

Coronary Ectasia – An Angiographic Survey

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Summary:

Coronary ectasia is defined as a localized dilatation of coronary artery 1.5 times or more of the adjoining normal vessel (CASS). The report prevalence ranges from 1.5% to 4.5% in various angiographic series. Exact aetiology of the condition is not known, though a generalized cause (like protease - protease inhibitors imbalance) and local factors have been proposed. It is observed more commonly in males, smokers, persons with familial hypercholesterolemia and coronary artery disease. The entity is generally considered benign, but atypical angina, coronary spasm and myocardial infarction may occur. Treatment is generally dictated by associated coronary artery disease. We studied 1214 angiograms from 1996 series and found 23 cases (1.89%) of ectatic coronary morphology. All were males with age 48.6 ± 5.61 years. 26.1% were smokers, 34.8% non-smokers and 39.1% had quit smoking; 13% were diabetics, 18% had family history of IHD, 35% were hypertensives and 8.7% had abnormal lipid profile. Vessels involvement was: RCA 56.5%, LAD+RCA 17.4% while LCX and LAD accounted for 13% each. 74% of cases had some degree of stenosis in ectatic vessel and 92% of the cases had stenotic lesions in other vessels. 50% of the ectatic segments were proximal, 25% had lesions in proximal and distal segments while 25% had involvement of proximal and mid segments. 50% of the lesions were localized, fusiform type, 8% were mixed and 42% had linear lesions.

Key Words:

Coronary Ectasia, Coronary angiography, Familial Hypercholesterolemia, coronary thrombosis, Coronary artery by-pass grafting (CABG).

Introduction:

Coronary ectasia is a localized dilatation of one or more coronary segments as compared to adjacent normal vessels. CASS definition requires a dilatation of at least 1.5 times normal to be labelled as ectasia¹. The condition is recognized on angiography usually performed for evaluation of suspected coronary artery.

The reported prevalence ranges between 1.5 - 4% of angiograms. Though of unknown aetiology, certain associations have been observed which include male sex, smoking, coronary artery disease, familial hypercholesterolemia, and α -1 anti trypsin excess. The condition generally runs a benign course.

We are reporting the prevalence of coronary ectasia and its various attributes in Pakistani population.

Materials & Methods:

We reviewed the reports of all angiograms performed at our institute during the year 1996 (1214 in total), and identified the angiograms which had mentioned presence of ectatic morphology. Angiography films of the cases thus identified were obtained and reviewed by us for confirmation of the findings and

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qualitative as well as quantitative assessment. The results were tabulated and analyzed using SPSS on personal computer. Chi-square method was used to test significance of observed values. A p value of 0.05 was taken as significant and 0.01 or less as highly significant.

TABLE 1.

**Risk factors for coronary ectasia
(Total angiograms 1214)
(n = 23)**

Parameters	Percentage (%0)	P Value
Males	100%	.001 (HS)
Smokers (Present+Past)	65.2%	.06 (NS)
High Lipids	8.7%	.001 (HS)
Ischemic Heart Disease	92%	.001 (HS)
Diabetes Mellitus	13%	.001 (HS)
Hypertension	35%	.06 (NS)

Results:

A total of 1214 angiograms were included in the study. A total of 25 cases were identified, two out of which were discarded because of minor degree of ectasia, not fulfilling the diagnostic criteria. 23 cases were used for analysis. The prevalence of ectasia, thus, was found to be 1.89%.

Our results show that all cases of ectasia were males with age 48.6 ± 5.61 (Mean \pm SD) years. Of them, 26.1% were smokers, 34.8% non-smokers and 39.1% had quit smoking. 13% were diabetics while 87% were non diabetics. 82% had no family history of IHD while 18% had it. 65% were normotensives and 35% hypertensives. 8.7% were known to have abnormal lipid profile.

Review of angiograms revealed frequent involvement of RCA (56.5%). LAD+RCA involvement occurred in 17.5% while LCX and LAD accounted for 13% each. RCA was the dominant coronary artery in 82% of cases, LCX in 13% and LAD in 5%. 74% of cases had some degree of stenosis in ectatic vessel, either proximally or distally, while 26% had no lesions in affected vessels. 92% of the cases had stenotic lesions in other vessels. 50% of the ectatic segments were found

in proximal parts only, 25% had lesions in proximal and distal segments while 25% had involvement of proximal and mid segments. 50% of the lesions were localized, fusiform type, 8% were mixed and 42% had linear lesions. 56.5% had good LV function while 43.5% had moderate LV function as judged by LV cine.

Treatment options were mostly based upon the associated stenotic lesions. 39.1% were offered CABG surgery, 34.7% medical treatment and 26.1% were advised PTCA. The results are summarized in table 1 - 3.

Discussion:

Coronary artery ectasia, an interesting angiographic finding, is a localized dilatation of coronary artery with at least 1.5 times the diameter of non-involved vessel¹. It has been reported in various series with a prevalence rate ranging from 1.4% to 6% of angiograms^{2,3}. In our study, an observed incidence of 1.89% is in conformity with other reported series.

Exact aetiology of the condition is not known; both local as well as generalized factors have been implicated⁴. The disease may be a separate entity in itself due to underlying biochemical abnormality (α -1 anti-trypsin excess)⁵, a manifestation of vasculitic involvement of coronary arteries in a plethora of vasculitides⁶, a result of mechanical forces (post stenotic dilatation) or a combination of these⁴. Unfortunately, almost all reported series were retrospective analyses, and did not specifically rule out

TABLE 2.

**Pattern of vessel involvement
(P = .008 HS)**

Vessel Involvement	Percentage
RCA	56.5%
RCA + LAD	17.5%
LAD	13%
LCX	13%

Key:

RCA Right Coronary Artery
LAD Left Anterior Descending Artery
LCX Left Circumflex Artery

all these possibilities. A very strong association with stenotic coronary artery disease has been observed by all investigators. In our series, 74% of cases had stenosis in related vessel while 92% had lesions in other, non-related vessels. Males predominate in all series, and smoking has emerged as a strong risk factor; though not in our study ($p=0.06$). A high incidence of ectasia is also seen in patients with familial hypercholesterolemia (18% vs. 2%)^{7,8}. The interpretation of above results, however, may be difficult as there are two potential

with ectasia (82%). And incidentally, RCA involvement, alone or in combination also occurred in 74% of the cases. This would mean that ectasia has something to do with vessel dominance; greater flow, higher hemodynamic stress or other unknown local factors may be at play in concert with generalized underlying predisposing abnormality. Significance of hemodynamic factors is also underscored by the fact most of ectatic lesions occur in proximal segments (75% in our series as well as other series) where hemodynamic stress is likely to be greater.

TABLE 3.

Morphological patterns of Coronary ectasia

Pattern of Involvement	Percentage
Fusiform	50%
Linear	42%
Mixed	8%
Proximal Involvement Only	50%
Proximal & Distal Involvement	25%
Mid Segment & Proximal Involvement	25%
Proximal Stenosis Due to Atherosclerosis	74%
Stenosis in Non-Ectatic Vessels	92%

sources of selection bias, namely, 1) in all the reported series angiography was primarily performed for detection/quantitation of coronary artery disease and 2), all above mentioned risk factors predispose to coronary artery disease per se. It would be interesting to study the incidence of coronary ectasia in normal, asymptomatic people without coronary artery disease. It would also be preferable to exclude vasculitis-associated ectasia, as this would represent an ectatic 'phenotype' rather than true ectasia. At present, it seems plausible to assume that localized morphological variety of coronary ectasia may occur in association with stenotic lesions in predisposed individuals, while linear variety may occur with quantitatively greater underlying biochemical abnormality like protease-anti protease imbalance.

An other intriguing aspect of the matter is uniformly reported much higher incidence (46% to 75%) of involvement of right coronary artery for which no explanation has been offered. Could it be related to higher incidence of RCA dominance? Possibly yes - as in our total series of 1214, the incidence of RCA dominance was 80%, almost the same as in patients

Coronary ectasia generally runs a benign course. Lesions may remain static or increase in size, and new lesions may appear during follow-up⁹. In contrast to the belief, that ectatic segment is less likely to go into spasm because less muscle, spasm has been well documented^{10,11}. Ectatic segments pose another threat: in-situ thrombosis without atheroma, possibly because of eddy currents or altered flow characteristics. This may present as atypical chest pain and may respond to anticoagulation¹².

Presence of ectasia does not seem to influence management strategy, which is largely dictated by extent and nature of co-existing coronary artery disease. There can be a case for ligation distal to ectatic segments to prevent thrombo-embolic phenomenon when CABG is being performed, though its value has not been documented.

Conclusion:

Coronary ectasia is not a rare entity in our patient population. Various characteristics of the condition in our population are generally similar to other international series. Its recognition in a patient may justify novel modifications to management decision making.

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References:

1. Emond-M; Mock-MB; Davis-KB; Fisher-LD; Holmes-DR Jr; Chaitman-BR; Kaiser-GC; Alderman-E; Killip-T-3rd. Long-term survival of medically treated patients in the Coronary Artery Surgery Study (CASS) Registry. *Circulation*. 1994 Dec; 90(6): 2645-57.
2. Hartnell-GG; Parnell-BM; Pridie-RB: Coronary artery ectasia. Its prevalence and clinical significance in 4993 patients. *Br-Heart-J*. 1985 Oct; 54(4): 392-5.
3. Aparici-M; Peteiro-J; Fernandez-de-Almeida-CA; Hidalgo-R; Alzamora-P; Barba-J; Alegria-E. Coronary ectasis: another form of atherosclerosis. *Med-Clin-Barc*. 1989 Oct. 7; 93(10): 368-71.
4. Williams-MJ; Stewart-RA. Coronary artery ectasia: local pathology or diffuse disease? *Cathet-Cardiovasc-Diagn*. 1994 Oct; 33(2): 116-9.
5. Benchimol-D; Bonnet-J; Gouverneur-G; Oysel-N; Bernadet-P; Moreau-C; Crockett-R; Larrue-J; Bricaud-H. Protease inhibitors and ectasia in coronary atherosclerosis. *Arch-Mal-Coeur-Vaiss*. 1988 Sep; 81(9): 1053-8.
6. Noma-M; Kikuchi-Y; Yoshimura-H; Yamamoto-H; Tajimi-T. Coronary ectasia in Takayasu's arteritis. *Am-Heart-J*. 1993 Aug; 126(2): 459-61.
7. Mabuchi-H; Michishita-I; Sakai-Y; Ikawa-T; Genda-A; Takeda-R. Coronary ectasia in a homozygous patient with familial hypercholesterolemia. *Atherosclerosis*. 1986 Jan; 59(1): 43-6.
8. Genda-A; Nakayama-A; Shimizu-M; Nunoda-S; Sugihara-N; Suematsu-T; Kita-Y; Yoshimura-A; Koizumi-J; Mabuchi-H; et-al. Coronary angiographic characteristics in Japanese patients with heterozygous familial hypercholesterolemia. *Atherosclerosis*. 1987 Jul; 66(1-2): 29-36.
9. Farto-e-Abreu-P; Mesquita-A; Silva-JA; Seabra-Gomes-R. Coronary artery ectasia: clinical and angiographic characteristics and prognosis. *Rev-Port-Cardiol*. 1993 Apr; 12(4): 305-10.
10. Bove-AA; Vlietstra-RE. Spasm in ectatic coronary arteries. *Mayo-Clin-Proc*. 1985 Dec; 60(12): 822-6.
11. Suzuki-H; Takeyama-Y; Hamazaki-Y; Namiki-A; Koba-S; Matsubara-H; Hiroshige-J; Murakami-M; Katagiri-T. Coronary spasm in patients with coronary ectasia. *Cathet-Cardiovasc-Diagn*. 1994 May; 32(1): 1-7.
12. Perlman-PE; Ridgeway-NA. Thrombosis and anticoagulation therapy in coronary ectasia. *Clin-Cardiol*. 1989 Sep; 12(9): 541-2.