Original Article

A Morphological Study Of Common Carotid Arteries in Atherosclerosis

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Objective: To review the pattern of atherosclerosis in common carotid arteries

Material & Methods: The pattern of atherosclerotic lesions was studied in common carotid arteries in 30 human autopsy cases. This study was conducted in the mortuary of King Edward Medical College, Lahore. Both the right and left carotids of all 30 cadavers were opened and reviewed both grossly and microscopically.

Results: The fatty streaks were present in 10 cases in the right common carotid artery and 10 cases in the left common carotid artery. The fibrolipid plaques were seen in 9 cases in the right common carotid artery and 9 cases in the left common carotid artery. The complicated lesions were present in 5 cases in the right common carotid artery and 6 cases in the left common carotid artery. In the right common carotid artery 4 cases showed ulceration and one case showed intimal vascularization and haemorrhage alongwith thrombus formation. In the left common carotid artery 4 cases showed ulceration and 2 showed intimal vascularization and haemorrhage alongwith thrombus formation. The calcified lesions were seen in 4 cases in the right common carotid artery and 5 cases in the left common carotid artery. The morphological changes in media and elastic were present in 6 cases in the right common carotid artery and 7 cases in left common carotid artery.

Conclusion: This study shows that raised atherosclerotic lesions are reasonably common in our population and stresses the need to conduct larger studies to comment on the relationship with age and sex.

Key Words: Atherosclerosis, Carotid arteries, Plaques

Introduction

Depolymerisation of acid-mucopolysaccharides involved in the plaque formation results in the loss of metachormasia of the ground substance. After that the visible fibers crumble and dissolve completely and 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 mis-zone of media. This provides the stimulus for the ingrowth 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 fiber system in the inner layer of the medial coat.[4]

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. Right and left common carotid arteries were taken out and opened lengthwise. One to four sections were taken from each common carotid artery 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

Gross Appearances

The fatty streaks were present in 10 of the 30 cases in the right common carotid artery and 10 of the 30 cases in left common carotid artery. They were distributed along the long axis of the vessel wall. The fibrolipid plaques were seen in 12 cases in the right common carotid and in 12 cases in left common carotid artery. The complicated lesions were present in 3 cases in the right common carotid artery and 4 cases in the left common carotid artery. In the right common carotid artery the ulceration was seen in 2 cases and thrombus formation in one case. In left common carotid artery the ulceration was seen in only 2 cases, intimal vascularization and haemorrhage in one and thrombus formation in one. The calcified

lesions were seen in 3 cases in the right common carotid artery and 4 in left common carotid artery. The number of raised lesions in the right common carotid artery was 1-3 and in the left common carotid artery was 2-3. The size of the largest raised lesion was 3x5mm and that of the smallest raised lesion was 3x3 mm. The raised lesions were distributed irregularly within 1 cm of the beginning of the right common carotid artery and of the ostia in left common carotid in these cases **(Table No.1).**

Microscopic Changes

The fatty streaks were present in 10 cases in the right common carotid artery and 10 cases in the left common carotid artery. On histological examination of the fatty streaks the foam cells along with the increase of fluid was present in the intima. Lipid was present both intra-cellularly and extra-cellularly along with the connective tissue changes. The fibrolipid plaques were seen in 9 of the 12 cases found on gross appearance in the right common carotid artery and 9 of the 12 cases found on gross examination in the left common carotid artery. The fibrolipid plaques showed fibrous degeneration and regeneration with mucoid changes **(Fig. No.1).** There was a meta chromatic change and hyalinization in the atherosclerotic lesion. Number of foam cells were prominent and the number of fibrocytes was also increased. Fat was present in the form of fatty pool and the needle-shaped cholesterol crystal clefts were also demonstrated. Variable number of foam cells was present with the necrotic area at the base of the lesions. The complicated lesions were present in 5 cases in the right common carotid artery and 6 cases in the left common carotid artery. In the right common carotid artery 4 cases showed ulceration and one case showed intimal vascularization and haemorrhage alongwith thrombus formation. In the left common carotid artery 4 cases showed ulceration and 2 showed intimal vascularization and haemo-rrhage along with thrombus formation. In ulcerated lesions the lipid contents were less in amount. Foam cells with fibrin were present abundantly. A lymphocytic reaction with granulation tissue was seen in the lesion. In cases showing intimal vascularization and haemorrhage, deposits were also present at the junction of media and atherosclerotic lesions **(Fig No. 2)**. In atherosclerotic lesions showing thrombus formation t

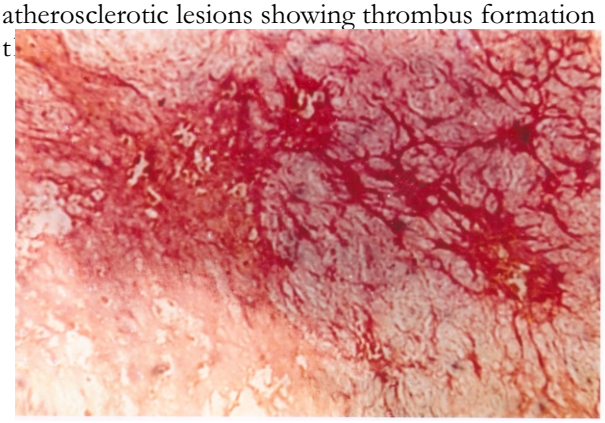

Fig-1: Photomicrograph showing regeneration of collagen fibrils in fibrolipid plaque in athersclerotic lesion.

Table-1: Atherosclerotic lesions in common carotid arteries in relation to age and sex (gross findings) (30 cases).

Age in years	Fatty Streaks		Fibrolipid Plaques		Complicated Lesions		Calcified Lesions	
	R	L	R	L	R	L	R	L
	M:F	M:F	M:F	M:F	M:F	M:F	M:F	M:F
6 - 15	-	-	-	-	-	-	-	-
16 - 25	-	-		-	-	-	-	-
26 - 35	3:1	3:1	1:0	1:0	-	-	-	-
36 - 45	3:3	3:33	1:1	1:1	-	-	-	-
46 - 55	-	-	3:0	3:0	-	-	-	-
56 - 65	-	-	2:2	2:2	1:0	2:0	1:0	2:0
66 - 75	-	-	1:1	1:1	1:1	1:1	1:1	1:1
76 - 85	-	-		-	-	-	-	-
Total	6:4	6:4	8:4	8:4	2:1	3:1	2:11	3:1
Percentage	20:13.33	20:13.33	26.66:13.33	26.66:13.33	6.66: 3.33	10:3.33	6.66:3.33	10:3.33

R=Right, L = Left M= Male, F=Female

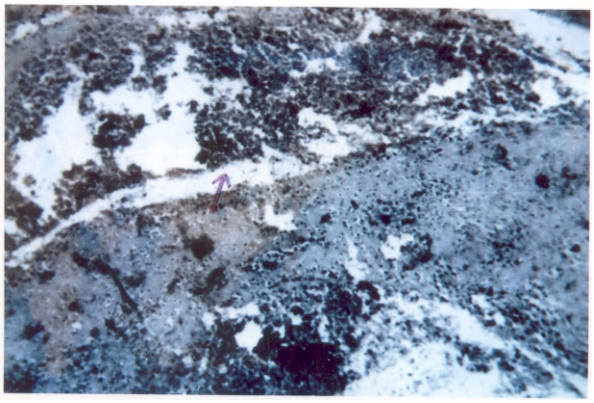

Fig-2: Photomicrograph of an atherosclerotic lesion showing haemorrhagic area (arrow). Hematoxylin and eosin ×80.

periphery and in between the platelet aggregates **(Fig. No. 3)**. The calcified lesions were seen in 4 cases in the right common carotid artery and 5 cases in the left common carotid artery. 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 ulcers in atherosclerotic lesions. The morphological changes in media and elastic were present in 6 cases in the right common carotid artery and 7 cases in left common carotid artery.

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. The fragmented internal elastic lamina was separated. The fragmented internal elastic lamina was separated apart and was totally deficient over wide areas at the base of large plaques **(Table no. 2).**

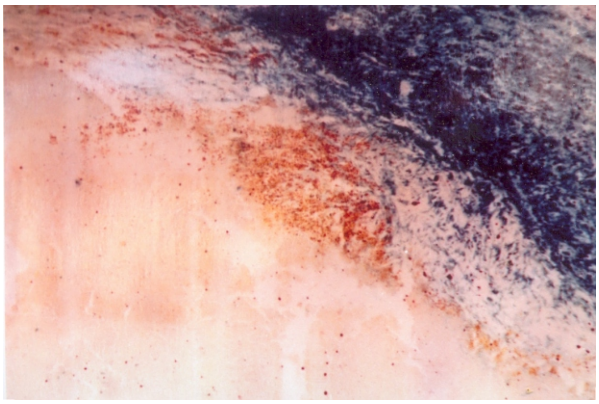

Fig-3: Photomicrograph showing areas of haemorrhage and massive haemosiderin deposition in a thrombotic common carotid artery. Perl's Prussian blue stain ×350.

Discussion

Gross morphology of Atherosclerotic lesions

The fatty streaks were distributed along the long axis of the vessel wall. The number of raised lesions in the right common carotid artery was 1-3 and in the left common carotid artery was 3 x 5 mm and that of the smallest raised lesions was 3x3 mm. The raised lesions were distributed irregularly within 1cm of the beginning of the right common carotid artery and of the ostia in left common carotid in these cases.

Table-2: Atherosclerotic lesions in the common carotid arteries in relation to age and sex (microscopic findings) (30 cases).

Age in years	Fatty Streaks		Fibrolipid Plaques		Complicated Lesions		Calcified Lesions	
	R	L	R	L	R	L	R	L
	M:F	M:F	M:F	M:F	M:F	M:F	M:F	M:F
6 - 15	-	-	-	-	-	-	-	-
16 - 25	-	-		-	-	-	-	-
26 - 35	3:1	3:1	1:0	1:0	-	-	-	-
36 - 45	3:3	3:3	1:1	1:1	-	-	-	-
46 - 55	-	-	1:0	1:0	1:0	1:0	1:0	1:0
56 - 65	-	-	2:1	2:1	1:1	2:1	1:0	2:0
66 - 75	-	-	1:1	1:1	1:1	1:1	1:1	1:1
76 - 85	-	-		-	-	-	-	-
Total	6:4	6:4	6:3	6:3	3:2	4.2	3:11	4:1
Percentage	20:13.33	20:13.33	20:10	20:10	10:6.666	13.33:6.66	10:3.33	13.33:3.33

R=Right, L = Left M= Male, F=Female

Microscopic Appearance of Atherosclerotic Lesions

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 deposition.[5] 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. Probably local adherence of the platelets at the endothelium releases Mitogenic Platelet factors into the arterial wall and causes some intimal smooth muscle cells proliferation.[7] In fibrolipid 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 muco poly saccharides. In addition there was a meta chromatic change in the ground substance along with hyalinization. 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.[10]

The accumulation of foam cells has been demonstrated in experimentally induced atherosclerosis.[11] The number of fibrocytes is increased during plaque formation. 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).[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-vascularization 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 hypercoagulability of platelets again is associated with hyperfibrinogenaemia 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 hyalinized collagen tissue in the intima.[17]

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. 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.[1]

Conclusion

This study shows that raised atherosclerotic lesions are reasonably common in our population and stresses the need to conduct larger studies to comment on the relationship with age and sex.

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