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Case Report

Acute Myocardial Infarction in Adolescents. Case Report

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Abstract

Introduction: The diagnosis of myocardial infarction (MI) typically involves cardiac biomarker elevation and evidence of myocardial ischemia. However, a subset of cases, known as myocardial infarction with non-obstructive coronary arteries (MINOCA), poses challenges in diagnosis and management. This study aims to present a case of MINOCA in a young male and discuss its clinical implications.

Case Presentation: a 16-year-old male experienced sudden chest pain radiating to the right arm. Cardiac evaluation revealed ST segment elevation on an electrocardiogram (ECG), leading to admission. Subsequent coronary angiography ruled out obstructive coronary disease. Further assessments indicated probable viral myopericarditis as the cause. The patient's symptoms improved, troponin levels decreased, and cardiac imaging results were normal.

Discussion: MINOCA, characterized by elevated cardiac biomarkers, non-obstructed coronary arteries, and ischemic clinical features, accounts for 6 - 15% of acute myocardial infarction (AMI) cases. This diagnosis is reached by excluding other potential causes of myocardial injury. The case underscores the importance of thorough diagnostic evaluation, including cardiac imaging, viral panels, and troponin monitoring, to differentiate MINOCA from other cardiac and non-cardiac conditions.

Conclusion: MINOCA, although rare in the pediatric population, remains a diagnostic challenge due to its unique presentation. Thorough evaluation is crucial to exclude other causes of myocardial injury and accurately classify the type of MI. Early identification and appropriate management are essential to ensure optimal outcomes in pediatric patients with suspected cardiac causes. Increased awareness and further research are needed to enhance understanding and management of MINOCA in pediatric cardiology practice.

Keywords: *Acute; Myocardial; Infarction; Adolescents*

Introduction

The clinical definition of myocardial infarction denotes the presence of acute myocardial injury detected by abnormal cardiac biomarkers in the setting of evidence of acute myocardial ischemia [1]. To diagnose AMI at least one of the troponin values must be above the 99th percentile upper reference limit [2]. Cardiac troponins I (cTnI) and T (cTnT) are components of the contractile apparatus of myocardial cells and are expressed almost exclusively in the myocardium, although, on some occasions cTnT is produced by skeletal muscle and is

therefore less specific; Troponin I being the most precise biomarker. Other biomarkers such as CK-MB, are less sensitive and less specific [1].

Epidemiology

It has been established that more than 30% of reported deaths in the world are caused by cardiovascular diseases; in 2013 there were 17.5 million deaths according to the World Health Organization (WHO), of which 7.4 million were due to ischemic heart disease [3]. In Mexico, the National Institute of Statistics and Geography INEGI reported one death every 4.3 minutes from ischemic heart disease [4]. However, in pediatric population the figures change, with myocardial diseases being an infrequent group of lethal anomalies in children and young people; Recent epidemiological studies have shown the relationship of myocardial diseases with sudden unexpected death in apparently healthy people under 35 years of age [5]. The incidence of sudden death in children and adolescents ranges from 0,8 to 6,2/100,000 people/year; 90% are of cardiovascular origin and among the most frequent causes are hypertrophic cardiomyopathy (25%), arrhythmogenic right ventricular cardiomyopathy (20%), coronary anomalies (12%), long QT syndrome (20%) and myocarditis (12%); Within other causes drugs and stimulants such as cocaine are found [6]. Although acute myocardial infarction also appears in people over 45 years of age, young people also suffer from it [7].

Etiology

In Pediatrics, amongst the causes of an AMI is the anomalous origin or aberrant course of a coronary artery, Kawasaki disease, congenital heart disease (pre and post-surgery) and dilated cardiomyopathy. Less frequently, it can be associated with hypertension, lupus, myocarditis, cocaine intake, and use of adrenergic drugs [8]. A frequently recognized cause of AMI is obstructive coronary artery disease; however, studies conducted in patients with ST-segment elevation AMI in the first 24 hours after the onset of pain, have shown that up to 10% of these do not present obstructive coronary lesions [9].

Electrocardiogram

Normally in children, the ST segment should not be seen elevated more than 1 mm or depressed less than 0.5 mm in any lead. An exception to the above is the "early repolarization", a normal variant in healthy adolescents where ST elevations of 2 to 4 mm may appear accompanied by tall T waves. Abnormal ST changes present in any situation that causes damage to the myocardium, such as congenital changes in the coronary arteries, but they can also occur in pericarditis, myocarditis, severe right or left ventricular hypertrophy, myocardial ischemia, and hydroelectrolytic disorders [10].

Among the most frequent electrocardiographic findings seen in an AMI in children include: Wide Q waves (> 0.035s) of new appearance a few hours later, ST segment elevation (> 2 mm) in the first few hours, biphasic T waves in the first few days (inverts sharply, then normalizes over time), prolonged QTc interval (> 0.44s) with abnormal Q waves [8].

Clinical manifestations

The imbalance between the supply and demand of oxygen in the myocardium is the determining event of the ischemic lesion that will develop into myocardial infarction. Ischemic symptoms that may present include: chest, upper extremity, jaw, or epigastric pain during exercise or at rest, or an anginal equivalent such as dyspnea or fatigue. Myocardial infarction (MI) can also occur with atypical symptoms, such as palpitations or cardiac arrest, or even without presenting any symptoms.

Classification

- Type 1 MI: MI caused by atherothrombotic coronary artery disease.
- Type 2 MI: Mismatch between oxygen supply and demand without acute rupture of an atherothrombotic plaque.
- Type 3 IM: Patients presenting with symptoms of AMI, ECG changes or ventricular fibrillation and dying before cTn alterations occur.

- Type 4 IM: Related to revascularization procedures.
    - Type 4a: Associated with percutaneous coronary intervention (signs and symptoms of a myocardial infarction with cTn values > 5 × the 99th percentile of the upper limit).
    - Type 4b: Associated with documented thrombosis of endovascular prosthesis (stent).
- Type 5 IM: Related to coronary artery bypass grafting. (signs and symptoms of a myocardial infarction with cTn values > 10 × 99th percentile of the upper limit) [1].

Differential diagnosis of ST elevation

The detection of ST segment elevation in a patient with chest pain should be considered STEMI, nevertheless, since only a minority of patients with chest pain and ST elevation have a final diagnosis of AMI, alternative non-ischemic causes of ST elevation such as those listed below may also be considered:

- **ST elevation myocardial infarction (STEMI):** Transmural ischemia appears due to sudden blockage of a coronary artery. Ischemia counteracts the function of ion channels in both repolarization (diastole) and depolarization (systole). ST-segment elevation is recorded in leads that reflect activity in the ischemic region. Opposing leads may show inverted STE. The presence of simultaneous reciprocal ST-segment depression in opposing leads is highly specific for a STEMI, so it is always crucial to search for it, sometimes by using additional recordings of the right precordial leads (V3R and V4R) or posterior leads (V7-V9). The weakening of electrical activity in the ischemic myocardium causes a decrease in the amplitude of the R wave. If the myocardial cells are no longer active, as is the case with a more extensive infarct, only activity in the opposite areas appears on the ECG as Q waves.

  After an infarction, the weakened transmural necrotic myocardium can potentially transform into an aneurysm, most commonly in the apex and anterior wall. An aneurysm might be identified as persistent ST elevation, T wave inversion and loss of R waves in the precordial leads. The morphology of the ST segment varies from slightly concave to broadly convex.

- **Pericarditis/myocarditis:** These conditions can interrupt the action potential in the epicardium. Most leads will show ST elevation, except for aVR and V1, which may exhibit ST depression., due to its position distant and opposite to the normal axis of the heart. The involvement of the atria causes the depression of the PR segment indicating a strong likelihood of pericarditis. Repolarization disorders go through various chronological phases; In the first phase, which can last for a maximum of two weeks after the symptoms start, a concave ascending ST elevation with positive T wave and PR depression can be seen (with opposite findings in lead aVR and V1). Following the initial few days to multiple weeks, the PR and ST segments normalize and the T wave may flatten. The last phase consists of a symmetrical inversion of the T wave that will gradually disappear over the following few weeks or months. The ST in pericarditis/myocarditis has mainly a concave morphology and unchanged R wave amplitude.

- **Takotsubo cardiomyopathy:** It is a transient condition with decreased contractility of the mid and often apical portions of the left ventricle and compensatory hyperkinesis of the basal segments. Generally, ECG changes do not correspond to a specific coronary territory and usually lack reciprocal abnormalities without evidence of myocardial necrosis on subsequent cardiac imaging. The first finding on the ECG is ST elevation, mainly in anteroseptal leads (V2-V4). This disappears in a few days and gets substituted by T wave inversion and a prolonged QT interval.

- **J waves syndromes:** This term is used to describe both Brugada syndrome and early repolarization syndrome in which the presence of J waves is observed; A J-point elevation in lead V1-V3 (BrS) or a notch or slur in the second half of the R wave in the inferior and/or lateral leads (ERS).

    - **Brugada Syndrome:** The type 1 Brugada pattern is characterized by a high take-off of QRS-ST elevation of 2 mm in the right precordial leads (V1-V2) without a clear r' wave and with an ascending concave or rectilinear ST followed by a negative T

wave and a slightly longer QRS duration in the right precordial leads. Type 2 has an r' wave ≥ 2 mm in leads V1-V2 with subsequent ST elevation of ≥ 0.5 mm and a positive or biphasic T wave in V2 creating a 'saddle back' morphology.

- **Early repolarization:** A J wave beginning 1 mm above baseline in at least 2 contiguous leads except leads V1-V3. ST segment amplitude is measured at the end of the J wave (Jt) and it is evaluated whether it is horizontal, sloping up or down 100 ms after Jt. The concave upsloping ST segment and high R wave amplitude in the precordial leads should be considered a normal pattern in the absence of a personal or family history of malignant arrhythmia.

Secondary repolarization abnormalities

- **Left bundle branch block:** The left ventricle depolarizes later due to a nonconducted stimulus in the left bundle of His, recognized as a prolonged QRS duration (>120 ms) and left axis deviation. The delayed activation of the epicardium reverses the direction of repolarization, now starting in the (sub)endocardial cells and causing the typical discordant pattern: repolarization (ST segment and T wave) reverses into depolarization (QRS complex). Therefore, a concave ST elevation is present in leads with negative QRS complex (V1-V3, inferior leads), and vice versa (ST depression and T-wave inversion with positive QRS complex in lateral leads).

- **Ventricular pacing:** In right ventricular pacing, repolarization abnormalities are almost the same as in LBBB due to the similar delay in the depolarization of the left ventricle and epicardial cells. The most notable difference with LBBB is a QS pattern mainly observed in V5-V6, while a clear R wave is present in LBBB.

- **Left ventricular hypertrophy:** Depolarization of the epicardium is delayed in hypertrophy due to the thickened muscle wall, leading to a reversed direction of repolarization and discordant repolarization abnormalities best observed in leads showing the highest QRS amplitude (especially lateral leads). Right and septal precordial leads may resemble a concave ST elevation with prominent T waves. Lateral leads often exhibit a concave ST depression and an asymmetric negative T wave with slow descent and rapid return to the baseline, sometimes with a small final positive deflection leading to a biphasic T wave.

Electrolyte disorders

- **Hyperkalemia:** Intercellular conduction is delayed in hyperkalemia, leading to a broad T wave and a shortened QT interval in precordial leads. Furthermore, there is a decrease of the P wave amplitude, widening of the QRS complex, and ST elevation in the right precordial leads (V1-V3). In the case of higher T waves than R waves, it is essential to rule out hyperkalemia. Reciprocal abnormalities, ST elevation, or a broad-based T wave can serve as significant AMI.

- **Hypercalcemia:** Severe hypercalcemia will lead to a shorter duration of the action potential due to decreased activity of Ca^2 channels during the plateau phase and an earlier onset of repolarization. The interval between the onset of the QRS complex and the onset of the T wave will decrease, and the T wave will "move forward". This can give the impression of ST elevation [11].

Case Presentation

We present a 16 years and 10 months old male, with a normal weight for his age and a history of having started taking L-arginine 1000 mg tablets in January 2022 (taking one tablet every 24 hours with irregular adherence), with the last intake on May 2, 2022. Occasional tobacco use and reported a single instance of cannabis consumption in April 2022. Initiated sexual activity at the age of 15, having had a total of 3 sexual partners. Additionally, he reported an episode of syncope in April 2022 without an identifiable triggering factor.

History of present illness: The symptoms initiated on the night of May 6th, 2022, featuring a headache that subsided with a 500 mg dose of paracetamol. Upon awakening on May 7th, 2022, he experienced pronounced precordial pain with radiation to the right arm, with no other symptoms present, for which they decided to go to our hospital unit for evaluation, where he exhibited a painful expression, was conscious, oriented, and adequately hydrated. Cardiac auscultation revealed rhythmic heart sounds of good tone and intensity without

any murmurs. The electrocardiogram showed ST segment elevation in leads DII, DIII, AVL, AVF, V5, and V6 (Figure 1), leading to the decision for admission to this unit.

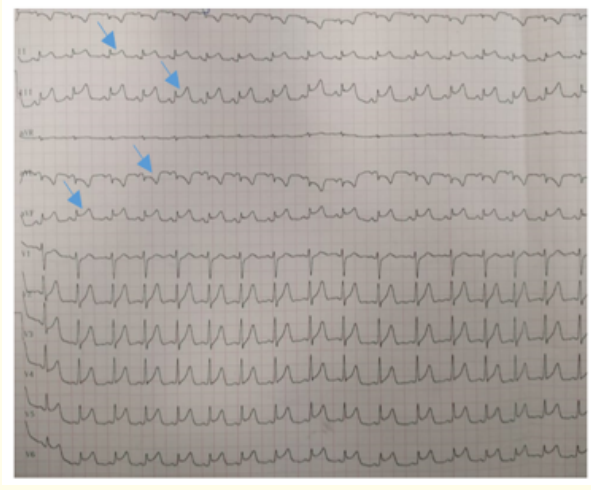

Figure 1: *The electrocardiogram showed ST segment elevation in leads DII, DIII, AVL, AVF, V5, and V6.*

Intravenous solutions were initiated, along with pain management using paracetamol and ketorolac. This led to partial improvement of symptoms. However, at 2:00 p.m., he experienced a sudden increase in precordial pain. A new electrocardiogram was performed (Figure 2), which showed that the previously mentioned ST elevation persisted; The laboratories reported increased of the cardiac enzymes (Table 1), a dose of IV morphine and acetylsalicylic acid is administered and it is decided to transfer to the hemodynamic area to perform coronary angiography (Figure 3 and 4) which reports epicardial arteries without angiographic or ultrasonographic lesions; due to a suspected myopericarditis of probable viral origin, treatment was initiated with acyclovir 400 mg orally every 8 hours, along with intravenous methylprednisolone. Following treatment, the patient remained asymptomatic.

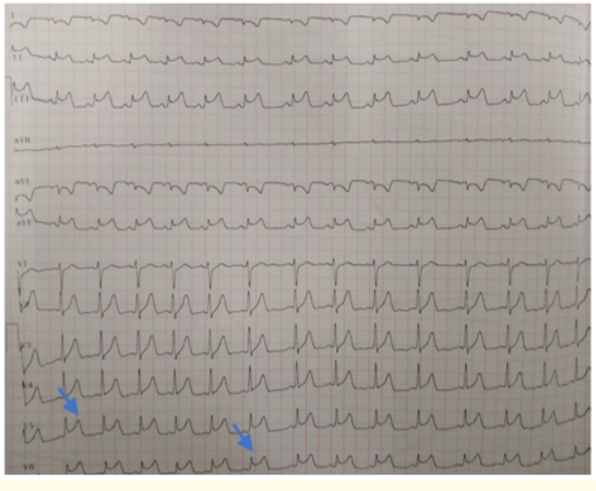

Figure 2: *The second electrocardiogram continued to show persistent ST elevation.*

Laboratories 05.07.22	
Leukocytes	14700/uL
ALT	54 UI/L
AST	310 UI/L
LDH	552 UI/L
CK-MB	398 U/L
Myoglobin	436 ng/ml
BNP	27.9 pg/ml
Troponin I	> 30 ng/ml

Table 1

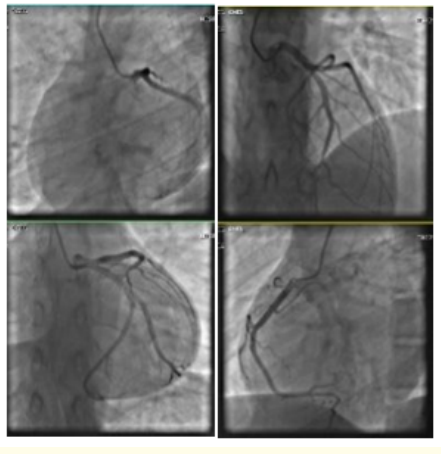

Figure 3

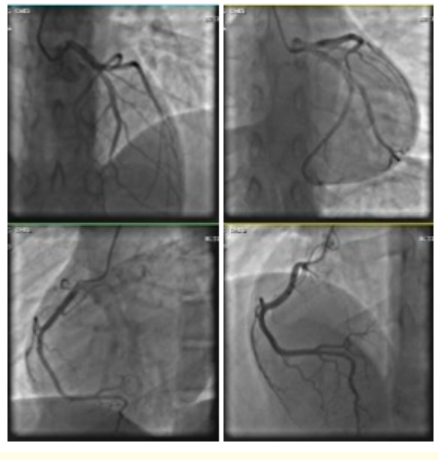

Figure 4

Laboratories from 05.08.2022 (Table 2) reported D-dimer less than 100, decrease of CK-MB and myoglobin and reported an increase of the natriuretic peptide. On 5.9.2022 he presented tachycardia of up to 159 bpm, the electrocardiogram continued with ST elevation (Figure 5); an echocardiogram is performed in which a structurally healthy heart is reported with data of myocarditis, reiterating that the cause is probably viral, therefore, the study protocol began by performing the TORCH panel test, Antibodies against *Trypanosoma cruzi*, HIV and VDRL, which were negative, a toxicological profile was also carried out with negative results. As indicated by Pediatric Cardiology, administration of human immunoglobulin was started at a dose of 2 grams per kilogram to reduce the risk of dilated cardiomyopathy, management with propranolol at a dose of 20 mg every 12 hours is also indicated.

Laboratories 05.08.22	
Leukocytes	8910/uL
Total Cholesterol	87 mg/dl
Triglycerides	80 mg/dl
ALT	55 UI/L
AST	274 UI/L
LDH	659 UI/L
CK-MB	80 U/L
Myoglobin	103 ng/ml
BNP	212 pg/ml
Troponin I	30 ng/ml
D-dimer	< 100

Table 2

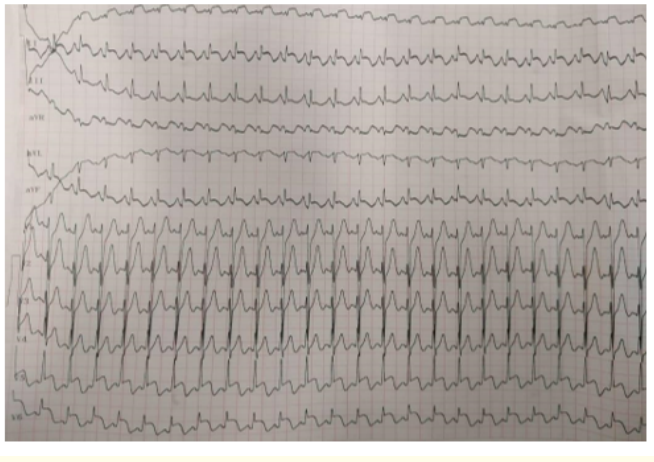

Figure 5: *Electrocardiogram 05-09-2022.*

The electrocardiogram from 05/11/2022 did not show ST elevations and decreased amplitude of the QRS complexes (Figure 6). On 05.12.22, a thyroid profile and cardiac enzymes was performed (Table 3).

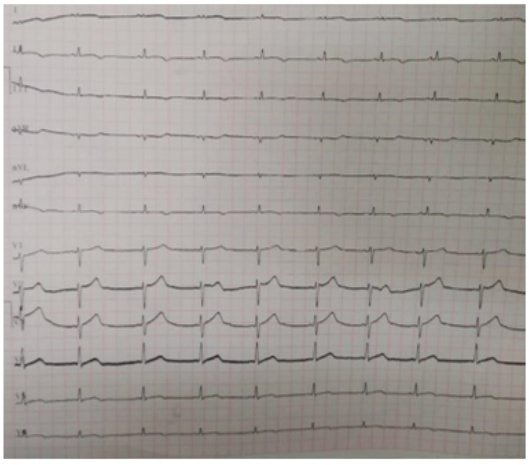

Figure 6: *Electrocardiogram 05-11-2022.*

Laboratories 05.12.23	
Free T3	1.950
Free T4	25.74
Total T3	0.40
Total T4	115.2
TSH	0.05
CK-MB	1 ng/ml
Myoglobin	106 ng/ml
BNP	135 pg/ml
Troponin I	0.10 ng/ml

Table 3

A control echocardiogram was performed on 05.13.2022 in which a structurally healthy heart was reported, myocarditis and tricuspid and mild pulmonary regurgitation. Laboratories from 05.16.22 reported in table 4. Electrocardiogram on 05.17.11 showed inversion of T waves in DII, DIII, aVF and v4-v6 (Figure 7).

On 05.18.2022, he was evaluated by the Pediatric Endocrinology service, where due to the previously mentioned thyroid profile values, he is diagnosed with mild hyperthyroidism without determining this diagnosis as a direct cause of the current cardiac pathology, emphasizing that the presence of cardiac failure or arrhythmias are more frequent in states of severe hyperthyroidism. The patient is discharged on 5.18.2022 asymptomatic, for subsequent monitoring by pediatric cardiology.

On 06.03.2022, he went for an assessment by pediatric cardiology where he was asymptomatic, without having had syncope, dyspnea and/or palpitations, in NYHA functional class I; an electrocardiogram is performed showing sinus rhythm, with HR 76 bpm, aP +79, aQRs +63, aT -105, PR 125 ms, QTc 434 ms, with vector transition in V3, negative T wave Symmetrical in DII, DII, aVF and V6, changes sug-

Laboratories 05.16.22	
Leukocytes	7,390/uL
Hemoglobin	16.7 g/dl
Platelets	295,000
Total Cholesterol	134 mg/dl
Triglycerides	192 mg/dl
HDL	41 mg/dl
LDL	54.6 mg/dl
ALT	38 UI/L
AST	19 UI/L
LDH	236 UI/L
PT	12.1 seconds
PTT	22.9 seconds
INR	1.07
D-dimer	1430
Free T3	2.37pmol/L
Free T4	19.64 pmol/L
Total T3	0.41 nmol/L
Total T4	81.44 nmol/L
TSH	0.23 uUi/ml
Procalcitonin	0.5 ng/ml

Table 4

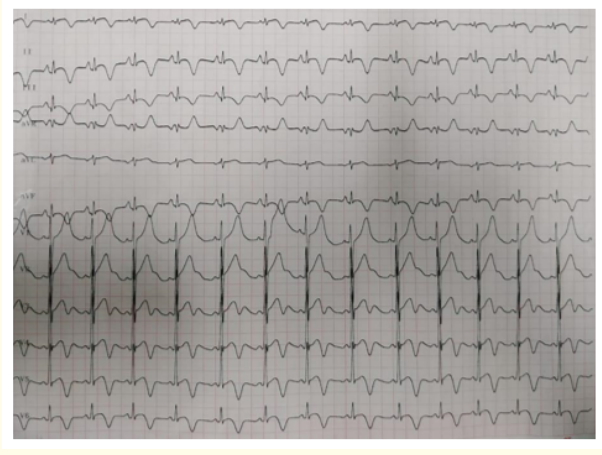

Figure 7: *Electrocardiogram 05-17-2022.*

gestive of inferolateral ischemia. No necrosis data. A control echocardiogram was also performed, reporting a structurally healthy heart, preserved biventricular systolic and diastolic function, normal pericardium, no spillage, thrombi, or image suggestive of vegetations, with no data suggesting the subacute stage of myopericarditis.

Discussion

The term MINOCA was first introduced by John Beltrame in 2013 to replace the previous terminology for myocardial infarction with normal coronary arteries (MINOCA) which included only patients without atherosclerosis of the epicardial vessels and not patients with angiographic stenosis between 1% and 50%.

MINOCA is reported in 6 - 15% of patients with AMI, who are generally relatively young with a lower prevalence of traditional cardiovascular risk factors, and is more frequent in women and in black, Maori and Hispanic ethnic groups [12], this being a very rare pathology, so its diagnosis is one of exclusion, since to diagnose it, other causes of myocardial ischemia had to be ruled out, such as myocarditis, septic cardiomyopathy, hypovolemic shock due to trauma or burns, and kidney or lung diseases [13]. It is estimated that it has a hospital mortality rate of 0.8% and a risk of death or rehospitalization after one year of 11.5%; the incidence of cardiovascular death, AMI, heart failure and CVD after seven years is 17.4%; making it an entity with an adverse prognosis [9].

In 2018, the introduction of the fourth universal definition of AMI required an adjustment of the previous diagnostic criteria for MINOCA and suggested restricting the term MINOCA to patients with an ischemic cause for their clinical presentation. As such, the diagnosis of MINOCA is now achieved after excluding clinically evident causes of troponin elevated heart rate, inadvertent obstructive CAD, and non-ischemic mechanisms of myocytes that can simulate an AMI [12].

The diagnosis of MINOCA, like the diagnosis of MI, indicates that there is an ischemic mechanism of the myocyte damage (i.e. non-ischemic causes are excluded, such as myocarditis). Furthermore, the diagnosis of MINOCA implies that the obstructive CD has not been missed. Rupture of an atherosclerotic plaque and coronary thrombosis may be a cause of MINOCA, i.e. type 1 MI. However, coronary spasm and spontaneous coronary dissection may also be involved, i.e. type 2 MI, along with other possible causes.

MINOCA diagnosis criteria

Diagnosis is made after the coronary angiography on a patient presenting clinical features consistent with an AMI:

- **AMI criteria**
  - Positive cardiac troponin defined as a rise or fall in serial values, with at least one value greater than the 99th percentile of normality.
  - Clinical evidence of infarction due to at least one of the following indicators:
    - Symptoms of ischemia.
    - New or presumably new significant changes of ST-T, or new LBBB.
    - Development of pathological Q waves.
    - Imaging tests with new loss of viable myocardium.
    - Intracoronary thrombus evident on angiography or autopsy.
- **Non-obstructive coronary arteries on angiography:** Absence of obstructive coronary disease on angiography (i.e. 50% no coronary stenosis), in any possible artery related to the infarction.
- **Absence of specific clinical cause for acute presentation:** At the time of angiography the specific diagnosis is not apparent. Therefore, there is a need to continue evaluating the patient for the underlying cause of MINOCA [9].

In 2020, the updated European Society of Cardiology (ESC) guidelines for patients with acute coronary syndromes without persistent ST-segment elevation included a section dedicated to MINOCA. As previously proposed by the AHA, nonischemic causes of acute myocardial injury were excluded from the MINOCA nomenclature [12].

The following class I recommendations were issued:

- To follow a diagnostic algorithm to differentiate true MINOCA from alternative diagnoses.
- Perform Cardiac Magnetic Resonance (CMR) in all MINOCA patients without an obvious underlying cause.
- Treat patients with an initial diagnosis of MINOCA and a conclusively established underlying cause according to the specific disease [12].

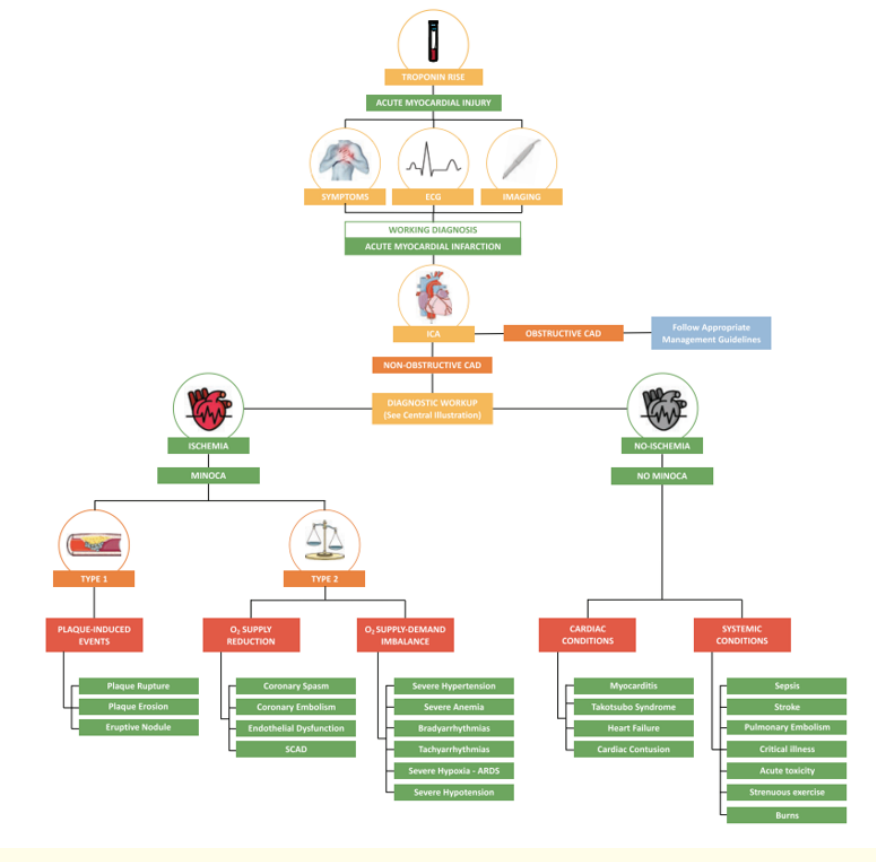

Figure 8: *Diagnostic Algorithm. Source: Occhipinti, Giovanni., et al. "Diagnostic Pathways in Myocardial Infarction with Non-obstructive Coronary Artery Disease (MINOCA)". European Heart Journal. Acute Cardiovascular Care, vol. 10, no. 7, Oxford UP, June 2021, pp. 813–22. https://doi.org/10.1093/ehjacc/zuab049.*

Conclusion

Given the clinical picture, the electrocardiographic manifestations and the elevation of troponins expressed in the patient, the initial approach was for Acute Myocardial Infarction; however, when ruling out obstruction of the epicardial arteries by means of coronary angi-

ography and, due to the suspicion of myopericarditis of probable viral etiology, it was imperative to carry out the appropriate paraclinical tests to confirm or rule out such diagnostic suspicion. Under this premise, the various studies described above were carried out to monitor cardiac function and structure, monitor the evolution of the patient and clarify the etiology of the pathology that we were facing. Once having the reports of the viral panels, which were reported negative; the different echocardiograms, reported without abnormalities; together with a gradual decrease until reaching normal levels of troponins and indirect markers of cardiac injury; along with a significant improvement expressed by the patient, and based on the classification of types of Myocardial Infarctions proposed by the AHA in the fourth universal definition of Myocardial Infarction of 2018, at the same time that the clinical conditions in which he was found, where there were no data of heart failure or any structural anomaly, it was classified as a Type 2 myocardial infarction, since, by definition, the acute rupture of an atherothrombotic plaque is not characteristic of this type of infarction and, above all, it met the criteria consistent with symptoms of acute cardiac ischemia, electrocardiographic changes, development of pathological Q waves and, most importantly, the existence of an atheromatous plaque was ruled out by coronary angiography, correlating it with the initial manifestations of the patient with the pathophysiology of this type of myocardial infarction which is described as a mismatch in the demand and supply of myocardial oxygen, MINOCA being the cause of all the clinical picture of our patient once ruling out structural anomalies, coronary obstruction and infectious processes.

Although MINOCA is a little-recognized pathology, with a higher incidence in the adult stage and with a predominance in the female gender, therefore making the incidence even more rare in the pediatric age group, turning this pathology into a diagnostic challenge for pediatric medicine, since it requires multiple studies and resources to exclude other entities, there is a great need, therefore, to pay more attention and training in the management of chest pain of probable cardiac etiology in adolescents, in order to identify and study early those patients who could present it.

Conflict of Interest

Authors reclaim to have no conflict of interest in this article.

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