

Research Article

Coronary Artery Dominance and the Pathology of Ischaemic Heart Disease: Dominance-Related Differences in Infarct Size and Collateral Circulation in a Cadaveric Series

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ABSTRACT

Background: Left coronary dominance has been linked to adverse outcomes in ischaemic heart disease, but direct anatomical evidence relating dominance to infarct size and collateral development is scarce. We examined these relationships in a cadaveric series.

Methods: Ninety-five formalin-fixed adult human hearts were dissected and classified by dominance. Atherosclerosis was graded, myocardial infarction was characterised by location, size (percentage of left ventricle), and histological age, and the collateral circulation was assessed. Infarct size was compared across dominance groups by ANOVA and categorical variables by the chi-square test.

Results: Coronary atherosclerosis was present in 75 hearts (78.9%) and myocardial infarction in 43 (45.3%). Infarcts were larger in left-dominant hearts than in right-dominant hearts ($32.2 \pm 8.3\%$ vs $18.4 \pm 5.4\%$ of the left ventricle; ANOVA $p < 0.001$), consistent with the greater myocardial territory subtended by the dominant circumflex. Collateral circulation was present in 47.8% of right-dominant, 66.7% of balanced, and 87.5% of left-dominant hearts, a gradient that approached but did not reach significance ($p = 0.054$), likely reflecting the small left-dominant subgroup. Healed infarcts predominated ($n = 26$).

Conclusions: Coronary dominance is associated with infarct size, left-dominant hearts sustaining larger infarcts, with a parallel trend toward greater collateral development. These direct anatomical findings support the adverse prognostic significance attributed to left dominance and the systematic consideration of dominance in ischaemic heart disease.

Keywords: Coronary Dominance, Myocardial Infarction, Collateral Circulation, Atherosclerosis, Ischaemic Heart Disease, Cadaver.

INTRODUCTION

Ischaemic heart disease is the leading cause of death worldwide and imposes a rising burden in India, where it presents earlier and more aggressively than in Western populations. Its anatomical substrate is the coronary circulation, whose configuration varies between individuals; foremost among these variations is coronary dominance—the pattern determining the origin of the posterior descending artery and thus the arterial supply to the diaphragmatic myocardium, posterior septum, and atrioventricular node.

Dominance is increasingly recognised as a determinant of clinical outcome. A meta-analysis of 255,718 patients found left dominance independently associated with increased mortality in acute coronary syndrome [1], and left or co-dominant

circulations have been linked to worse outcomes after percutaneous coronary intervention [2]. Because the dominant vessel subtends a larger myocardial territory, its occlusion places more myocardium at risk—a mechanism proposed to explain the adverse prognosis and the declining prevalence of left dominance with age [3]. Dominance has further been related to the incidence of inferior myocardial infarction [4] and to the angiographic severity of coronary disease [5]. These inferences, however, derive largely from clinical and imaging studies; direct anatomical and pathological evidence linking dominance to infarct size and collateral development is scarce, and cadaveric study alone permits histological quantification of infarction and direct assessment of collateral vessels. The present study examined 95 adult cadaveric

hearts to characterise the relationship between coronary dominance and the pathology of ischaemic heart disease—the distribution of atherosclerosis, the location, size, and histological age of myocardial infarction, and the extent of collateral circulation.

MATERIALS AND METHODS

Study design and specimens. This descriptive, cross-sectional cadaveric study was approved by the Institutional Ethics Committee of Malwanchal University, Indore (reference MU/IEC/[YYYY]/[NNN]). Ninety-five formalin-fixed adult human hearts were examined; hearts with gross traumatic disruption, advanced autolysis, or prior cardiac surgery were excluded. Dominance was classified by the origin of the posterior descending artery as right, left, or balanced. Pathological assessment. The coronary arteries were examined for atherosclerosis, graded as none, minimal, mild, moderate, or severe on the basis of luminal narrowing and plaque morphology, and the maximum percentage stenosis was recorded. Transverse myocardial slices were examined for infarction; infarcts were characterised by location, by size expressed as the percentage of left-ventricular

myocardium involved, and by histological age (acute, organising, or healed) on representative haematoxylin–eosin sections. The collateral circulation was assessed on gross dissection and graded by the degree of anastomotic development. Statistical analysis. Analyses were performed in SPSS v26. Categorical variables are presented as frequencies with percentages and compared by the chi-square test; continuous variables are presented as mean ± SD and compared across dominance groups by one-way ANOVA, with the independent-samples t-test for pairwise comparison. A two-tailed p<0.05 was considered significant.

RESULTS

Study population and atherosclerosis. Among the 95 hearts (61 male, 34 female; mean age 59.7 ± 12.8 years), coronary atherosclerosis was present in 75 (78.9%) and was graded severe in 10 hearts (Figure 1). Baseline characteristics by dominance group are shown in Table 1; the groups were comparable in age and sex. Moderate-to-severe atherosclerosis was proportionally more frequent in left-dominant and balanced hearts than in right-dominant hearts, although this did not reach significance (χ² p=<0.001).

Table 1. Baseline Characteristics and Atherosclerotic Burden by Dominance Pattern.

Variable	Right (n=69)	Balanced (n=18)	Left (n=8)
Age, years (mean ± SD)	59.59 ± 13.10	60.78 ± 12.00	57.88 ± 12.94
Male, n (%)	46 (66.7%)	10 (55.6%)	5 (62.5%)
Atherosclerosis present, n (%)	49 (71.0%)	18 (100.0%)	8 (100.0%)
Max. stenosis, % (mean ± SD)	24.42 ± 15.22	63.39 ± 9.77	84.00 ± 5.13

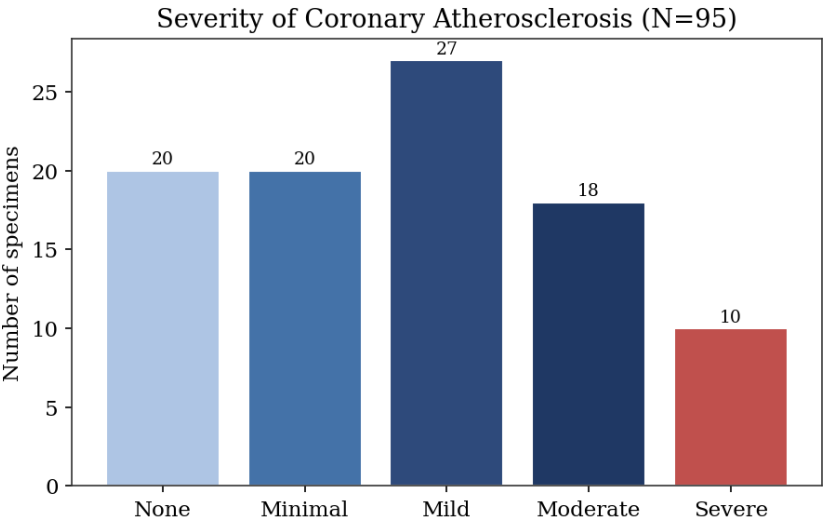


Figure 1. Severity Distribution of Coronary Atherosclerosis across the 95 Cadaveric Hearts.

Myocardial infarction. Myocardial infarction was identified in 43 hearts (45.3%). Infarcts most frequently involved the inferior/diaphragmatic and anterior territories (Table 2), and healed infarcts predominated

(26 of 43), with organising and acute infarcts less frequent. The distribution of infarct location corresponded to the dominance-determined territory, inferior infarction predominating in right-dominant hearts.

Table 2. Myocardial Infarction: Location and Histological Age (N = 43 Infarcted Hearts).

Characteristic	n	%
Location		
Inferior	29	67.4
Anterior	9	20.9
Lateral	5	11.6
Histological age		
Acute	5	11.6
Organising	12	27.9
Healed	26	60.5

Percentages are of the 43 infarcted hearts.

Infarct size by dominance. The size of infarction differed significantly across dominance patterns (Table 3, Figure 2). Left-dominant hearts sustained the largest infarcts ($32.2 \pm 8.3\%$ of the left ventricle), significantly greater than right-dominant hearts ($18.4 \pm 5.4\%$; ANOVA $F=12.8$,

$p<0.001$; left vs right $p<0.001$), with balanced hearts intermediate ($22.7 \pm 9.7\%$). This gradient is consistent with the larger myocardial territory subtended by the dominant circumflex artery in left-dominant hearts.

Table 3. Myocardial Infarct Size (% of Left Ventricle) by Dominance Pattern

Dominance	Infarcted hearts (n)	Infarct size, % LV (mean $\pm$ SD)	ANOVA p
Right	30	18.35 ± 5.37	<0.001
Balanced	6	22.73 ± 9.68	
Left	7	32.20 ± 8.34	

One-way ANOVA across the three groups; left vs right pairwise $p<0.001$.

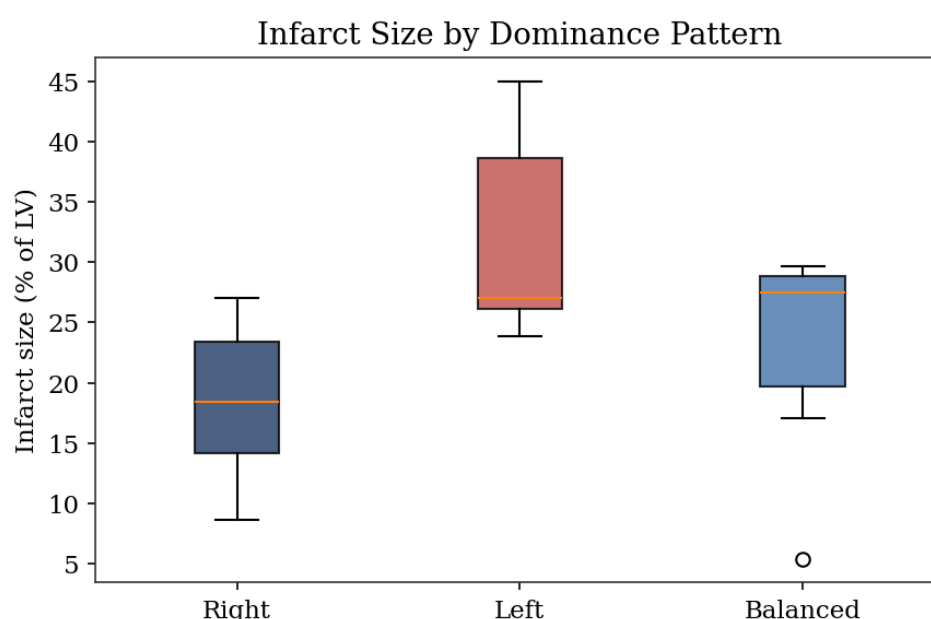


Figure 2. Myocardial Infarct Size (Percentage of Left Ventricle) By Dominance Pattern. Left-Dominant Hearts Sustained Significantly Larger Infarcts (ANOVA $P<0.001$).

Collateral circulation. Collateral circulation was present in 52 hearts (54.7%) overall and was graded more developed in hearts with severe disease and infarction. By dominance, collaterals were present in 47.8% of right-dominant, 66.7% of balanced, and 87.5% of left-dominant hearts (Table 4, Figure 3); this graded increase approached but did not reach

statistical significance (χ^2 for trend, $p=0.054$), a result likely constrained by the small number of left-dominant hearts. The direction of the association—greater collateral development where the dominant vessel subtends a larger territory—is consistent with a compensatory response to a greater ischaemic burden.

Table 4. Collateral Circulation by Dominance Pattern.

Dominance	Hearts (n)	Collateral present, n (%)
Right	69	33 (47.8%)
Balanced	18	12 (66.7%)
Left	8	7 (87.5%)
Total	95	52 (54.7%)

χ^2 for trend across groups, $p=0.054$.

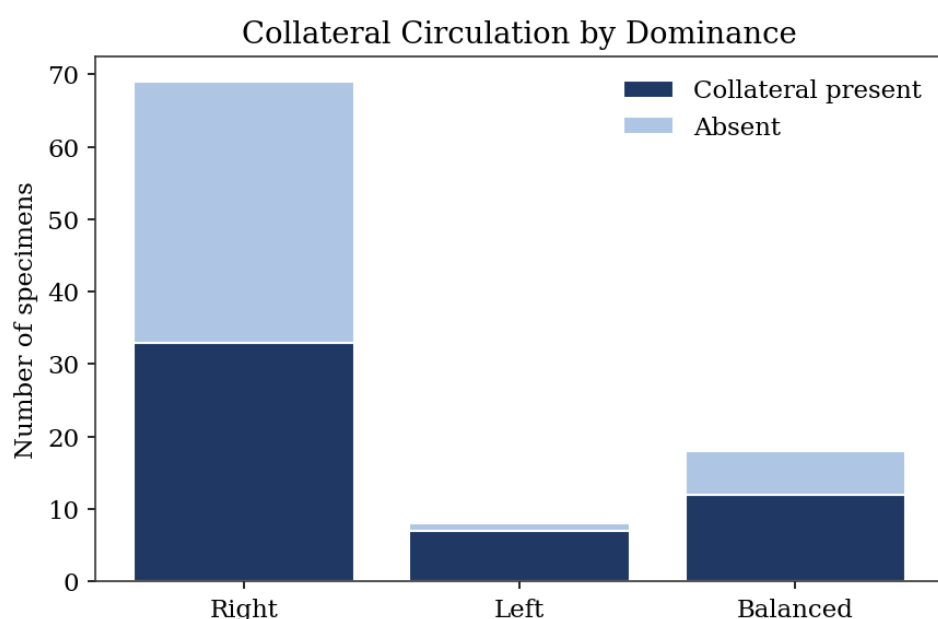


Figure 3. Presence of Collateral Circulation by Dominance Pattern, Showing a Graded Increase from Right-To Left-Dominant Hearts

DISCUSSION

This cadaveric study provides direct anatomical and pathological evidence bearing on the clinical significance of coronary dominance. Its central finding is that left-dominant hearts sustain significantly larger myocardial infarcts than right-dominant hearts, with balanced hearts intermediate. Because the dominant circumflex artery of a left-dominant heart supplies not only the lateral and posterolateral walls but also the diaphragmatic surface and part of the interventricular septum, its occlusion places a substantially larger territory at risk. The present demonstration of larger infarcts in left-dominant hearts provides a direct anatomical correlate for the increased mortality attributed to left dominance in large clinical cohorts and

meta-analyses [1,2], and complements the observation that left dominance represents a less well-balanced circulation [3].

The parallel, though non-significant, trend toward greater collateral development in left-dominant hearts is of mechanistic interest. It is consistent with the stimulation of collateral growth by a greater chronic ischaemic burden, and it identifies dominance as one determinant of the collateral response. The apparent paradox—greater collateralisation yet larger infarcts in left-dominant hearts—resolves in the recognition that both are consequences of the larger territory subtended by the dominant vessel; the net clinical effect depends upon the balance between the territory at risk and the compensatory collateral supply, which may

vary between individuals. This nuance may contribute to the divergent findings of clinical studies of dominance and outcome [2,5].

The observed correspondence between infarct location and the dominance-determined territory further supports the anatomical logic of these findings and accords with clinical studies relating dominance to the site of infarction [4]. Taken together, the results support the systematic documentation of dominance in the assessment of ischaemic heart disease and its incorporation into anatomical and clinical teaching.

Limitations. The cadaveric design precludes clinical correlation, and the number of left-dominant hearts (n=8), though consistent with population prevalence, limits statistical power—most evident in the collateral analysis, where a clinically appreciable gradient did not reach significance. Infarct size was estimated on gross and histological examination rather than by volumetric imaging. Single-centre sampling constrains generalisability, and larger multi-centre studies are warranted to confirm these associations.

CONCLUSION

In this cadaveric series, coronary dominance was associated with the size of myocardial infarction, left-dominant hearts sustaining significantly larger infarcts, with a parallel trend toward greater collateral development. These direct anatomical findings provide a pathological correlate for the adverse prognostic significance attributed to left dominance and support the systematic consideration of the dominance pattern in the evaluation of ischaemic heart disease.

Declarations

Ethics Approval and Consent to Participate: The study was approved by the Institutional Ethics Committee of Malwanchal University, Indore (approval reference MU/IEC/[YYYY]/[NNN]). All cadaveric material was obtained and used in accordance with the institutional and statutory provisions governing body donation for education and research; specimen anonymity was maintained throughout.

Consent for Publication: Not applicable.

Availability of Data and Materials: The datasets generated and analysed during the current study are available from the corresponding author on reasonable request.

Competing Interests: The authors declare that they have no competing interests.

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Authors' Contributions: Both authors contributed to the study design; JSKRK performed the dissections, pathological assessment, and analysis and drafted the manuscript; AB supervised the study, interpreted the data, and critically revised the manuscript. Both authors read and approved the final version.

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