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REVIEW ARTICLE

Coronary Artery Dominance and Myocardial Infarction: A Review of Anatomical Variations, Physiological Impacts, and Clinical Implications in Inferior Myocardial Infarction.

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Abstract

Background: Coronary artery dominance defined by the vessel supplying the posterior descending artery (PDA) plays a critical role in the prognosis and management of myocardial infarction. While right dominance is the most common anatomical pattern, left dominance is associated with a higher risk of adverse outcomes, particularly in inferior wall myocardial infarctions (MI).

Methodology: This narrative literature review was conducted using scientific databases including PubMed, NCBI, Google Scholar, as well as publisher platforms such as ScienceDirect and SpringerLink. Peer-reviewed English-language articles published from 1940 to 2025 were reviewed. Foundational anatomical concepts were supplemented by standard anatomical textbooks. Clinical relevance was ensured by synthesizing literature based on pre-formulated research questions.

Results: Right coronary dominance is present in approximately 75% of individuals, while left and co-dominant patterns account for the remainder. Right and co-dominance are generally associated with better anatomical and clinical outcomes due to more robust collateral circulation. In contrast, left dominance is linked to an increased risk of ischemia in inferior wall MIs due to insufficient perfusion to the PDA territory. Although coronary flow reserve may be influenced by dominance type, this remains an underexplored area. Clinical outcomes can be improved by aligning ECG findings with coronary anatomy and tailoring surgical or interventional strategies accordingly. Routine documentation and confirmation of coronary dominance either incidentally or deliberately via imaging can aid clinical decision-making.

Conclusion: Coronary artery dominance is a critical anatomical and physiological factor that significantly influences outcomes in inferior STEMI. Integrating coronary dominance assessment into diagnostic and treatment protocols using ECG, angiography, and emerging tools such as AI can help personalize patient management, reduce complications, and improve prognosis. Future research should focus on quantifying the physiological effects of dominance types and optimizing therapeutic strategies accordingly.

Keywords

Inferior STEMI, Coronary Artery Dominance, Left Dominance, Coronary Flow Reserve, CXCL12-CXCR4 Signaling.

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Introduction

Coronary artery dominance refers to the anatomical configuration by which the posterior descending artery (PDA) is supplied most commonly by the right coronary artery (RCA), less frequently by the left circumflex (LCx) artery, and occasionally by both. These configurations are known as right-dominant, left-dominant, and co-dominant systems, respectively. In the general population, approximately 80–85% exhibit right dominance, 10% left dominance, and 5–10% co-dominance[1,2]. This anatomical variation holds substantial clinical relevance, particularly in a myocardial infarction (MI), as it determines the extent and location of myocardium at risk during ischemic events.

In left-dominant systems, occlusion of the LCx artery compromises a broader myocardial area, including the posterior wall, interventricular septum, and atrioventricular node leading to more severe clinical presentations. In contrast, right dominance more frequently results in inferior wall infarctions, which are generally less extensive and more hemodynamically stable due to better collateral development[3]. Co-dominant systems, although less well studied, may offer a protective advantage through dual perfusion of the PDA territory.

Despite extensive documentation of coronary artery dominance in anatomical literature, its integration into clinical frameworks remains underdeveloped. While numerous studies have explored dominance patterns, infarct size, ECG findings, and clinical outcomes, the results are often inconsistent and fail to reach a consensus. Additionally, there remains a lack of comprehensive synthesis that combines anatomical knowledge with clinical application in a manner that supports individualized treatment planning.

The interpretation of ECG findings, for example, could be enhanced by considering the underlying dominance pattern yet this integration is not routinely practiced. Similarly, decisions during percutaneous coronary intervention (PCI), such as catheter selection and procedural approach, are not consistently guided by coronary dominance despite its potential impact on procedural success and patient outcomes. Even the potential role of coronary flow reserve (CFR) in patients with angiographically normal arteries and suspected microvascular dysfunction has not been fully explored in relation to dominance type.

To address these gaps, this review aims to explore key questions surrounding coronary artery dominance. These include its anatomical and embryological basis; its prevalence in the general population; its clinical significance, especially in inferior MI; how ECG and angiographic findings can be used in tandem to guide PCI and CABG strategies; whether CFR offers reliable risk prediction in relation to dominance; and what additional risks are posed by left-dominant circulation patterns. By synthesizing these domains, this review seeks to enhance the clinical utility of coronary dominance assessment and inform future research and patient care.

Methodology

Methodological Approach

This review adopted a structured narrative review approach to synthesize current evidence on coronary artery dominance and its clinical implications in myocardial infarction (MI). The review included peer-reviewed journal articles, anatomical and embryological texts, and clinical resources published between 1940 and 2025.

A systematic literature search was conducted using electronic databases including PubMed, Google Scholar, NCBI Bookshelf, as well as publisher platforms such as ScienceDirect, SpringerLink, and Wolters Kluwer. Additional resources included authoritative anatomical and embryological textbooks such as Snell's Clinical Anatomy and Langman's Medical Embryology. Reputable clinical and imaging repositories such as StatPearls, Radiopaedia, and Radiology Assistant were also consulted to support the anatomical and imaging components of the review.

In total, 45 peer-reviewed articles were referenced. Keywords and MeSH terms used during database searches included: “coronary artery dominance,” “normal anatomy of coronary arteries,” “inferior STEMI,” “left coronary dominance mortality,” “CT coronary angiography,” “coronary anatomy,” “cardiac MRI coronary anatomy,” “coronary flow reserve,” “CXCL12-CXCR4 signaling,” “right and left coronary dominance,” “ECG patterns,” and “coronary artery remodeling.”

Inclusion and Exclusion Criteria

Studies were included if they were peer-reviewed and published in English, focused on human coronary anatomy, physiology, or pathology, and addressed coronary dominance in terms of prevalence, anatomical variation, imaging assessment, embryological basis, genetic determinants, or clinical outcomes, particularly in the setting of myocardial infarction (MI). Studies were excluded if they were conference abstracts without accessible full-text articles or isolated case reports that lacked generalizable findings or broader clinical relevance.

Screening and Data Extraction

Titles and abstracts were first screened for relevance. Eligible studies then underwent full-text review. Key data were extracted relating to:

- The prevalence and anatomical types of coronary artery dominance.
- The clinical significance of dominance in myocardial infarction outcomes.
- Imaging modalities used to assess coronary anatomy (e.g., CT angiography, cardiac MRI).
- Embryological and genetic factors influencing coronary development.
- The role of coronary flow reserve (CFR) in ischemic risk assessment.
- Overall prognostic implications in patients with different dominance types.

This methodological approach was chosen to provide a clinically meaningful synthesis that bridges anatomical foundations with practical cardiovascular care.

Anatomy of coronary circulation and its relation to dominance types

General Overview of Coronary Circulation

The coronary circulation comprises the arterial supply and venous drainage responsible for perfusing the myocardium. The two principal arteries the right coronary artery (RCA) and the left coronary artery (LCA) originate from the right and left aortic sinuses of the ascending aorta, respectively[4]. This section will focus exclusively on the arterial component of coronary circulation, as the venous drainage lies outside the scope of this discussion.

Branches of the Left Coronary Artery

The LCA typically bifurcates shortly after its origin into two primary branches: the left anterior descending artery (LAD) and the left circumflex artery (LCx)[4,5]. The LAD descends through the anterior interventricular groove toward the apex of the heart, giving rise to septal perforators and diagonal branches that supply the anterior interventricular septum and the cardiac apex[6].

The LCx courses through the left atrioventricular groove, giving off obtuse marginal (or left marginal) branches that perfuse the lateral wall of the left ventricle (LV)[6]. It also gives rise to atrial branches that supply the left atrium (LA). In approximately 22.1% of individuals, the sinoatrial nodal artery (SANa) originates from the LCx, contributing to the perfusion of the sinoatrial (SA) node. However, in the majority (68%), the SANa arises from the RCA, establishing the RCA as the dominant supplier of the SA node[7].

Branches of the Right Coronary Artery

The RCA originates from the right aortic sinus and travels between the right auricle and pulmonary trunk, then descends in the right atrioventricular groove before curving around the heart’s acute margin[4-8]. Its major branches include the SANa (in 68% of cases), right conus artery, right marginal artery, anterior and posterior ventricular branches, atrioventricular nodal branches, atrial branches, and the posterior descending artery (PDA)[4].

Coronary Artery Dominance Types

Coronary artery dominance is defined by the artery that gives rise to the PDA, which supplies the posterior third of the interventricular septum and the inferior walls of both ventricles[9]. Understanding dominance patterns is clinically relevant, particularly in the context of myocardial infarction (MI), as they influence prognosis and treatment planning.

Three main anatomical dominance patterns exist:

- Right Dominance: The PDA arises from the RCA.
- Left Dominance: The PDA arises from the LCx artery.
- Co-Dominance: The PDA receives contributions from both the RCA and LCx.

Table 1: Prevalence of each anatomical dominance pattern.

Dominance Type	Prevalence Range	References
Right Dominance	70–85%	10, 11, 12
Left Dominance	8–15%	13, 14, 16
Co-Dominance	5–10%	10, 15, 16

Coronary arteries are classified as functional end arteries, meaning they lack sufficient anastomoses to compensate for an occlusion through collateral flow[17].

As a result, an occlusion in the artery supplying the PDA not only compromises the perfusion of its direct territory (inferior walls and posterior septum) but also exacerbates ischemic injury in the regions normally perfused by its parent artery.

Patients with left dominance are at higher risk for adverse outcomes because the LCx supplies a larger myocardial territory. Thus, its occlusion can result in extensive infarction. In contrast, co-dominant circulation provides a form of redundancy, if one artery is occluded, the other may partially maintain perfusion of the PDA territory, thereby reducing infarct size and improving prognosis.

These anatomical insights underscore the importance of individualized clinical strategies. Tailoring diagnostic and therapeutic interventions based on coronary dominance may enhance the accuracy of MI localization and the effectiveness of treatment, ultimately leading to better patient outcomes.

Embryology of coronary dominance

Introduction to the Embryological Basis of Coronary Dominance

Understanding the embryological development of coronary dominance is essential for appreciating its implications in myocardial infarction prognosis. The coronary arteries originate from epicardial cells, which in turn are derived from the proepicardial organ, a structure located in the caudal region of the dorsal mesocardium[18]. The coronary vasculature begins to form after heart looping and ventricular chamber development. During early cardiogenesis, the embryonic myocardium is spongy in nature and is nourished by a network of intramural capillary sinusoids and intramyocardial blood-filled spaces.

As myocardial compaction progresses, the subepicardial vascular plexus takes over the role of perfusion. This plexus eventually gives rise to the major coronary arteries RCA, LCA and the primary veins of the cardiac venous system[19]. By the 5th to 8th week of gestation, the branches of the subepicardial plexus invade the aortic root and establish connections with the right and left aortic sinuses, forming the right and left coronary ostia. This process is tightly regulated by CXCL12–CXCR4 signaling, which guides the vascular branches to their correct locations on the aortic root[20].

Experimental models in mice have demonstrated that the deletion of either CXCL12 or CXCR4 leads to the misplacement of coronary ostia, underscoring the critical role of this signaling pathway in proper coronary patterning[21].

Remodeling of the Coronary Arteries

Initially, the coronary system is symmetrical, with both the RCA and LCA contributing equally to the supply of the ventricles, interventricular septum, and posterior interventricular groove. This early symmetry suggests that either artery has the potential to develop into the dominant supplier of the posterior descending artery (PDA).

The eventual establishment of dominance arises through a process of vascular remodeling, during which:

- Inefficient capillary branches regress,
- Major coronary arteries mature and stabilize,
- A definitive coronary arterial tree is formed[22].

This remodeling phase, which finalizes between 8 and 12 weeks of gestation, is influenced by three key factors, molecular signaling, flow dynamics and myocardial metabolic demands.

Molecular signaling plays a pivotal role, with localized expression of CXCL12 being particularly influential. Imaging studies of human fetal hearts (12–23 weeks gestation) have shown region-specific CXCL12 expression, especially in the right ventricular myocardium[23].

This molecular expression is itself affected by hemodynamic forces. In fetal life, the right heart receives and ejects more blood than the left due to:

- Presence of the foramen ovale, which shunts blood from the right to the left atrium,
- High pulmonary vascular resistance, increasing pressure load on the right side[22,23].

As a result, the right ventricular myocardium undergoes more mechanical stress and becomes more trabeculated. This increased trabeculation and activity raise oxygen demands, causing localized hypoxia, which triggers hypoxia-inducible factors (HIFs) to activate CXCL12 transcription. The resulting expansion of right-sided vasculature suppresses the development of the left-sided arterial system, establishing right dominance.

In contrast, left dominance may result from lower CXCL12 expression in the right ventricle, allowing relatively higher expression and vascular development on the left. The same mechanisms are mirrored, with the LCx taking over PDA supply.

In cases of co-dominance, approximately equal levels of CXCL12 expression are observed in both ventricles. This balance may be attributed to symmetrical flow dynamics in the interventricular region during fetal development, enabling both RCA and LCx to develop well-formed posterior branches that jointly contribute to the PDA.

Physiology of coronary blood flow

Coronary blood flow (CBF) exhibits unique physiological characteristics distinct from other organ systems, primarily due to the cyclic nature of cardiac contraction and relaxation. In particular, the left ventricular myocardium receives the majority of its perfusion during diastole, the phase of myocardial relaxation. This contrasts with most other organs, where peak perfusion often occurs during systole.

Approximately 80% of coronary perfusion to the left ventricle occurs during diastole, when vascular resistance is lowest and oxygen delivery is most efficient[24,25].

This diastolic predominance is critical for sustaining myocardial oxygenation especially within the subendocardial layers, which are the most vulnerable to ischemia. These inner myocardial layers are subjected to the highest intracavitary pressures and exhibit the greatest oxygen demand.

During tachycardia, diastolic duration shortens, exacerbating the risk of hypoperfusion in these regions. Hence, efficient regulation of CBF becomes essential in physiologic states like exercise and pathologic conditions such as myocardial infarction.

The body employs several intrinsic mechanisms to regulate coronary perfusion according to metabolic demands:

- Autoregulation: Maintains stable coronary flow across a range of perfusion pressures.
- Metabolic control: Local vasodilatory substances (e.g., adenosine, lactate) respond to hypoxia and increased myocardial work, augmenting flow.
- Endothelial signaling: Molecules such as nitric oxide and prostacyclin are released by the endothelium to induce smooth muscle relaxation and vasodilation[26,27].
- Myogenic response: Vascular smooth muscle constricts or relaxes in response to changes in transmural pressure, stabilizing perfusion.
- Autonomic nervous system modulation:
- β-adrenergic stimulation promotes vasodilation and increases coronary flow;
- α-adrenergic activation leads to vasoconstriction, particularly under stress[28].

A central concept in coronary physiology is Coronary Flow Reserve (CFR) the ratio between maximal achievable and resting coronary blood flow. In healthy individuals, CFR typically ranges from 3 to 5. A reduced CFR indicates impaired vasodilatory capacity and is a marker for increased risk of myocardial ischemia, even in the absence of obstructive coronary disease[29,30].

Clinical significance of CFR includes:

- Detection of subclinical or microvascular coronary disease, especially when traditional imaging is inconclusive;
- Informing therapeutic decision-making, particularly regarding the need for revascularization in patients with intermediate coronary lesions;
- Risk stratification, as impaired CFR is a robust predictor of adverse cardiovascular outcomes.

Traditionally, CFR is assessed using invasive pressure wires or advanced imaging techniques such as positron emission tomography (PET), cardiac MRI, or Doppler echocardiography. However, emerging technologies such as intracoronary sensors and AI-driven perfusion modelling are paving the way for dynamic, real-time CFR assessment during procedures, with the potential to personalize interventions based on continuous physiological feedback.

This physiological framework becomes especially relevant in left-dominant coronary circulation, where the LCx artery supplies a larger myocardial territory, often without significant collateral support from the RCA. In such individuals, tachycardia or flow-limiting lesions in the LCx can severely compromise perfusion across a broad area of the myocardium. The absence of collateral backup in left-dominant systems renders them particularly susceptible to large infarct sizes and poor outcomes in ischemic events.

Despite growing interest in coronary physiology, variation in CFR across different coronary dominance types remains poorly understood, particularly in acute ischemic settings. This represents a critical gap in the literature and underscores the need for further investigation. A deeper understanding of how anatomical dominance affects physiological reserve could significantly improve our ability to assess ischemic risk and guide tailored treatment strategies.

Dominance-specific patterns of myocardial infarction

The anatomical pattern of coronary artery dominance significantly influences the presentation, extent, and clinical outcomes of myocardial infarction (MI)[31,32]. As dominance determines the origin of the posterior descending artery (PDA) which perfuses the posterior interventricular septum and inferior ventricular walls the implications for ischemic injury vary depending on whether the circulation is right-dominant, left-dominant, or co-dominant.

Right-Dominant Circulation

Right-dominant anatomy is the most prevalent pattern, observed in approximately 70–85% of individuals. In this configuration, the RCA gives rise to the PDA, supplying the inferior wall of the left ventricle and, in some cases, the right ventricle as well. Occlusion of the RCA, particularly proximally, often results in inferior wall MI, and may extend to the right ventricular myocardium depending on lesion location.

Clinical manifestations include bradyarrhythmias and hypotension, especially when the SA or AV nodes are affected due to their typical RCA supply. Proximal RCA occlusions are more likely to impair right ventricular function, while distal occlusions may be better tolerated.

Studies have demonstrated that individuals with right dominance exhibit a 2.4-fold increased risk of inferior MI compared to non-right dominant individuals[33]. However, the presence of robust collateral circulation in some patients may mitigate infarct size and improve outcomes.

Left-Dominant Circulation

Left-dominant circulation, present in approximately 8–15% of the population, carries significantly higher clinical risk. In this pattern, the LCx artery gives rise to the PDA, making it responsible for perfusing both the posterior wall and critical components of the conduction system, including the AV node.

Occlusion of the proximal LCx artery can lead to extensive infarction and severe outcomes:

- Infarct volumes may be up to 2.1 times greater, as measured by cardiac MRI[34].
- In-hospital mortality is significantly higher—36.4% in left-dominant STEMI cases versus 7.1% in right-dominant patients[35].
- Patients often experience increased long-term complications, including arrhythmias, heart failure, and recurrent ischemic events.

Despite this heightened risk, coronary dominance is rarely incorporated into routine risk assessment or therapeutic decision-making, such as selecting targets for revascularization or determining urgency in STEMI intervention. This oversight underscores a critical knowledge and practice gap in contemporary cardiology.

Co-Dominant Circulation

In co-dominant anatomy present in approximately 5–10% of individuals the PDA receives dual blood supply from both the RCA and LCx arteries. This configuration is hypothesized to provide partial protection against ischemic injury, as alternate flow pathways may compensate in the event of a single-vessel occlusion.

Observational data suggest that patients with co-dominant circulation typically experience:

- Intermediate-sized infarcts relative to right or left-dominant individuals,
- A 22% lower 30-day mortality following percutaneous coronary intervention (PCI) compared to left-dominant counterparts[35].

However, co-dominant patterns remain underrepresented in both imaging research and interventional cardiology literature. Their potential role in modulating infarct size, preserving conduction function, or guiding interventional strategies has not been explored in depth through large-scale, prospective trials.

Coronary dominance exerts a significant influence on myocardial infarction characteristics, including infarct location, size, and prognosis. Despite its clinical relevance, dominance is not routinely evaluated or integrated into therapeutic algorithms. Given the emerging data linking dominance patterns with MI outcomes, future research should prioritize:

- Incorporation of dominance assessment into risk stratification tools;
- Evaluation of dominance-specific interventional strategies;
- Validation through prospective, multicenter studies.

Such advancements may enable more personalized and anatomically informed management of ischemic heart disease.

Clinical importance of coronary artery dominance patterns

Understanding coronary artery dominance has significant implications across the diagnosis, management, and interventional strategies of patients presenting with inferior myocardial infarction (MI).

Dominance affects not only the region of ischemic involvement but also electrocardiographic interpretation, interventional planning, and surgical outcomes.

Dominance Patterns in ECG Interpretation

The electrocardiogram (ECG) remains a cornerstone in diagnosing inferior wall MI, primarily using ST-segment elevations in leads II, III, and aVF. However, coronary dominance significantly modulates ECG patterns, sometimes leading to diagnostic uncertainty or misinterpretation, particularly between right and left dominance.

Traditionally, the lead III > lead II ST-segment elevation (STE) pattern is associated with right coronary artery (RCA) occlusion (i.e., right-dominant), while lead II > lead III STE suggests left circumflex artery (LCx) occlusion (i.e., left-dominant)[36,37].

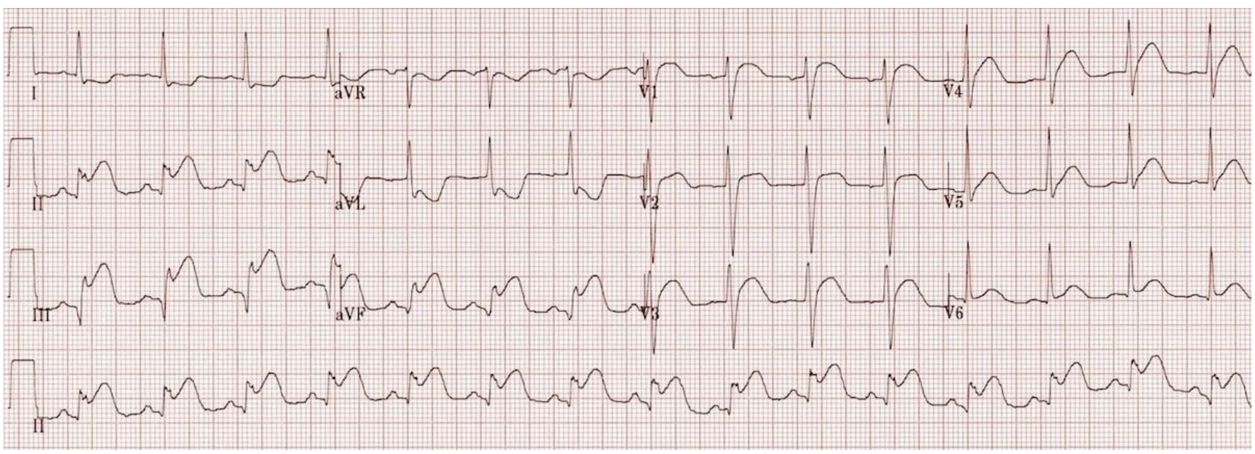

Figure 1: STE in II, III, and aVF, with greater elevation in III than II, suggestive of RCA occlusion. Reciprocal changes in leads I and aVL and greater ST depression in aVL vs. aVR reinforce this diagnosis[38].

While this rule of thumb is widely taught, its specificity is limited. In a cohort study of 135 patients, Zhan et al. found that although STE in lead III > II occurred in 87% of RCA infarctions, it was also present in 61% of LCx infarctions, yielding a specificity of only 17%[39]. Thus, coronary dominance must be considered if previously documented, especially given the prevalence of right dominance (Table 1).

Additional lead comparisons particularly aVL and aVR enhance diagnostic accuracy. Sahi et al. found that[40]:

- ST depression in aVL > aVR strongly indicated RCA occlusion (Sensitivity: 98%, Specificity: 82%).
- ST depression in aVR ≥ aVL pointed toward LCx occlusion (Sensitivity: 83%, Specificity: **98%).

These patterns should always be interpreted in conjunction with reciprocal changes in lead I and aVL to improve diagnostic precision. However, even with sophisticated interpretation, electrocardiography alone is insufficient, particularly in co-dominant anatomies, where dual PDA supply can blunt classical ECG findings[41-43].

Moreover, dominance influences atrial electrophysiology. Left dominance, via LCx supply to the AV node and posterior myocardium, increases vulnerability to conduction abnormalities and atrial fibrillation (AF).

A comparative study of 394 patients (194 left-dominant, 200 right-dominant) with normal coronary angiograms found significantly longer P-wave durations (Pmax) and greater P-wave dispersion (PWD) in left-dominant patients ($p = 0.001$), especially in lead II[42].

These findings suggest that left dominance predisposes to AF even in structurally normal hearts, emphasizing its subclinical electrophysiological relevance.

Role of Coronary Imaging

a. Coronary Angiography: Coronary angiography remains the gold standard for identifying the culprit artery in inferior MI and for determining coronary dominance. This is crucial in left-dominant patients, where larger infarct sizes confer higher morbidity.

Selective injection of contrast into the left and right coronary ostia reveals the origin of the PDA from the RCA in right dominance, or the distal LCx in left dominance. The dominant artery typically exhibits a larger caliber, which aligns with embryological patterns[44].

b. Angiography-Guided Catheter Selection and PCI Planning: A study involving over 8,000 patients found that catheter selection varies by dominance type. In right-dominant anatomy:

- Judkins Right (JR) is used for RCA,
- Judkins Left (JL) for the left coronary artery (LCA).

In left-dominant patients, Extra Backup (EBU) catheters provide enhanced support for navigating the larger LCx, especially when proximal lesions or takeoff angles demand stability. Amplatz Left (AL) catheters are used selectively, particularly for anomalous origins or when EBU fails[45].

In co-dominant anatomies, dual access (JR and JL/EBU) is often required to assess full inferior wall perfusion. Incomplete visualization could result in under-revascularization. Given the frequency of right dominance, there is a risk of false assumption if dominance is not explicitly determined.

Dominance also affects arterial diameter and intervention strategy. For example, the LCx diameter may increase from 2.0 ± 0.37 mm (non-dominant) to 3.0–3.5 mm (dominant)[45,46]. This necessitates tailored:

- Stent sizing,
- Bifurcation strategies,
- Angulation views (e.g., "spider" view for LCx-PDA),
- Plaque assessment using IVUS or OCT[47,48].

Such intravascular imaging helps identify calcified plaques and determines if plaque-modification tools (e.g., rotablation) are needed during PCI.

Impact of Dominance on CABG Outcomes

Coronary dominance also influences outcomes in Coronary Artery Bypass Grafting (CABG). In a cohort of 100 CABG patients:

- 21% were left-dominant,
- 69% right-dominant,
- 10% co-dominant.

Left-dominant patients had significantly higher rates of incomplete revascularization (42.9% vs. 20.3%) and mortality (42.9% vs. 0.9%)[49].

Table 2: CABG Outcomes by Dominance.

Dominance	Incomplete Revascularization (%)	p-value (IR)	Mortality (%)	p-value (Mortality)
Right	20.3	0.033	0.9	0.003
Left	42.9	—	42.9	—

These differences stem from complex anatomy in left-dominant patients, where the LCx, obtuse marginal (OM), and PDA branches are posteriorly located, deeper in the atrioventricular groove, and harder to visualize or graft. In contrast, right-dominant anatomy presents more superficial vessels, such as the RCA and its AV nodal branches, making grafting and PCI more straightforward.

In co-dominant patients, outcomes tend to mirror right-dominant anatomy. Selçuk et al. found no significant increase in complexity or complications in co-dominant versus right-dominant CABG procedures[50]. This is attributed to dual perfusion pathways providing a backup in case of graft failure.

Right-dominant patients may also benefit from collateral flow via the well-developed LCA and LCx. However, in left-dominant patients, the hypoplastic RCA lacks sufficient capacity to compensate if LCx branches are not revascularized, increasing the risk of ischemia in critical myocardial territories.

Findings from the literature

This review confirms that right coronary dominance is the most prevalent pattern in the general population, followed by left dominance and then co-dominance. Beyond prevalence, right dominance is often anatomically advantageous during inferior ST-elevation myocardial infarction (STEMI), partly due to hemodynamic, metabolic, and developmental asymmetries. These differences originate in the embryonic heart, where higher right-sided blood flow and metabolic demand guide the preferential development of the RCA. This flow-mediated patterning results in larger vessel caliber and more robust collateral support on the right side.

Although right-dominant patients are more likely to suffer an inferior MI, they typically have better outcomes due to functional collateralization and less extensive myocardial jeopardy compared to left-dominant individuals.

In contrast, left dominance is associated with poorer perioperative prognosis, owing to the larger perfusion territory of the left coronary system and the RCA's inability to compensate during LCx occlusion. Moreover, the anatomical depth and angulation of left-dominant branches, such as the obtuse marginal and posterior descending arteries, complicate surgical access. These challenges often result in longer cardiopulmonary bypass (CPB) times, elevating the risk of complications such as stroke, atrial fibrillation (AF), or the need for postoperative extracorporeal membrane oxygenation (ECMO).

Recent surgical benchmarks recommend CPB durations under 240 minutes (4 hours), as durations beyond 270 minutes correlate with increased postoperative morbidity[51]. These time constraints may discourage complete revascularization in left-dominant patients, where complex LCx anatomy could prolong operative time beyond safe limits. Consequently, grafting is often selective, prioritizing patient safety over completeness.

Diagnostic planning, therefore, must begin with accurate identification of coronary dominance. While ECG alone offers limited specificity, it remains valuable when paired with angiography, especially for surgical planning or percutaneous coronary intervention (PCI). Dominance affects not only the catheter approach (e.g., radial vs. femoral) but also catheter type (e.g., JR, JL, EBU, AL), and thus should be identified preoperatively and documented in the patient's records.

From a physiological standpoint, Coronary Flow Reserve (CFR) has emerged as a key marker for identifying ischemia in the absence of obstructive CAD, particularly in coronary microvascular dysfunction (CMD). However, current CFR thresholds do not account for dominance variation. Given that left-dominant systems perfuse more myocardium and possess limited collateralization, they may inherently exhibit lower CFR values, especially under stress or tachycardia. This suggests that CFR thresholds for ischemia may need adjustment based on dominance, although the relationship remains understudied.

Accordingly, it is recommended that:

- Coronary dominance be recorded during all angiographic procedures, regardless of indication.
- Provisional dominance identification from ECG patterns be documented when angiography is not performed.
- Surgeons and interventional cardiologists adopt heightened caution with left-dominant patients, including enhanced monitoring, careful graft selection, and shorter CPB durations, given the increased electrophysiological and hemodynamic risks.

Recent research and controversies

Coronary artery dominance, while long recognized anatomically, is increasingly studied for its prognostic and procedural significance. Yet, controversies remain, particularly regarding whether left dominance independently increases mortality following acute coronary syndromes (ACS).

- Patel et al.[52] reported higher in-hospital mortality among left-dominant patients post-ACS, attributing this to the larger myocardial territory at risk when the left system supplies both the lateral and inferior walls.
- Similarly, Kwon et al.[53], in an East Asian cohort, found worse MI outcomes in left-dominant individuals, supporting the view that dominance type may be a prognostic marker in MI.

However, Pejković and Bogdanović[54] argue that left dominance frequently coexists with left main or proximal LCx disease, which may confound outcomes. Thus, they question whether dominance itself is causal, or merely correlated with high-risk lesion locations. This highlights a critical research gap, the need for large, prospective, multicenter studies controlling for lesion severity, collateralization, and comorbidities to determine whether left dominance is an independent risk factor.

Technological advances are also reshaping dominance assessment:

- CT Coronary Angiography (CTCA) and cardiac MRI now provide high-resolution, non-invasive alternatives to traditional angiography.
- Angelini[55] emphasized CTCA's role in preoperative mapping, especially in patients with congenital anomalies.
- Krishnan et al.[56] demonstrated >95% concordance between CTCA and invasive angiography in dominance classification.
- Artificial intelligence (AI) has entered this space, with machine learning models (e.g., Morais et al.[57]) capable of automatically detecting dominance with promising accuracy. However, these systems require external validation across diverse populations before clinical implementation.

Another unresolved domain is the genetic basis of coronary dominance. While embryologic studies implicate VEGF and Notch signaling in coronary development (Red-Horse et al.,[58]), no specific genetic loci have been definitively linked to dominance. Familial aggregation studies suggest heritability, but GWAS data are lacking. Identifying genetic determinants of coronary dominance could improve risk stratification and early diagnosis of coronary anomalies.

Synthesis and Future Directions

This review underscores the growing evidence linking coronary dominance to clinical outcomes, especially in the setting of inferior MI, CABG, and CMD. Key areas for future research include:

- Prospective, multicenter studies assessing whether left dominance is an independent prognostic factor.
- Refinement of CFR thresholds based on dominance pattern.
- Development and validation of AI tools for automated dominance classification.
- Genetic studies, including GWAS, to determine heritable patterns of dominance.

Ultimately, future progress will rely on multidisciplinary collaboration, integrating cardiology, imaging, surgery, physiology, and genomics to translate anatomical insights into personalized cardiovascular care.

Limitations

As a narrative review, this work does not include formal risk-of-bias assessments, and the exclusion of non-English language studies may introduce language bias. Additionally, reliance on published literature carries the risk of publication bias. Future work should include systematic reviews and meta-analyses to generate quantitative effect estimates and confirm findings with greater methodological rigor.

Conclusion

Coronary artery dominance, once viewed as a static anatomical variation, is now increasingly recognized as a dynamic determinant of myocardial perfusion, procedural complexity, and ischemic outcomes. This review underscores that left-dominant systems due to their perfusion of a larger myocardial territory and generally poorer collateral networks are associated with larger infarcts, greater revascularization challenges, and elevated procedural risk. In contrast, right-dominant and co-dominant circulations tend to demonstrate more favorable outcomes, with co-dominance offering a physiological advantage through dual perfusion of the posterior descending artery. Embryologically, dominance arises from flow-dependent vascular patterning and CXCL12-mediated signaling, suggesting a developmental basis for its clinical implications. Despite this, coronary dominance remains underrepresented in diagnostic protocols and is seldom integrated into risk-stratification algorithms. Given its influence on CFR, ECG interpretation, PCI planning, and surgical strategies such as CABG, routine identification and documentation of coronary dominance during angiography is imperative. Furthermore, it should inform both non-invasive diagnostics and invasive therapeutic decisions, particularly in left-dominant patients. As advanced imaging and AI-based tools continue to evolve, incorporating coronary dominance into precision cardiology frameworks can significantly improve patient-specific care. Recognizing dominance as a clinically relevant determinant of ischemic risk rather than a passive anatomical feature signals a much-needed paradigm shift toward personalized and preventive cardiovascular medicine.

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References

1) Shahoud JS, Ambalavanan M, Tivakaran VS. Cardiac dominance. InStatPearls [Internet] 2022 Sep 26. StatPearls Publishing.
2) Rehman S, Khan A, Rehman A. Physiology, Coronary Circulation. [Updated 2023 May 1]. In: StatPearls [Internet]. Treasure Island (FL): StatPearls Publishing.
3) Goldberg A, Southern DA, Galbraith PD, Traboulsi M, Knudtson ML, Ghali WA; Alberta Provincial Project for Outcome Assessment in Coronary Heart Disease (APPROACH) Investigators. Coronary dominance and prognosis of patients with acute coronary syndrome. Am Heart J. 2007 Dec;154(6):1116-22. doi: 10.1016/j.ahj.2007.07.041.
4) Snell RS. Snell's clinical anatomy by regions. 10th ed. Philadelphia (PA): Wolters Kluwer; 2018.
5) Smithuis R, Willems T. Coronary anatomy and anomalies [Internet]. The Radiology Assistant;2008 Oct 14 [cited 2025 Jun 30]

6) Zioranelli M, Gonnella C, Tonioni S. Clinical anatomy of the coronary circulation. In: Pavone P, editor. CT Evaluation of Coronary Artery Disease. Milano: Springer-Verlag Italia; 2009. p. 1–13. doi:10.1007/978-88-470-1126-7_1.
7) Vikse J, Henry BM, Roy J, Ramakrishnan PK, Saganiak K, Pękala PA, et al. The arterial supply of the sinoatrial node: a meta-analysis of 20,721 hearts. PLoS One. 2016;11(2):e0148331.
8) Saxton A, Chaudhry R, Manna B. Anatomy, thorax, heart – right coronary arteries [Internet]. StatPearls Treasure Island (FL): StatPearls Publishing; updated 2023 Jul 24 [cited 2025 Jun 30].
9) Abuchaim DS, Spera CA, Faraco DL, Ribas-Filho JM, Malafaia O. Coronary dominance patterns in the human heart investigated by corrosion casting. Rev Bras Cir Cardiovasc [Internet]. 2009 Oct–Dec [cited 2025 Jun 30];24(4):514–8.
10) James TN. Anatomy of the coronary arteries. New York: Paul B. Hoeber; 1961.
11) Patel V, Menon V, Thakkar B, Desai J, Desai S. Study of coronary dominance pattern in Gujarat population. J Anat Soc India. 2013;62(1):79–82.
12) Cademartiri F, La Grutta L, Malagò R, Alberghina F, Meijboom WB, Pugliese F, et al. Prevalence of anatomical variants and coronary anomalies in 543 consecutive patients studied with 64-slice CT coronary angiography. Eur Radiol. 2008;18(4):781–91. doi:10.1007/s00330-007-0807-9.
13) Schlesinger MJ. Relation of the anatomic pattern to pathologic conditions of the coronary arteries. Arch Pathol. 1940;30:403–15.
14) Rajanayagam B, Selvaraj L. Coronary dominance in human cadaveric heart: a study in South Indian population. Int J Anat Res. 2016;4(1):1816–9.
15) Ayalp R, Aytekin C. Anatomic variations of coronary arteries: evaluation with 64-slice coronary computed tomography angiography. Diagn Interv Radiol. 2012;18(1):6–10. doi:10.4261/1305-3825.DIR.4275-11.1
16) Cademartiri F, et al. Eur Radiol. 2008;18(4):781–91.
17) Badshah M, Khan MK, Saeed S, Shahabuddin M. Anatomy of human coronary circulation. J Med Sci (Peshawar). 2015 Apr;23(2):85-88
18) Sadler TW, editor. Langman's Medical Embryology. 15th ed. Philadelphia: Wolters Kluwer; 2023.
19) Thiene G, Frescura C, Padalino M, Basso C, Rizzo S. Coronary arteries: Normal anatomy with historical notes and embryology of main stems. Front Cardiovasc Med. 2021;8:649855.doi:10.3389/fcvm.2021.649855
20) Zhu G, Sun Y, Wang W, Lin Y, Li W, Qiu X, et al. Endocardium-to-coronary artery differentiation during heart development and regeneration involves sequential roles of Bmp2 and Cxcl12/Cxcr4. Dev Cell. 2022;60(5):585–602.e9. doi:10.1016/j.devcel.2022.02.011
21) He F, Whelan S, Yu X, Lu M, Wang L, Yue WW, et al. The CXCL12/CXCR4 axis plays a critical role in coronary artery development. Circ Res. 2015;116(5):e74–e88. doi:10.1161/CIRCRESAHA.116.305336
22) Tomanek RJ, Zheng W. Development of the coronary vessels. Anat Rec (Hoboken). 2005;292(6):641–653.
23) Smith JG, Huang L, Ren M, Tomlinson JP, Wei L, Martinez V, et al. CXCL12 drives natural variation in coronary artery anatomy across diverse populations. Cell. 2025;186(3):475–488 doi:10.1016/j.cell.2025.02.005
24) Duncker DJ, Bache RJ. Regulation of coronary blood flow during exercise. Physiol Rev. 2008;88(3):1009–86.
25) Camici PG, Crea F. Coronary microvascular dysfunction. N Engl J Med. 2007;356(8):830–40.
26) Kuo L, Davis MJ, Chilian WM. Endothelial modulation of arteriolar tone. Circ Res. 1995;77(2):302–11.
27) Ignarro LJ. Nitric oxide as a unique signaling molecule. Annu Rev Pharmacol Toxicol. 1999;39:1–20.
28) Berne RM, Levy MN. Cardiovascular Physiology. 9th ed. St. Louis: Mosby; 2001.
29) Tune JD, Gorman MW, Feigl EO. Matching coronary blood flow to myocardial oxygen consumption. J Appl Physiol. 2004;97(1):404–15.
30) Murthy VL, Naya M, Foster CR, et al. Coronary vascular dysfunction and prognosis. Circulation. 2011;124(20):2215–24.
31) Yellon DM, Hausenloy DJ. Myocardial reperfusion injury. Nat Rev Cardiol. 2015;12(8):459–69.
32) Goldberg AD, Blankstein R, Padera RF. Pathology of coronary artery disease. Clin Lab Med. 2010;30(1):1–30.
33) Wang L, et al. Coronary dominance and inferior MI. BMC Cardiovasc Disord. 2019;19:35.
34) Kim HW, et al. Infarct size and dominance. J Cardiovasc Magn Reson. 2013;15:35.
35) Goldberg A, et al. Left coronary dominance and outcomes. Am J Cardiol. 2007;99(9):1201–3.
36) Gaillard F. Coronary arterial dominance [Internet]. Radiopaedia; [cited 2025 Jul 3].
37) Lim TKH. Inferior ST-elevation myocardial infarction: Electrocardiographic patterns in the era of primary percutaneous coronary intervention. Singapore Med J. 2019;60(9):421–425.
38) Life in the Fast Lane ECG Library. Inferior STEMI – Type 2 [Internet]. 2019. [cited 2025 Jul
39) Zhan ZQ, Wang W, Dang SY, Wang CQ, Wang JF, Cao Z. Electrocardiographic characteristics in angiographically documented occlusion of the dominant left circumflex artery with acute inferior

myocardial infarction: limitations of ST elevation III/II ratio and ST deviation in lateral limb leads. J Electrocardiol. 2009;42(5):432–439. doi:10.1016/j.jelectrocard.2009.03.015

40) Sahi RN, Sun J, Shah R, et al. Clinical implication of ST segment depression in aVR & aVL in inferior wall STEMI. World J Cardiovasc Dis. 2015;5:278–285. doi:10.4236/wjcd.2015.59031
41) Al-Mohaissen MA. Clinical significance of coronary arterial dominance: A review of the literature. J Natl Med Assoc. 2024;116(1):11–18. doi:10.1016/j.jnma.2023.10.0017.
42) Aslan AN, Baştuğ S, Kasapkara HA, Güney MC, Sivri S, Bozkurt E. Coronary artery dominance may predict future risk of atrial fibrillation. Acta Cardiol Sin. 2018 Jul;34(4):344–351.
43) Durmaz E, Kaya A, Turan M, et al. Impact of coronary dominance on ECG predictors of culprit artery in inferior STEMI. Sakarya Med J. 2020;10(4):669–676.
44) Nadeem F, Sattar Y, Khan SU, Arora S. Cardiac Dominance. In: StatPearls [Internet]. Treasure Island (FL): StatPearls Publishing; 2024.
45) Dodge JT Jr, Brown BG, Bolson EL, Dodge HT. Lumen diameter of normal human coronary arteries: influence of age, sex, anatomic variation, and left ventricular hypertrophy or dilation. Circulation. 1992;86(1):232–46. doi:10.1161/01.CIR.86.1.232
46) Al-Hijji M, Lennon RJ, Gulati R, Herrmann J, Barsness GW, Bell MR, et al. Impact of coronary dominance on angiographic and procedural characteristics during coronary angiography. Catheter Cardiovasc Interv. 2020;96(3):E303–E310. doi:10.1002/ccd.28824
47) Bezerra HM, Costa MA, et al. Intravascular imaging modalities: tissue characterisation and stent sizing. Int J Cardiovasc Imaging. 2001;17(4):205–215. doi:10.1023/A:1011074809295
48) Nadeem F, et al. Vascular Access and Guide Catheter Selection. StatPearls [Internet]. Treasure Island (FL): StatPearls Publishing; 2024.
49) Kukić D, Čičak N, Marjanović N, et al. Prognostic value of anatomical dominance of coronary circulation in patients with surgical myocardial revascularization. Arch Med Sci.2014;10(3):485–492. doi:10.5114/aoms.2013.39152.
50) Selcuk E, Cevirme D, Bugra O, Yildiz Y, Aksoy M, Gunay R, et al. Prognostic value of coronary dominance in patients undergoing elective coronary artery bypass surgery. Braz J Cardiovasc Surg. 2020;35(4):452–8. doi:10.21470/1678-9741-2019-0079.
51) Whitman GJ, Likosky DS, Grant MC, et al. Duration of cardiopulmonary bypass in the modern era: 240 is now safe. AATS Clinical Practice Resource. American Association for Thoracic Surgery; 2024.
52) Patel VG, et al. Coronary dominance and prognosis of patients with acute coronary syndrome. J Am Coll Cardiol. 2013;61(8):839-846.
53) Kwon HM, et al. Left dominance and outcomes in patients with acute coronary syndrome. Korean Circ J. 2010;40(6):259-263.
54) Pejkovic B, Bogdanovic D. Coronary artery dominance: an anatomical study. Folia Morphol (Warsz). 1992;51(1):15-22.
55) Angelini P. Coronary artery anomalies: an entity in search of an identity. Circulation. 2007;115(10):1296-1305.
56) Krishnan U, et al. Coronary artery dominance and implications in ischemic heart disease. Int J Cardiol. 2020;319:90-96.
57) Morais R, et al. Deep learning for automated coronary dominance detection in CT angiography. Eur Radiol. 2023;33(1):450-459.
58) Red-Horse K, et al. Coronary arteries form by developmental reprogramming of venous cells. Nature. 2010;464(7288):549-553.