ORIGINAL ARTICLE

A MORPHOLOGICAL STUDY OF CELIAC, SUPERIOR MESENTERIC AND INFERIOR MESENTERIC ARTERIES IN ATHEROSCLEROSIS

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Background: Depolymerisation of acid-mucopolysaccharides results in the loss of metachormasia of the ground substance followed by visible fibre crumbling, complete dissolution and replacement by lipid droplets and cholesterol. The objective of this study was to assess the distribution of different atherosclerotic lesions in celiac, superior mesenteric and inferior mesenteric arteries relation to age and sex. **Methods:** A prospective descriptive observation study was conducted at Mortuary of King Edward Medical University Lahore, and Department of Pathology Allama Iqbal Medical College Lahore. A total of 30 human autopsies were carried out. Celiac, Superior mesenteric and inferior mesenteric arteries were taken out and opened length-wise. One to four areas of tissue were taken from each artery for histological examination. Slides were prepared from each paraffin block. Sections were stained with haematoxylin and eosin. Special stains were performed on sections to display each component of atherosclerosis. **Results:** The fibrolipid plaques were seen in 6 cases. The complicated lesions were present in 5 cases. Of these, 4 showed ulceration and 1 showed thrombus formation. The calcified lesions were observed in 5 cases. The morphological changes in media and elastica were present in 4 cases. In superior mesenteric artery fatty streaks were present in 8 cases. The fibrolipid plaques were confirmed in 5 cases. Three cases showed ulceration, 1 case showed intimal vascularisation, haemorrhage, and thrombus formation. The calcified lesions were present in 2 cases. The morphological changes in media and elastica were seen in 4 cases in anterior mesenteric artery. Fatty streaks were present in 8 cases. The fibrolipid plaques were grossly observed in 6 cases. The complicated lesions were present in 4 cases; of these, 3 cases showed ulceration, and 1 showed intimal vascularisation and haemorrhage along with thrombus formation. The calcified lesions were present in 3 cases. The morphological changes in media and elastica were present in 4 cases. **Conclusion:** This data indicates the incidence of ischemic changes in abdominal viscera due to atherosclerotic narrowing.
Keywords: Celiac, Superior, Inferior, mesenteric, atherosclerosis, arteries

INTRODUCTION

Depolymerisation of acid-mucopolysaccharides involved in the plaque formation results in the loss of metachormasia of the ground substance. After that the visible fibres crumble and dissolve completely and it is replaced by lipid droplets and cholesterol.[1] In ulcerated atheroma extensive foma cell are formed that are connected by fibrin-mesh.[2] Intimal thickening causes hypoxia of mid-zone of media. This provides the stimulus for the in-growth of capillaries from the adventitial vessels into the thickened intima. Thrombosis may occur on an ulcerating atheroma.[3] In atherosclerosis fine granules of Calcium appear in the ground substance and the necrotic tissues at the marginal layer of ulcers. The relative attenuation of the media is due to the disintegration of the elastic fibre system in the inner layer of the medial coat.[4] The objective of this study was to assess the distribution of different atherosclerotic lesions in celiac, superior mesenteric and inferior mesenteric arteries relation to age and sex.

MATERIAL AND METHODS

A total of thirty human autopsies were carried out during this study. The autopsies were done in the Mortuary of the King Edward Medical College, Lahore. Celiac, superior mesenteric and inferior mesenteric arteries were taken out and opened lengthwise. One to four sections were taken from each Celiac, superior mesenteric and inferior mesenteric arteries for histological examination. Tissue processing was done. On the average 7–8 slides were prepared from each block by taking ribbons of tissues. The paraffin sections were stained using Haematoxylin and Eosin stain, Curtis's Picro-ponceau stain, Verhoeff's elastic tissue stain, Von Kossa's staining technique, periodic acid Schiff (PAS) reaction, Toludine blue stain and Peral's Prussian blue stain.

RESULTS

The fatty streaks were present in 7 of the 30 cases and they were distributed along the long axis of vessel wall. The fibrolipid plaques were present in 10 cases, and complicated lesions were seen in 2 cases. Out of these complicated cases 1 showed ulceration and 1 showed thrombus formation. The intimal vascularisation and haemorrhage were present in 2 cases. Number of the raised lesions was 1–2 in these cases. Size of the largest raised lesion was 3×3 mm. All raised lesions were distributed irregularly within 0.5 Cm of the ostia (Table-1).

Table-1: Atherosclerotic lesions in celiac artery in relation to age and sex (Gross findings) (n=30)

Age in years	Fatty Streaks		Fibrolipid Plaques		Complicated Lesions		Calcified Lesions	
	M	F	M	F	M	F	M	F
6–15	-	-	-	-	-	-	-	-
16–25	-	-	-	-	-	-	-	-
26–35	1	1	1	-	-	-	-	-
36–45	3	2	2	-	-	-	-	-
46–55	-	-	-	-	-	-	-	-
56–65	-	-	4	-	1	-	1	-
66–75	-	-	1	2	1	-	1	-
Total	4	3	8	2	2	-	2	-

The fatty streaks were present in 7 cases. On histological examination of the fatty streaks the foam cells along with increase of fluid was present in the intima. Lipid was present both intracellularly and extracellularly along with the connective tissue changes. The fibro-lipid plaques were seen in 6 of the 10 cases found on gross appearance. The fibro-lipid plaques showed fibrous degeneration and regeneration with mucoid changes. There was a metachromatic change and hyalinization in the atherosclerotic lesions (Figure-1).

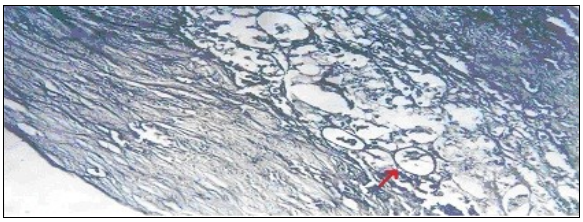

Figure-1: Photomicrograph of superior mesenteric artery showing well formed foan cells with regenerating fibrous tissue. H & E ×420

Number of foam cells was prominent and the number of fibrocytes was also increased. The fat was present in the firm of fatty pool and the heedle-shaped cholesterol crystal clefts were also demonstrated. Variable number of foam cells was present with the necrotic area at the base of the lesion (Figure-2). The complicated lesions were present in 5 cases; of these 4 showed ulceration and one showed thrombi formation. In ulcerated lesions the lipid contents were less in amount. Foam cells with fibrin was present abundantly. A lymphocytic reaction with granulation tissue were seen in the lesion. In cases showing intimal vascularisation and haemorrhage there was novascularisation in the intima. In addition to that the red blood cells and haemosiderin deposits were also present at the junction of media and atherosclerotic lesions. In atherosclerotic lesions showing thrombus formation the fibrin strands were present at the periphery and in between the platelet aggregates. The calcified lesions were observed in 3 cases. The calcified masses were deposited in degenerated debris and hyalinized collagen tissue in the intima. Deposits of calcium were particularly present around the necrotic areas, lipid pool and marginal layers of the ulcers in atherosclerotic lesions (Figure-2). The morphological changes in media and elastica were present in 4 cases. The medial coat was relatively attenuated below the sclerotic plaque and was one half or less of the thickness of the media in the adjacent part of the artery. The fibres on the inner third of media were severely degenerated.

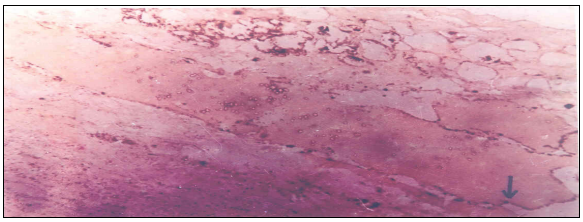

Figure-2: Photomicrograph of atherosclerotic lesion in celiac artery showing metachromatic change. Toludine Blue stain ×450

The fragmented internal elastic lamina was separated apart and was totally deficient over wise areas at the base of large plaques (Table-2).

Table-2: Atherosclerotic lesions in celiac artery in relation to age and sex (Microscopic findings) (n=30)

Age in years	Fatty Streaks		Fibrolipid Plaques		Complicated Lesions		Calcified Lesions	
	M	F	M	F	M	F	M	F
6–15	-	-	-	-	-	-	-	-
16–25	-	-	-	-	-	-	-	-
26–35	1	1	1	-	-	-	-	-
36–45	3	2	1	-	1	-	-	-
46–55	-	-	-	-	-	-	-	-
56–65	-	-	2	-	2	-	2	-
66–75	-	-	1	1	1	1	1	-
Total	4	3	5	1	4	1	3	-

The fatty streaks were present in 8 out of 30 cases. They were arranged along the long axis of the vessel wall. The fibrolipid plaques were seen in 6 cases. The complicated lesions were present in 3 cases; of these, 2 cases showed ulceration and 1 showed thrombus formation. No intimal vascularisation and haemorrhage was present in any of the cases. The calcified lesions were seen in 2 cases. The number of the raised lesions was 1–2 in all these cases. Size of the largest raised lesion was 3×4 mm and that of the smallest was 3×3 mm. The colour of fatty streaks was yellowish white. The raised lesions were distributed irregularly within 1 Cm of ostia in all cases (Table-3).

Table-3: Atherosclerotic lesions in superior mesenteric artery in relation to age and sex (Gross findings) (n=30)

Age in years	Fatty Streaks		Fibrolipid Plaques		Complicated Lesions		Calcified Lesions	
	M	F	M	F	M	F	M	F
6–15	-	-	-	-	-	-	-	-
16–25	-	-	-	-	-	-	-	-
26–35	1	1	1	-	1	-	-	-
36–45	3	3	-	3	-	-	-	-
46–55	-	-	1	-	1	-	1	-
56–65	-	-	1	-	1	-	1	-
66–75	-	-	-	-	-	-	-	-
Total	4	4	3	3	3	-	2	-

On microscopic examination the fatty streaks were present in 8 cases. The fibrolipid plaques grossly observed in 6 cases. The complicated lesions were present in 4 cases of these, 3 cases showed ulceration, and one showed intimal vasucularisation and haemorrhage along with thrombus formation. The calcified lesions were present in 3 cases. The morphological changes in media and elastics were present in 4 cases. The histological features in these atherosclerotic lesions were the same as mentioned previously (Table-4).

Table-4: Atherosclerotic lesions in superior mesenteric artery in relation to age and sex (Microscopic findings) (n=30)

Age in years	Fatty Streaks		Fibrolipid Plaques		Complicated Lesions		Calcified Lesions	
	M	F	M	F	M	F	M	F
6–15	-	-	-	-	-	-	-	-
16–25	-	-	-	-	-	-	-	-
26–35	1	1	1	-	1	-	-	-
36–45	3	3	-	2	-	1	-	-
46–55	-	-	1	-	1	-	1	-
56–65	-	-	1	-	1	-	1	-
66–75	-	-	-	-	-	-	-	-
Total	4	4	3	2	3	1	2	-

The fatty streaks were present lesions were present in 2 cases who showed ulceration. The calcified lesions were seen in 2 cases. The number of the raised lesions was 1–2 in each case. The size of the largest raised lesion was 4×3 mm and that of the smallest raised lesion was 3×3 mm. the raised lesions were distributed irregularly along the vassal within 1 Cm of the ostia (Table-5).

Table-5: Atherosclerotic lesions in inferior mesenteric artery in relation to age and sex (Gross findings) (n=30)

Age in years	Fatty Streaks		Fibrolipid Plaques		Complicated Lesions		Calcified Lesions	
	M	F	M	F	M	F	M	F
6–15	-	-	-	-	-	-	-	-
16–25	-	-	-	-	-	-	-	-
26–35	1	1	1	-	1	-	-	-
36–45	3	3	-	2	-	-	-	-
46–55	-	-	2	-	-	-	1	-
56–65	-	-	3	-	-	-	-	-
66–75	-	-	-	1	-	1	-	1
Total	4	4	6	3	1	1	1	1

The fatty streaks were present in 8 cases. The fibrolipid plaques grossly observed in 6 cases. The complicated lesions were present in 4 cases; of these, 3 cases showed ulceration, and one showed intimal vascularisation and haemorrhage along with thrombus formation. The calcified lesions were present in 3 cases. The morphological changes in media and elastics were present in 4 cases. The histological feature sin these atherosclerotic lesions were the same as mentioned previously (Table-6).

Table-6: Atherosclerotic lesions in inferior mesenteric artery in relation to age and sex (Microscopic findings) (n=30)

Age in years	Fatty Streaks		Fibrolipid Plaques		Complicated Lesions		Calcified Lesions	
	M	F	M	F	M	F	M	F
6–15	-	-	-	-	-	-	-	-
16–25	-	-	-	-	-	-	-	-
26–35	1	1	1	-	1	-	-	-
36–45	3	3	-	2	-	-	-	-
46–55	-	-	1	-	1	-	1	-
56–65	-	-	1	-	1	-	1	-
66–75	-	-	-	1	-	1	-	1
76–85	-	-	-	-	-	-	-	-
Total	4	4	3	3	3	1	2	1

DISCUSSION

On the light microscopy, the fatty streaks showed the presence of foam cells beneath the endothelial lining. There was increase of fluid in the ground substance. In addition to these changes, the connective tissue was arranged in the form of loose mesh with some fibrin depositions. It seems likely that lipoproteins are transported across intact endothelial cells by micro-pinocytosis.[6] Lipid was present both intra-cellularly and extra-cellularly. Foam cells are smooth muscle cells containing lipids.[5] Probably local adherence of the platelets at the endothelium releases Mitogenic platelets to the endothelium releases Mitogenic Platelet factors into the arterial wall and causes some intimal smooth muscle cells proliferation?[7] In fibro-liqid plaques both connective tissue and lipid changes were prominent. These changes were visible as mucoid swelling due to the presence of protein molecules and acid-mucopolysaccharides. In addition there was a metachromatic change in the ground substance along with hyalinisation. This change has previously been related to the increased amount of the ground substance.[2] Alteration in intrinsic composition and molecular size of proteoglycans occurs in atherosclerotic lesion.[8] The increase in the number of foam cells in fibrolipid plaques was probably due to increase in the smooth muscle cell proliferation and vacuolated forms.[9] In such vacuolated cells the lipid containing inclusions have been associated with the structural elements of smooth muscle cells. To Foam cells accumulation have been demonstrated in experimentally induced atherosclerosis. The number of fibrocytes is increased during plaque formation.[11] It is associated with increased formation of collagen and elastic fibres. These connective tissue components are probably derived from the proliferating smooth muscle cells in the intima. There was high concentration of fibrin in developing atherosclerotic lesion.[8] It was established that there is an association between accumulation of fibrin and binding of low density lipoproteins (LDL).[10,12] On the other hand it was proposed that the process of smooth muscle cell

proliferation is related to the tumour formation initiated by mutation. The lipids were seen in the form of fatty pool and needle-shaped cholesterol crystal clefts.[13,14] LDL is important to the initiation and probably the progression of atherosclerotic lesions.[15,16] In the ulcerated lesions the lipid contents were markedly less in amount. On the other hand foam cells were extensively present at the base and fibrin was seen intervening these cells.[2] The blood vessels were found in the intima. RBCs and haemosiderin deposits were present at the junction of media and atherosclerotic lesion. It was also explained that neo-vascularisation in the intima may lead to haemorrhage because they run the tissue that does not support them adequately.[4] In thrombus formation Platelet aggregation at the exposed sub endothelial tissue was seen. The fibrin strands were present at the periphery and in between the platelet aggregates. The collagen rich atherosclerotic lesion initiates thrombosis, because it exposes the blood to powerful platelet aggregating (collagen), and coagulation activating (traumatic surface and lipids) factors that are not found in normal vessel wall. Fibrinogen leads to the Platelet aggregation associated with release of vasoconstrictor, thromboxane A2. This hyper-coagulability of platelets again is associated with hyper-fibrinogenemia and thrombosis. Lack of PG12 due to endothelial injury may lead to thrombus formation, because PG12 is powerful anti-aggregating vasodilator.[12] Contrary to above mentioned observations it was described that Fibrous plaque is fibrinoid or organized thrombus.[4] This study was supported by the observations that calcified granules were presented around the degenerated debris and hyalinised collagen tissue in the intimate. They also observed that deposits of calcium were particularly present at the periphery of necrotic areas, lipid pool and marginal layer of ulcers in atherosclerosis.[17] The fibres on the inner third of media were severely degenerated. Internal elastic lamina was fragmented and was totally deficient over wide areas at the base of large plaques due to rigid pressure.

CONCLUSION

This study shows distributes of different atherosclerotic lesions in celiac, superior and inferior mesenteric arteries in relation to age and sex. This limited and basic data indicates the incidence of ischemic changes in abdominal viscera due to atherosclerotic narrowing.

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