Luminal Dimensions of Left Anterior Descending Coronary Artery in a Black Kenyan Population

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Abstract

Luminal diameter and percent stenosis of left anterior descending coronary artery are considered indicators of early atherosclerosis, arterial occlusion, myocardial infarction, and are important in quantitative estimation of coronary disease severity. These parameters show ethnic variation but there are hardly any data from African populations. This study examined luminal diameter and percent stenosis of 108 proximal left anterior descending coronary arteries. The specimens were obtained from adult individuals [72 males, 36 females; mean age 34.8 years (range 18 – 72 years) who had died of non-cardiovascular causes. The specimens were processed for routine paraffin embedding and sectioning. Seven-micron thick sections were stained with Mason’s trichrome and examined with light microscope. Luminal diameter and percent stenosis were determined on the microscopic sections using multiscan software. The dimensions were corrected for heart weight and analyzed for age and gender differences.

The mean luminal diameter was 2.72 ± 0.018 mm, with insignificant age and gender difference after correcting from heart weight. It increased with number of branches of left coronary artery. Mean percent stenosis was 34.6%. Percent stenosis of over 50% was present in 25% of individuals, all males. Of these, 8% were under 20 years. There was visible and focal intimal thickening. The mean luminal diameter is comparable to those reported from Caucasian and Asian populations, and there is significant percent stenosis in a substantial number of individuals. These findings suggest comparable vulnerability to atherosclerotic occlusion of left anterior descending.

Keywords: Luminal diameter; Stenosis; Coronary artery

Introduction

Luminal diameter and percent stenosis, like intimalmedial thickness and atherosclerotic plaque are considered indicators of early atherosclerosis and are important in quantitative estimation of coronary disease severity [1,2]. Myocardial infaracts and cardiac failure are commonly associated with relatively narrow coronary arteries, but seldom found in hearts with wider coronary arteries [3,4]. These parameters also influence size of stents to be inserted incase of occlusion [5]. The luminal diameter and associated percent stenosis show ethnic differences [6]. Knowledge of these features is important in understanding the progress and complications of atherosclerosis, hence formulation of mitigation strategies. There is, however, little data from black African populations. Since the left anterior descending (LAD) is one of the most frequently affected arteries in atherosclerosis [7] we studied its luminal diameter and percent stenosis in a black Kenyan population.

Materials and Methods

Materials were obtained from 108 cases [72 male; 36 females; Mean age 34.6 years range 18 – 72 years] during autopsy at Department of Human Anatomy, University of Nairobi. Gender, and age were recorded and hearts weighed. The number of branches of left coronary artery was counted and recorded. Specimens for microscopy were taken within 48 hours of death, to avoid overt postmortem damage to the tissues. Two-millimeter long specimens were taken from the proximal segment of LAD. They were fixed by immersion in 10% formaldehyde solution and processed routinely for paraffin embedding. Seven-micron thick sections were stained with Mason’s trichrome and Haematoxylin/Eosin stains for demonstration of the general organization of the mural components.

To determine luminal diameter, the region around the lumen was traced using Multiscan software and the collapsed artery transformed into a dilated ‘physiological’ state, which is nearly a circle. The length obtained was taken as the circumference of the circle (C) and the diameter (D) of the lumen computed using the mathematical formula D = C/π. The diameters were corrected for heart weight and differences between genders, age and number of LCA branches determined. Luminal stenosis was used to assess the extent of intimal cushioning or intimal hyperplasia in males and females and different age groups. This was defined as the area of intima on the luminal side of the arterial media divided by the computed luminal area of the artery.

The area enclosed by the lumen and the area of the intima enclosed by the internal elastic lamina (IEL) was also measured. The ratio of intima: lumen was used to assess percent stenosis/ degree of luminal narrowing (Figure 1).

Results

The mean luminal diameter was 2.72 ± 0.018 mm, higher in males (2.79 ± 0.05 mm) than in females (2.52 ± 0.06 mm). The age and gender differences were not significant, after correcting for heart weight (p=0.89, 0.63 respectively). The diameter increased with number of branches from 2.68 ± 0.05mm in bifurcation to 4.92 ± 0.05mm in pentafurcation (Table 1).

Mean percent luminal stenosis was 34.6%. It was caused by
generalized intimal hyperplasia and also by focal intimal thickening (Figure 2a). These thickenings were highly cellular (Figure 2b). There was asymmetrical intimal hyperplasia (Figure 2c), which in some cases was severe enough to cause partial luminal occlusion (Figure 2d). Percent stenosis above 50% was present in 25% of the individuals, all male: 8% in 11-20 years; 5% in 20-40 years; 4% in 40-60 years and 8% in those over 60 years.

Discussion

Observations of the present study have revealed a mean proximal LAD diameter of 2.72 ± 0.018 mm. These results are similar to those of an earlier Kenyan study [8] and those of an Indian study [9] and only slightly lower than those reported in other studies [10,11] (Table 1).

Closer similarity of the results to those of Saikrishna et al., [9] may be due to similarity in sampling protocol while some of the other differences can be attributed to methodology like grid counting [12]. A luminal diameter of less than 2.5mm is associated with a higher likelihood of CAD [9]. This implies that the black Kenyan population is vulnerable to developing CAD. The studies cited in (Table 2) reveal that there are only minor ethnic variations in luminal diameter of LAD. This suggests that differences in mortality rates due to CAD [17] are attributable to other factors, most likely extrinsic risk factors.

Further, observations of the present study show that males had slightly larger luminal diameter (2.79 mm) than females (2.52 mm). Notably, however, after controlling for heart weight, the gender differences were no longer significant (p=0.89). Previous studies on gender differences were equivocal with some reporting similar findings [18] while others [12] found females to have larger lumens after controlling for heart weight. The closest comparison to heart size in other studies has been to body size. Macalpin et al., (1973) reported that differences in lumen diameter between men and women could be acceptably resolved when total coronary area was normalized to body size [19]. On the contrary, Dodge et al., (1992) reported such differences

**Table 1:** Variation of mean diameter of LAD with branching pattern of LCA among black Kenyans.

<table>
<thead>
<tr>
<th>Branching Pattern</th>
<th>Gender</th>
<th>Diameter Mean ± SE</th>
<th>Pvalue</th>
</tr>
</thead>
<tbody>
<tr>
<td>Bifurcation</td>
<td>Overall</td>
<td>2.68 ± 0.054</td>
<td>0.01</td>
</tr>
<tr>
<td></td>
<td>Male</td>
<td>2.69 ± 0.052</td>
<td></td>
</tr>
<tr>
<td></td>
<td>Female</td>
<td>2.52 ± 0.058</td>
<td></td>
</tr>
<tr>
<td>Trifurcation</td>
<td>Overall</td>
<td>3.54 ± 0.043</td>
<td>0.01</td>
</tr>
<tr>
<td></td>
<td>Male</td>
<td>3.55 ± 0.042</td>
<td></td>
</tr>
<tr>
<td></td>
<td>Female</td>
<td>3.36 ± 0.025</td>
<td></td>
</tr>
<tr>
<td>Quadrifurcation</td>
<td>Overall</td>
<td>4.23 ± 0.056</td>
<td>N/A</td>
</tr>
<tr>
<td>Pentafurcation</td>
<td>Overall</td>
<td>4.92 ± 0.055</td>
<td>N/A</td>
</tr>
</tbody>
</table>

N/A: There were no females.

**Table 2:** Luminal diameter of LAD in different populations.

<table>
<thead>
<tr>
<th>Author</th>
<th>Technique</th>
<th>N</th>
<th>Population</th>
<th>Luminal diameter (mm)</th>
</tr>
</thead>
<tbody>
<tr>
<td>Current Study, 2013</td>
<td>Histomorphometry</td>
<td>108</td>
<td>Kenyan</td>
<td>2.72 ± 0.018</td>
</tr>
<tr>
<td>Saidi et al., 2002 [8]</td>
<td>Rulers and Dividers</td>
<td>100</td>
<td>Kenyan</td>
<td>2.70</td>
</tr>
<tr>
<td>Saikrishna et al., 2006 [9]</td>
<td>Coronary Angiography</td>
<td>94</td>
<td>Indian</td>
<td>2.85 ± 0.59</td>
</tr>
<tr>
<td>Kiviniemi et al., 2004 [11]</td>
<td>Angiography Color Doppler TTE</td>
<td>30</td>
<td>Finland</td>
<td>3.4 ± 0.22</td>
</tr>
<tr>
<td>Dhall et al., 2003 [12]</td>
<td>Grid counting</td>
<td>50</td>
<td>Indian</td>
<td>6.2</td>
</tr>
<tr>
<td>Zindrou et al., 2006 [10]</td>
<td>Coronary Angiography</td>
<td>53</td>
<td>Indo – Asians Caucasians</td>
<td>3.5 ± 0.8</td>
</tr>
<tr>
<td>Dodge et al., 1992 [13]</td>
<td>Angiography</td>
<td>83</td>
<td>American</td>
<td>3.7 ± 0.4</td>
</tr>
<tr>
<td>Kaimkani et al., 2004 [14]</td>
<td>Angiography</td>
<td>220</td>
<td>Pakistani</td>
<td>3.22 ± 0.74</td>
</tr>
<tr>
<td>Eleu teri et al., 2002 [15]</td>
<td>Echo - Doppler</td>
<td>115</td>
<td>Italian</td>
<td>1.8 ± 0.4</td>
</tr>
<tr>
<td>Kimball et al., 1990 [16]</td>
<td>Angiography</td>
<td>18</td>
<td>Canadian</td>
<td>3.32 ± 0.54</td>
</tr>
</tbody>
</table>
to persist [13]. The lack of gender differences after controlling for heart weight suggests that it is probably not gender that determines luminal size but the myocardial mass and inherent need for perfusion.

Luminal stenosis of over 70% is considered clinically significant [20-23]. In the current study, over 15% of cases had severe intimal hyperplasia, corresponding to ≥70% luminal stenosis. This implies that this proportion of the population is likely to suffer ischemic heart disease. A further observation of the current study, in support of this high vulnerability to CAD, is the existence of substantial percent stenosis of over 20% from the second decade. Significant percent stenosis, that is, over 40% is positively associated with atherosclerotic lesions [24]. In the present study, 6 males had significant stenosis, one being in the 2nd decade, two in the 3rd and 4th decades and three in over 50 years. This concurs with literature reports from Caucasian populations that atherosclerosis starts in the first decade and progresses to peak after 40 years [25].

Higher diameter in cases of variant branching is hardly reported. It is nonetheless consistent with adaptive response to changes in wall shear stress concomitant with turbulent flow [1,26] at branching points. It constitutes a dynamic remodeling response to changes in wall thickness and wall shear stress [27-29].

Conclusion

Mean luminal diameter of LAD in black Kenyan population is comparable to those of Caucasian and Asian populations, and there is significant percent stenosis in a substantial number of individuals from an early age. This suggests comparable vulnerability to atherosclerosis and occlusion of LAD. Control measures should be instituted from early age.

References