

From Infarction to Conduction Collapse: Complete Sino-Atrial Block Following Inferior ST-Elevation Myocardial Infarction: A Case from a Non-PCI Center

Christrian Ngongang Ouankou^{1*}, Djibrilla Siddikatou², Mbua Larissa Kongnyuy³,
Archange Nzali⁴, Michelle Ngono³, Patrick Ateba³, Yemele Honoré Kemnang⁵, Ba Hamadou^{3,6}

¹Faculty of Medicine and Pharmaceutical Sciences, University of Dschang, Dschang, Cameroon

²Faculty of Medicine and Pharmaceutical Sciences, University of Douala, Douala, Cameroon

³Faculty of Medicine and Biomedical Sciences, University of Yaounde 1, Yaoundé, Cameroon

⁴Centre de Cardiologie Interventionnel de Douala, CCID, Douala, Cameroon

⁵Centre hospitalier Simone veils de Blois, Blois, France

⁶Garoua General Hospital, Garoua, Cameroon

Email: *ngongangco@gmail.com

How to cite this paper:

Ngongang Ouankou, C., Siddikatou, D., Kongnyuy, M.L., Nzali, A., Ngono, M., Ateba, P., Kemnang, Y.H. and Hamado, B. (2026) From Infarction to Conduction Collapse: Complete Sino-Atrial Block Following Inferior ST-Elevation Myocardial Infarction: A Case from a Non-PCI Center. *World Journal of Cardiovascular Diseases*, 16, 304-311. <https://doi.org/10.4236/wjcd.2026.165031>

Received: March 6, 2026

Accepted: May 23, 2026

Published: May 26, 2026

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Abstract

Background: ST-elevation myocardial infarction (STEMI) is frequently complicated by bradyarrhythmias, including sinoatrial block (SAB). The sinoatrial nodal artery, which supplies the sinoatrial (SA) node, originates from the right coronary artery (RCA) in approximately 60% of cases. Consequently, most cases of SA node dysfunction in the context of ischemia occur in inferior myocardial infarction (MI) and are potentially reversible. We report a case of inferior STEMI associated with a third-degree sino-atrial block. **Case Report:** A 61-year-old woman was admitted to the emergency department with a persistent epigastric burning sensation lasting 20 hours, followed by chest tightness for 2 hours prior to presentation, associated with dyspnea. Her cardiovascular risk factors included hypertension, hypercholesterolemia, and obesity. Emergency electrocardiography confirmed inferior and apicolateral STEMI complicated by a third-degree sinoatrial block with a junctional escape rhythm at 34 beats/min. Thrombolysis was performed, and the patient was subsequently referred for percutaneous coronary intervention (PCI). PCI was carried out 48 hours later with temporary cardiac pacing due to persistent conduction disturbances despite atropine therapy. Coronary angiography revealed significant stenosis of the RCA in its second segment, and thrombectomy successfully restored coronary blood flow. The temporary pacemaker was removed after monitoring showed stable sinus bradycardia at approximately 50beats/min.

Follow-up ECG demonstrated significant regression of ST-segment elevation. **Conclusion:** Complete SAB in MI is less common than AV block but may be life-threatening. Prompt monitoring and early intervention are essential to prevent adverse outcomes. Thrombolysis, when indicated, should be considered in settings where PCI is not immediately available, with arrangements for timely and adequately equipped referral.

Keywords

ST-Elevation Myocardial Infarction, Sino-Atrial Block, Temporary Cardiac Pacing, Thrombolysis

1. Introduction

ST-elevation myocardial infarction (STEMI) is frequently associated with conduction disorders, including sinoatrial block (SAB), although it is less commonly reported than atrioventricular block in this context [1].

Most cases of SAB related to myocardial ischemia occur in inferior STEMI. This is explained by the fact that the right coronary artery supplies the sinoatrial node in approximately 60% of individuals via the sinoatrial nodal artery [2]-[4]. The mechanisms involved include ischemic injury to the sinoatrial node and/or increased vagal tone mediated by the Bezold-Jarisch reflex. The latter typically responds to atropine, whereas ischemic damage does not [1] [2] [5].

Complete SAB in inferior STEMI is associated with increased morbidity and mortality. Therefore, early reperfusion therapy is essential to prevent poor outcomes and reduce the need for temporary or permanent cardiac pacing [1] [4].

We report a case of inferior STEMI complicated by complete sinoatrial block and hemodynamic instability.

2. Case Presentation

A 61-year-old retired nurse presented to the emergency department with a 20-hour history of burning epigastric pain radiating to the back, associated with dyspnea and resistant to proton pump inhibitors. Two hours prior to admission, she developed a precordial tightening sensation without radiation, syncope, or presyncope.

Her modifiable cardiovascular risk factors included grade III obesity (BMI: 42.7 kg/m²), long-standing hypertension (untreated for the past three months), dyslipidemia treated with atorvastatin 20 mg/day, sedentary lifestyle, and an unhealthy diet.

At admission, the epigastric pain had resolved, but chest pain persisted with moderate intensity,

2.1. Clinical Evaluation

The patient was stable on admission with bradycardia at 34 beats/min, afebrile,

respiratory rate 18 breaths/min, and oxygen saturation 95% on room air. Thirty minutes later, she developed hemodynamic instability with blood pressure 88/61 mmHg without signs of cardiogenic shock. She then began to desaturate, with oxygen saturation 90% on room air, and had mild bilateral basal crackles.

2.2. Electrocardiography

An emergency 12-lead ECG (**Figure 1**) showed absence of atrial activity with a junctional escape rhythm at approximately 34 beats/min. There was ST-segment elevation of about 10 mm in the inferior leads (DII, DIII, aVF), 5 mm in lateral leads (V4 - V5), and 2 mm in V4, with reciprocal ST depression in DI, aVL, aVR, and V1 - V3.

2.3. Echocardiography

Bedside transthoracic echocardiography showed segmental hypokinesia predominantly affecting the inferior and inferolateral walls, with a left ventricular ejection fraction of 51% and grade 1 diastolic dysfunction. Right ventricular function was preserved and there was no mechanical complication.

2.4. Laboratory Findings

Laboratory investigations revealed elevated cardiac troponin I (0.66 ng/mL), normal serum electrolytes, and renal impairment (GFR: 29 mL/min). Inflammatory markers were elevated (CRP: 24 mg/L). Lipid profile showed hypercholesterolemia (LDL: 1.76 g/L). HbA1c was 6.5%, NT pro BNP: 788 pg/ml, mildly elevated liver enzymes < 2 N, and normal Full blood count.

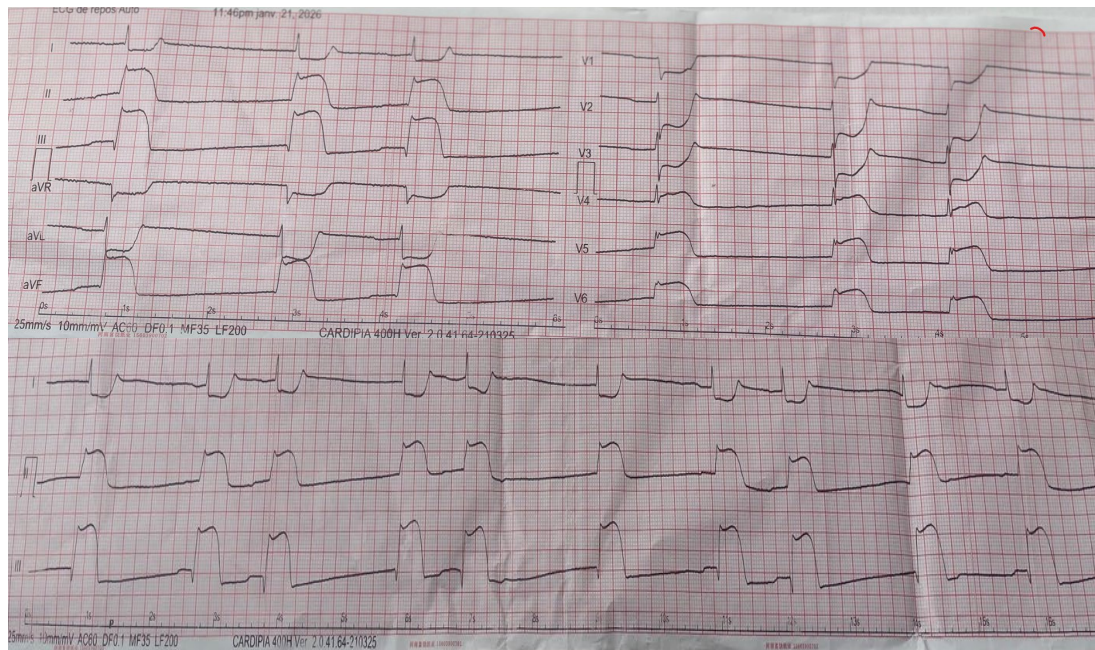


Figure 1. Emergency ECG showing 3rd degree sino-atrial block with Junctional escape rhythm, an inferior and apico-lateral STEMI.

2.5. Management

Initial management included; Oxygen therapy, with target SpO₂ at 94%, dual antiplatelet therapy (aspirin 300 mg and clopidogrel 600 mg with maintenance dose at 100 and 75 mg respectively), anticoagulation with enoxaparin, 10,000 UI/12 hr S/C, high-dose statin therapy: atorvastatine 80 mg, proton pump inhibitor, pantoprazole, analgesia, and, atropine (administered at 1 mg per dose with a cumulative dose of 3 mg which proved ineffective), fibrinolysis with streptokinase (after administration of hydrocortisone 100 mg slow IV then Streptokinase 1.5 MUI diluted to 50 cc with Dextrose 5% given within 60 min) was performed after exclusion of contraindications.

2.6. Clinical Course

After thrombolysis, clinical improvement was noted with stabilization of blood pressure (120/68 mmHg) and reduction in bradycardia.

Serial ECGs showed 2 hrs, 6 hrs and 36 hours after thrombolysis respectively showed.

An accelerated idioventricular rhythm at about 90 beats/min, a regression of about 70% of the ST segment elevation at the apico lateral territory, and of about 40% at the inferior leads (**Figure 2**).

Sino atrial bloc, insignificant ST elevation at the apico lateral territory without Q necrosis wave, regression of the ST segment elevation of about 70% at the inferior leads but with presence of pathological Q wave as well as regression of the ST depression in the anteroseptal, and upper lateral leads, isolated ventricular ectopic beats (**Figure 3**).

Regular Sinus bradycardia at about 50 bpm, Q waves at the inferior leads with repolarization disorders (**Figure 4**).

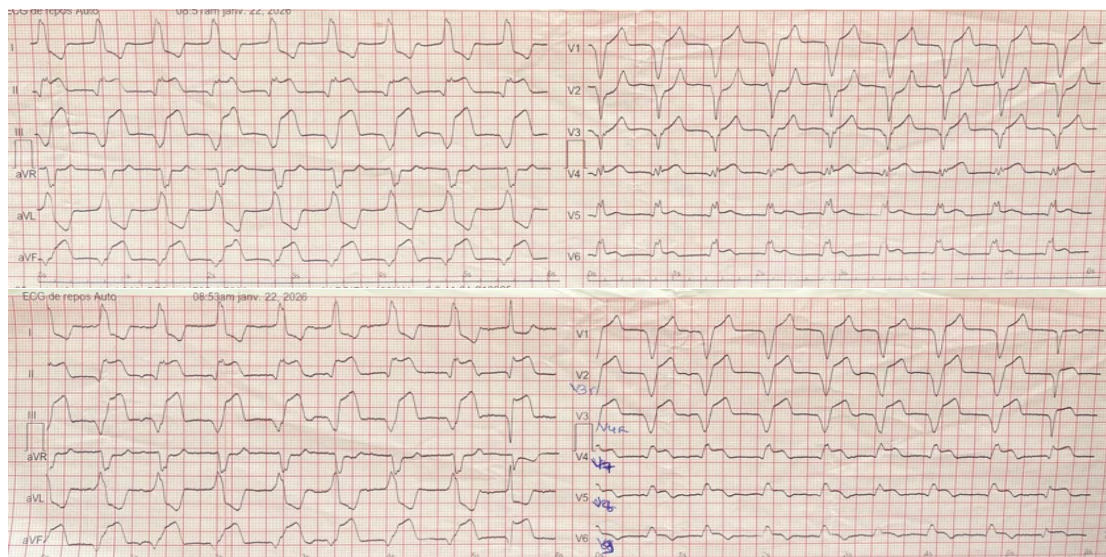


Figure 2. Two hours ECG post Fibrinolysis, showing an accelerated idioventricular rhythm at about 90 beats/min.

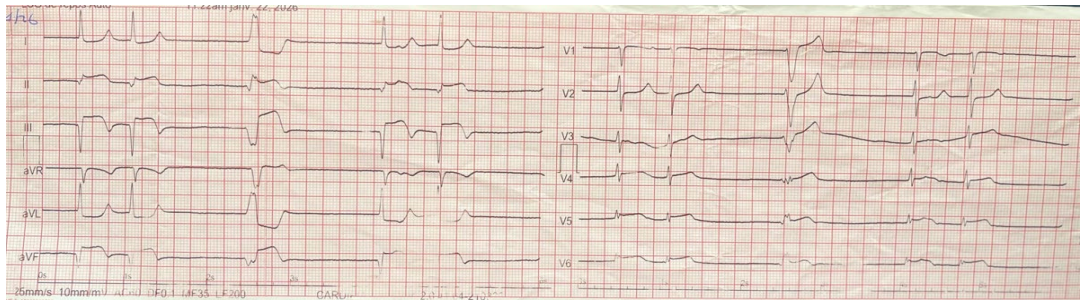


Figure 3. Persisting complete Sino-atrial block with significant regression of ST segment elevation.

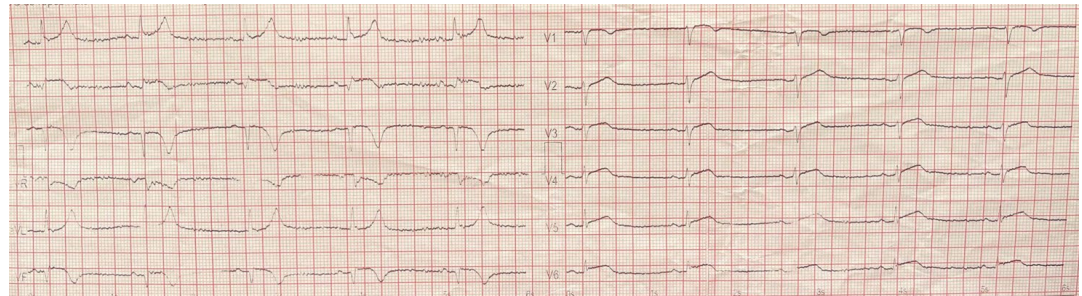


Figure 4. Return to sinus rhythm with sinus bradycardia at 50 beats/min.

Subsequent ECGs showed great variations fluctuating from sinus rhythm to a sinoatrial bloc.

The patient was later referred with medical assistance on Oxygen, to the nearest available PCI center about 720 km away by flight.

2.7. Per Cutaneous Coronary Intervention with Temporary Pacing Catheter

A temporary pacing catheter was placed just before the PCI through the right femoral vein.

2.7.1. Procedure and Findings

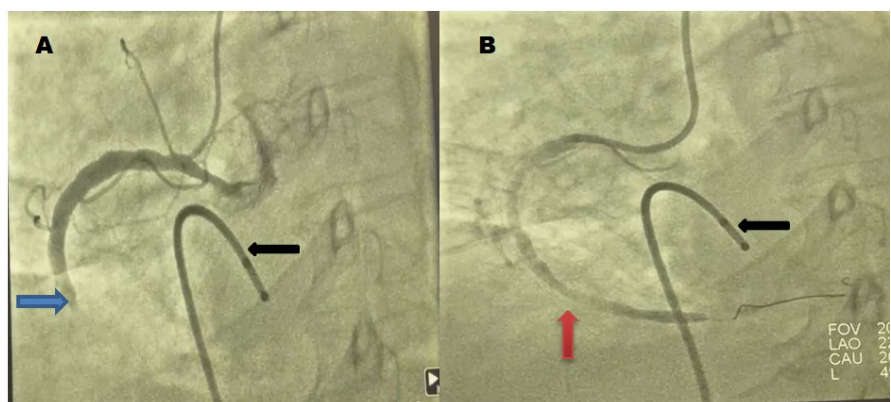


Figure 5. Coronary images before (A) and after thrombolysis (B); A very large RCA with significant stenosis in its second segment (Blue arrow) and restoration of RCA flow after thrombolysis (red arrows); Temporary pacing visible (black arrows).

Left Femoral Approach

Coronary network: Very large Right dominant.

Right Coronary Artery (RCA): Significant stenosis of the RCA in its second segment with extensive thrombosis involving all RCA2 and RCA3 (**Figure 5(a)**).

Left Main Coronary Artery: Normal length, 3.5 mm caliber, giving rise to Left Anterior Descending (LAD) and Circumflex (Cx) arteries.

LAD: Rudimentary with myocardial bridging, no significant stenosis (**Figure 6**).
Circumflex: Mildly atheromatous without significant stenosis, small caliber.

The patient underwent thrombectomy/aspiration, retrieving multiple thrombi (**Figure 7**), restoring flow in RCA2 (**Figure 5(b)**) and RCA3. Tirofiban was administered with intracoronary bolus and IV relay 6 hours post-procedure.

2.7.2. Evolution and Follow-Up

In the Cardiology ICU, the patient's general condition improved with good ambient air saturation and regression of ST elevation in all concerned leads. She equally developed an Atrial fibrillation with spontaneous return to sinus rhythm.



Figure 6. Hypoplastic LAD artery (blue arrow).



Figure 7. Multiple thrombi aspirated.

The outpatient treatment consisted of apixban 2.5 mg bid, clopidogrel 75 mg od, Rosuvastatine/Ezetimibe 20/10 mg 1 tab in the evening, Ramipril 1.25 mg od,

Dapaglifozine 10 mg od, Pantoprazole 40 mg od.

Also, lifestyle changes was requested, and consultation with a Dieteticien with HbA1C control in 3 months, Nephrology follow-up (kidney function improved)

As therapeutic project, the patient was scheduled for control PCI one month later and eventually cardiac readaptation

3. Discussion

Our patient presented 20 hours after the onset of epigastric pain, which was initially misinterpreted as peptic ulcer disease. Atypical presentations of acute coronary syndrome, particularly epigastric pain, are more common in women and are frequently associated with delayed hospital presentation [6] [7]. This delay can complicate therapeutic decision-making, especially in settings where access to percutaneous coronary intervention (PCI) is limited, which was our case.

Inferior myocardial infarction complicated by severe conduction disorders such as complete sinoatrial block carries a high risk of right ventricular involvement, increased mortality, and potential need for permanent pacing. In our case, management was particularly challenging due to the lack of immediate access to a PCI center. The indication for thrombolysis was debated, as the patient presented beyond the conventional 12-hour window [8]. However, some authors suggest that thrombolysis may still be beneficial within the first 24 hours in selected patients under 65 years of age without contraindications [9]. Given that our patient met these criteria and had experienced recurrent chest pain less than two hours prior to presentation, and developed hemodynamic instability, thrombolytic therapy was administered. The presence of persistent third-degree sinoatrial block with junctional escape rhythm raised concern for irreversible conduction system damage in the absence of prompt reperfusion, potentially requiring permanent pacemaker implantation [1] [4]. All these parameters influenced our therapeutic decisions. Following thrombolysis, the patient showed significant clinical improvement, including resolution of chest pain and gradual recovery of sinus rhythm, although intermittent conduction disturbances persisted. No major hemorrhagic complications were observed.

Percutaneous coronary intervention demonstrated a hypoplastic left anterior descending artery and a dominant, well-developed right coronary artery supplying the majority of myocardial perfusion. Delayed revascularization could have precipitated more severe ischemic complications.

The need to transfer the patient over a distance of approximately 720 km by flight to access PCI highlights the challenges of managing acute coronary syndromes in resource-limited settings. This case underscores the importance of adaptability and timely decision-making in such environments.

4. Conclusions

Acute coronary syndromes, particularly inferior ST-elevation myocardial infarction, may be complicated by severe conduction disorders such as complete sino-

atrial block. These abnormalities can become irreversible in the absence of early reperfusion therapy.

In settings where immediate access to PCI is not available, thrombolytic therapy should be promptly considered in the absence of contraindications, while organizing timely transfer to a specialized center. Such an approach is essential to improve clinical outcomes and reduce morbidity and mortality.

Conflicts of Interest

The authors declare no conflicts of interest regarding the publication of this paper.

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