# *Type 2 Myocardial Infarction Unmasking Critical Left Main Coronary Artery Stenosis in Severe Anemia due to Addison-Biermer Disease: A Case Report*

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**ABSTRACT**

***Type 2 Myocardial Infarction Unmasking Critical Left Main Coronary Artery Stenosis in Severe Anemia due to Addison-Biermer Disease: A Case Report***

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*To this day, myocardial infarction (MI) remains the leading cause of mortality worldwide. While its etiology is multifactorial, anemia – regardless of its underlying cause – may either accompany MI as a comorbidity or serve as a direct precipitant of myocardial ischemia, particularly in cases of type 2 MI. Importantly, anemia diagnosed at hospital admission has been recognized as an independent predictor of adverse cardiovascular outcomes, including both short- and long-term mortality, irrespective of its role in the pathogenesis of MI.*
*In the case presented, severe macrocytic anemia caused by vitamin B12 deficiency resulted in acute coronary syndrome without ST-segment elevation (NSTE-ACS). Because of typical chest pain and deep ST-segment depression in multiple leads, the patient was initially qualified for urgent coronary angiography. In the meantime, however, morphology results revealed severe anemia, and a noninvasive strategy was chosen. During hospitalization, the patient's blood deficiency was corrected, the cause of anemia was identified, and treatment was initiated.*

**STRESZCZENIE**

***Zawał serca typu 2 ujawniający krytyczne zwężenie pnia lewej tętnicy wieńcowej w przebiegu ciężkiej niedokrwistości spowodowanej chorobą Addisona-Biermera – opis przypadku***

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*Zawał mięśnia sercowego (MI) w dalszym ciągu pozostaje główną przyczyną zgonów na całym świecie. Anemia – niezależnie od przyczyny – może współistnieć z zawałem jako choroba współtowarzysząca, ale również być bezpośrednim czynnikiem wywołującym niedokrwienie mięśnia sercowego, szczególnie gdy mówimy o zawale typu 2. Co istotne, niedokrwistość stwierdzona w chwili przyjęcia do szpitala jest uznawana za niezależny czynnik prognostyczny niekorzystnych zdarzeń sercowo-naczyniowych, wliczając w to zarówno krótkoterminową, jak i długoterminową śmiertelność.*
*W przedstawionym przypadku ciężka niedokrwistość doprowadziła do wystąpienia ostrego zespołu wieńcowego bez uniesienia odcinka ST (NSTE-ACS). Ze względu na objawy kliniczne i zmiany niedokrwienne w EKG przy przyjęciu pacjent początkowo zakwalifikowany został do pilnej koronarografii. Jednak w międzyczasie wyniki morfologii krwi ujawniły ciężką niedokrwistość makrocytarną, co skłoniło lekarzy do podjęcia decyzji o wstrzymaniu zabiegu i wdrożeniu leczenia zachowawczego. Podczas hospitalizacji dokonano transfuzji preparatów krwiopochodnych i wdrożono substytucję witaminy B12.*

*Coronary angiography was performed two months later, once blood counts had normalized, revealing significant stenosis of the distal segment of the left main coronary artery (LMCA) and critical ostial stenosis of the left anterior descending artery (LAD). This case report provides a starting point for discussing the optimal timing of coronary angiography in the setting of NSTE-ACS with concomitant anemia and urges deliberate consideration of any potential delay, as well as follow-up treatment.*

**Keywords:** *myocardial infarction, macrocytic anemia, vitamin B12, atherosclerosis*

*Koronarografia przeprowadzona dwa miesiące później po unormowaniu parametrów morfologii krwi, ujawniła istotne zwężenie dystalnego odcinka pnia lewej tętnicy wieńcowej (LMCA) oraz krytyczne zwężenie ujścia gałęzi przedniej zstępującej (LAD). Przypadek ten stanowi punkt wyjścia do dyskusji nad optymalnym momentem wykonania koronarografii w kontekście NSTE-ACS współistniejącego z niedokrwistością oraz skłania do rozważnego przemyślenia kwestii potencjalnych opóźnień w diagnostyce i dalszym leczeniu.*

**Słowa kluczowe:** *zawał mięśnia sercowego, niedokrwistość makrocytarna, witamina B12, miażdżyca*

## Introduction

Myocardial infarction (MI) remains one of the leading causes of morbidity and mortality worldwide, posing significant diagnostic and therapeutic challenges in clinical practice. By definition, MI is classified into five types based on underlying pathophysiology, which correspond to different treatment strategies and varying prognoses. Type 1 MI is caused by atherothrombotic events, whereas type 2 MI encompasses a broader group of conditions characterized by secondary ischemia resulting from a mismatch between oxygen supply and demand [1, 2].

After establishing the diagnosis of MI, it is crucial to differentiate between type 1 and type 2 in order to select the most effective treatment [3]. High-risk type 1 MI usually requires reperfusion by invasive revascularization (percutaneous coronary intervention or, far less frequently, coronary artery bypass grafting), whereas in type 2 MI the treatment approach should focus on alleviating the primary underlying causes of ischemia, which can be diverse [4].

Anemia can either coexist with any type of MI as a comorbidity or act as a direct cause of ischemic myocardial injury, leading to type 2 MI. In the first scenario, it is often secondary to bleeding associated with the administration of antiplatelet or anticoagulant therapy. Upper gastrointestinal bleeding may be further promoted by stress ulcer formation. In addition, hematuria can result from traumatic or improperly placed urinary catheters in severely ill patients.

Regardless of its role in precipitating MI, anemia measured on hospital admission for acute coronary syndrome has been identified as an independent predictor of adverse cardiac events, as well as increased short- and long-term mortality. In one study, patients with moderate to severe anemia (Hb concentration < 11 g/dL) had a twofold increase in long-term mortality risk [5]. Several explanations have been proposed in the literature to account for this association, including impairment of myocardial remodeling and excessive inflammation [2].

According to the WHO definition, severe anemia is diagnosed if the hemoglobin (Hb) level falls below 8 g/dL and requires immediate blood transfusion [6]. Another approach to categorizing anemia utilizes the mean size of erythrocytes, denoted by mean corpuscular volume (MCV). An MCV under 80 fL signifies microcytic anemia, and an MCV over 100 fL is considered macrocytic; values in between are classified as normocytic. Microcytic anemia results from iron deficiency and accompanies chronic bleeding; it is especially frequent in gastrointestinal tract cancers. In contrast, normocytic anemia is typical of acute anemia (mainly bleeding, less often hemolysis), as well as chronic kidney disease and bone marrow disorders. Macrocytic anemia is generally related to vitamin B12 and folic acid deficiency.

## Macrocytic anemia

Macrocytic anemia can be further subdivided into megaloblastic and non-megaloblastic types based on the presence of enlarged nucleated red blood cells (macroovalocytes) in a peripheral blood smear [7]. The most common cause of megaloblastic anemia is vitamin B12 deficiency [8], which itself can be caused by various factors, such as pernicious anemia (also known as Addison–Biermer disease), chronic overconsumption of alcohol, or *Diphyllobothrium latum* infection.

Pernicious anemia is a relatively rare condition, with general population prevalence estimates ranging from 1/10,000 to 1/1,000 [8]. It is caused by the presence of autoantibodies directed against parietal cells (specifically their H+/K+ ATPase) and/or intrinsic factor, which is required for vitamin B12 absorption. The consequences of vitamin B12 deficiency are hematological (megaloblastic anemia) and neurological (peripheral neuropathy). Neurological manifestations result from impaired myelination, most prominently affecting the posterior and lateral columns of the spinal cord, leading to sensory deficits in the lower limbs (symmetric paresthesia, numbness, and unsteady

gait). Additionally, the accumulation of methylmalonate in the central nervous system—a neurotoxic metabolite—may further exacerbate neurological damage and can be associated with nonspecific psychiatric symptoms such as apathy, anxiety, confusion, memory loss, or cognitive decline.

The clinical course of pernicious anemia is typically insidious, with symptoms developing gradually over an extended period. As a result, patients may fail to recognize or report their manifestations, having adapted to the subtle, progressive nature of the condition. Most individuals with vitamin B12 deficiency present for medical evaluation due to nonspecific symptoms attributable to anemia, such as fatigue, pallor of the skin, and headaches.

The main objective of investigating the exact etiology of vitamin B12 deficiency is to identify patients who require lifelong cobalamin replacement therapy (e.g., those suffering from pernicious anemia), as opposed to those with inadequate intake (e.g., an improperly balanced vegan diet) or *Diphyllobothrium latum* infection. According to the British Committee for Standards in Hematology guidelines, anti-intrinsic factor antibodies (anti-IFAB) should be checked in all symptomatic patients. However, although highly specific (95%), their sensitivity is low (anti-IF antibodies are present in 40–60% of cases), which means that patients negative for anti-IFAB may still have pernicious anemia. The diagnosis of "antibody-negative pernicious anemia" is made if no other causes of vitamin B12 deficiency are found. Consequently, treatment should be initiated without delay to prevent further exacerbation of neurological deficits. Therefore, in clinical practice, anti-IFAB testing is often initially omitted, since the result does not influence the course of treatment and does not rule out pernicious anemia.

Moreover, gastroscopic biopsy may be performed to assess for atrophic gastritis, as it is often associated with pernicious anemia.

Although the treatment of vitamin B12 deficiency anemia is simple and based solely on supplementation, in patients with pernicious anemia, a lifelong parenteral regimen is required. It is worth noting that hematological abnormalities may be absent, so vitamin B12 deficiency may present exclusively with neurological manifestations. Nonetheless, early treatment is essential to avoid permanent neurological disability.

## Case

A 72-year-old male with a history of smoking and treated for arterial hypertension was admitted to the emergency department with a diagnosis of acute coronary syndrome without ST-segment elevation. The patient reported severe chest pain accompanied by torsions. On ECG, there were diffuse ST-segment depressions in most leads, with ST elevation in aVR (Fig. 1A).

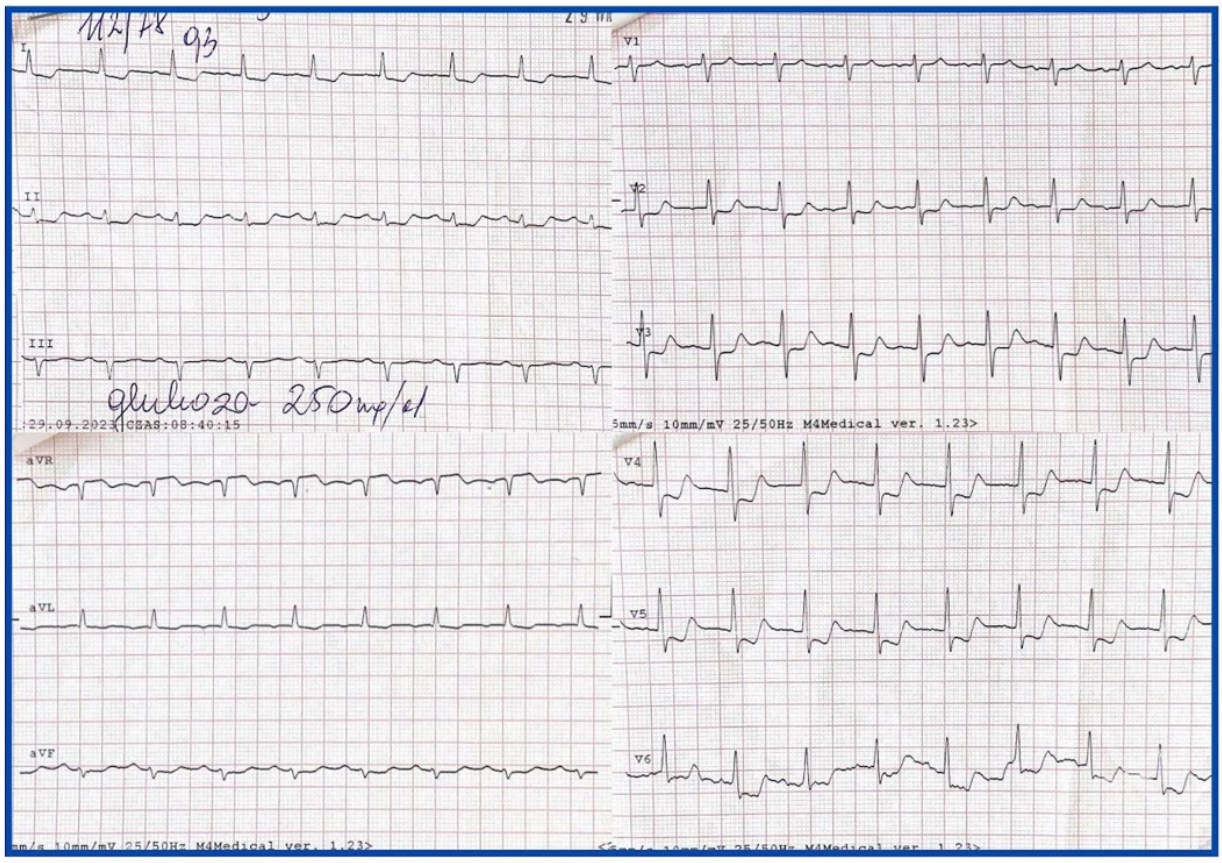
On admission, the patient was still experiencing chest pain. Vital signs were as follows: blood pressure 112/78 mmHg and heart rate 93 bpm. The patient was hemodynamically stable, and physical examination revealed no signs of heart failure (Killip class I).

Because of the clinical presentation and ischemic changes on ECG suggesting left main stenosis, proximal left anterior descending artery stenosis, or triple-vessel coronary artery disease [9], the patient was qualified for urgent coronary angiography [4]. In the meantime, however, laboratory results revealed severe anemia with Hb 5.7 g/dL (Tab. 1), and a decision was made to defer the procedure. The patient had no signs of active bleeding, was hemodynamically stable, and had no arrhythmias. He underwent an urgent red blood cell transfusion (two units of packed red blood cells), which relieved his chest pain. ECG performed after the transfusion showed significantly reduced ischemic changes (Fig. 1B), echocardiography revealed hypokinesis of the apex and apical segments of the left ventricle with an LVEF of 50%, and troponin was elevated with a dynamic profile consistent with MI (Tab. 1).

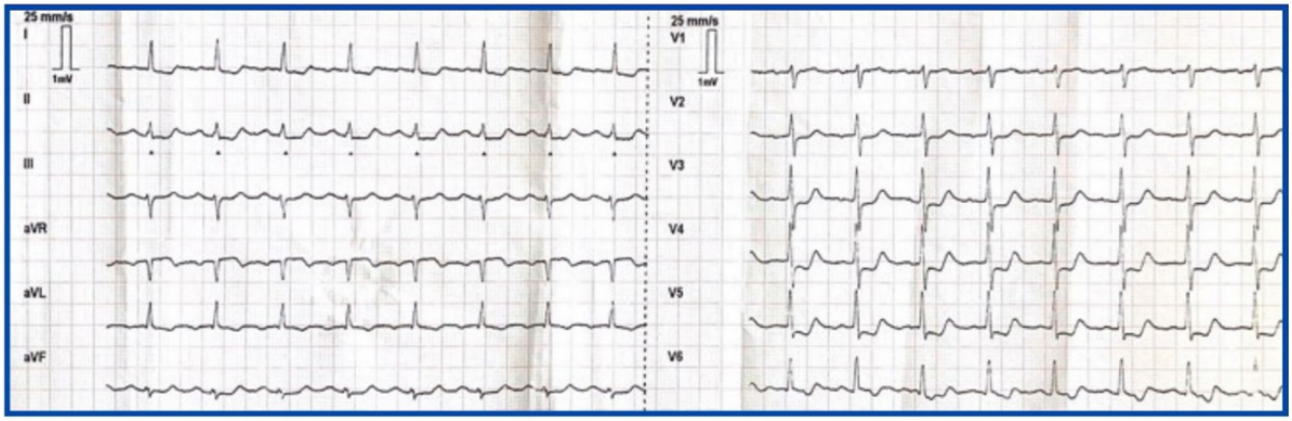
Further hematological evaluation revealed pancytopenia, macrocytic anemia, and decreased serum vitamin B12 concentration, with normal folic acid levels. During a comprehensive clinical interview, the patient reported neurological and psychiatric symptoms over the past year, including paresthesia, muscle weakness, and motor difficulties, memory problems, and a persistently low mood. Due to progressive cognitive decline and prolonged social withdrawal, the patient's family ultimately encouraged him to seek medical attention. Notably, during the physically demanding journey to the healthcare facility, the patient experienced chest pain and was admitted to the hospital.

As part of the diagnostic evaluation for anemia, an upper gastrointestinal endoscopy was performed. Histopathological analysis of biopsy specimens from the fundus and antrum revealed chronic atrophic gastritis with features typical of pernicious anemia—including glandular atrophy, intestinal metaplasia, and chronic lymphoplasmacytic infiltration. No evidence of *Helicobacter pylori* infection was found. Additionally, the patient was evaluated by a psychiatrist and a neurologist, who concluded that the neurological and psychiatric symptoms were attributable to vitamin B12 deficiency. The clinical presentation was consistent with Addison–Biermer disease, indicating it as the cause of anemia.

During hospitalization, the patient received four units of packed red blood cells and one unit of platelet concentrate. Moreover, intramuscular vitamin B12




A




B

Figure 1. ECG recorded at 25 mm/s. A. On admission, ST-segment depressions in leads I, II, aVF, V2–V6, negative T wave in lead aVL, and ST elevation in lead aVR. B. ECG taken on the same day after transfusion of two units of red blood cells—less severe ST-segment changes

injections were administered. A gradual improvement in hematological parameters was observed. The patient was mobilized and did not experience recurrence of angina. An LVEF of 50% was confirmed on follow-up examination. He was discharged home after 2 weeks of hospitalization and prescribed nebivolol 5 mg once daily, atorvastatin 5 mg once daily, folic acid 5 mg once daily, and vitamin B12 250 µg once weekly. However, due to severe ECG abnormalities on admission, markedly elevated troponin levels, and segmental wall motion abnormalities on echocardiography, he was scheduled for coronary angiography once his blood counts normalized. Coronary angiography was performed after two months, when blood counts had normalized. At that point, the patient had no psychiatric or neurological symptoms and was in

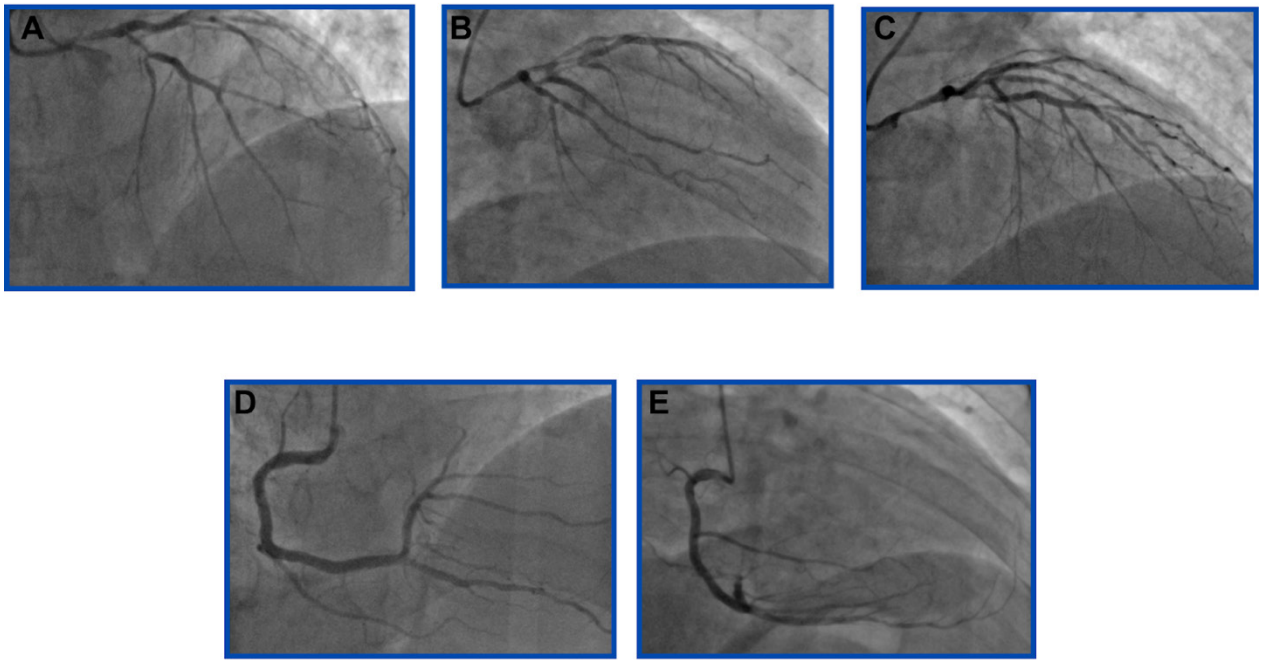



Figure 2. Coronary angiography prior to PCI. A: LCA: significant tight stenosis of the ostial LMCA and proximal segment of the left anterior descending (LAD) artery (left cranial view). B: LCA: significant tight stenosis of the proximal segment of the LAD with impaired TIMI 2 flow; circumflex artery without abnormalities. C: LCA: significant tight stenosis of both the LMCA and proximal segment of the LAD (right cranial view). D: RCA: no abnormalities. E: RCA: retrograde filling of the distal LAD segment

a good mood, active, and socially engaged. He denied chest pain during this period. On resting ECG, slight ischemic changes were still present.

The examination revealed right coronary artery dominance with retrograde filling of the periapical segment of the left anterior descending artery (LAD), visible in the RAO projection. In the left coronary artery, stenosis of the distal segment of the trunk was found (minimal lumen area of 3.38 mm² on IVUS), which was combined with critical ostial stenosis of the LAD (minimal lumen area of 4.36 mm² on IVUS).

A cardiothoracic surgery consultation was proposed, but the patient refused. Therefore, percutaneous coronary intervention (PCI) was performed using a single-stent technique with proximal optimization. The procedure restored normal angiographic parameters—full vessel lumen, patent circumflex artery, and no visible dissections. The results were confirmed by post-PCI IVUS, where the minimal lumen area of the trunk was 8 mm² and that of the LAD ostium was 6 mm². Dual antiplatelet loading was performed before the procedure.

## Conclusions

Anemia accompanying ACS is a well-established predictor of increased short- and long-term mortality [10]. Although severe anemia can independently precipitate type 2 MI, current evidence suggests that it is frequently not the sole underlying cause. In an analysis of the ACS registry by Sudarsky et al. [11], 95% of anemic patients who underwent coronary angiography demonstrated obstructive coronary artery disease, and 78% were deemed suitable for revascularization, indicating a multifactorial pathophysiology in most cases. This underscores the need for increased vigilance among clinicians, who should remain cautious about the possibility of significant underlying coronary artery disease, with anemia merely exacerbating ischemia.

Importantly, the underlying cause of anemia may further modulate ischemic risk. In vitamin B12 deficiency, impaired DNA synthesis leads to ineffective hematopoiesis, usually affecting all three cell lines, including reduced platelet counts. Moreover, the decreased erythrocyte count is accompanied by reduced red blood cell deformability, limiting their passage through stenotic vessels and the microcirculation. Together, these abnormalities exacerbate tissue hypoxia and may further complicate the clinical course in ACS.

In this context, early red blood cell (RBC) transfusion may serve as an appropriate initial strategy to enhance oxygen delivery, particularly in hemodynamically compromised patients. However, as highlighted by the present case, it remains essential to maintain a high index of suspicion for concomitant coronary artery disease, which may necessitate definitive diagnostic and therapeutic intervention.

Table 1. Changes in the patient's laboratory parameters

| Parameter (normal range) [units] | The day of admission | Day 2 of hospitalization | Discharge from the hospital | 2 months after |
|---|---|---|---|---|
| HGB (12.2–15.8)[g/dL] | 5.7 | 7.9 | 8.8 | 15.2 |
| HCT (37.0–45.0) [%] | 17.3 | 22.8 | 26.1 | 48.8 |
| MCV (80.0–96.0)[fL] | 123.6 | 108.1 | 107.9 | 85.2 |
| WBC (4.00–10.00) [$10^3$/μL] | 2.21 | 2.77 | 7.74 | 8.49 |
| PLT (130–400) [$10^3$/μL] | 34 | 37 | 246 | 223 |
| B12 (197–771) [pg/mL] | 62 | - | - | - |
| Folic acid (2.0–9.1) [ng/mL] | 4.2 | - | - | - |
| Fe (33–193) [μg/dL] | 112 | - | - | - |
| Troponin Ths (0.00–0.014) [ng/mL] | 0.035 -> 1.93 | 3.71 | 0.239 | - |
| CK-MB (0.1–4.87) [ng/mL] | 1.89 -> 81.51 | 58.82 | 0.63 | - |

In patients qualified for initial conservative management of ACS, high-dose statin therapy should be initiated, and antiplatelet treatment should be considered. In this case, failure to administer a high-dose statin and at least one antiplatelet agent constituted a management error—particularly after the cause of anemia had been identified and hematological parameters had improved, including normalization of the platelet count. This is especially important given the presence of significant ischemic changes on ECG, regional wall motion abnormalities on echocardiography, and elevated troponin levels, all of which strongly suggest an underlying coronary etiology.

Another important aspect is the timing of coronary angiography. According to the 2023 ESC guidelines, if angiography in the course of non–ST-elevation MI is not indicated urgently or within the early 24-hour window, its timing should be guided by clinical stabilization and optimization of comorbid conditions but performed prior to hospital discharge [4]. In our patient, however, given the incomplete recovery of cognitive function, persistently abnormal blood counts (Hb 8.8 g/dL at discharge), and the absence of clinical symptoms after mobilization on the ward, we elected to postpone coronary angiography until full clinical stabilization. The patient was advised to limit strenuous physical activity until the procedure was performed. In retrospect, after reviewing the coronary imaging, the decision to delay coronary angiography is questionable.

An entirely distinct issue is microcytic anemia (usually secondary to chronic blood loss), where invasive treatment of coronary artery disease—and the associated need for dual antiplatelet therapy (DAPT)—carries an increased risk of ongoing bleeding. In such cases, it is essential to identify and, if possible, eliminate the source of bleeding. In patients with a high likelihood of underlying coronary artery disease—evidenced by persistent symptoms despite blood transfusion and restoration of hemoglobin levels above 8 g/dL, such as resting angina, hemodynamic instability, or electrical disturbances—it is advisable to perform coronary angiography and consider the use of stents with the shortest possible duration of DAPT or, alternatively, drug-coated balloons.

An additional, somewhat peripheral yet important issue is the threshold at which red blood cell (RBC) transfusion should be prioritized. Currently, there is no clear consensus or universally accepted guideline defining a specific hemoglobin level that mandates transfusion in the setting of ACS. While the World Health Organization generally recommends transfusion when hemoglobin falls below 8 g/dL in stable patients, this threshold may not be appropriate in the context of myocardial ischemia, where oxygen delivery is critical. Several observational studies have suggested that liberal transfusion strategies (e.g., transfusion at Hb <10 g/dL) might be associated with better outcomes in patients with ACS, but randomized controlled trials have yielded conflicting results. For example, the REALITY trial [12] compared restrictive (Hb <8 g/dL) versus liberal (Hb <10 g/dL) transfusion strategies in patients with MI and anemia. It found no significant difference in major adverse cardiac events between the two groups, although some subgroups may benefit from a more liberal approach. Importantly, both undertransfusion and overtransfusion carry risks—the former due to inadequate oxygen delivery and the latter due to increased blood viscosity, volume overload, and potential prothrombotic effects. Therefore, transfusion decisions should be individualized, taking into account the patient's hemodynamic stability, extent of ischemia, ongoing bleeding, comorbidities, and clinical symptoms, rather than relying solely on a fixed hemoglobin value. Further research is needed to establish

evidence-based transfusion thresholds specifically tailored to patients with ACS.

In summary, the intricate interplay between anemia and myocardial ischemia highlights the need for intervention studies to establish evidence-based guidelines on whether and when to perform coronary angiography in MI and when to choose a conservative strategy, so that outcomes for these patients can be optimized. Additionally, it should be noted that anemia as the sole etiology is uncommon; more often, anemia serves to unmask underlying coronary artery disease.

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The authors declare no conflict of interest.

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