CARDIOMAG NEWSLETTER

Volume 3| Issue 31| 2022

IN THIS ISSUE

Effects of fat-free mass and body fat on prognosis in patients with Myocardial Infarction

Overweight and obese patients with coronary artery disease have a more favourable prognosis, which is referred as "obesity paradox". Body mass index may differ from true fatness in person to person. This differentiation may not be able to discriminate between an excess of body fat and an increase in fat-free mass leading to nutritional misclassification. However, some people with similar BMI may have different body compositions and different metabolic profiles. [1]

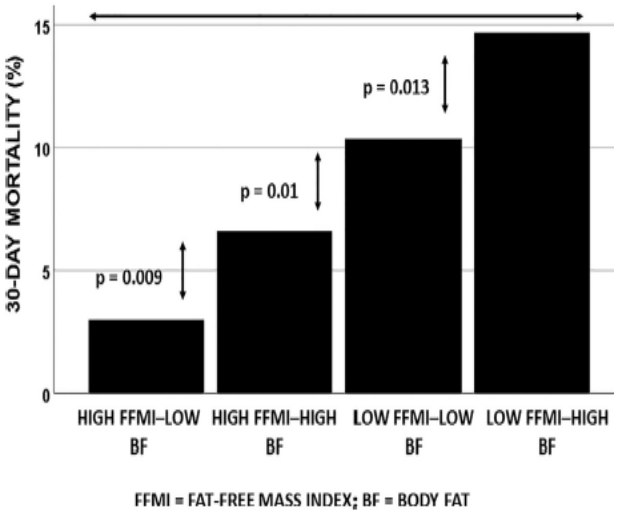
Researches have shown the protective effect of excess body fat that is counterintuitive and reversing causality.[2]Greater skeletal muscle strength provides better mobility, and cardiorespiratory fitness, which are known to be prognostic factors for a better outcome after coronary intervention.[2] In different people, the outcomes depend on the body composition. Studies have shown that both fat free mass and body fats have affected prognosis in patients with stable coronary artery disease, but no data is available in patients with myocardial infarction.[1]

Patients with low fat free mass index and low body fat are having high mortality risk, confirming the obesity paradox.

Kanic V.et al aimed to evaluate the effects of fat free mass and excess body fat on 30-day and long-term all-cause mortality in patients with MI undergoing percutaneous coronary intervention

This single-centre retrospective study enrolled 6,453 patients with MI and collected data. Enrolled patients were divided into 2 categories: Category high and Category low, according to the fat-free mass index and 2 categories: category low and category high, according to the body fat. Comparison between the 4 groups were done: High FFMI-Low BF, High-FFMI-High BF, Low FFMI-Low BF, and Low FFMI-High BF. In the HighFFMI-LowBF group, the lowest crude mortality after 30 days and in the long term was observed followed by the High FFMI-High BF group, the Low FFMI-Low BF group, and the Low FFMI-High BF group. (Fig:1) The differences between the group were significant.

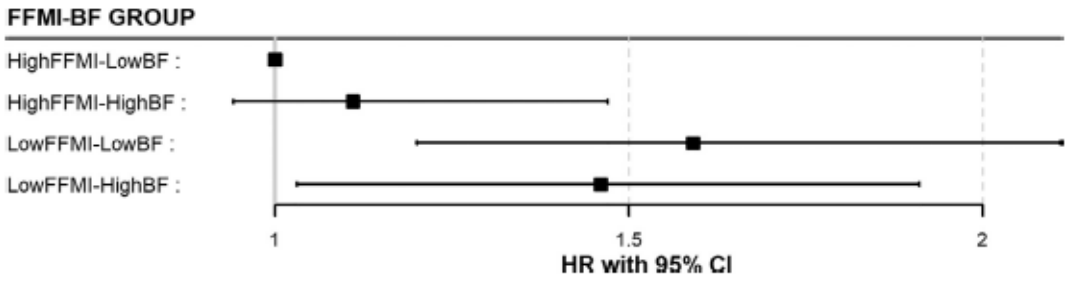
Fig:1 Observed 30-day mortality according to the fat-free mass index-body



FFMI = FAT-FREE MASS INDEX; BF = BODY FAT

Though the risk was similar in all groups, the FFMI-BF groups independently predicted 30-day mortality. The Low FFMI-Low BF and Low FFMI-High BF patients had a higher mortality risk but compared to the HighFFMI-LowBF group, the long-term mortality risk was similar in the High FFMI-High BF group.(Fig 2) In almost all the patients, body composition was able to predict mortality rate than body mass index. In patients with low FFM and low BF with high mortality risk, mortality appeared to be inversely related to FFM. Obesity paradox was observed in the body composition groups

Fig:2 Adjusted long-term mortality risk according to the fat-free mass index-body fat group



Conclusion: This study concluded that in patients with MI, long-term mortality is inversely related to fat-free mass, and body mass index does not accurately shows body fat and body composition. Though people have similar BMIs, their body compositions differs leading to alteration in the outcome. Patients with low FFMI and low BF are having high mortality risk during follow-up, confirming the obesity paradox.

References:

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Long-term Association between Estimated Pulse Wave Velocity and all-cause mortality in patients with stable angina undergoing elective coronary angiography

One of the strongest marker of cardiovascular disease is the arterial stiffness assessed by carotid-femoral pulse wave velocity. For the identification of organ damage related to hypertension, current hypertension guidelines have listed carotid-femoral pulse wave velocity as a major marker.[1] Carotid-femoral pulse wave velocity is an incremental predictive marker for future cardiovascular events and all-cause mortality beyond classical risk scores and BP.[2] However there are limited evidences suggesting that elevated carotid-femoral pulse wave velocity is associated with the poor response of BP to BP-lowering drugs and that carotid-femoral pulse wave velocity regression is related to improved survival.[2]Measurement of carotid-femoral pulse wave velocity not only requires specialized equipment but also trained staff. Hence, it has not been widely implemented in clinical practice.[1]However in patients with symptomatic stable angina, the prognostic potential of estimated pulse wave velocity during long-term follow-up is unclear.

Association between estimated pulse wave velocity with all-cause mortality and MI was observed in patients with stable angina pectoris beyond traditional risk factors.

Laugesen E et al. aimed to assess the association between estimated pulse wave velocity and all-cause mortality, myocardial infarction and stroke in patients with stable angina undergoing elective coronary angiography. Researchers enrolled 25,066 patients with a known case of myocardial infarction, stroke, heart failure, or valvular with stable angina pectoris undergoing elective coronary angiography. Patients having diabetes were excluded from the study. The association between incident all-cause mortality, MI, and stroke were calculated with the help of multivariable Cox models. A median follow-up period of 8.5 years reported 779 strokes, 1233 MIs, and 4112 deaths.

Result of this study highlighted the association between estimated pulse wave velocity with all-cause mortality and MI after adjusting for age, SBP, BMI, smoking, eGFR, Charlson Comorbidity Index score, antihypertensive therapy, statins, aspirin etc. The adjusted model with estimated pulse wave velocity was associated with a minor increase in discrimination for mortality

Fig3: Kaplan-Meier plots
A: Estimated pulse wave velocity and death

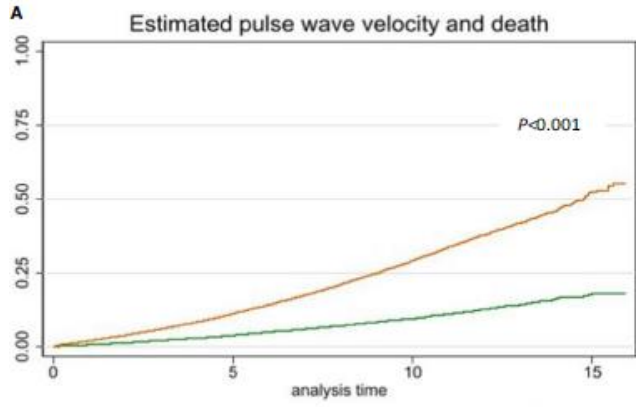


Fig4: Kaplan-Meier plots
B. Estimated pulse wave velocity and myocardial infarction.

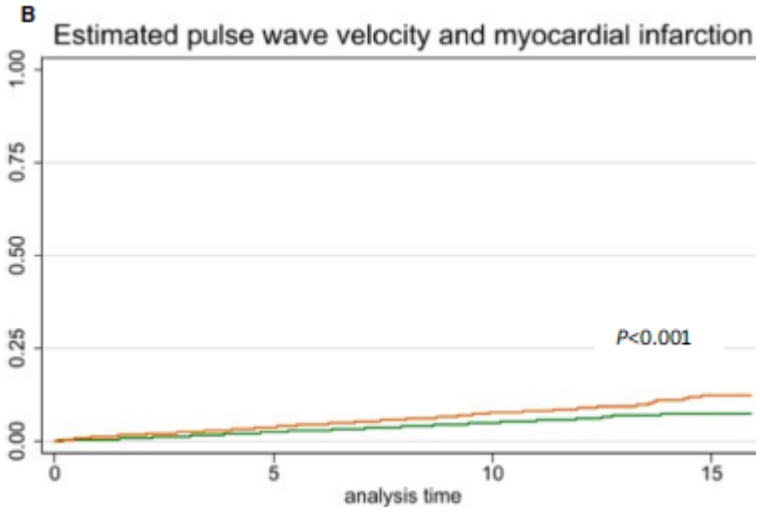
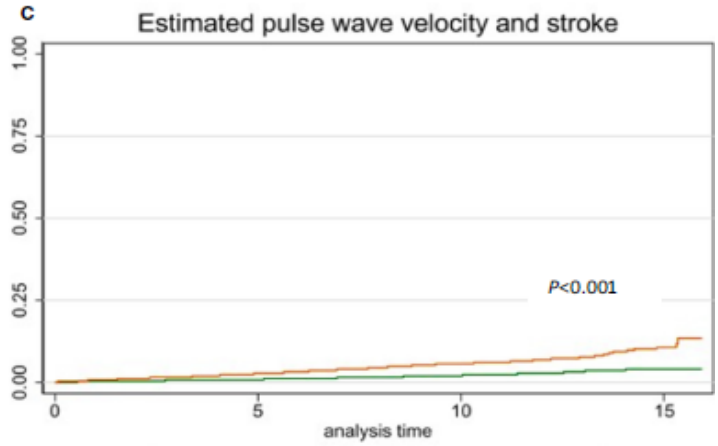


Fig5: Kaplan-Meier plots
C: Estimated pulse wave velocity and stroke



Conclusion: This study demonstrated an association between all-cause mortality and MI beyond traditional risk factors in patients with stable angina. However, there was no improvement observed in the added prediction of mortality to a clinically relevant extent

References

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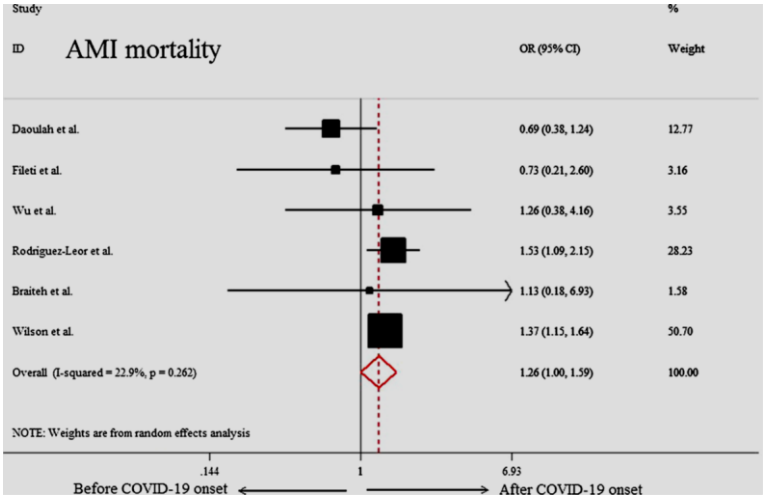
The rate of acute myocardial infarction admissions and major complication during the COVID 19 pandemic, in comparison with pre-COVID periods.

Globally, COVID-19 pandemic created an overwhelming pressure on the healthcare systems requiring reorganization and realignment to deal with the demanding workload associated with the rise of COVID-19 infection. During the pandemic, not only SARS-COV-2 related mortality has been documented, a substantial excess mortality rate related to non-COVID-19 diseases was observed. This raises concern about the public health consequences of the pandemic related to non COVID.[1]

During the COVID-19 pandemic, AMI related hospital admissions significantly decreased compared to pre-COVID period

During the COVID-19 pandemic, numerous data suggests decline in the acute myocardial infarction related hospital admissions. Hospitals were perceived as dangerous places, for which patients did not activate emergency medical systems regarding the infection risk. Moreover, patients with AMI had a significantly higher in-hospital mortality during the COVID-19 pandemic when compared to those admitted before COVID-19, which can be due to late arrival to the hospital.[2] COVID-19 led to cardiovascular complication included ST elevation myocardial infarction and non-STEMI. [1]Reduction in the AMI related hospital admissions could either because of a lower incidence of AMI at the time of the outbreak, or patients were seeking medical care when they become symptomatic. If the latter explanation is correct, this

Fig:6 In-hospital mortality Rate Forest Plot in AMI patients



can lead to catastrophic effects on the prognosis of the patient, as early management of AMI is crucial and life-saving

Pourasghari H et al. aimed to compare the rate of AMI admissions and major complication before and during the COVID pandemic. Patients data were collected through PubMed, Scopus, and Web of Science Core Collection and enrolled 41 studies into it. Original COVID-19 related studies with outcomes such as AMI admissions (STEMI or NSTEMI), AMI mortality rate (including in-hospital mortality), and major complications in patients with MI were included and compared with pre pandemic periods.

During COVID-19, AMI hospitalized patients were 35% less compared with pre-pandemic periods, which was statistically significantly. During COVID-19 periods, patients hospitalized for STEMI and NSTEMI were 29% and 34% respectively less compared with pre COVID periods, which was statistically significantly.

In-hospital mortality in AMI patients increased overall by 26% during COVID- 19 period, which was not statistically significant (Fig:6). At the same time, in-hospital mortality related to STEMI and NSTEMI patients increased by 15% and 26% respectively in the COVID-19 period, which was not statistically significant.

Conclusion: This study explains the challenges in the health-care systems with the impact of the COVID-19 pandemic. During the COVID-19 pandemic, AMI related hospital admissions significantly decreased. However, there were no significant differences in-hospital mortality rate between the pre and post-COVID- 19 periods. To avoid and prevent collateral damage in AMI patients during pandemic, or in future outbreaks, strategies should be improved.

References:

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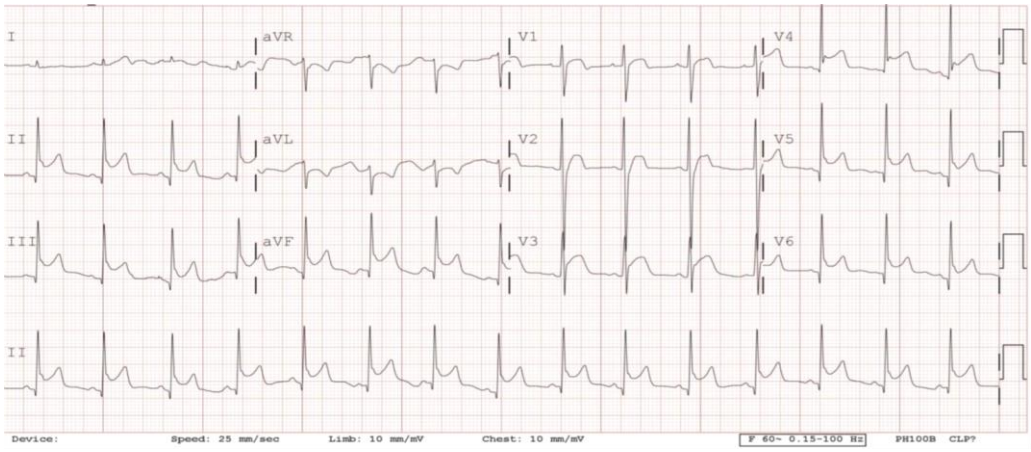
Myocarditis and Coronary Artery Spasm mimicking Acute Myocardial Infarction: A Case Report

Myocarditis is the inflammation of the myocardium, which can be an autoimmune disease or an infection that may or may not show symptoms. In angiography, Myocarditis and coronary artery vasospasm mimics STEMI with normal coronary arteries. However, acute myocarditis rarely causes coronary artery spasm.

Myocarditis and coronary artery spasm can mimick acute myocardial infarction leading to ST-segment elevation

Ko H et al report a case of coronary artery spasm with reversible electrocardiographic changes mimicking STEMI in 15-year-old male patient with acute myocarditis. A 15-year-old male patient was presented with sudden-onset chest pain at 02:30 AM characterized as squeezing on the left parasternal area for over 2 hours. Patient also complaint of having flu-like illness since 3 days. Patients did not have any comorbidities, such as hypertension, diabetes, or cardiovascular disease nor had the family history of coronary artery disease.

Fig7: 12-lead electrocardiogram showed sinus rhythm and ST-segment elevation in inferior lead (II, III, aVF), and precordial leads, accompanied by ST-segment depression in in aVL at the time of admission

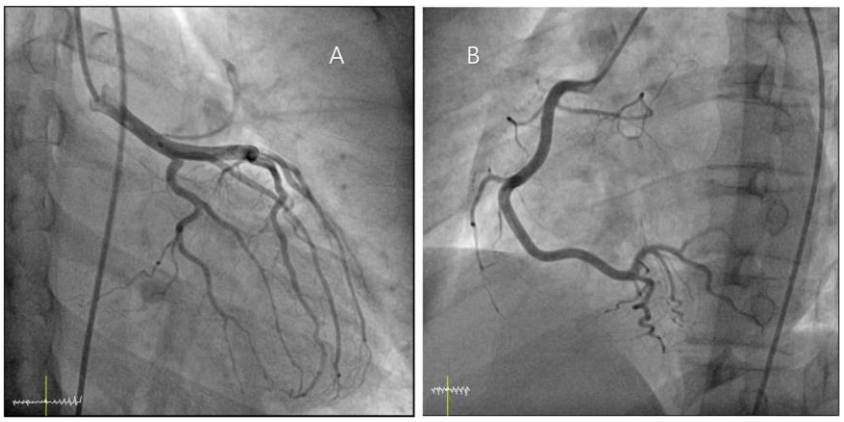


When asked, patient denied alcohol consumption and smoking. At the time of admission, Patient's creatine kinase MB fraction was increased to 49.2 ng/ml and Troponin-I was increased to 15.5 ng/ml. ST-segment elevation at I, aVL, and precordial leads were recorded. Blood pressure was 110/70 mmHg, heart rate was 86 bpm, respiratory rate was 15 b/min, body temperature was 38.5 °C and oxygen saturation at room air was 99%. Chest X-ray was done which showed normal heart size and clear lung fields. Sinus rhythm with ST-segment elevation in the inferior and

precordial leads were seen.(Fig 7) Normal left ventricular ejection fraction was recorded with very mild anteroseptal hypokinesis. After 23 hours, myoglobin was increased to 363.2 ng/mL, Troponin I increased to 21,538.68 pg/mL and creatine kinase MB fraction increased to 170.0 ng/mL. Coronary angiography revealed normal coronary arteries (fig 8). Treatment with immunoglobulin improved the cardiac markers and ECG. On the 3rd day of admission, patient experienced with another episode of chest pain during

sleeping that led to increased cardiac markers and ST-segment elevation. However, the chest pain got resolved without medication. These finding suggested the diagnosis of

Fig8:Coronary angiography demonstrated normal left (A) and right (B) coronary arteries



acute myocarditis and coronary artery spasm causing ST-segment elevation. cardiac magnetic resonance imaging showed myocardial oedema and subepicardial

enhancement in the anterolateral and infero-lateral wall

of mid base left ventricle. The patient got discharged on the 12th day with complete resolution of symptoms and normal ECG finding.

Conclusion: This case report demonstrated how myocarditis and coronary artery spasm can mimick acute myocardial infarction causing ST-segment elevation. As these diseases have different medical management strategies, importance should be given while evaluating cardiac MRI, past history, age, risk factors, subjective symptoms, cardiac markers, dynamic changes in ECG for differential diagnosis.

References

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