cardiovascular malfunction and ultimately result in clinical illnesses. The heart, the heart valves, and the vascular system are the primary components of the cardiovascular system that experience age-related change structurally. The aorta is a form of elastic artery and it is the biggest artery in the vascular system of the human body. It is also the most important artery. It begins at the level of the aortic valve in the left ventricle of the heart and travels all the way down to the level where the common iliac arteries split off into two separate arteries. The aorta's primary job is to transport oxygen-rich blood from the heart to the rest of the body, which it does by carrying blood along its length. The aorta is often split into three primary portions that are distinguished by its position. These sections are known as the ascending aorta, the arch of the aorta, and the descending aorta, respectively. The descending aorta branches out to form the thoracic aorta and the abdominal aorta respectively. The tunica intima, tunica medium, and tunica adventitia are the three separate layers that make up the artery wall on a microscopic level. These layers go from the inner surface to the outside layer. Alterations in the structural and functional features of the wall of the human aorta are typically what happen as a result of ageing of the

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aorta. Many research have been conducted into the age-dependent changes that occur in the aorta, and it has been discovered that the influence might be on the diameter, length, and thickness of the aortic wall, as well as the tissue composition inside the aortic wall. According to the findings of these research, the lumen diameters increased gradually with increasing age. [5,6] There are structural changes in the tunica intima and media that are related with ageing. These changes may have an effect on the thickness of the aortic wall. According to the findings of many studies, these two layers have steadily grown thicker over the years. [5] Moreover, changes occurred in the histology of the aorta wall as people became older. There are three primary types of tissue that make up the artery wall: collagen, elastin, and smooth muscle cells. Both the number of these tissues as well as their arrangement inside each tunica of the aorta wall had undergone significant transformations as a result of the disease.[6] The lengthening, widening, and stiffening of the artery wall are all factors that contribute to elevated blood pressure. The purpose of this research is to describe and compare the changes in arterial wall thickness, elastic fragmentation, the number of collagen fibres, and smooth muscle cells that occur in the walls of ascending aorta (AA) and pulmonary trunk (PT) at various ages.

MATERIALS AND METHODS

Throughout the course of the autopsies that were performed in the anatomy department, the ascending aorta and the pulmonary trunks were removed. There were a total of 50 samples taken, four of which belonged to the fetal group (one of 32 weeks and another of 34 weeks). All of the samples were taken during the first six hours after the subject's death to prevent any autolytic alterations. Our research did not take into account deaths that occurred as a consequence of chest crush injuries, burns, or cardiopulmonary causes of death. The participants, who ranged in age from twenty to eighty, were split into five distinct groups.

METHODOLOGY

The anterior walls of the AA and PT, located about two centimetres distal to the aortic and pulmonary valves, respectively, was the source of the rectangular tissue fragments that were recovered. After that, the specimens were promptly fixed with 10% formalin after being moved there. At the histology lab of the Anatomy Department, regular histological processing[7] was performed twenty-four hours after the tissue was fixed. Using a rotary microtome, successive portions with a thickness of 6 microns were cut off. Once the sections had been incubated for an hour, the hematoxylin and eosin stains were applied to them. The elastic fibres, collagen fibres, and smooth muscle cells in the vessel wall were seen with the use of specific stains called Orcein Van Gieson's and Masson's trichrome. After being mounted, the sections were inspected using a binocular microscope. The degree of the fragmentation of elastic fibres, number of smooth muscle cells, and quantity of collagen were all factors that were taken into consideration. The thickness of the overall vessel wall as well as that of each tunic was measured.

The thickness of each tunic of the vessel wall was measured using an ocular micrometre at x10 objective magnification. After gathering data from each of the three areas, an average was calculated for all of the information. It was determined that the thickness of the tunica medium was equal to the amount of the vessel wall that extended from the internal elastic lamina to the outer limit of the muscle layer. At a magnification of x40, the fragmentation of elastic fibres as well as the number of smooth muscle cells per one hundred millimetre squared in the tunica media were counted. Elastic fragmentation was thought to have occurred when there was a localised fracture of elastic fibre in the tunica media of the vessel wall. According to Schlatman and Becker's [8] criteria, a total of 8 grades were given based on the severity of the elastic fragmentation. For the purposes of grading, a tiny field with the greatest possible fragmentation was evaluated. It was considered grade 1 if there were two foci of elastic fibre breakup in one field, grade 2 if there were three to ten foci of breakup, grade 3 if there were ten foci of breakup, grade 4 if one third to one half of the vessel wall showed fragmentation with loss of lamellar pattern, and grade 5 if the fibres underwent severe fragmentation throughout the vessel wall with complete loss of lamellar pattern.

Using a net micrometre that had a square grid of 10 mm by 10 mm, smooth muscle cells were counted. Using a high-power lens, the average number of insects per 100 mm2 was determined for five different sites that were chosen at random.

STATISTICAL DATA

The data were input into Microsoft Excel, and SPSS version 25.0 was used to do the analysis. The mean, standard deviation, minimum, and maximum values of quantitative variables were used to define the variables. In this study, the percentage distribution was used to characterise qualitative factors. Analysis of Variance was used to conduct comparisons of quantitative variables involving more than two different groups (ANOVA). Quantitative factors were examined using the independent sample t-test so that a comparison could be made between the two groups. The threshold of significance was determined to be a p-value of 0.05, which was used.

RESULTS

The specimens that were gathered for the purpose of microscopic examination were partitioned into five distinct age categories, as shown in Table 1.

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Table 1: Age distribution

Group No.	Age Groups	No. of specimen
I	Fetus	4
II	below 20	8
III	20-40	8
IV	40-60	10
V	60-80	20

There was a lack of subendothelial connective tissue in group I (the foetal group) in both the AA and PT studies. It was found between the internal elastic lamina (IEL) and the endothelium beginning in group II and continuing forward. With increasing age, there was a continuous rise in the thickness of tunica intima of both the vessels, and statistically speaking, this was a significant finding (p 0.001). The average intimal thickness for AA ranged from 5.77 to 271.52 microns, with group I having 5.77 microns and group IV having 271.52 microns. When it came to PT, the average thickness grew from 2.11 to 150.25 microns from group I to group V.

Table 2. Comparison of mean intimal thickness of ascending aorta and pulmonary trunk

Group No.	AA	PT
I	5.77	2.11
II	61.85	55.69
III	129.85	117.28
IV	231.29	133.69
V	271.52	150.25

Tunica media thickness in PT was consistently lower across all age categories when compared to AA. The ratio of the thickness of the tunica media of PT to that of AA was found to be between 0.5 and 0.8 in all age groups, with the exception of group I, in which it was approximately equal to 1.

In Group 1 of AA, the average thickness of the tunica adventitia was 278.69, which corresponds to a percentage of the whole wall thickness of 40%, but in PT, it was 169.98, which corresponds to a percentage of the full wall thickness of 36%. Following a period of forty years, there was a discernible decline in the amount of tunica adventitia present in both vessels. It fell to 14% in group IV of AA and dropped to 10% in group IV of PT correspondingly.

The 40-60 year time period had the biggest increase in the average complete wall thickness of AA. The average thickness throughout this group was 1898 microns. According to the results of Student's T-test, the mean complete wall thickness of persons younger than 60 years old was 1369, but the mean thickness of those older than 60 years was 1879. There was a discernible and statistically significant increase in thickness (p = 0.003).

In the case of PT, the 20-40 range had the largest average complete wall thickness. It was 839µ. While there were variations in complete wall thickness that were statistically significant across various age groups (p 0.001), the comparison of people with ages less than or equal to 60 years and those older than 60 years did not indicate any statistical significance (p = 0.36).

Table 3. Comparison of average full wall thickness of ascending aorta and pulmonary trunk

Group No.	AA	PT
I	569	501
II	1211	711
III	1577	839
IV	1898	803
V	1879	798

The internal elastic lamina (IEL) in groups I and II of AA was continuous. There was some infrequent splitting in group III, and in groups IV and V there was a repetition of the IEL. The PT demonstrated the fragmentation of IEL beginning with group II and continuing ahead. In the tunica media of groups I to II of AA, we found long elastic laminae that were parallel to one another and compactly organised. During the PT, Group I demonstrated densely packed laminar elastic fibres, while fragmentation of Grade 3 was seen beginning in the first decade and continuing afterwards. In AA, it wasn't until the sixth decade that anybody saw it. In AA, no evidence of grade 5 fragmentation was found at all, however in PT, grade 5 fragmentation was found in 20 out of 50 tissues.

As compared to the foetal group, the average number of smooth muscle cells in the ascending aorta was highest in the foetal group, however in the pulmonary trunk, the number of smooth muscle cells was higher in the 0-20 year age range than in the foetal group. After then, degradation of smooth muscle cells took place, but to a much lesser amount compared to when AA was present. It was discovered that PT had a greater muscle mass than AA in all age categories, with the exception of the foetal group.

DISCUSSION

In spite of the fact that a great number of researchers have looked into the microscopic structure of both AA and PT, the literature only has a limited number of reports of comparative studies that include both arteries and how they relate to age. Due to the fact that large artery stiffness is a factor in determining the risk of cardiovascular morbidity and mortality, as well as due to the fact that both arteries have a similar embryological origin, a comprehensive investigation of age-related alterations in both vessels was conducted.

Throughout the course of our research, we found that as participants' ages increased, there was a commensurate rise in the average thickness of the intima. Yet, after the first decade of life in both veins, sub endothelial connective tissue could be seen between the endothelium and the internal elastic lamina. In the foetal AA and PT, this connective tissue was not present. Investigations conducted by Lakatta [9] and Crawford [10] found the same thing: there was no subendothelial connective tissue in the ascending aorta of newborns. According to Crawford, in early infancy, there was a loose meshwork of connective tissue that separated the internal elastic lamina and the endothelium. As time went on, this loose meshwork of

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connective tissue expanded in both quantity and density. Ross et al. [11] shown that there was a discernible thickening of the intima in older children, and that there was typically some stretching, splitting, or tearing of the underlying internal elastic lamina over the course of many years. Even in our research, we found that occasional splitting of the internal elastic lamina in AA started occurring in the second decade and that replication began in the sixth decade, but in PT, the process of splitting the internal elastic lamina began in the first decade itself.

When the thickness of the tunica media of AA was compared to that of PT, it was found that the ratio (P/A) was within a range of 0.5 to 0.9 in all age groups, with the exception of the foetal group, where the P/A ratio varied between 0.94 and 0.98. This finding was based on the comparison of the two. Heath et al. [12] did a study in which they compared the thickness of the tunica media of the alveoli of normal persons to the thickness of the tunica media of those who had pulmonary hypertension. Those who had pulmonary hypertension had a thick media similar to that of the aorta, and their P/A ratio was approximately 1, similar to what was seen in the foetal group. When the thickness of the tunica medium of persons with ages more than or equal to 60 years was compared to that of those with ages less than 60 years, a statistically significant increase in AA was seen, but not in PT. According to Mackay et al.[13]'s research on pulmonary arteries, there was no discernible increase or decrease in the thickness of the tunica media with increasing age.

Mario Saldana and colleagues14 reported that in the last three months of prenatal life, the elastic shape of the pulmonary trunk and aorta were comparable to that seen in newborns. This was shown to be the case when they compared the foetal heart to an adult heart. Elastic fibres in a baby were long and straight or undulating, parallel to one another, and densely packed together, similar to how they were throughout the third trimester of intrauterine life. This investigation came to similar conclusions as the previous one. The fragmentation of elastic fibres began in AA during their third decade of life and advanced to higher grades by the time they reached their sixth decade. In contrast, the PT demonstrated significant degrees of elastic fragmentation from the very beginning of the first decade of life itself. Mario Saldana and his colleagues saw a lot of the same similarities. [14] In the research that was carried out by L.S. Foster [15] on the elastic fibres of the aorta, degenerative alterations were noticeable after the age of around 50 years.

Both the foetal aa and the foetal pt had an abundance of smooth muscle cells sandwiched between long, straight, and densely packed lamellar elastic fibres. A considerable decline in the number of smooth muscle cells occurred in AA as the patient's age increased. In contrast, the PT demonstrated a rise in the number of smooth muscle cells during the first decade of observation, followed by a small deterioration throughout subsequent decades. Foster observed a gradual reduction of smooth muscle along with fatty alterations and weakly stained dispersed nuclei in the aorta of older patients as the disease progressed. [15]

Cattell[16] found that there was a correlation between a rise in collagen content in the wall of AA and an increase in age across the span of 14–90 years. In our research, quantitative measurements of collagen were not possible; nonetheless, it was shown that tissues with higher grades of elastic fragmentation included a greater quantity of collagen.

The fraction of tunica adventitia in AA and PT exhibited a decreasing trend beginning in the fourth decade and continuing forward. In the research that Sankar Dayal Gupta and colleagues conducted, they found that the tunica adventitia had become thinner. [17]

In light of this, the current research discovered that the percentage of tunica medium increased up until the fourth decade of life, after which it began to decrease. This was the case despite the fact that both AA and PT showed a consistent rise in the amount of tunica intima. At every age, the tunica media of the pulmonary trunk was thinner in comparison to that of AA, and elastic fibres experienced significant degrees of fragmentation beginning in the very first decade. This is because, towards the end of the first month of life outside the uterus, there is a drop in pulmonary arterial pressure, which thereafter stays about the same for the remainder of a person's life. There was no statistically significant difference seen between age groups older than 60 years and those younger than 60 years with regard to the thickness of the tunica media of the PT. The percentage of tunica adventitia was found to have decreased beginning in the third decade in both of the vessels. On the other hand, the average thickness of the whole wall tended to become thicker as the centuries progressed.

It is possible that an increase in elastic fragmentation, collagen fibres occupying the intervals between fragmented elastic fibres, and atrophy and degeneration of smooth muscle cells contributed to the decrease in the proportion of tunica media and adventitia after the fourth and third decades, respectively. The vessels become more rigid as a direct consequence of these modifications. As a result, the burden placed on the heart increases because a greater systolic pressure is necessary to stretch an artery that is more rigid. A pressure pulse wave is generated when a bolus of blood is expelled into the aorta during systole. This wave moves along the wall at a velocity that is determined by the material's stiffness. Since the pulse wave velocity is higher in the stiffer aorta, the reflected wave returns quickly enough to add to the wave that is generated by the heart during systole, which results in an increase in systolic pressure and a decrease in diastolic pressure.

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CONCLUSION

Both AA and PT get more rigid as one gets older. Yet, compared to AA, the degenerative alterations that occur in PT beyond the first decade of life are far less severe. This can be explained by the fact that pulmonary circulation is a system that operates at a low pressure, whereas systemic circulation is a system that operates at a high pressure. Because of the high pressure within the vessel wall, elastic fibres in the ascending aorta are constantly stretched. This constant stretching eventually leads to the fragmentation of these fibres, which in turn leads to the transfer of mechanical load to collagen and the arterial stiffening that is characteristic of ageing.

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