Coronary artery ectasia
– a study of 50 cases

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Introduction

Definition: 1.5 times dilatation of coronary artery compared to the normal adjacent segment.

Ectasia vs. aneurysm:

Ectasia is a diffuse affection of the arterial wall.

Aneurysm is a localized affection.
Histopathology of CAE

- Similar to coronary atherosclerosis: lipid deposition with foam cells, fibrous cap and significant loss of musculoelastic layer of the vessel wall.

- Extensive degeneration and hyalinization of the vascular media with signs of inflammation.

- Some cases are without atherosclerosis (minority).
Histopathology of CAE
Pathogenetic mechanism

- Chronic inflammation in the vessel wall. There is elevated plasma levels of CRP, IL-6 and V-VAM.
- Destruction of the vascular musculoelastic layer resulting in medial thinning.
- Chronic overstimulation of the endothelium by nitric oxide (NO).
- Breakdown of vascular extracellular matrix proteins by metalloproteinases.
Etiology of coronary ectasia/aneurysm

- Atherosclerosis (destruction of coronary media)
- Arteritis (Kawasaki’s disease, Takayasu’s disease, SLE, syphilis)
- Connective tissue disorders (Ehlers-Danlos, Marfan’s)
- Mycotic emboli
- Angioplasty
- Congenital
Markis classification of ectasia

- **Type I**: Diffuse ectasia of 2 or more vessels.
- **Type II**: Diffuse ectasia of one vessel and localized disease in another.
- **Type III**: Diffuse ectasia of one vessel only.
- **Type IV**: Localized affection of one vessel only.
Diagnosis of coronary ectasia

- Coronary angiography (gold standard)
- Intravascular ultrasound (IVUS)
- Coronary CT
- Coronary MRI
Flow alterations in coronary ectasia

- Turbulent and slow flow with stagnation of blood in the dilated segments are characteristic findings.
- Resting volumetric flow is increased but coronary flow reserve is diminished.
- There may be increased microvascular resistance.
Clinical sequelae and prognosis

- Depends on the severity of coexisting coronary lesions. About 85% cases accompany atherosclerotic coronary disease.

- Prognosis is not altered merely by CAE.

- In CASS study adjusted 5-year survival in patients with/without CAE was similar.

- Prognosis of isolated CAE is good (mortality rate 2%).
Treatment of coronary ectasia

- Chronic anticoagulation (not proven by evidence).
- Fibrinolysis and heparin in ACS.
- Aspirin for all cases. Combination of antiplatelets is not proven.
- Nitrates are discouraged (may worsen ischemia).
- PCI and CABG for accompanying stenosis.
- Stringent risk factor modification.
PCI in coronary ectasia

- Care about adequate stent expansion and wall apposition.
- Stent optimization by IVUS.
- Covered stent can offer superior acute angiographic result by excluding the ectatic segment (long term benefit not proven).
LAD showing a severe stenosis (black arrow) adjacent to a coronary ectatic segment, before stent placement (left), after stent placement (middle), and intracoronary ultrasound image (right) within the proximal ectatic segment exhibiting inadequate apposition of stent struts (asterisks) against the vessel wall (white arrows) despite stent overexpansion.
PTFE covered stent
Coronary artery ectasia – a study of 50 cases
Materials and methods

- Place of study: CMH, Dhaka
- 50 consecutive coronary artery ectasia (CAE) cases were identified through a search of CAG records over a period of Jan’ 02 to Nov’ 08.
- Angiographic and clinical characteristics were recorded and analyzed.
Aim

To study the angiographic and clinical characteristics of coronary artery ectasia (CAE) among our patients.
Results

- Total no. of CAG: 4975
- No. patients with CAE: 50
- Incidence of CAE: 1%
- Male - female ratio: 49 : 1
- Age: Range 34 to 80 years
  Median 54.4 years
## Results

Distribution of major coronary risk factors:

<table>
<thead>
<tr>
<th>Risk factors</th>
<th>No. (%)</th>
</tr>
</thead>
<tbody>
<tr>
<td>Cigarette smoking</td>
<td>23 (46%)</td>
</tr>
<tr>
<td>Hypertension</td>
<td>21 (42%)</td>
</tr>
<tr>
<td>Dyslipidemia</td>
<td>14 (28%)</td>
</tr>
<tr>
<td>Diabetes Mellitus</td>
<td>12 (24%)</td>
</tr>
<tr>
<td>Family H/O IHD</td>
<td>7  (14%)</td>
</tr>
</tbody>
</table>
## Results

### Distribution of lesions (ectasia):

<table>
<thead>
<tr>
<th>Artery</th>
<th>Count</th>
<th>Percentage</th>
</tr>
</thead>
<tbody>
<tr>
<td>RCA</td>
<td>45</td>
<td>90%</td>
</tr>
<tr>
<td>LAD</td>
<td>18</td>
<td>36%</td>
</tr>
<tr>
<td>LCX</td>
<td>16</td>
<td>2%</td>
</tr>
<tr>
<td>LM</td>
<td>3</td>
<td>6%</td>
</tr>
<tr>
<td>RI</td>
<td>1</td>
<td>2%</td>
</tr>
</tbody>
</table>
### Results

Treatment recommendation of patients with CAE:

<table>
<thead>
<tr>
<th>Intended therapy</th>
<th>No. patients (%)</th>
</tr>
</thead>
<tbody>
<tr>
<td></td>
<td>N = 50</td>
</tr>
<tr>
<td>CABG</td>
<td>14 (28%)</td>
</tr>
<tr>
<td>PCI</td>
<td>11 (22%)</td>
</tr>
<tr>
<td>Medical</td>
<td>25 (50%)</td>
</tr>
</tbody>
</table>
Results

Clinical presentations:

STEMI : 27 (54%)
UA/NSTEMI : 6 (12%)
Stable angina : 8 (16%)
Asymptomatic : 9 (18%)
Results

Ectasia with associated stenotic lesion : 44 (88%)

Ectasia without associated stenotic lesion: 6 (12%)
Ectasia de CD
Discussion
Discussion

Our incidence rate of 0.99%, is similar to incidence rates published in other series.

Our patients were predominantly male (98%). This gender difference was partially attributed to the lower incidence of coronary artery disease in women. CAE can occur in relatively younger people. Age of the patients ranged from 34 to 80 yrs median age was 54.5 yrs. This is consistent with others studies. In fact, CAE is suffered mainly by the males.
Discussion

Atherosclerosis is probably the main cause of primary coronary ectasia. In our series risk factors for coronary atherosclerosis were: smoking (46%), hypertension (42%), dyslipidaemia (28%), diabetes (24%) and positive family history (14%). The frequency of above risk factors is different from findings of Lahiri et al. who ranked the risk factors as: hypertension (51.7%), diabetes (34.5%), smoking (20.2%), dyslipidaemia (17.8%), family history 3.5%. Smoking ranks first in our study.
Discussion

In our series, 54% presented with myocardial infarction. According to Endoh et al and Naumu et al this figure is 65% and 25% respectively. In our study CAE and coexistent obstructive CAD was found in 88% cases which is consistent with other series.
Discussion

Our angiographic findings were similar to previous reports where CAE affected most commonly the RCA and LM the least.

In our study 28% patients were recommended CABG. They had either triple vessel disease or double vessel disease. Less severe cases were recommended PTCA (22%) or medical treatment (50%).
Discussion

Suitable management and treatment of CAE are not yet well established. Chronic anticoagulation is a conventional therapy in this disease. Recommendation for antiplatelet is universal. Benefit of double antiplatelets is not known. Trimetazidine may improve exercise induced angina and also improve tolerance to physical activity. In case of thrombotic occlusions thrombolysis is effective.
Conclusion

The angiographic incidence of CAE was 0.995% in our study. The majority of the patients were male in their 6th decade with concomitant history of smoking, hypertension, dyslipidemia or diabetes. The most common clinical presentation was STEMI. CAE was associated with obstructive coronary artery disease in majority of the cases. The RCA was most commonly affected and majority patients had single vessel involvement.
THANK YOU