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Right bundle-branch block (RBBB) and acute coronary syndrome (ACS) a narrative review

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ABSTRACT

Right bundle-branch block (RBBB) is a common electrocardiographic finding that may be benign or may signify acute myocardial ischemia, particularly in patients presenting with chest pain. This narrative review examines the pathophysiology, diagnostic challenges, and clinical implications of new-onset RBBB in acute coronary syndrome (ACS). Acute RBBB often indicates proximal left anterior descending artery occlusion, leading to larger infarcts, higher rates of cardiogenic shock, ventricular arrhythmias, and mortality. We discuss mechanisms involving septal perforator ischemia, differentiate benign RBBB from pathological causes through clinical history, ECG features (e.g., rsR' pattern, appropriate discordant T-wave changes), and advanced testing. Key ECG red flags for occlusion myocardial infarction include concordant ST elevation, excessive discordant ST elevation (>25 % of QRS amplitude), reciprocal changes, and new bifascicular block. Evidence from meta-analyses, cohorts, and guidelines (e.g., 2017 ESC STEMI, 2022 ACC Chest Pain) supports treating new RBBB with ischemic symptoms as a STEMI equivalent, warranting urgent reperfusion. Limitations include observational data and lack of RBBB-specific validated criteria. Clinicians should use serial ECGs, troponins, and low-threshold angiography. This review underscores the need for vigilant ECG interpretation in emergency settings to improve outcomes. (198 words).

1. Introduction

Right bundle-branch block (RBBB) is a common electrocardiographic conduction abnormality that may be encountered as an incidental finding or may appear acutely in the setting of myocardial ischemia. In the general population, the prevalence of RBBB ranges from 0.2 % to 1.3 %, with higher rates observed in men (1.4 %) compared to women (0.5 %) and a marked increase with advancing age—from approximately 1 % at age 50 years to 17 % at age 80 years. Among patients with structural heart disease, such as those with heart failure, the prevalence is substantially higher, approaching 10–11 %, reflecting underlying conduction system involvement or comorbidities like pulmonary disease. In individuals with ischemic heart disease, RBBB is also more frequent and carries prognostic implications, with studies indicating 64 % increased odds of in-hospital mortality associated with its presence [1,4].

The electrocardiographic appearance of incidentally discovered nonacute RBBB is typically benign and consistent, featuring a prolonged QRS duration exceeding 120 milliseconds, a triphasic rsR' or rSR' pattern in the right precordial leads (V1-V2) due to delayed right ventricular activation, and a broad, slurred S wave in the lateral leads (I, aVL, V5-V6) (Fig. 1 and 2). Secondary repolarization abnormalities are common but nonspecific, manifesting as ST-segment depression and T-wave inversion in the right precordial leads (V1-V3), which result from the altered sequence of ventricular depolarization rather than ischemia.

New or acute RBBB in the context of ischemic symptoms, however, is associated with larger infarct size, higher rates of cardiogenic shock, ventricular arrhythmia, and increased short- and long-term mortality, often serving as a marker of proximal left anterior descending artery (LAD) involvement. Multiple observational studies and meta-analyses show that AMI patients with new RBBB have worse outcomes than patients without RBBB, and new RBBB is frequently associated with occlusion of the infarct-related artery (commonly the LAD or its septal branches) [1,4,6]. Early recognition of an occlusion-myocardial infarction (OMI) beneath RBBB is therefore critical for timely reperfusion.

Because RBBB alters repolarization, it can mask or mimic ST-segment changes; therefore, clinicians should apply structured criteria (analogous to Sgarbossa/Smith rules used for LBBB) and look for concordant ST elevation, excessive discordant ST elevation, reciprocal changes, and dynamic ECG evolution [2,3]. This review discusses the underlying mechanisms, key ECG findings distinguishing benign RBBB from ischemic RBBB, common culprit vessels, supporting clinical evidence, and

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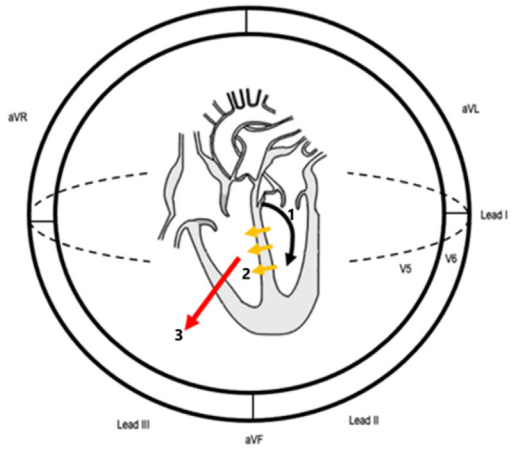


Fig. 1. Sequence of conduction in RBBB
1) Left ventricular activation via the left bundle (black arrow 1) occurs normally producing the S wave in V1 and R wave in V6 (Fig. 2).
2) Septal depolarisation (yellow arrows) is thus unaffected, producing a normal early QRS complex – Figure to showing corresponding 2 wave in V1 and the Q wave in V6 marked as 2 in Fig. 2.
3) Activation of the RV originates across the septum. The resultant depolarisation vector (red arrow) produces delayed R waves in leads V1–3, and S waves in V6 marked as 3 in Fig. 2.
(Image Fig. 1 adapted from Life in the Fast Lane (LITFL), ECG Library – Cardiac Conduction System, accessed October 12, 2025. Used under CC BY 4.0 license.)

the implications for early diagnosis and reperfusion management in acute coronary occlusion.

2. Pathophysiology of RBBB in ACS

- **Anatomy & conduction**: The right bundle branch runs along the interventricular septum and is supplied primarily by septal perforators arising from the left anterior descending (LAD) artery in most patients. Ischemia of the septum (proximal LAD occlusion) or direct injury to the conduction system can produce a new RBBB. (Fig. 3)
- **Mechanisms during AMI**: acute ischemia of the right bundle (septal branch territory), transmural anterior septal infarction, or extensive myocardial injury producing conduction slowing can produce RBBB. RBBB therefore can be a marker of larger infarct size or proximal occlusion [1].

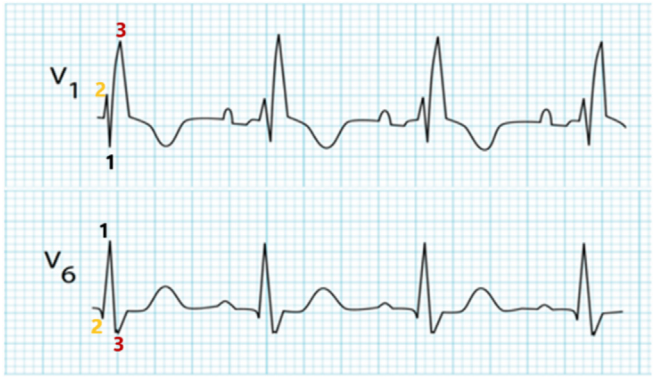


Fig. 2. Sequence of conduction in RBBB
1) Left ventricular activation via the left bundle (black arrow 1) occurs normally producing the S wave in V1 and R wave in V6 (Fig. 2).
2) Septal depolarisation (yellow arrows) is thus unaffected, producing a normal early QRS complex – Figure to showing corresponding 2 wave in V1 and the Q wave in V6 marked as 2 in Fig. 2.
3) Activation of the RV originates across the septum. The resultant depolarisation vector (red arrow) produces delayed R waves in leads V1–3, and S waves in V6 marked as 3 in Fig. 2.

3. Causes of RBBB other than ACS

RBBB occurs when there's a delay or block in the electrical conduction through the right bundle branch of the heart's conduction system, leading to a widened QRS complex on ECG (typically ≥120 ms for complete RBBB). It can be complete or incomplete (QRS 100–120 ms), and causes are broadly categorized as cardiac, degenerative, iatrogenic/traumatic, or other/miscellaneous (Table 1). Below is your list with minor clarifications or additions based on common associations for completeness:

3.1. Cardiac causes

- Congenital heart disease:
  - Atrial septal defect (secundum type – classically associated)
  - Tetralogy of Fallot, other repaired CHD: Post-surgical scarring can contribute.
- Right-sided pressure/volume overload:
  - Pulmonary embolism (acute cor pulmonale)
  - Pulmonary hypertension / chronic cor pulmonale: Leads to right ventricular hypertrophy (RVH).
- Cardiomyopathy:
  - Dilated cardiomyopathy
  - Arrhythmogenic right ventricular cardiomyopathy (ARVC): Involves fibrofatty replacement in the right ventricle.
- Valvular heart disease (tricuspid, pulmonary): Can cause right ventricular strain.
- Myocarditis, endocarditis, post-cardiac surgery: Inflammatory or infectious processes damaging the conduction tissue.

3.2. Degenerative / conduction system disease

- Fibrosis or calcification of conduction system (Lenègre/Lev disease): Age-related sclerosis or fibrosis specific to the conduction pathways.
- Age-related conduction degeneration: Common in older adults without other pathology.

3.3. Iatrogenic / trauma

- After right heart catheterization: Direct mechanical disruption.
- Post valve replacement or septal myectomy: Surgical trauma to the bundle.
- Chest trauma: Blunt force impacting the heart.

3.4. Others

- Electrolyte disturbance (rare – hyperkalemia): Can transiently affect conduction.
- Idiopathic (isolated RBBB in healthy individuals, especially incomplete RBBB, often benign): No identifiable cause; considered a normal variant in some cases.
- Additional rare causes not in your list: Infiltrative diseases (e.g., sarcoidosis, amyloidosis), rheumatic heart disease, or high blood pressure leading to structural changes

4. How to identify benign RBBB in healthy individuals vs. from other causes

Benign (or isolated) RBBB is often an incidental finding on ECG in asymptomatic people without underlying heart disease, and it's

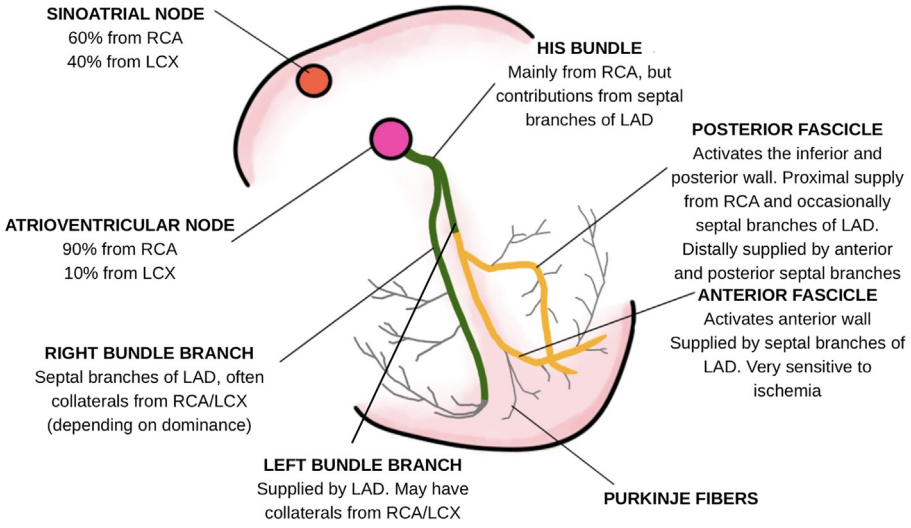


Fig. 3. Cardiac conduction system and their blood supply. – (Image courtesy Dr. Gokul Nath R, used with permission).

generally not associated with increased risk of adverse outcomes in the absence of other factors. It's more common in younger individuals (e.g., athletes or those under 40) and can be incomplete rather than complete. Distinguishing it from pathological causes requires a systematic approach focusing on clinical context, ECG details, and further evaluation to rule out structural or progressive issues. Here's a step-by-step guide based on clinical practice:

4.1. Clinical history and symptoms

- **Benign**: Asymptomatic or found incidentally (e.g., during routine ECG for sports clearance or check-up). No history of chest pain, shortness of breath, syncope, palpitations, or family history of sudden cardiac death.
- **Pathological:** Associated symptoms like dyspnea, fatigue, or edema suggesting underlying issues (e.g., heart failure, pulmonary embolism). History of heart disease, recent trauma, surgery, or risk factors like hypertension, diabetes, or smoking.

4.2. Physical examination

- **Benign**: Normal exam with no signs of heart failure (e.g., no jugular venous distension, edema, or abnormal heart sounds).
- **Pathological**: Signs of right heart strain (e.g., loud P2 heart sound in pulmonary hypertension) or other cardiac abnormalities.

4.3. ECG characteristics:

Benign: Isolated RBBB with typical features—rsR' or rSR' pattern in V1-V2, wide/slurred S wave in I/V5-V6, QRS duration 120–140 ms for complete (or < 120 ms for incomplete). No additional abnormalities like ST-segment changes, Q waves, or axis deviation. Discordant T-wave changes are appropriate (inverted in V1-V3).

5. Expected ECG appearance with isolated RBBB (not ischemic)

- QRS duration ≥120 ms with typical **rsR'** pattern in V1–V3 (terminal R′) and wide S waves in lateral leads (I, V6).

Table 1
Causes of RBBB.

Category	Specific Causes	Notes / Mechanism
Cardiac Causes	Ischemic heart disease / ACS (proximal LAD occlusion)	Infarction affecting the right bundle branch
	Congenital heart disease	ASD (secundum type), Tetralogy of Fallot, other repaired CHD; post-surgical scarring may contribute
	Right-sided pressure/volume overload	Pulmonary embolism (acute cor pulmonale), Pulmonary hypertension, chronic cor pulmonale → RVH
	Cardiomyopathy	Dilated cardiomyopathy, ARVC (fibrofatty replacement in RV)
	Valvular heart disease	Tricuspid or pulmonary valve disease → RV strain
	Myocarditis / Endocarditis / Post-cardiac surgery	Inflammatory or infectious damage to conduction tissue
Degenerative / Conduction System Disease	Fibrosis or calcification (Lenègre/Lev disease)	Age-related sclerosis/fibrosis of conduction pathways
	Age-related conduction degeneration	Common in older adults without other pathology
Iatrogenic / Trauma	Right heart catheterization	Mechanical disruption of the right bundle
	Valve replacement / Septal myectomy	Surgical trauma to conduction system
	Chest trauma	Blunt cardiac injury affecting the bundle
Other / Miscellaneous	Electrolyte disturbances (rare – e.g., hyperkalemia)	Transient conduction abnormalities
	Idiopathic / isolated RBBB	Often incomplete RBBB in healthy individuals; usually benign
	Infiltrative diseases	Sarcoidosis, amyloidosis → conduction tissue infiltration
	Rheumatic heart disease	Structural changes affecting RV conduction
	Hypertension / Structural remodeling	LV or RV hypertrophy may indirectly affect conduction

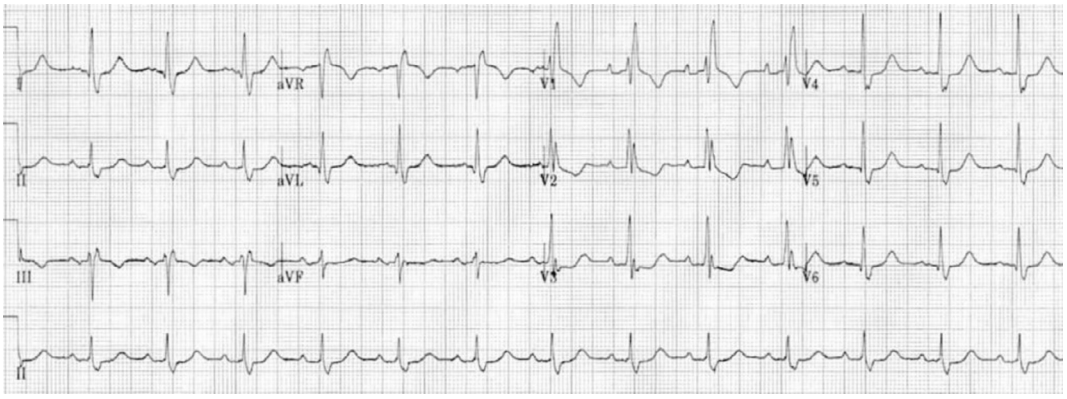


Fig. 4. Typical RSR' pattern in V1–2, Widened S waves again demonstrated in lateral leads, especially V4–6, Appropriate discordance in leads V1–2.

- Secondary ST-T **changes** in leads V1–V3: modest **ST depression** and **T-wave inversion** (discordant with the terminalR′). These are typical repolarisation changes and are usually stable over time (Fig. 4).

6. ECG features that should raise concern for ACS/occlusion in the presence of RBBB

(Apply clinical context — chest pain, haemodynamic instability, arrest — and look for these ECG red flags.) (Table 2).

- **New or presumed-new RBBB** in a patient with ischemic symptoms — treat as STEMI equivalent, as it often reflects proximal LAD occlusion. Acute RBBB has associated with higher mortality than LBBB STEMI equivalent [9,10].
- **New Onset Bifascicular Block New** RBBB and LAFB is highly associated with proximal LAD occlusion and negative outcomes. It should always raise the suspicion for OMI Occlusion MI and you should look for subtle ST Changes which may be more difficult to discern [3]. (Fig. 7).
- **Concordant ST-segment elevation** ST-elevation in the same direction as the QRS complex in a lead (e.g., ST elevation in V1–V3 despite R′) is highly specific for acute occlusion (analogous to concordant Sgarbossa finding) [2,3]. (Fig. 8).
- **Excessive discordant ST elevation** — large ST elevation that is out of proportion to the QRS (Smith-modified Sgarbossa style argument: e.g., ST elevation >25 % of preceding S-wave amplitude in discordant leads) suggests occlusion and is more sensitive than the original discordant rule. Though derived for LBBB/paced rhythms, the proportionality concept can be applied to any conduction abnormality when interpreting for OMI [3,8]. (Fig. 9).
- **ST elevation in contiguous non-anterior leads** (e.g., inferior or lateral leads) that cannot be explained by RBBB secondary changes. Clear ST elevation in II/III/aVF or V5–V6 should prompt reperfusion evaluation.
- **Reciprocal ST depression** in opposing leads supports true STEMI rather than benign RBBB repolarisation.
- **Dynamic changes on serial ECGs** (evolving ST changes, new Q waves) increase the likelihood of acute occlusion.

7. ECG features that should raise concern for other pathologies in the presence of RBBB

Pathological QRS >140 ms suggests additional conduction issues or ventricular hypertrophy. Look for confounders like:

- **Brugada pattern**: coved ST elevation (>2 mm) and J-point elevation in V1-V2 (type 1) or saddleback ST (type 2). (Fig. 5).
- **ARVD or RV enlargement**: Additional right-axis deviation, epsilon waves, or T-wave inversions beyond V1-V3. (Fig. 6).
- **Incomplete RBBB (iRBBB)** is often benign in healthy young people but requires differentiation from these patterns.

7.1. Further diagnostic tests

Benign: Normal results on echocardiography (no RVH, normal ejection fraction, no valvular issues), Holter monitor (no arrhythmias), or exercise stress test (if indicated for athletes).

Pathological: Abnormal findings like RV dilation on echo (suggesting cor pulmonale), reduced ejection fraction (cardiomyopathy), or evidence of ischemia on stress testing/angiography. Blood tests for electrolytes, troponins, or BNP if acute causes suspected.

Advanced imaging (e.g., cardiac MRI) if ARVC or infiltrative disease is a concern.

7.2. Risk stratification and follow-up

Benign: No treatment needed; periodic ECG monitoring if young or athlete. Prognosis is excellent with low risk of progression.

Pathological: Address underlying cause (e.g., anticoagulation for PE, beta-blockers for cardiomyopathy). May have higher risk of arrhythmias or heart failure, warranting closer follow-up.

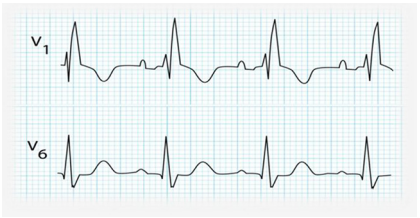
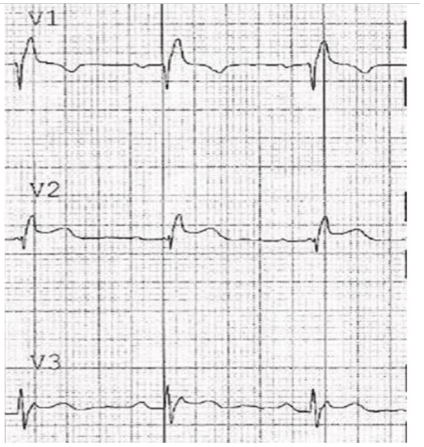
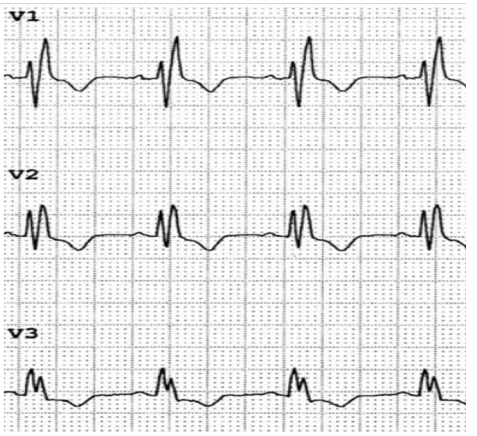
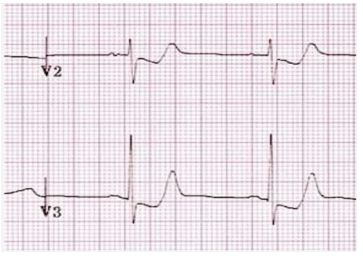
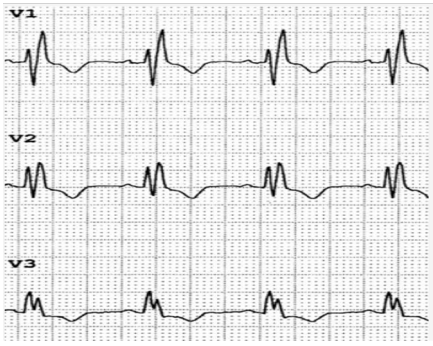
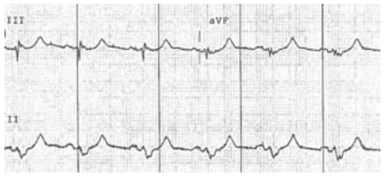
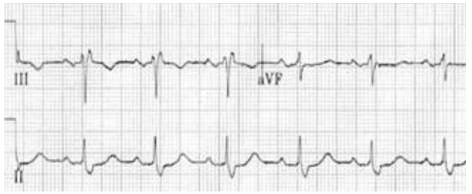
In summary**, benign RBBB is a diagnosis of exclusion** after ruling out structural, ischemic, or other causes through history, ECG scrutiny, and targeted tests. Consult a cardiologist for personalized evaluation, especially if there's any doubt.

8. ECG effects of RBBB: expected (non-ischemic) vs abnormal (suspicious for ACS)

8.1. Culprit vessels and mechanistic correlations

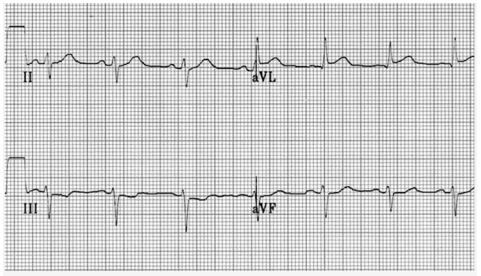
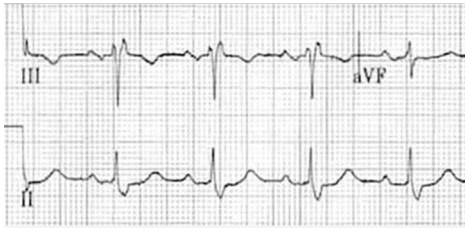
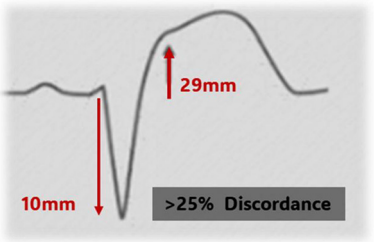
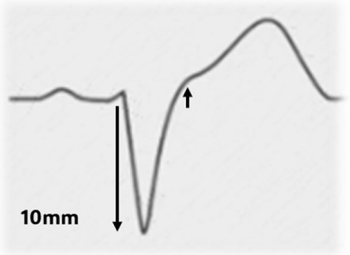
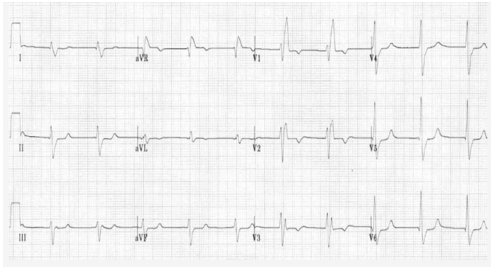
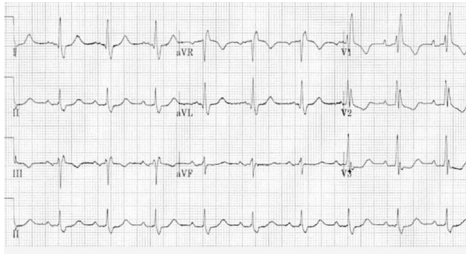
- **Proximal LAD / first septal perforator**: most commonly implicated when RBBB appears with anterior ischemia; septal branches supply the right bundle in many hearts, so proximal LAD occlusion often produces RBBB. Multiple case series and pooled analyses have found a frequent association between new RBBB in AMI and LAD involvement [1,5].
- **Left main or multivessel disease** may also present with conduction disturbances including RBBB, particularly when infarction is extensive [11].

Table 2
ECG effects of RBBB: expected (non-ischemic) vs abnormal (suspicious for ACS).

ECG Feature	Suggestive of STEMI/ACS (high risk)	Incidental (Non-Ischemic) RBBB	Illustration: Acute RBBB + OMI	Illustration: Incidental RBBB
QRS morphology	Same pattern, but if new RBBB with chest pain is a STEMI equivalent	Stable, chronic rsR' or rSR' pattern in V1-V2 with QRS >120 ms; no dynamic widening or fragmentation		
ST-T in V1-V3	ST elevation (concordant with QRS or excessive >2 mm),	Secondary ST depression and T-wave inversion discordant to terminal QRS (typically <1 mm, proportionate)		
ST T in V1- V3	upright T waves (loss of discordance)	Secondary ST depression and T-wave inversion		
ST changes in other leads	Clear ST elevation in ≥2 contiguous leads (inferior: II, III, aVF; lateral: I, aVL, V5-V6)	No primary ST elevation; may have subtle secondary changes but stable and nonspecific	**Hyper-acute T waves inferiorly** 	
Reciprocal	Present (e.g., ST depression in inferior	Absent or minimal; no reciprocal depression		

(continued on next page)

Table 2 (*continued*)

ECG Feature	Suggestive of STEMI/ACS (high risk)	Incidental (Non-Ischemic) RBBB	Illustration: Acute RBBB + OMI	Illustration: Incidental RBBB
changes	leads when anterior STEMI suspected)	beyond secondary repolarization effects		
Proportionality	Excessive discordance (ST elevation >25 % of S wave depth in any lead, similar to Sgarbossa criteria)	Proportionate discordance (ST deviation <25 % of S wave depth, typically <1–2 mm)		
Bifascicular Block	RBBB + Left Axis Deviation i.e., LAFB – Bifascicular Block.	RBBB have normal Axis. Any Abnormal Axis is suggestive of a fascicular Block		

(Use serial ECGs and clinical gestalt — treat suspicious cases as possible STEMI and activate reperfusion pathways when appropriate.)

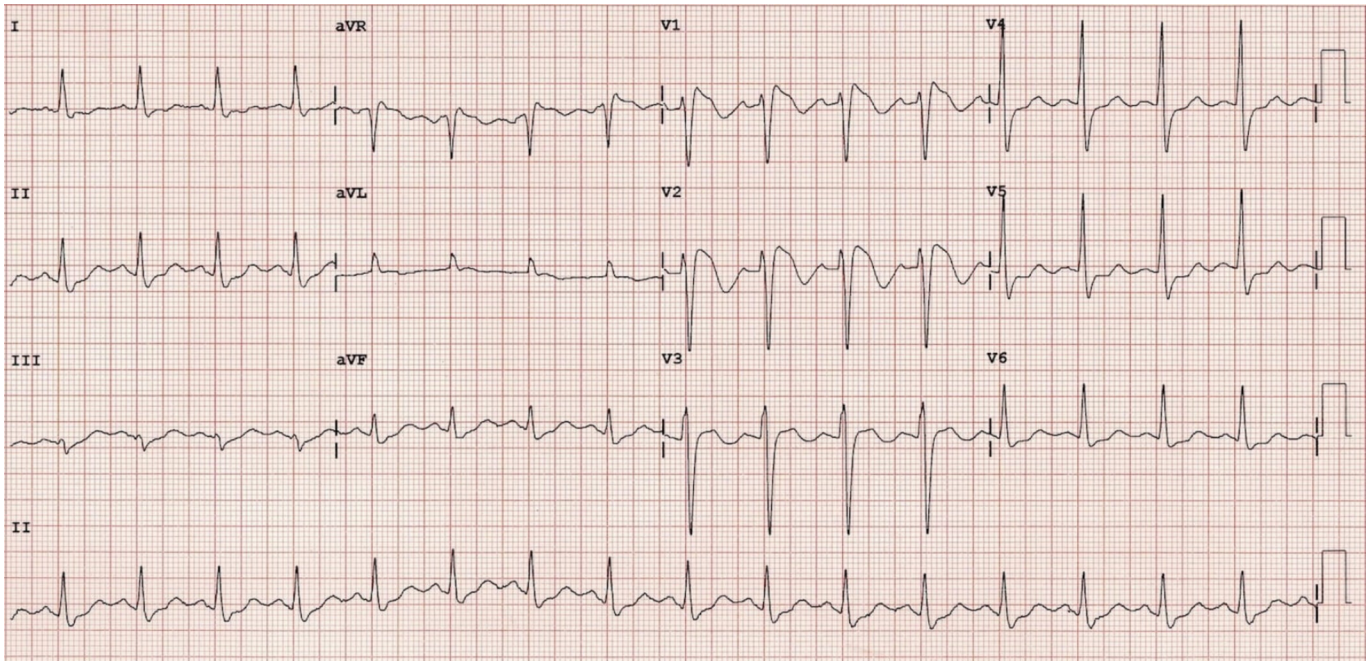


Fig. 5. Type 1 Brugada - QRS may be normal or only mildly prolonged (not as wide as typical RBBB). Instead of a clean rSR', the terminal R' is "coved" or "saddleback-shaped" with associated ST elevation, Coved ST segment elevation >2 mm in >1 of V1-V3 followed by a negative T wave.

9. Algorithmic approach to RBBB

9.1. Evidence

- **AHA/ACC 2021 Chest Pain and ACS guidelines:** also acknowledge that new or presumed new RBBB in a patient with ischemic symptoms carries a high risk of acute coronary occlusion and adverse prognosis, and should be managed with urgent reperfusion similar to STEMI [9].

- **ESC STEMI guidelines (2017):** state that new or presumed new RBBB in the presence of typical ischemic symptoms should be treated as a STEMI equivalent, because it is frequently associated with acute occlusion of the proximal LAD and larger infarcts [10].

- **Meta-analysis and cohort data:** A 2018 pooled analysis/meta-analysis and several cohort studies demonstrate that **new-onset RBBB in AMI is associated with increased long-term mortality, ventricular arrhythmias, and cardiogenic shock compared with AMI patients without RBBB.** New permanent RBBB after STEMI correlated with worse long-term outcomes in PCI cohorts [1,4].

- **Single-centre cohorts & registry analyses:** Studies report RBBB prevalence in AMI around 5–10 % with higher in-hospital mortality compared with patients without BBB. RBBB accompanying anterior STEMI has particularly poor prognosis [6,7].

- **Case reports & ECG case collections**: Multiple reported cases describe proximal LAD occlusion presenting with new RBBB and either

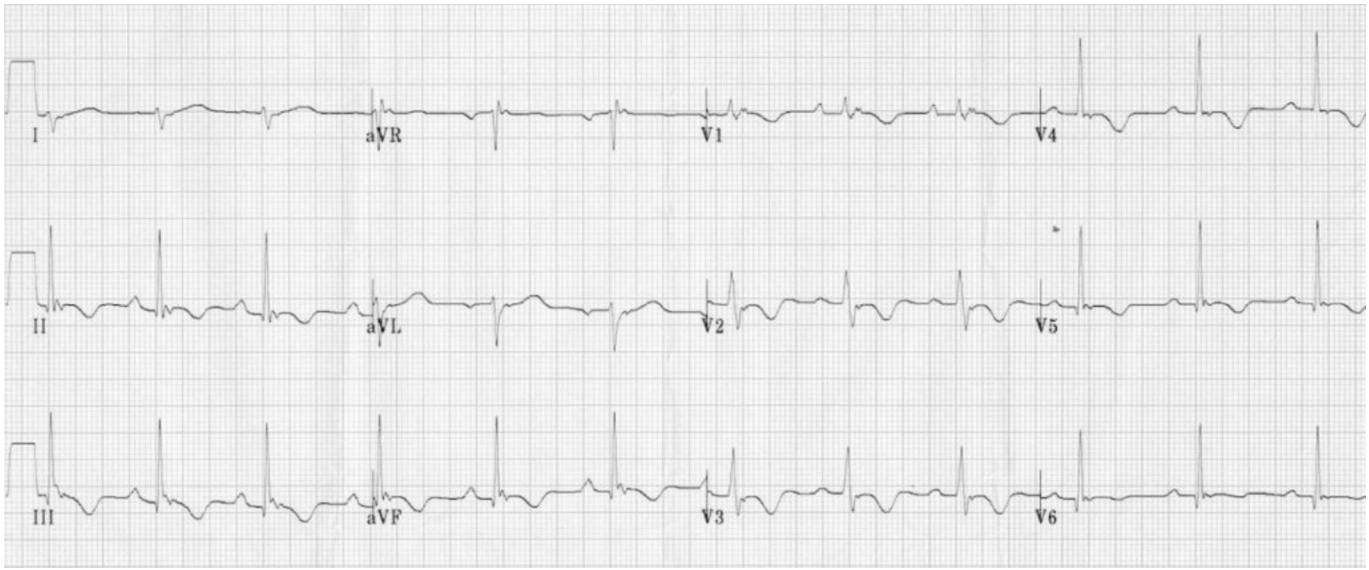


Fig. 6. ARVD - **Arrhythmogenic Right Ventricular Dysplasia - Localised** widening of QRS in V1–2, Incomplete RBBB–like pattern, but the terminal R' is often **low-amplitude** and not as sharp as classic RBBB, **Epsilon wave** (small positive deflection at the end of QRS in V1–V3) is **pathognomonic** for ARVC. **Deep T-wave inversions in V1–V3 (sometimes beyond to V4–V5)** in the absence of complete RBBB are a **major diagnostic criterion**.

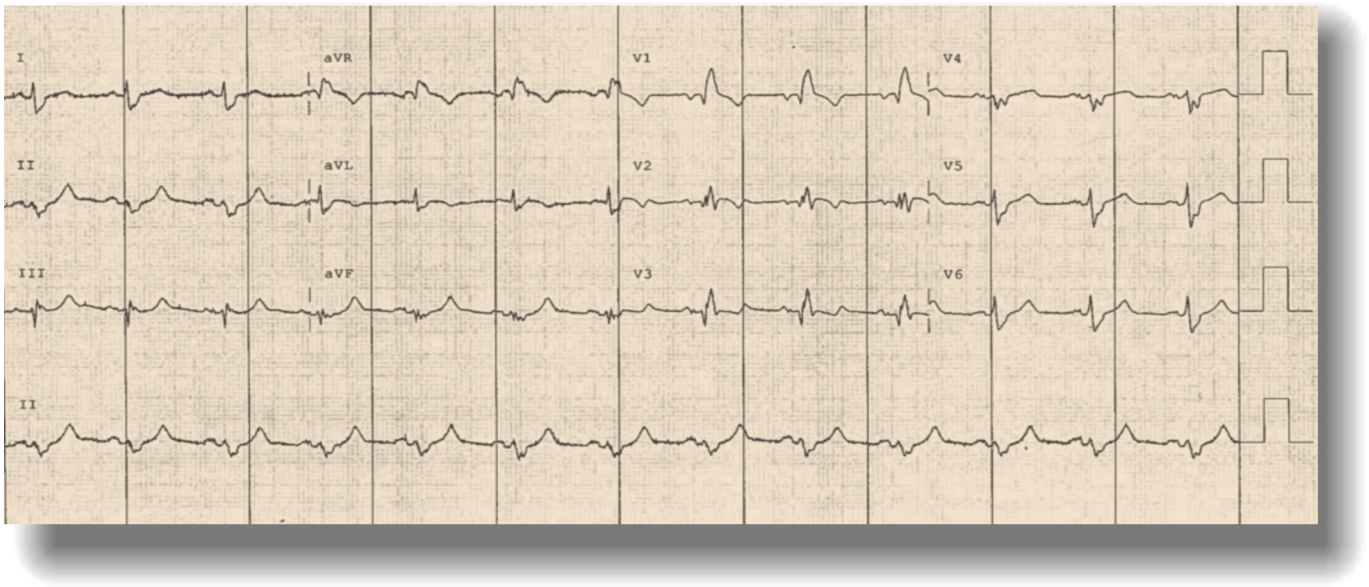


Fig. 7. ECG shows Bifascicular Block - RBBB + Left Axis Deviation i.e., LAFB – Bifascicular Block, and there is concordant STE in lead V2, all concerning for a proximal LAD occlusion.

subtle or overt ST elevation (several small series and FOAMed ECG case collections highlight that some RBBB patients have occlusion MI that would be missed without careful evaluation). These reports illustrate the range of ECG presentations and support low threshold for angiography when the ECG is suspicious [5].

9.2. High-risk clinical and ECG features — summary (actionable)

Treat as **high probability of OMI / activate reperfusion** when RBBB coexists with:

- **New or presumed-new RBBB with ischemic symptoms** (chest pain, syncope, dyspnoea, hypotension).
- **Concordant ST-elevation** in any lead (highly specific).
- **Excessive discordant ST-elevation** by proportionality rules (Smith-type logic) [3].
- **ST elevation in ≥ 2 contiguous non-anterior leads** (inferior or lateral) not explained by RBBB.
- **Reciprocal ST depression or dynamic serial changes.**

Clinical implication: Do not dismiss ST changes in RBBB as mere secondary repolarisation without considering the context. When the clinical picture is suggestive and ECG red flags are present, urgent reperfusion strategy (primary PCI when available) should be strongly considered. Guidelines and expert consensus emphasise integrating ECG and clinical findings rather than relying on conduction abnormality alone [9,10].

9.3. Practical diagnostic approach (algorithmic)

1. Evaluate symptoms & haemodynamic status. If unstable, proceed emergently to reperfusion pathways.
2. Determine whether RBBB is new or chronic (old ECGs if available). New = higher risk.
3. Look for concordant ST elevation, excessive discordant ST elevation, reciprocal changes, or ST elevation in contiguous leads outside expected RBBB pattern. Apply Smith-style proportional assessment for discordant changes [3] (Fig. 10 and 11) (Table 2).

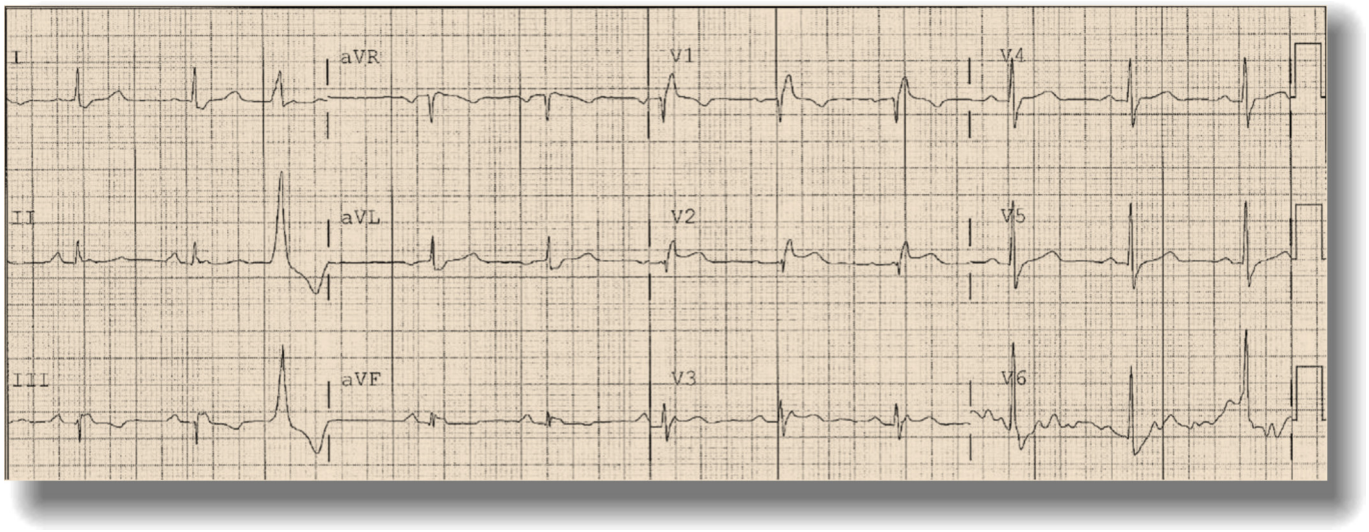


Fig. 8. ECG shows STE in V2 and V3 concordant with the R' wave with RBBB Morphology. This was an acute LAD occlusion.

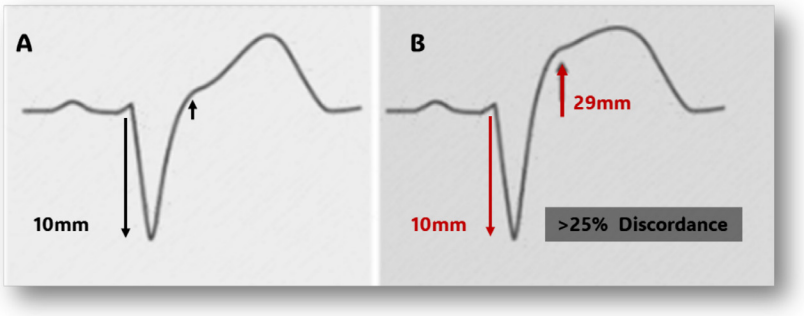


Fig. 9. Excessive discordant ST elevation- A- RBBB showing secondary repolarization changes with minimal discordant ST-segment depression, B- RBBB with excessive discordant ST-segment elevation exceeding 25 % of the preceding S-wave depth in leads V1-V3 (modified Smith criteria), indicative of underlying occlusion myocardial infarction.

4. If ECG suspicious + clinical features → activate cath lab / thrombolysis per local pathways. If uncertain → serial ECGs, bedside troponin, point-of-care ultrasound, and low threshold for angiography.

9.4. Limitations of existing evidence and research gaps

- Much evidence is observational, registry-based or case-series; randomized data are not available [1,4,6].
- The Sgarbossa/Smith criteria were derived for LBBB and paced rhythms; while the conceptual rules (concordance, proportionality) are useful, they are not formally validated specifically in RBBB populations — this is an area for prospective study [2,3].

10. Conclusion

New-onset RBBB in the setting of chest pain is an important high-risk marker that often indicates more extensive ischemia, frequently implicates LAD/septal perfusion territories, and carries worse outcomes [1,7]. Clinicians should actively search for concordant ST elevation, excessive discordant ST elevation, reciprocal changes and dynamic ECG evolution. When these features occur with a compatible clinical picture, the ECG should be treated as a possible STEMI equivalent and reperfusion pathways should be activated promptly [9,10]. Future prospective studies are needed to better quantify diagnostic thresholds and validate proportional criteria specifically in RBBB.

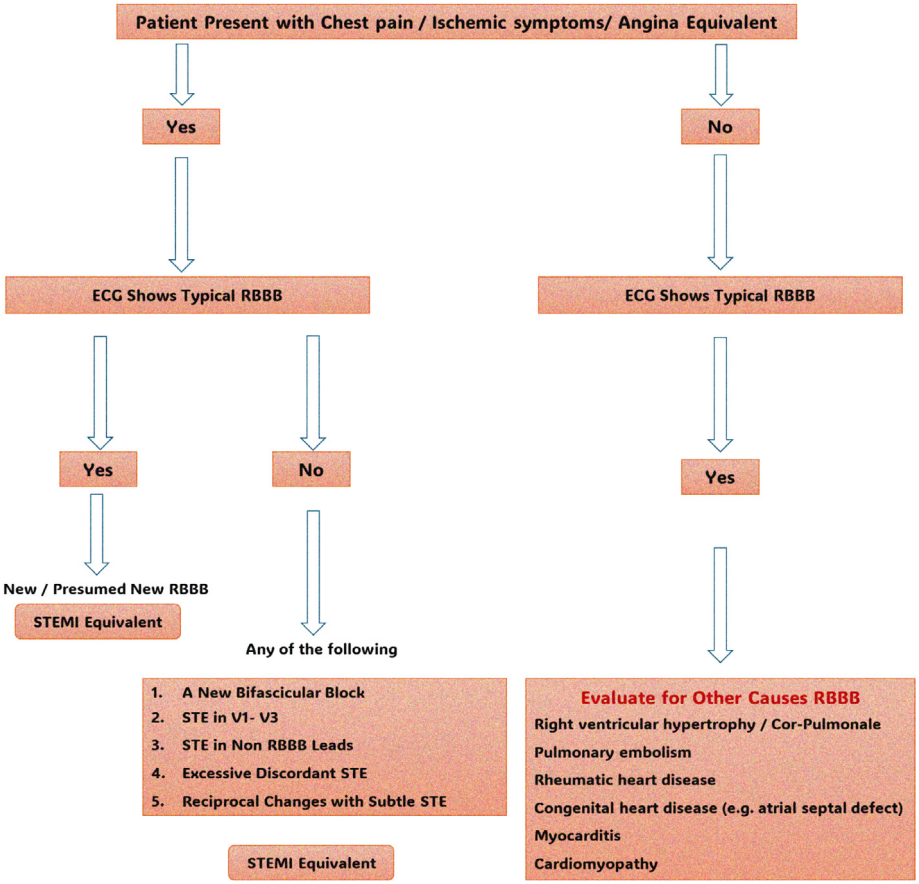


Fig. 10. The evaluation of RBBB in ER.

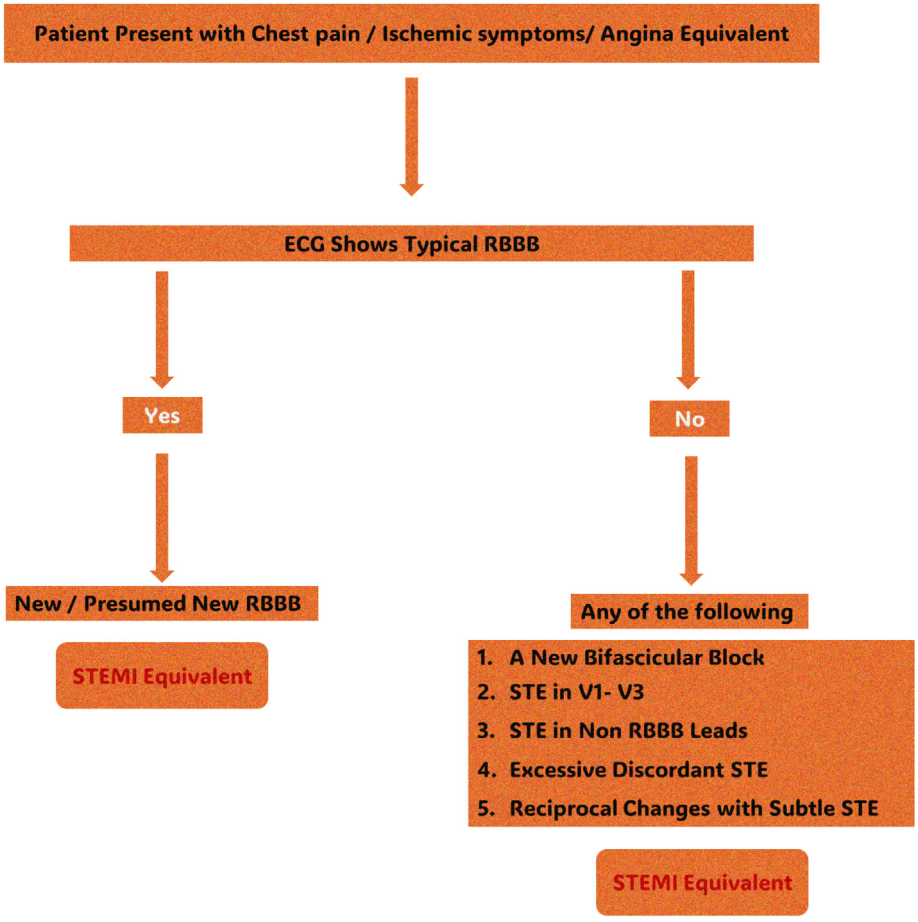


Fig. 11. - Evaluation RBBB in patients with Chest pain in ER.

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Use of generative AI in scientific writing

During the preparation of this work, the we have used AI-assisted tools for drafting and editing portions of the text. After using these tools, the author(s) reviewed and edited the content as needed and take(s) full responsibility for the content of the published article.

Declaration of competing interest

The authors of the review article declare that we have no known competing financial interests or personal relationships that could have appeared to influence the work reported in this paper.

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