Medical and Research Publications
International Open Access

Review Article **Journal of MAR Cardiology (Volume 5 Issue 2)**

Acute Myocardial Infarction in the COVID-19 Era: A Narrative Review

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Received Date: March 20, 2023

Published Date: April 01, 2023

Abstract

Myocardial infarction is part of a group of cardiovascular disorders characterized by cardiac ischemia resulting from an occluded coronary artery. An infarction can manifest clinically as chest pain, neck pain, jaw pain, dyspnoea, and dizziness. Ischemic heart disease is the precipitating condition of myocardial infarction and is the leading cause of death in the world, killing nearly 9 million people annually.

Interestingly, since the start of the Covid-19 pandemic, there has been a significant decrease in myocardial incidence globally. Although research on the reasons surrounding the marked reduction in myocardial infarction is limited, this review explores potential causal factors. This narrative review also investigates the pathogenic effect of SARS-CoV-2 on myocardial infarction.

Myocardial infarction can present differently between the sexes and being aware of this can improve quality of care given to patients. Myocardial infarction is associated with a plethora of well researched risk factors including obesity, smoking, physical activity, and diet. Disturbingly, despite these risk factors being highly preventable, the global prevalence of myocardial infarction is steadily increasing. As such, further exploration into the diagnosis, pathophysiology, management, and prevention of myocardial infarction is warranted, which defines the aim of this narrative review.

Keywords: *Myocardial Infarction, Acute Coronary Syndrome, Ischemic Heart Disease, Covid-19 pandemic, SARS-CoV-2*

Introduction

Myocardial Infarction (MI), the clinical manifestation of ischemic heart disease (IHD), is the leading cause of death worldwide. With a reported 8.9 million deaths in 2019, MI was responsible for an astonishing 16% of the world's total deaths (The top 10 causes of death 2020). In the United States alone, someone suffers a myocardial infarction every 40 seconds (CDC 2021).

Covid-19, which has claimed the lives of approximately 5 million people worldwide (WHO 2021), is a contagious virus transmitted via the respiratory tract that was first identified in December of 2019 in Wuhan, China (Islam, Kundu et al. 2021). Caused by severe acute respiratory syndrome coronavirus 2 (SARS-CoV-2), the ongoing Covid-19 pandemic was officially declared a pandemic by the WHO on March 11th, 2020, resulting in more than half the worlds population going into partial or complete lockdown (Farooq, Khan et al. 2020).

However, the Covid-19 Pandemic has seen a marked reduction in the reported number and hospitalizations of Acute Myocardial Infarctions (AMI). This phenomenon has been observed worldwide in countries including, but not limited to France, Italy, The US, and China (Fardman, Zahger et al. 2021). Some reports show the incidence of MI being nearly halved relative to pre-pandemic numbers (Bhatt, Moscone et al. 2020). These findings appear somewhat contradictory to the nature of AMI, as stressful events have been shown to be a risk factor (Rathore 2018)

Despite the overall decline in incidence, an Israeli study published in 2021 observed that AMI patients during the pandemic were almost twice as likely to have severe complications from the ischemic episode than the control group from 2018 (Fardman, Zahger et al. 2021). They also identified a significant time delay in the provision of treatment for patients who suffered from an MI during the pandemic. Furthermore, studies conducted by the University of Catanzaro, Italy (Pelle, Tassone et al. 2020) and Jin Yin-tan Hospital, Wuhan (Huang, Wang et al. 2020), identified increased incidence and severity of myocardial infarction in patients who contracted Covid-19. Beyond its psychosocial implications, the danger of Covid-19 to AMI susceptible individuals stems from its ability to cause high levels of systemic inflammation, resulting in a cytokine storm and exacerbating prognosis (Pelle, Tassone et al. 2020).

Also known as acute coronary syndrome or heart attack, MI has a variety of risk factors including blood pressure, age, BMI, stress, and sex (Rathore 2018). In IHD, plaque builds up in the coronary arteries leading to narrowing of the vessels, and ultimately reduced blood flow to the heart. In acute presentations, this plaque ruptures causing a thrombus to form, occluding the vessels, and results in an infarction. MI can be subdivided into three main classifications including STEMI (ST-segment

elevation myocardial infarction**)**, NSTEMI **(**non-ST-segment elevation myocardial infarction**)** and MINOCA **(**Myocardial infarction in the absence of obstructive coronary stenosis**)** that result in myocardial ischemia **(**Mayo Clinic 2020b**)**. Both NSTEMI and STEMI were comparably affected by the pandemic **(**Fardman, Zahger et al. 2021**)**.

The Aim of this study is to outline various theories explaining the reduction in AMI incidence since the start of the pandemic, as well as propose psychosocial factors that may have contributed to this decline. This study will also explore both AMI and AMI complicated with Covid-19, discussing their presentation, diagnosis, pathophysiology, treatment, and prevention.

Methods (Search Strategy)

This narrative review was conducted by exploring contemporary literature on myocardial infarction and Covid-19. Only English resources were included in this study, whereas sources predating 2015 were excluded. Pre-pandemic numbers cited in this narrative review refer to those obtained before the year 2020.

Data was sourced using the key words: Myocardial Infarction OR MI OR Acute Myocardial Infarction OR AMI OR Heart Attack OR Acute Coronary Syndrome OR Coronary Artery Disease OR CAD AND Symptoms AND Diagnosis AND Prevalence OR Incidence AND Pathophysiology AND Management AND Prevention AND Covid-19 OR Coronavirus OR SARS-CoV-2 OR Covid-19 pandemic OR Lockdown.

PubMed, EBSCO, Medline and The UNIC Library were the primary databases used in this narrative review. Data was also obtained from other institutions including the WHO, the CDC, and NICE.

Disease Presentation

Signs & Symptoms

The CDC lists the major symptoms of MI as Chest pain, light-headedness, and shortness of breath. Patients presenting with MI may also have pain in their jaw, back or arms **(**Heart Attack Symptoms, Risk Factors, and Recovery 2021**)**. These symptoms can be accompanied by diaphoresis, abdominal pain, pleuritic pain, and indigestion, but are less commonly seen **(**Basit, Malik et al. 2021**)**.

An elevated troponin level is a specific marker for cardiac injury that is widely regarded as a key sign of MI **(**Pagidipati, Peterson 2016**)**. Biomarkers such as CK-Mb, which is more often elevated in men, and CRP, which is more often elevated in women, are also indicative of MI **(**Liakos, Parikh 2018**)**

Variation Between Men and Women

MI can present differently between men and women, with women often experiencing more atypical symptoms such as emesis and pain in their neck, back and jaw **(**Pagidipati, Peterson 2016**)**. Conversely, men more commonly experienced chest pain and diaphoresis. In fact, women suffering from MI were found to be 12% more likely to present without chest pain compared to men **(**Liakos, Parikh 2018**)**. It is crucial to recognize the different ways men and women present with MI as the diagnosis is more often missed in women, who are therefore less likely to receive life saving treatment **(**Lee, Liu et al. 2021**)**.

Variations in the Covid-19 Era

During the Covid-19 pandemic however, the number of patients classified as Killip class III and IV nearly doubled compared to pre-pandemic records **(**Fardman, Zahger et al. 2021**)**. Clinically, these patients were more likely to present with lung crackles, cardiogenic shock, and severe hypotension accompanying their infarction, worsening their prognosis**(**Vicent, Velásquez-Rodríguez et al. 2017**)**.

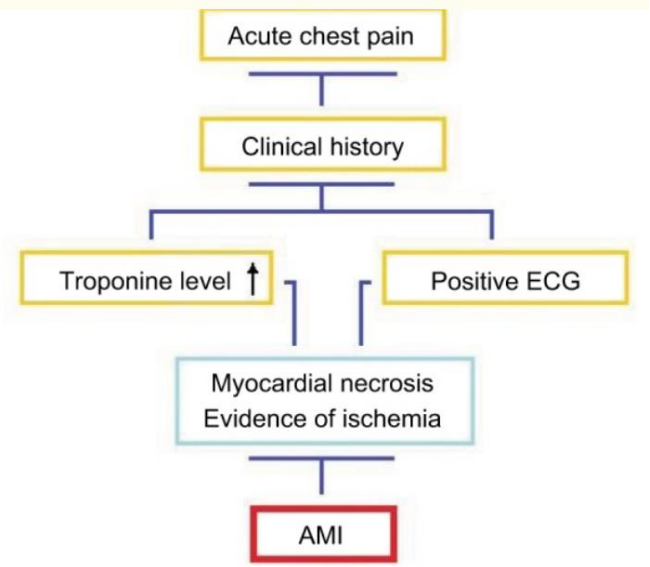
An individual case study by the University of Catanzaro on AMI complicated with Covid-19, reported classical symptoms of MI in their study subject, as well as the typically elevated cardiac biomarkers. However, the study did find high levels of IL-6, and other inflammatory markers linked to the viral infection **(**Pelle, Tassone et al. 2020**)**.

Diagnosis

Physical Examination

MI can be diagnosed by a variety of investigations including physical assessment, electrocardiograms, blood tests for cardiac biomarkers **(**Mayo Clinic 2020a**)** and coronary artery angiography **(**CAG**)**; often regarded as the gold standard **(**Liu, H., Zhang et al. 2021**)**. Figure 1 demonstrates the basic diagnostic steps taken when suspecting AMI **(**Aydin, Ugur et al. 2019**)**. Diagnosing patients begins with the clinical manifestations of the condition, including the risk factors and history associated with MI. Chest

pain, along with the male sex, obesity, increased age, and history of smoking all aid in diagnosing AMI **(**Rathore 2018**)**. Clinically, men diagnosed with MI are more likely to be smokers, have PVD and a previous MI, whereas women are typically older, have more comorbidities and a higher Killip class **(**Liakos, Parikh 2018**)**. Patients admitted for MI during the Covid-19 pandemic were tested for SARS-CoV-2 via nasopharyngeal swab and, in a particular study by the University of Catanzaro, positive cases were further investigated via chest X-ray **(**Pelle, Tassone et al. 2020**)**.



(Figure 1 was obtained from the paper Biomarkers in acute myocardial infarction: current perspectives by Aydin, Ugur et al. 2019**)**

Figure 1: Diagnostic Flow Chart for Patients with MI

Electrocardiogram

Electrocardiograms **(**ECG**)** are the first investigation done when suspecting MI. The ECG passes electric signals through the heart muscle and indicates myocardial damage when there is abnormal signal transmission **(**Heart attack -Diagnoses and treatment 2020**)**. ECGs are useful in distinguishing between STEMI and NSTEMI. However, a study published by the Canadian Journal of Cardiology reported ECGs are less effective in diagnosing NSTEMI due to a high degree of nondiagnostic results and is therefore principally used to identify MI with ST-segment elevation. ECG results showing ST wave depression or inversion are indicative of ischemia, but not of coronary artery occlusion, and are interpreted as NSTEMI **(**Miranda, Lobo et al. 2018**)**. If ST-elevation or anterior ST-depression is observed, the condition should be treated as a STEMI until further tests confirm or decry the diagnosis

(Basit, Malik et al. 2021). A negative ECG is not sufficient to dismiss AMI as a potential diagnosis and are usually repeated in 15–30-minute intervals in symptomatic patients. Continuous ECG investigations during the first hour has been shown to increase ECG sensitivity (Miranda, Lobo et al. 2018).

Cardiac Biomarker Levels Obtained by Blood Test

Troponin is the principal cardiac biomarker in MI diagnosis. It is not only a highly specific and sensitive cardiac marker, but the absence of troponin elevation essentially excludes MI in most patients. A negative blood tests for cardiac biomarkers can rule out MI, an advantage over an ECG for which a negative test is not exclusionary. Troponin blood levels begin to increase between 2-4 hours following the infarct and can remain elevated for up to 3 weeks (Basit, Malik et al. 2021). The peak blood level occurs 24 hours after the ischemic attack, and a 20-50% increase should be observed to confirm MI. The magnitude of the troponin elevation is proportional to the extent of myocardial damage in the patient (Aydin, Ugur et al. 2019).

However, despite the high diagnostic value provided by troponin, it has its limitations. On average, men tend to present with higher troponin levels compared to women following an MI. Consequently, sex-specific biomarkers such as CK-MB and CRP may provide more accurate diagnoses and result in fewer missed cases of MI (Pagidipati, Peterson 2016).

Creatine Kinase-MB (CK-MB), an alternative cardiac biomarker, has been found to be more commonly elevated in men (Liakos, Parikh 2018). Like troponin, CK-MB will reach peak levels around 24 hours post-infarction, but it takes twice as long, between 4-9 hours, to become elevated. CK-MB will also return to normal levels considerably sooner than troponin; within 2-3 days. The relative increase of CK-MB is related to the size of the infarction but is unable to assess minor myocardial damage due to its high molecular weight. Another shortcoming of CK-MB is its lack of specificity. CK-MB is found throughout the body, in skeletal muscle and various organs, and can be elevated due to injury outside the heart. Therefore, elevated CK-MB levels must be paired with positive ECG finding to diagnose MI (Aydin, Ugur et al. 2019).

C-Reactive Protein (CRP) is another commonly used cardiac biomarker in the diagnosis of MI that is more likely to be elevated in women. The sex-specific characteristic of CRP is beneficial as it can help prevent the misdiagnoses of women (Liakos, Parikh 2018). CRP has been shown to effectively monitor inflammation and coronary artery disease (CAD) in MI patients (Aydin, Ugur et al. 2019). Both CRP's

sex-specificity and the ability to recognise CAD is consistent with a study done by the University Malaya Medical Centre, finding that women are considerably more likely to present with dyslipidemia, diabetes, and hypertension **(**Lee, Liu et al. 2021**)**.

Coronary Artery Angiography

Coronary artery angiography **(**CAG**)** is regarded as the best diagnostic investigation for MI. CAG is performed by inserting a catheter into the coronary arteries and injecting a dye that will allow the vessels to appear on X-ray. This imaging modality can clearly show blockages and ruptures, and the results are then interpreted by a CAG scoring system. There are currently a wide variety of scoring systems being used, however, a recent study in 2021 conducted by the Chinese Medical Journal developed and assessed a novel Coronary Artery Tree Description and Lesion Evaluation technique termed CatLet. The CatLet system was designed to incorporate anatomic variations of the coronary vessels and demonstrated a superior predictive ability in long-term clinical outcomes compared to other scoring systems that neglect to take anatomic variations into consideration **(**Liu, J., He et al. 2020**)**.

Interestingly, an Israeli study implementing CAG on MI patients in the Covid-19 era saw eight times fewer cases of non-obstructive coronary artery MI **(**MINOCA**)** compared to their control group. **(**Fardman, Zahger et al. 2021**)**. This finding is consistent with the increased mortality observed among MI patients during the pandemic **(**Fardman, Zahger et al. 2021**)** as a diagnosis of MINOCA is associated with a lower in-hospital mortality **(**Niccoli, Camici 2020**)**. The exact reason for this decline is unclear.

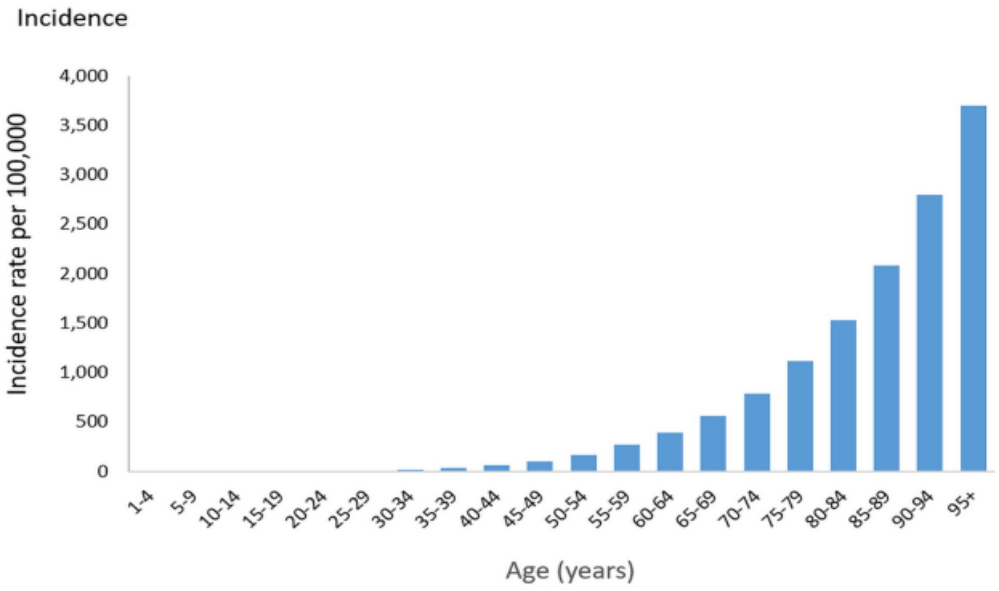
Epidemiology

Prevalence and Incidence of Myocardial Infarction

MI is part of a group of cardiovascular diseases **(**CVD**)** that is the leading cause of death worldwide and is responsible for a third of the world's yearly death toll. Of the deaths due to CVDs, IHD was the more prevalent, accounting for approximately half. IHD is the leading cause of MI **(**NHS 2019**)** and was ranked by the WHO in 2019 as the world's single biggest killer.

A study published in 2020 reported the prevalence of IHD to be 1655 per 100,000, affecting roughly 1.72% of the world's population and it is rising **(**Khan, Hashim et al. 2020**)**. The number of people

with IHD has more than tripled since the year 2000 **(**WHO 2020**)**. By 2030, the prevalence is expected to rise to 1845 per 100,000 due to the increasing presence of MI risk factors in the population. The ageing world population and increasing prevalence of obesity are the main contributors to the rising incidence of MI. Increased age is directly related to increased incidence of IHD as shown in Figure 2 **(**Khan, Hashim et al. 2020**)**. In the United States there are roughly 1.5 million MIs every year, approximating an incidence rate of 600 per 100,000 per year. INTERHEART, a Canadian study, identified 9 risk factors accounting for 90% of the risk of developing an MI. They included obesity, smoking, hyperlipidemia, hypertension, diabetes, sedentary lifestyle, consuming alcohol, fatty diet, and psychosocial factors. Smoking between 1-5 cigarettes a day was associated with a 40% increased risk on its own **(**A Maziar Zafari, Eric H Yang 2019**)**.



(Figure 2 was obtained from the paper Global Epidemiology of Ischemic Heart Disease: Results from the Global Burden of Disease Study by Khan, Hashim et al. 2020**)**.

Figure 2: Age Distributed Incidence Rate of IHD in 2017

Global Distribution of Myocardial Infarction

The global prevalence of IHD varies greatly based on region. Statistics obtained in 2017 reported Russia as the region with the highest prevalence of MI at more than 4000 per 100,000 and Africa with the lowest, below 900 per 100,000. IHD prevalence in Europe for instance is more than doubled compared to the global average. Conversely, low socioeconomic status regions such as India and

Africa are well below the average. This is likely due to their relatively young populations compared to the high life expectancy in European nations. However, when the populations are adjusted for age, the regions more similarly resemble the global average **(**Khan, Hashim et al. 2020**)**. These disparities are expected to decrease in the coming years due to increasing life expectancy in developing nations. As a result, the global incidence of MI is expected to rise as well **(**A Maziar Zafari, Eric H Yang 2019**)**.

Myocardial Infarction Distribution Between Men and Women

MI significantly affects men more than women. Two separate studies by the University Malaya Medical Centre **(**Lee, Liu et al. 2021**)** and Shahid Beheshti University of Medical Sciences **(**Mozaffarian, Etemad et al. 2021**)** both found that between 70-80 percent of patients presenting with both STEMI and NSTEMI are male. Men who present with MI are on average younger than women and have fewer comorbidities. Consequently, women have a markedly worse prognosis after suffering an MI. Table 1, obtained from the University of Malaya Medical Centre, shows the in-hospital, 30-day, and 1 year mortality rates of men and women between the years of 2012 and 2016. By all metrics, women had a higher unadjusted mortality rate for STEMI and NSTEMI. **(**Lee, Liu et al. 2021**)**. Mozaffarian, Etemad et al had similar findings, reporting that 1-month, 6-month, and 1-year mortality rates were consistently higher in women. It is important to note that upon adjusting for increased age and comorbidities, the discrepancy between men and women's mortality rate became insignificant.

	STEMI			NSTEMI		
	Men	Women	*p* value	Men	Women	*p* value
In-hospital mortality	1411 (9.80)	456 (19.63)	< 0.001	503 (7.10)	210 (9.93)	< 0.001
30-day mortality	1640 (13.53)	533 (26.80)	< 0.001	670 (11.24)	287 (16.02)	< 0.001
1-year mortality	2166 (19.07)	698 (37.39)	< 0.001	1300 (23.22)	520 (30.70)	< 0.001

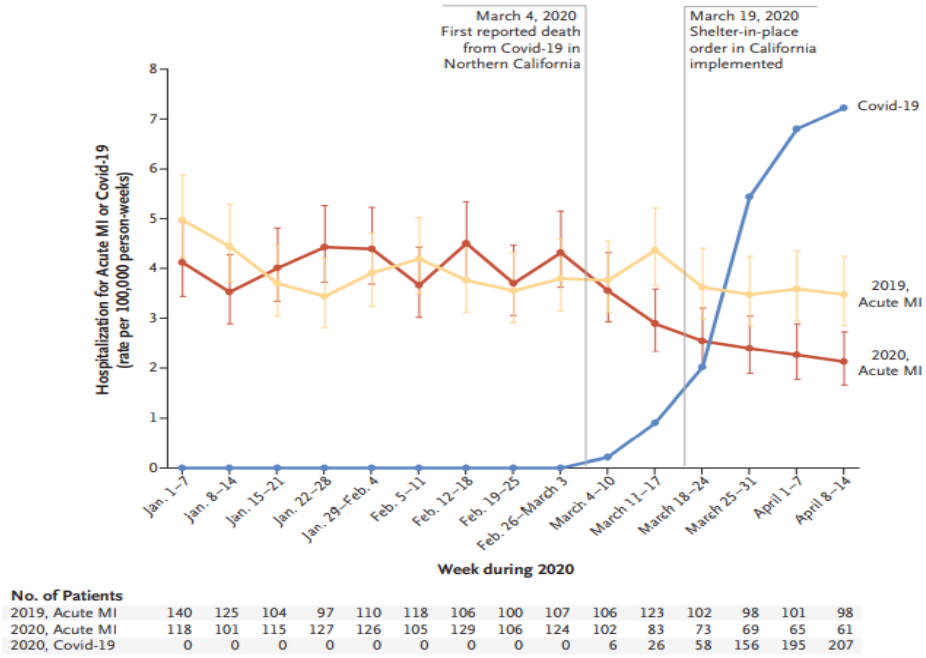
(Table 1 was obtained from the paper Sex and gender differences in presentation, treatment, and outcomes in acute coronary syndrome, a 10-year study from a multi-ethnic Asian population: The Malaysian National Cardiovascular Disease Database—Acute Coronary Syndrome **(**NCVD-ACS**)** registry by Lee, Liu et al. 2021**)**

Table 1: Comparing Various MI Mortality Rates Between Men and Women

The Effect of the Covid-19 Pandemic on Myocardial Infarction Incidence

The ongoing pandemic caused by SARS-CoV-2 had a drastic impact on the incidence of MI. Reports vary between countries, ranging from a 30% decline in France to as high as 80% in Italy. A nation-wide study in France observed that the decline proportionally affected age and gender, however, was amplified in NSTEMI compared to STEMI which were reduced by 35% and 25% respectively **(**Mesnier, Cottin et al. 2020**)**. A similar study conducted in northern California involving hundreds of clinics reported a 48% decline in hospitalizations compared to their 2019 control group. Perhaps even more notable was the decreased incidence rate of MI observed within 2020, which was reduced from 4.1 per 100,000 person weeks between January 1st to March 3rd to 2.1 per 100,000 persons weeks between April 8th and 14th. Interestingly, the incidence of hospitalization for AMI in northern California began to decline directly following the first reported death of Covid-19 in that area as presented in Figure 3 **(**Ambrosy, McNulty et al. 2020**)**. Although the reasons for the decline have been largely unexplored, the most likely reason is that patients feared contracting the novel virus and would forgo admitting themselves to a hospital despite being symptomatic **(**Mesnier, Cottin et al. 2020**)**.

However, patients who were hospitalized for MI during the pandemic experienced more adverse complications and had a slightly increased in-hospital mortality according to an Israeli study involving 13 intensive cardiac care units. Compared to the control group in 2018, MI patients in 2020 were 2 times as likely to go into cardiac arrest, and 4 times as likely to experience mechanical complications. It is worth noting that Covid-19 tests were only available for 25% of cohort, and therefore it is not possible to exclude SARS-CoV-2 as a contributing factor to the worsened clinical manifestations observed **(**Fardman, Zahger et al. 2021**)**.



No. of Patients															
2019, Acute MI	140	125	104	97	110	118	106	100	107	106	123	102	98	101	98
2020, Acute MI	118	101	115	127	126	105	129	106	124	102	83	73	69	65	61
2020, Covid-19	0	0	0	0	0	0	0	0	0	6	26	58	156	195	207

(Figure 3 was obtained from the paper The Covid-19 Pandemic and the Incidence of Acute Myocardial Infarction by Ambrosy, McNulty et al. 2020**)**

Figure 3: Hospitalization Incidence Rate of AMI Before and During the Pandemic Relative to that of Covid-19

Pathophysiology

Common Aetiologies of Myocardial Infarction

The pathogenesis of MI can be precipitated by a variety of factors. The most common aetiologies are typically classified as either type 1 MI or type 2 MI. Type 1 MI is due to CAD that progresses to rupture of the atherosclerotic plaque, occluding the coronary vessels, and reducing blood flow to the myocardium. A Type 2 MI occurs when the oxygen demand of the heart is not being met, but the ensuing myocardial ischemia is due to factors other than atherothrombosis. Although there are overlapping characteristics of some MI aetiologies, STEMIs and NSTEMIs manifest as type 1 MI **(**Kingma 2018**)**, whereas MINOCAs can present as either type 1 or type 2 **(**Niccoli, Camici 2020**)**.

A STEMI arises from the complete blockage of a coronary vessel, whereas a partial vessel occlusion is termed NSTEMI. Both cause ischemia and can afflict irreversible damage to the myocardium if adequate blood flow is not restored within 15 minutes. Under prolonged ischemic conditions the cardiac myocytes will undergo ultrastructural damage including swelling, mitochondrial damage, and

lysis of myocardial fibres by inflammatory cells. Myocytes are either killed by this damage or become apoptotic if they are unable to function normally, leading to cardiac necrosis **(**Kingma 2018**)**. Risk factors that augment CAD will in turn increase the damage done to the myocardium upon MI. Smoking for example, has been shown to precipitate haemorrhaging of the infarct area, as well as exacerbate inflammation to the point of vascular injury **(**Haig, Carrick et al. 2019**)**.

For an MI to be considered non-obstructive, the vessels must not be narrowed more than 50%. MINOCA, depending on the underlying pathology, may be caused by CAD, myocarditis, coronary spasms as well as other disorders. Coronary spasms account for 25% of all MINOCA presentations and are brought about by excessive response to vasoconstrictor stimulation that in turn reduce blood flow to the heart, causing ischemia. Myocarditis, especially when cause by parvovirus B-19, can cause direct damage to cardiac myocytes. Systemic inflammation may also trigger the compression of edematous coronary vessels, further restricting blood flow to the heart **(**Niccoli, Camici 2020**)**.

Myocardial Infarction Triggered by SARS-CoV-2

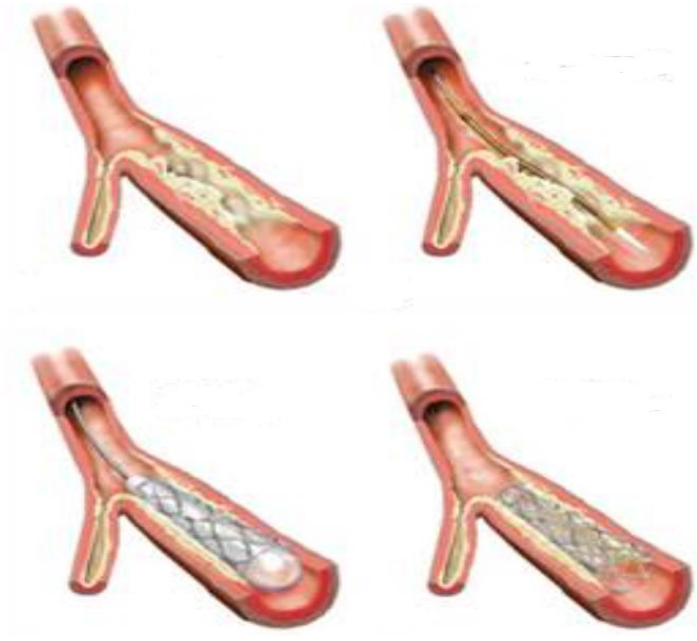
Multiple studies have identified a causal relation between Covid-19 and MI. In severe presentations of Covid-19 widespread systemic inflammation, known as a cytokine storm, has been observed **(**Pelle, Tassone et al. 2020**)**. Inflammation of this magnitude can rupture plaque already present in the coronary arteries, leading to an infarct. A study published in the Journal of the American College of Cardiology observed elevated biomarkers indicative of cardiac injury, including Troponin and CRP, in their Covid-19 patients. They also found increased levels of Interleukin-6 **(**IL-6**)**, a procoagulant that may predispose patients to thrombi formation **(**Bikdeli, Madhavan et al. 2020**)**. These findings are consistent with a study conducted by Johns Hopkins University prior to the pandemic which identified a causal relation between excessive IL-6 stimulation and CVD. Furthermore, the University of Catanzaro concluded that can SARS-CoV-2 can directly trigger MI, as seen in their study subject **(**Pelle, Tassone et al. 2020**)**.

Management

Acute Therapy

AMI patients admitted to the hospital less than 12 hours following the onset of their symptoms are indicated for reperfusion therapy **(**NICE 2020**)**. For patients to be eligible for percutaneous coronary intervention **(**PCI**)** the American Heart Association recommends that the total ischemic time **(**time from symptom onset to PCI**)** does not exceed 120 minutes **(**Wah, Pek et al. 2017**)**. This reperfusion technique involves the insertion of a catheter equipped with a stent into the stenosed artery. Once in place, a balloon is inflated which expands the stent and allows reperfusion of the ischemic area to occur **(**Heart and Stroke Foundation 2021**)**. This technique is demonstrated in Figure 4. It is worthy to note that the shorter the symptom-to-door time **(**S2D**)**, the better the prognosis seen in AMI patients **(**Fardman, Zahger et al. 2021**)**.

Incidentally, the Covid-19 pandemic saw a substantial increase in S2D time compared to pre-pandemic numbers. Fardman, Zahger et al observed a 60% increase in time between symptom onset and reperfusion, from 180 minutes in 2018 to 290 minutes in 2020. These findings were in accordance with the markedly worse prognosis observed in the 2020 cohort, and further stresses the importance of rapid administration of reperfusion therapy **(**Fardman, Zahger et al. 2021**)**.



(Figure 4 was obtained from the Heart and Stroke Foundation of Canada. Source: Percutaneous Coronary Intervention 2021**)**

Figure 4: PCI Procedure

Pharmacologic Treatment

A variety of pharmacological therapies are available for the treatment of AMI. The specific indications and contraindications will need to be individually assessed for maximal safety and efficacy.

PCI is typically performed with adjuvant dual antiplatelet therapy **(**DAPT**)** and administration of unfractionated heparin. For patients at high risk of thromboembolism, triple antithrombotic therapy is implemented **(**Iwańczyk, Skorupski et al. 2020**)**, consisting of:

- Clopidogrel at a loading dose of 300-600mg with a maintenance dose of 75mg per day.
- 300mg of aspirin is taken orally followed by a maintenance dose of 100mg a day.
- An anticoagulant such as heparin or warfarin

IV morphine is a common choice for symptomatic relief of MI associated angina, dyspnea, and anxiety. For highly anxious patients, benzodiazepines may be administered. Beta-blockers are another treatment option, useful in decreasing heart rate and blood pressure, thereby reducing the heart's oxygen demand. However, beta-blockers are contraindicated in MI caused by coronary vasospasm **(**Ojha, Dhamoon 2021**)**.

Lifestyle Modifications

Managing the risk factors associated with MI are an effective way to improve its prognosis. For example, quitting smoking is an inexpensive way to decrease the likelihood of developing MI **(**Ojha, Dhamoon 2021**)**. Smoking has been consistently shown to increase serum cholesterol, form atherosclerotic plaques, and exacerbate CAD. In addition to this, the nicotine content of cigarettes has sympathetic stimulative properties and can increase oxygen demand on the heart **(**Rathore 2018**)**.

Diets play a major role in managing the risk of developing MI. Consuming ow amounts of saturated fats, such as in pescatarian and plant-based diets, have reportedly cardioprotective qualities **(**Ojha, Dhamoon 2021**)**. Sedentary lifestyles and obesity are causally related to MI, but low intensity exercises can help to reduce BMI as well as the risk of heart attack **(**Rathore 2018**)**. NICE guidelines currently recommend between 20-30 minutes of physical activity every day **(**NICE 2020**)**.

The presence of chronic stress in a patient's life increases their risk of having an MI. The physiological mechanism behind emotional distress is less understood but is thought to be able to cause MI by precipitating the rupture of atherosclerotic plaque. Therefore, stress-free environments can have a positive affect on patients at risk of MI **(**Rathore 2018**)**. It is plausible that people during the pandemic

were able to avoid stressors in their life while in quarantine, which would provide an explanation for the marked decline of heart attacks seen throughout the pandemic. However, this remains conjecture as the topic is largely unexplored and could benefit from additional research.

Prevention

Primary preventive measures refer to steps individuals can take to reduce their probability of developing CAD. These typically include health lifestyle choices like maintaining an ideal body weight, avoiding saturated fats as well as abstaining from smoking and excessive alcohol consumption. The goal of primary prevention is to minimize exposure to CAD risk factors, effectively halting disease progression before it begins **(**Tamam N Mohamad 2021**)**.

If primary prevention fails due to immutable risk factors the patient may have, the next step is to employ secondary prevention measures. Secondary prevention aims to recognize CAD in its early stages to limit its development. However, these measures are more costly compared to those in primary prevention. Beyond continuing to make positive health decisions, secondary prevention involves implementing drugs to aid in risk reduction. In this case, long term medication like ACE inhibitors, statins and beta-blockers are prescribed. There is substantial evidence supporting the ability of secondary prevention measure to reduce MI mortality **(**Kirsch, Becker et al. 2020**)**. Statins alone have reportedly yielded 15-20 percent drops in MI fatality in the context of secondary prevention **(**Karunathilake, Ganegoda 2018**)**.

Tertiary prevention often translates into invasive procedures designed to extend the life expectancy of the patient. These procedures include coronary bypass surgeries, stent insertions and pacemaker attachments. The extensive recovery period and financial burden associated with these procedures is what makes recognizing early risk factors of MI so critical. Ideally, primary and secondary measures will halt progression of MI before tertiary prevention measures become indicated **(**Karunathilake, Ganegoda 2018**)**.

Conclusion

Although MI is a serious adverse health event, the comprehensive data surrounding its pathogenesis makes the disease manageable. Primary prevention measures remain the most effective methods in decreasing MI prevalence and require increased awareness to combat the increasing incidence of MI. Treatments surrounding acute onset of MI is lacking and alternative measures of reperfusion need to

be developed for patients unable to access medical treatment within 120 minutes, who are therefore not eligible to receive PCI.

Furthermore, the MI mortality disparities between men and women have been attributed to the higher rates of misdiagnosis in women. With better education surrounding sex-specific signs and symptoms, we can attempt to reduce this.

However, there is a significant gap in the literature regarding the massive reduction in MI incidence seen during the Covid-19 pandemic. Theories explaining this significant reduction need to be further explored. Understanding the reasons behind such a decline may prove beneficial if we hope to produce a similarly small MI prevalence post-pandemic.

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