lection. Aero

-19th Edition,

during emercombat. Aero

962-66, Aero

patterns and of India 16:

cents in the 5 - Jun 1947.

A Histopathological Study on Hearts in Ischaemic Heart Disease Fatalities

SK ADAVAL, GN KUNZRU, RN VERMA, RN DIWAN, VM ALURKAR

A histopathological study on 43 hearts recovered during autopsies conducted on Ischaemic Heart Disease (IHD) fatalities has been carried out with a view to determine the acclusive status of coronary arteries and its effect on the myocardium, 74.5% of the hearts were from fatalities of age group between 31 and 50 years. Commonest vessel involved was left coronary artery and its anterior descending branch followed by right coronary artery and circumflex branch of left coronary artery, in that order. Commonest mode of occlusion was thrombosis associated with atherosclerotic changes (22 cases) progressive Juminal narrowing followed by (15 cases). Haemorrhage in atheromatous plaque and rupture of the plaque comprised the rarest cause of occlusion (5 cases). Histological evidence of infarction was discernible in only 39.9% of hearts. All infarctions involved only left ventricle, commonest being anterior. Mechanism of occlusive pathology of coronary arteries with special reference to changes in the myocardium, is discussed.

CORONARY atherosclerosis is the commonest cause of Ischaemic Heart Disease³. The prevalence and severity of the lesion, however, is reported to show marked geographical variation which has often been linked with nutritional and other factors. Occlusion

due to atherosclerotic process involves progressive luminal narrowing which may be associated with thrombosis, haemorrhage into the plaque or rupture of an atheromatous plaque leading to embolism. Incidence of atherosclerotic process is reported to be higher in the anterior descending branch of left coronary artery followed by right coronary artery and then the circumflex branch of left coronary. In association with atherosclerosis, thrombosis is a major cause of coronary occlusion consequence of which may or may not manifest in the form of an infarction. However, when the infarctions occur the commonest site is left ventricle.

Present study has been undertaken to assess the atherosclerotic status of the hearts available during autopsies conducted at Pathology Laboratory of Command Hospital AF, Bangalore on IHD fatalities during 1968-1978.

Material and Method

43 specimens of heart recovered during autopsies on patients who died of IHD at CHAF, Bangalore were studied. Coronary arteries were studied by means of serial cross sections of the vessels at different intervals. Histological study was carried out on blocks taken at 1 cm intervals from the entire length of left and right coronary arteries and their major branches. In each case, the blocks included portions

of myocardium, so that sections from many areas of atrial and venticular wells as well as interventricular septum were available. Atherosclerotic changes in coronary arteries were graded according to Mason's classification (1959) mentioned below and the findings on gross examination were histologically confirmed.

Grade 0 — Normal arteries

Grade 1 — Atheromatous plaquing leaving adequate lumen (50% or less narrowing)

Grade II — Atheromatous plaquing with severe restriction of lumen.

Grade III — Atheromatous plaquing with degenerative changes particularly calcification and/or complete occlusion of lumen.

Observations

Age groups of the fatalities studied are shown in Table—I.

TABLE—I

Age group distribution of 43 cases

Group	Age	No. of cases	Percentage
1	21 to 30 years	1	2.3
11	31 to 40 years	17	39.5
111	41 to 50 years	15	34.9
IV	51 to 60 years	10	23.3

Table I shows that out of 43 patients studied, only one case (2.3%) is in the age group of 21 to 30 years. 32 cases (74.4%) are in the age group of 31 to 50 years while 10 cases (23.3%) are in the age group of 51 to 60 years.

Prevalence of atherosclerosis in different coronary arteries is shown in Tables II and III.

Table II shows that in 3 cases only left coronary artery and its descending branch is involved. In 20 cases left and right coronary arteries are involved. In two cases left coronary artery and its left circumflex branch is involved. In 18 cases, all the three branches are involved. It is, thus observed that left coronary artery and its anterior descending branch is commonest to be involved by right coronary arteries. Last to be involved is circumflex branch of left coronary artery.

Table III shows that in 32 hearts occlusion is in the left coronary artery and its anterior descending branch followed by 10 hearts with occlusion in the right coronary artery. Only in one case occlusion is in the left circumflex artery and in two cases (Table III) occlusion is both in the left right coronary arteries.

It is observed from Table III that the most common cause of occlusion (22 cases) is thrombosis with atherosclerosis.

In 15 cases only atherosclerosis with progressive lumen narrowing, was observed while in 3 cases haemorrhage in atheromatous plaques was seen. Two cases showed rupture of atheromatous plaques and one occlusion was due to haemorrhage in organising thrombus with atheromatous plaque.

Table—II

Prevalence of atherosclerosis in different coronary arteries.

Age Group	No. of cases	Left coronary artery and its anterior descending branch	ement of Rt coronary artery	Lt circumfiex artery
	3	3 20	20	- 11/2
H HI	20 2	2	18	18
iV	18	18	38	18
	43	43		100

s studied, p of 21 to group of are in the

t coronary

only left involved, teries are bry and its cases, all observed escending

escending n in the clusion is s (Table coronary

branch of

he most s throm-

ogressive 3 cases een. Two ques and ganising

rcumfiex y

8

DICINE

TABLE—III

Prevalance of coronary atherosclerosis with or without complication

Branh of coronary artery	No. of cases	Grade I & II athero- sclerotic changes	Grade III changes with occlusion of lumen	Atherosc- lerosis with thrombosis	Athero- sclerosis with haemorrage	Athero- sclerosis with embolism.	Total
Lt coronary artery & its left anterio descending branch		11	11+(2)*	15+(1)@	2+(1)@	1	32
Rt coronary artery	38	26	2+(2)*	6	1	1	10
Lt circumflex artery.	20	19		1			1
Cause of coronary			13+(2)*	22+(1)@	3+(1)@	2	43

@ One case is common with both thrombosis and haemorrhage.

* Two are common with occlusion in both Rt & Lt coronary arteries.

TABLE—IV

Prevalence of myocardial infarction in different age group

Age group	No. of cases	Without evidence of infarction.	Recent Infarction upto 3 weeks old	Old infarction	Recent and old infarction	No. of cases showing infarction	Percentage
1	1	1	_	-			TEN MEXICA
	17	12	3	2	10-3	5	29.4
107	15	8	4	2	1	7	46.6
IV	10	5	1	4	_	5	50.0
Total	43	26	8	8	1	17	
	2.000						

TABLE—V

Prevalence of infarction with occlusion of different coronary arteries

SI. No.	Branch of coronary artery involved	No. of cases	Recent infarction	Old	Recent and old infarction.
1.	Lt coronary arteryand its	30	6	5	Nil.
2.	anterior descending branch Right coronary artery alone	10	2	2	Nil ed
3.	Lt circumflex artery alone	1	Nil	1	Nilse
4.	Lt & Rt coronary artery	2	Nil	Nil	- H - 1-11

Evidence of myocardial infarction in different age groups is shown in Table IV.

Table IV shows that infarction is present in only 17 cases out 43. In 8 cases recent infarction, in 8 cases old infarction and in 1 case both recent and old infarction were encountered. It is also observed that evidence of infarction increases with the increase in age and overall percentage is 42.

Infarction with associated occlusion of different vessels is shown in Table-V.

Out of 8 cases of recent infarction 6 showed only anterior infarction and 2 both anterior and posterior infarction. Out of 8 cases of old infarction, in 5 cases there is only anterior infarction, in 2 cases both posterior and anterior infarction and in one case both anterior and lateral infarction.

In one case there is evidence of old anterior infarction and fresh posterior infarction. None of the cases show infarction of right ventricle.

Discussion

Coronary atherosclerosis has assumed importance not only in the affluent population of the West, but also in developing countries such as India, Poor economical nutritional status and dietary habits might be responsible for the relatively low incidence and mild nature of atherosclerosis in general and coronary artery atheroma in particular in the people of India. Just as heart disease is the leading cause of death in the United States, coronary heart disease. especially the atherosclerotic type, is the chief form of fatal cardiac disease in our country also. This disease was not considered a significant factor in young adults in earlier studies^{2,9,18,16}. French & Dock⁵ reviewed military personnel and discovered 80 young soldiers between age of 20 and 30 years having died of uncomplicated coronary artery disease. Though coronary heart disease is most prevalent in men past 45 years of age8" deaths from this disease outnumber those from any other type of heart disease even in early stages like 25 to 34 years of age. Our study includes only serving defence personnel mostly in the age group of 25 to 45 years of age and findings in Table I may not give the true picture of the age incidence. None of the female hearts could be studied as permission for autopsy could not be obtained from next of kin. In our series, 74.4% of cases were in the age group of 31 to 50 years. Only one case died due to coronary occlusion at the age of 22 years which has been included in Group I.

Most authors agree that atherosclerotic lesions occur more frequently in the left coronary artery and its anterior descending branch, than in the right coronary artery, because this is the continuation of main branch which provides the greatest number of arterial radicles and supplies the major myocardial mass in males. It is, therefore, anatomically and functionally the predominant artery to bear the main thrust⁷, 14. In our series we have found that all the 43 hearts show involvement of left coronary artery and its anterior descending branch. Incidence is less in the right coronary artery and still less in the circumflex branch of the left coronary artery. (Refer Table II). The area of severest involvement is about 2 cm to 3 cms from the coronary ostia. This happens because haemodynamic forces near the bifurcation, viz eddying of the blood stream during the diastolic recoil of the dilated coronary arteries, is an important factor in the machanism of plaque formation. In a survey of 1495 cases of occlusion Plotz11 found that distribution of occlusion was most common in left and descending branch followed by right coronary artery, left circumflex artery and left main artery, in that order.

The atherosclerotic plaques are usually patchy and situated eccentrically along one side of the lumen.

One of the complications of coronary atherosclerosis is gradual encroachment of the lesion upon the lumen of the artery, leading to luminar narrowing followed by complete occlusion. Other complications are coronary thrombosis, intramural haemorrhage, rupture of an atheromatous plaque, and rarely, aneurysm formation and embolism of atheromatous material in association with atherosclerosis and embolism of atheromatous tissue, coronary thrombosis is a major cause of coronary occlusion. We have also observed that the commonest cause of coronary occlusion is thrombosis with atherosclerosis, followed by progressive atherosclerotic narrowing (Table III). Haemorrhage in atheromatous

In our of 31 to oronary is been

lesions ary and a right of ocardial ly and the 43 pry and less in the strum-

r Table
t 2 cm
appens
cation,
astolic
cortant
In a
d that
in left
ronary
artery,

upon owing ations hage, neuratous

atchy

of the

and irom-We se of sclerotic

CINE

plaque and embolism due to rupture of atheromatous plaque is rare cause of coronary occlusion in our series. In one case there was evidence of haemorrhage in organising thrombus. Atheromatous lesion without thrombosis and haemorrhage can lead to myocardial ischaemia⁶ as in our series also it is the second most common cause of coronary occlusion. In our series, only in 2 cases there was complete occlusion of left and right coronary artery though Horn et al⁶ have observed severe narrowing in two or three of the major arterial trunks in 81% of the hearts.

A consequence of coronary occlusion is myocardial infarction, a Ithough this need not invariably happen. The collateral circulation may be adequate so that muscle necrosis is prevented or the patient may die before any visible changes of an infarct develop. Conversely infarction of the myocardium can occur in the absence of sudden occlusion, In these cases, myocardial ischaemia is the result of coronary insufficiency brought on by certain factors that increase the demand of the myocardium for more oxygen4. A suggestion has been made that myocardial infarction unassociated with acute coronary thrombosis may be the clinical equivalent of Selye's experimentally produced electrolyte-steroid cardiopathy with necrosis38. This is characterised by large infarctoid necrosis caused by the combined action of certain steroids and sodium salts in the absence of coronary occlusion. In our series, in majority of cases, there was no evidence of infarction but percentage of infarction increases with increase in the age as per Table IV.

In younger age group, activity is a precipitating factor, fatal attacks occur mostly without warning and prognosis is more severe. Old infarctions are more with the increase in age because patients might have survived the fresh infarctions.

Almost all infarcts occur in the left ventricle. The most frequent site is the area supplied by the branch of left coronary artery. The next most common site is the area supplied by the right coronary artery and less commonly in the areas supplied by the left circumflex artery. In our series also all infarcts have occured in the left ventricle mostly in the areas supplied by the anterior descending

branch of left coronary artery. Right ventricle which is relatively thin walled, is sufficiently nourished by blood directly from the lumen of the heart through the luminal vestels and its metabolic requirements are not very high, thus decreasing the possibility of infarction¹.

References

- Anderson, W.A.D. Pathology: CV Mosby Company, St. Louis, 1971, p. 610-611.
- Appelbaum, E and Nicolson, GHB. Amer Heart J 10: 522, 1935.
- Blumgart, H.L. Schlesinger, MJ and Davis, D. Studies on relation of clinical manifestations of angina pectoris, coronary thrombosis and myocardial infarction to pathologic findings with particular reference to significance of collecteral circulation, Am Heart J. 19, 1.1940.
- Edward, JE. Pathologic spectrum of occlusive coronary artery disease. Lab Invest. 5, 475-491, 1956.
- French, AJ and Dock, W. J Amer Med Ass. 124: 1233, 1944.
- Horn, R.C.Jr. and Fine, G. In James TN and Keyes, J.W., Editors: The Etiology of myocardial infarction, Boston, Little, Brown and Co., 1963 p. 229.
- James TN. Anatomy of the coronary arteries. New York, Paul B. Hoeber, Inc. 1961.
- Lew E.A. Mortality from coronary heart disease. J. Chronic Dis. 6: 192-209, 1957.
- 9. Miller WS, and Woods, WW. Brit. Heart, J. 5: 101, 1943
- 10. Nathenson, M.H.: Amer. J Med Sci. 170: 240, 1926.
- 11. Plotz M. Coronary heart disease, New York, Paul B. Hoober, Inc. 1957.
- Schlesinger, M.J. Anatomic patterns of coronary arteries. Arch. Path (Chicago), 30: 403-415, 1940.
- Selye H. The pluricasual cardiopathies, Spring field III Charieo C Thomas, Publisher 1961.
- Truex R.C. Distribution of coronary arteries. In Likoff, W and Moyer, JH Editors, Coronary heart disease New York, 1963, Grune & Stratton Inc.
- 15. Wright Smith RJ Roy Melb, Hospclin Rep. 7: 71, 1936.