ECRONICON OPEN ACCESS

EC CARDIOLOGY
Opinion

Current Criteria for the Diagnosis and Treatment of Acute Myocardial Infarction

Claribel Plain Pazos[1]*, Anel Pérez de Alejo Alemán[2], Carmen Rosa Carmona Pentón[1], Nereyda Caraballo Moya[3], Isis Esther Martín Alonso[4], Anisbel Pérez de Alejo Plain[5] and Nereida Quintero García[6]

[1]Specialist of II Degree in Comprehensive General Medicine, Assistant Professor, Medical Sciences Branch "Lidia Doce Sánchez", Sagua la Grande, Villa Clara, Cuba

[2]Specialist of I Degree in Internal Medicine, Assistant Professor, Teaching General Teaching Hospital "Mártires del 9 de Abril", Sagua la Grande, Villa Clara, Cuba

[3]Specialist of I Degree in Comprehensive General Medicine, Assistant Professor, Medical Sciences Branch "Lidia Doce Sánchez", Sagua la Grande, Villa Clara, Cuba

[4]Specialist I Degree in Comprehensive General Medicine, Assistant Professor, University Polyclinic "IdalbertoRevuelta", Sagua la Grande, Villa Clara, Cuba

[5]1st Year Student of Medicine, Medical Sciences Branch "Lidia Doce Sánchez", Sagua la Grande, Villa Clara, Cuba

[6]Licentiate in Nursing, Assistant Teacher, Polyclinic Mario A Pérez, Sagua la Grande, Villa Clara, Cuba

***Corresponding Author:** Claribel Plain Pazos, Specialist of II Degree in Integral General Medicine, Assistant Professor, Branch of Medical Sciences "Lidia Doce Sánchez", Sagua la Grande, Villa Clara, Cuba.

Received: June 15, 2018; **Published:** September 25, 2018

Abstract

Cardiovascular diseases are a serious epidemiological problem and the main cause of death in the contemporary world, with acute myocardial infarction being responsible for the majority of deaths. Due to the importance of this topic, a review of 40 bibliographies was carried out in order to describe the current criteria for the diagnosis and treatment of acute myocardial infarction, finding that it has been one of the leading causes of death in the world in recent years. and in Cuba, its diagnosis includes precordial pain, electrocardiographic findings and serological markers, and it is confirmed with the positivity of two of these 3 elements. As a more effective treatment, early revascularization was determined by invasive or chemical methods, which reduced complications and deaths for this cause.

Keywords: *Myocardial Infarction; Heart Attack; Heart Attacks; Myocardium*

Introduction

Cardiovascular diseases are a serious epidemiological problem in the contemporary world [1]. Ischemic heart disease has been the biggest health problem and the main cause of death in many countries of the world for several decades. At the beginning of the 20th century, these caused less than 10% of all deaths in the world and in this century, they are responsible for almost half of the deaths in developed countries, as well as 25% in the countries in of development [2]. In 2015, it is estimated that 17.7 million people died, of these more than three quarters occurred in developing countries [3]. The report of the Ministry of Health of the Americas (NHANES), published In 2016, the number of patients with coronary heart disease was estimated at 15.5 million, with a prevalence of 6.2% in subjects over 20 years of age (7.6% men and 5% women) [4]. In the United States, it is estimated that 600,000 new Acute Infarctions occur. of the

Myocardium (IMA) each year, of which 25% present with a silent clinical profile and 320,000 (53.33%) as episodes of exacerbation of ischemic heart disease (AMI and acute unstable angina). Of the totality of AMI that occur annually in the United States, mortality is reported around 25% [2].

Thus, coronary heart disease continues to be the leading cause of death in developed countries (three out of every four deaths are cardiovascular causes and it is estimated that in 2020 it will be the first cause of death worldwide [5].

In Ecuador, cardiovascular diseases occupy the first place among the causes of mortality, and among them the most feared disease is AMI, its incidence is close to 40,000 people per year, which would mean that every 12 minutes an Ecuadorian suffers a infarction [2].

In Cuba, in 2016, heart disease occupied the first cause of death with a total of 24 462 deaths, 66.05% due to ischemic diseases; and of these, 44.42% due to acute myocardial infarction. The provinces with the highest incidence were Havana, Santiago de Cuba, Matanzas, Holguín and Villa Clara [6]. In Villa Clara and Sagua la Grande, heart disease was also the main cause of death. And in Sagua in 2016 there were 77 hospital admissions for this cause with 8 intra-hospital deaths to 10,38% of deceased's [7].

The incidence of coronary disease is more frequent in men, being the proportion with respect to women of 2:1 or more [8].

Because of the importance of the diagnosis and early treatment of acute myocardial infarction to avoid complications and death to a large part of the population, it is decided to carry out an investigation with the objective of updating this topic.

Development

The definition of acute coronary syndrome (SICA) encompasses the spectrum of conditions compatible with acute myocardial ischemia and/or infarction, due to the abrupt reduction of coronary blood flow [9] and is classified as having no ST-segment elevation (SICASEST) or elevation of the ST segment (SICACEST), when there is no expression of markers of myocardial necrosis, is called unstable angina (AI) [10]. Different studies have shown that the most frequent is SICACEST [1,2,4-6,10]. AMI is the necrosis of myocardial cells as a consequence of prolonged ischemia caused by the sudden reduction of coronary blood supply, which involves one or more areas of the myocardium [9-12]. The authors agree that the main cause of AMI is the rupture or erosion of an atheromatous plaque which causes an acute thrombosis with or without concomitant vasoconstriction [2,3,5,8-10]. The rupture of the plaque exposes atherogenic substances that can produce an extensive thrombus in the artery related to the infarction, if the obstruction is not complete it can lead to asymptomatic episodes of coronary occlusion and if it is complete they provoke transparietal lesion of the ventricular wall in the myocardial bed irrigated by the affected coronary artery and usually elevates the ST segment in the Electrocardiogram (ECG) [8,9]. In the universal classification of myocardial infarction invoked by Thygesen K., *et al.* in the third universal definition of myocardial infarction [10] that appears in The Third Global AMI Task Force, 5 types of infarcts are described, based on pathological, clinical and prognostic differences, along with different treatment strategies. These 5 types are described in table 1 [10].

Type 1: Spontaneous myocardial infarction Spontaneous myocardial infarction related to atherosclerotic plaque rupture, ulceration, fissuring, erosion, or dissection with resulting intraluminal thrombus in one or more of the coronary arteries leading to decreased myocardial blood flow or distal platelet emboli with ensuing myocyte necrosis. The patient may have underlying severe CAD but on occasion non-obstructive or no CAD.
Type 2: Myocardial infarction secondary to an ischaemic imbalance In instances of myocardial injury with necrosis where a condition other than CAD contributes to an imbalance between myocardial oxygen supply and/or demand, e.g. coronary endothelial dysfunction, coronary artery spasm, coronary embolism, tachy-/brady-arrhythmias, anaemia, respiratory failure, hypotension, and hypertension with or without LVH.
Type 3: Myocardial infarction resulting in death when biomarker values are unavailable Cardiac death with symptoms suggestive of myocardial ischaemia and presumed new ischaemic ECG changes or new LBBB, but death occurring before blood samples could be obtained, before cardiac biomarker could rise, or in rare cases cardiac biomarkers were not collected.
Type 4a: Myocardial infarction related to percutaneous coronary intervention (PCI) Myocardial infarction associated with PCI is arbitrarily defined by elevation of cTn values > 5 x 99th percentile URL in patients with normal baseline values (≤ 99th percentile URL) or a rise of cTn values > 20% if the baseline values are elevated and are stable or falling. In addition, either (I) symptoms suggestive of myocardial ischaemia, or (II) new ischaemic ECG changes or new LBBB, or (III) angiographic loss of patency of a major coronary artery or a side branch or persistent slow-or no-flow or embolization, or (IV) imaging demonstration of new loss of viable myocardium or new regional wall motion abnormality are required.
Type 4b: Myocardial infarction related to stent thrombosis Myocardial infarction associated with stent thrombosis is detected by coronary angiography or autopsy in the setting of myocardial ischaemia and with a rise and/or fall of cardiac biomarkers values with at least one value above the 99th percentile URL.
Type 5: Myocardial infarction related to coronary artery bypass grafting (CABG) Myocardial infarction associated with CABG is arbitrarily defined by elevation of cardiac biomarker values > 10 x 99th percentile URL in patients with normal baseline cTn values (≤ 99th percentile URL). In addition, either (I) new pathological Q waves or new LBBB, or (II) angiographic documented new graft or new native coronary artery occlusion, or (III) imaging evidence of new loss of viable myocardium or new regional wall motion abnormality.

Table 1: *Universal classification of myocardial infarction.*

Several studies have listed some risk factors that predispose to the occurrence of AMI, such as Hyperlipoproteinemia, Hypertension, Diabetes mellitus, Smoking, Obesity and Stress [2,5,8]. Numerous epidemiological studies have not only been able to demonstrate the effect of risk factors on the occurrence of cholera disease However, the synergistic interaction between them and the effect on primary and secondary prevention of their modification and control is also important [14]. Knowledge of these risk factors for the family doctor is of vital importance since this would work in the preventive work with the objective of diminishing the influence of the IAM in the community. Acute myocardial infarction (AMI) can be recognised by clinical features, including electrocardiographic (ECG) findings, elevated values of biochemical markers (biomarkers) of myocardial necrosis, and by imaging, or may be defined by pathology [10]. The presence of two of first three of these elements allows the diagnosis to be made [9]. Thygesen K., *et al.* [10] in the third universal definition of myocardial infarction that appear in The Third Global AMI Task Force inform that in 2000, the First Global AMI Task Force presented a new definition of AMI, which implied that any necrosis in the setting of myocardial ischaemia should be labelled as AMI. These principles were further refined by the Second Global MI Task Force, leading to the Universal Definition of Myocardial Infarction Consensus Document in 2007, which emphasized the different conditions which might lead to an AMI. This document, endorsed by the European Society of Cardiology (ESC), the American College of Cardiology Foundation (ACCF), the American Heart Association (AHA), and the World Heart Federation (WHF), has been well accepted by the medical community and adopted by the WHO.

However, the development of even more sensitive assays for markers of myocardial necrosis mandates further revision, particularly when such necrosis occurs in the setting of the critically ill, after percutaneous coronary procedures or after cardiac surgery. The Third Global AMI Task Force has continued the Joint ESC/ACCF/AHA/WHF efforts by integrating these insights and new data into the current document, which now recognizes that very small amounts of myocardial injury or necrosis can be detected by biochemical markers and/or imaging recognizes that very small amounts of myocardial injury or necrosis it can be detected by biochemical markers and/or imaging. Clinical manifestations may appear pain of appearance with effort or at rest. This pain usually lasts at least 20 minutes, but may be shorter [9,11-13] and may last, in the opinion of other authors, up to at least 15 minutes [15]. It can occur in the center or left of the thorax and radiate to arms, jaw, back or shoulders. It may be associated with dyspnea due to left ventricular failure, nausea, vomiting, diaphoresis, or syncope. These symptoms may be in association with chest pain or present in the absence of chest pain [9]. Pain may present primarily atypically in the epigastrium (often confused with digestive pathology), arms, shoulders, wrist or back without occurring in the chest. chest. It is usually oppressive, burning or burning [9,15], although several bibliographies report that in 25% of infarcted patients there are no symptoms (silent AMI) [2]. It is very important to take these aspects into account in attention to the patients in the emergency services that is the 1st link of attention where an opportune and immediate diagnosis of this disease must be made.

Changes in the electrocardiogram (ECG) in relation to AMI can be observed in the 12-lead registry. Dynamic changes in the ECG waveforms during acute myocardial ischaemic episodes often require acquisition of multiple ECGs, particularly if the ECG at initial presentation is non-diagnostic. Serial recordings in symptomatic patients with an initial non-diagnostic ECG should be performed at 15 - 30 min intervals or, if available, continuous computer-assisted 12-lead ECG recording. Recurrence of symptoms after an asymptomatic interval are an indication for a repeat tracing and, in patients with evolving ECG abnormalities, a pre-discharge ECG should be acquired as a baseline for future comparison [10]. Among the electrocardiographic findings of AMI are: new ST-segment elevation greater than 1 mm (0.1 mV) in two or more contiguous leads: in leads V2-V3 ≥ 2.5 mm (0.25 mV) in men under 40 years, ≥ 2 mm in those aged 40 years or older and ≥ 1.5 mm (0.15 mV) in women. BRI of the Beam of His (BRIHH) of new appearance and suggestive history of IAM.ST-segment depression of V1-V4 and history suggestive of AMI that may correspond to an inferobasal (posterior) AMI [9,12,16-21]. The appearance of giant T waves (hyperacute) must be considered in the early phase of AMI), even without ST segment elevation (they can benefit from thrombolysis) [9]. Therefore, before an electrocardiogram with these giant T and a characteristic clinical picture, thrombolytic treatment should be started even without having the enzymatic results. It should also be remembered that a normal ECG does not exclude AMI in the presence of a characteristic clinical picture [9], an aspect of great importance when making decisions in emergency services, since the patient with characteristic symptoms of AMI and negative electrocardiogram it must be admitted to the hospital wards with follow-up, because in this case if we must wait for the enzymatic results or some electrical change in the evolutionary EKG. It is also necessary to take into account that there are electrocardiographic findings that can confuse the diagnosis of the IMA, Thygesen K., *et al.* in the third universal definition of myocardial infarction that appears in The Third Global AMI Task Force [10] alludes to this topic. A QS complex in

lead V1 is normal. A Q wave < 0.03 seconds and < 25% of the R wave amplitude in lead III is normal if the frontal QRS axis is between -30° and 0°. A Q wave may also be normal in aVL if the frontal QRS axis is between 60° and 90°. Septal Q waves are small, non-pathological Q waves < 0.03 seconds and < 25% of the R-wave amplitude in leads I, aVL, aVF, and V4 - V6. ECG abnormalities that mimic myocardial ischaemia or MI are presented by Thygesen K., *et al.* [10] (Table 2).

False positives
• Early repolarization
• LBBB
• Pre-excitation
• J point elevation syndromes, e.g. Brugada syndrome
• Peri-/myocarditis
• Pulmonary embolism
• Subarachnoid haemorrhage
• Metabolic disturbances such as hyperkalemia
• Cardiomyopathy
• Lead transposition
• Cholecystitis
• Persistent juvenile pattern
• Malposition of precordial ECG electrodes
• Tricyclic antidepressants or phenothiazines
False negatives
• Prior MI with Q-waves and/or persistent ST elevation
• Right ventricular pacing
• LBBB

Table 2*: Common ECG pitfalls in diagnosing myocardial infarction.*

Myocardial injury is detected when blood levels of sensitive and specific biomarkers such as cTn or the MB fraction of creatine kinase (CKMB) are increased. Cardiac troponin I and T are components of the contractile apparatus of myocardial cells and are expressed almost exclusively in the heart. Although elevations of these biomarkers in the blood reflect injury leading to necrosis of myocardial cells, they do not indicate the underlying mechanism [9,10].

Myoglobin is the first to rise at 1.5 - 2 hours and remains elevated for 8 - 12 hours [18], some studies agree that Troponin Te I: They are the most sensitive and specific marker, rising 3 - 4 hours after the onset of AMI and remaining there for 7 days at 14 days [9,10,18]. CPK-MB rises 2-3 hours and remains high for 1 - 2 days [17], CPK rises 4 - 6 hours after AMI and normalizes in two or three days [9,18], although Thygesen K., *et al.* in the Third Global AMI Task Force raises its measurement is not recommended for the routine diagnosis of AMI, due to the wide tissue distribution of this enzyme. If it needs to be used, it must be combined with troponin or CPK-MB for a more accurate diagnosis of AMI [14]. If quantitative Troponin T is available, a value equal to or greater than 0.1 ng/ml would be considered positive. The result of cardiac enzymes should be requested at the time of admission to the emergency room; if it is negative and there is a high index of suspicion, it will be repeated at 6 and 12 o'clock [9]. Once the diagnosis is suspected, it should be treated as a red code and start treatment immediately, as previously discussed, time is muscle, when more The treatment is started faster and the less time is spent in the diagnosis, the lower the muscle damage that the heart will suffer and the greater the survival.

The treatment consists of:

a) Horizontal rest with continuous electrical monitoring, placing a defibrillator monitor near the patient, peripheral intravenous access and non-invasive monitoring of oxygen saturation.

b) Pain relief: Opiates. Morphine of choice (10 - 20 mg ampule) 4 - 8 mg intravenously (IV); Repeatable doses of 2 mg every 5 - 15 minutes. In the practical order, a 10 mg (1 cc) morphine chloride ampule is diluted in 9 cc of physiological saline and a solution of 1 mg of morphine is obtained for each cc. If an important vagotonic effect is obtained (bradycardia, hypotension) after administration of morphine, administer atropine (0.5 mg ampules) 0.5 - 1 mg IV to a dose of 2 mg if necessary. If there is known hypersensitivity to morphine or if the AMI is lower with large vagal discharge, hypotension or bradyarrhythmia, meperidine (50 - 100 mg ampulla) 25 mg IV is preferred; Repeatable dose every 5 - 15 minutes. If significant vomiting occurs, antiemetics should be used (metoclopramide 5 - 10 mg IV that can be given at the same time as opioids). Avoid intramuscular injections.

c) Oxygen: 2 - 4 liters per nasal catheter or mask. Mainly in case of lack of air, hypoxia (oxygen saturation < 90%) or other signs of heart failure (HF), it has been proven that a hyperoxia can be harmful because of its vasoconstrictor effect that causes the necrosis of the cardiac muscle to spread.

d) Nitrites: Nitroglycerin: 1 tablet (0.5 mg) sublingual entry that can be repeated every 5 minutes for 3 - 4 doses. It cannot be used if the systolic arterial pressure (SBP) is less than 90 mmHg or heart rate (HR) less than 50 or greater than 100 beats per minute (bpm) and suspicion of right ventricle (RV) AMI.

e) Antiplatelets: Aspirin: 160-325 mg dose chewed and ingested as soon as symptoms start. It is preferred without enteric coating and an initial average dose of 250 mg. Should not be used if there is known allergy to salicylates, then use Clopidogrel: (75 mg tablets) loading dose if the patient will receive thrombolytic therapy with recombinant streptokinase (SKR), 300 mg in patients younger than 75 years and 75 mg in greater. If not indication of SKR dose of 75 mg for any age.

f) Beta-blockers (BBA): Oral BBAs should be administered as early as possible in all patients without contraindications. There are contraindications in severe ventricular dysfunction, HR less than 60 per minute, atrioventricular block (BAV) of greater degree than the first, and a history of bronchospasm. The benefit of indefinite treatment with beta-blockers after STEMI is well established, but not intravenous administration of these drugs systemically in the acute phase. The drug to be used is Atenolol: initial dose orally according to the clinical situation: 50 - 100 mg orally every 24 hours. BBA IV: initial dose 5 mg IV followed by the oral dose at the time, in certain clinical situations (hypertension, tachyarrhythmias, non-tolerance of the oral route) [9,10,17].

g) Restoration of coronary flow and reperfusion of myocardial tissue: Early pharmacological or mechanical reperfusion should be performed during the first 12h of the onset of symptoms in patients with clinical STEMI and with persistent ST-segment elevation or with new or suspicious a new complete blockade of the left branch [9,10,17,19-22]. Prehospital care of patients with suspected STEMI with fibrinolytic drugs is a crucial element, which directly conditions the probability of survival and can be safely performed in Intensive municipal areas, especially if the expected time for arrival at the hospital is greater than 30 minutes, less than two hours after the onset of symptoms and clear absence of contraindications. Recombinant streptokinase has been produced in Cuba since the 1990s and its safe use has been validated in medical practice. This thrombolytic drug is available in all hospitals, municipalities and in primary health care [23] and is the unique thrombolytic drug available in Cuba till now. Streptokinase of 1,500,000 pcs. It is diluted in 100 ml of 0.9% physiological saline solution or 5% dextrose, to pass in 30 - 60 minutes through a peripheral vein, preferably in an infusion pump [9,11]. By lysing the thrombus, the coronary flow is restored, with which is guaranteed to decrease ventricular dysfunction and with it a reduction in mortality [16,18-20]. If we start from the theoretical concept that 15% of the myocardium at risk becomes non-viable every 30 minutes of persistent occlusion, 5 support the notion that the reduction of delays and thus an earlier reperfusion, improve the patient's prognosis. In the vast majority of care units there are no means available for revascularization by invasive procedures, which is why drug reperfusion therapy is the most widely used worldwide, based on the principle that revascularization should begin immediately diagnosed the patient and not delay its start with the transfer to units that have the conditions for these procedures. In Cuba, with the use of emergency ambulances equipped with state-of-the-art technology, the immediate start of Streptokinase treatment is guaranteed even in remote places where they can access.

Only patients with contraindications are exempt from this. Several authors classify absolute: previous intracranial hemorrhage of any type, ischemic stroke in the last 3 months, suspected aortic dissection, active bleeding or bleeding diathesis (except menstruation), structural vascular brain injury known intracranial neoplasm malignant known. Closed head injury, in the last three months. Of concerning: History of ischemic stroke more than three previous months, or intracerebral disease other than those in absolute contraindications Refractory hypertension (systolic blood pressure > 180 mmHg and/or diastolic blood pressure > 110 mmHg), traumatic or prolonged Resuscitation, internal bleeding recent (in 2 - 4 weeks), Previous exposure (between 5 days and 6 months) to Streptokinase or previous allergic reaction [9,11,18,20]. Among the most frequent complications are: arrhythmias, post-AMI angina and shock cardiogenic [21]. Currently, the latter is the one that causes more deaths after an AMI [21,23-25].

Conclusions

AMI continues to be one of the leading causes of death in the world and in Cuba, its diagnosis is made by the clinic, electrocardiographic findings and serological markers, with two of these 3 positive elements and can confirm the same, the most effective treatment it is the early revascularization by invasive or chemical methods which has reduced the complications and deaths due to AMI.

Bibliography

1. Poll Pineda JA., *et al*. "Clinical and epidemiological characterization of patients with acute coronary syndrome according to sex". *Medisan* 21.10 (2017): 3003-3010.

2. Moreno Martín G., *et al*. "Incidencia y manejo del infarto agudo miocárdico con elevación del segmento ST, Hospital José María Velasco Ibarra, 2014". *Enfermería Investiga* 1.4 (2016): 151-157.

3. Dirección de Información Documentación. Escuela Latinoamericana de Medicina. "Colesterol elevado y riesgo cardiovascular". *Boletín Bibliográfico Cardiosalud* 1.3 (2017): 14.

4. Llancaqueo M. "Manejo del síndrome coronario agudo en el paciente adulto mayor". *Revista Médica Clínica Las Condes* 28.2 (2017): 291-300.

5. Balmori Marín BL., *et al*. "Caracterización clínico-epidemiológica del infarto del miocardio trombolizado en la unidad de cuidados intensivos de adultos". *Mediciego* 19.1 (2013).

6. Dirección Nacional de Registros Médicos y Estadísticos de Salud. "En línea: Anuario Estadístico de la Salud 2016". La Habana: Ministerio de Salud Pública (2017).

7. Plain Pazos C., *et al*. "Analysis of the Behavior of Acute Myocardial Infarction during 2016. Hospital "Mártires del 9 de Abril". Sagua la Grande. Villa Clara. Cuba". *EC Cardiology* 5.7 (2018).

8. Moldes M., *et al*. "Comportamiento del infarto agudo del miocardio en Cuidados Intensivos. Centro Diagnóstico Integral Simón Bolívar. Venezuela". *Revista Médica Electrónica* 39.1 (2017): 43-52.

9. Coll Muñoz Y., *et al*. "Infarto agudo del miocardio. Actualización de la Guía de Práctica Clínica". *Revista Finlay* 6.2 (2016): 170-190.

10. Thygesen K., *et al*. "Third universal definition of myocardial infarction". *European Heart Journal* 33.20 (2012): 2551-2567.

11. Martínez Sánchez C., *et al*. "Panorama del Infarto Agudo del Miocardio en México". In: Martínez Ríos MA. Infarto Agudo del Miocardio. Documento de postura. Colección de aniversario. 150 años. México: Academia Nacional de Medicina (2014): 3-7.

12. Steg G., *et al*. "Guía de práctica clínica de la ESC para el manejo del infarto agudo del miocardio en pacientes con elevación del segmento ST". *Revista Española de Cardiología* 66.1 (2013): 53.

13. Vilches Izquierdo E., *et al*. "Tratamiento a largo plazo del infarto agudo del miocárdico con elevación del segmento ST: actualización". *Revista Cubana de Investigaciones Biomédicas* 33.3 (2014): 322-333.

14. Alvarado Izquierdo OU. "Comportamiento del sindrome coronario agudo ST elevado en el Hospital Regional Honorio Delgado Espinoza año 2015" (2017).

15. Paolasso E., *et al*. "Manejo y Tratamiento del InfartoAgudo de Miocardio con Supradesnivel del Segmento ST". 2do Congreso Virtual de Cardiología (2001).

16. Baltodano Flores HF. "Trombólisis en los pacientes con síndrome coronario agudo con elevación del segmento ST, Hospital Escuela Antonio Lenín Fonseca, Enero 2014-Diciembre 2015". Doctoral dissertation, Universidad Nacional Autónoma de Nicaragua, Managua (2017).

17. Ibañes B., *et al*. "Guía ESC 2017 sobre el tratamiento del infarto agudo del miocardio en pacientes con elevación del segmento ST". *Revista Española de Cardiología* 70.12 (2017): 1082.

18. Borrayo Sánchez G., el al. "Protocolo para la atención de infarto agudo del miocardio en urgencias: Código Infarto". *Revista Médica del Instituto Mexicano del Seguro Social* 55.2 (2017): 233-246.

19. Senior JM., *et al*. "Guía de práctica clínica para pacientes con diagnóstico de síndrome coronario agudo: atención inicial y revascularización". *Revista Colombiana de Cardiología* 20.2 (2013).

20. Escudero García G. "Tendencias en el Tratamiento de Reperfusión en Pacientes con Infarto Agudo de Miocardio con Elevación del Segmento ST". Estudio de 16 años de Duración. Tesis Doctoral, Universidad de Murcia (2016).

21. Tolego Valverde LD. "Infarto Agudo delMiocardio: Relación edad-reperfusión, tiempo-Reperfusiónenel servicio de emergencias Hospital San Francisco deAsís. Grecia". *Revista Clínica Escuela de Medicina UCR-HSJD* 4.1 (2014): 19-26.

22. Martínez García G., *et al*. "Complicaciones intrahospitalarias del infarto del miocardio con elevación del segmento ST". *Revista Cubana de Medicina Militar* 45.3 (2016): 332-343.

23. Olivera Escalona AL. "Reperfusión coronaria en el infarto agudo del miocardio con elevación del segmento ST: importancia del contexto y tiempo de aplicación". *Medisan* 19.10 (2015): 1259-1267.

24. Sánchez Abalos V.M., *et al*. "Morbilidad y mortalidad por infarto agudo del miocardio". *Medisan* 18.4 (2014): 516-522.

25. Szarfer Jorge L., *et al*. "Reperfusión mecánica simple en el infarto agudo de miocardio: Determinantes, implicaciones en el pronóstico y relación con la histología del trombo". *Medicina* 77.5 (2017): 358-364.

Volume 5 Issue 10 October 2018