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Incidence of In-Hospital MACE in Patients Presented with Acute STEMI in the Presence of CTO in Non-IRA

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Abstract

Background: In STEMI patients, the presence of CTO in non-IRA has been associated with worse prognosis and adverse outcomes in many studies, whether in the short term or long term. This has been thought to be due to many factors including more major comorbidities in CTO patients, higher ischemic burden, and the "double Jeopardy theory".

Aim of Study: The aim of our study is to evaluate the incidence of in-hospital MACE in patients presenting with STEMI and CTO in non-IRA.

Patients and Methods: We analyzed 111 patients (52 patients with CTO in non-IRA and 59 without CTO in non-IRA) to compare their clinical outcomes in Ain Shams University Hospitals.

Although it was not found to be statistically significant, we did find that the incidence of In-hospital MACE is higher in cases with CTO in non-IRA in comparison to cases without CTO in non-IRA including single vessel disease patients and multi-vessel disease patients. Acute Pulmonary Edema and Cardiogenic Shock were found to be significantly higher in cases with CTO in non-IRA. 50% of cases with CTO developed MACE; and 100% of the CTO cases that developed MACE had reduced ejection fraction.

Results: This study showed that the majority of patients were males, smokers, hypertensive, and that all the involved patients in the study were dyslipidemic. In the majority of the cases the culprit was LAD as they mostly presented with Anterior STEMI.

Conclusion: In patients presenting with STEMI, acute pulmonary oedema and cardiogenic shock incidence was significantly higher in cases undergoing primary PCI with CTO in non-IRA compared to cases without CTO in non-IRA (either single vessel disease patients and multi-vessel disease). In-hospital MACE incidence was still higher yet not statistically significant in patients with CTO in non-IRA.

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Introduction

ST elevation myocardial infarction (STEMI) is caused by total thrombotic coronary artery occlusion. Patients presenting with STEMI require rapid diagnosis and emergent revascularization to the acutely occluded coronary artery (ie, culprit artery). This strategy is to reduce the risk of death and the extent of permanent myocardial injury associated with MI **[1].**

Primary percutaneous coronary intervention (PCI) refers to taking a patient presenting with STEMI directly to the cardiac catheterization laboratory to undergo mechanical revascularization using balloon angioplasty and coronary stents aiming at restoring epicardial infarct-related artery patency and achieving microvascular reperfusion as early as possible. PCI when performed within 120 minutes and ideally within 90 minutes, is the optimum therapy for patients presenting with Acute MI **[2].** However, subgroups of patients continue to have high morbidity and mortality rates due to major adverse Cardiovascular events (MACE).

Previous observational studies have shown that approximately 40%–60% of patients with acute STEMI have multivessel diseases (MVD). A concurrent chronic total occlusion (CTO) in a non-IRA is incidentally found in about 8%–15% of patients with STEMI. The presence of concurrent CTO in non-IRA can impose a higher short- and long-term risk of MACE in these patients following successful PCI due to the "double jeopardy" **[3,4].**

Chronic total occlusion (CTO) is a complete or nearly complete blockage of one or more coronary arteries. The blockage, typically present for at least three months, is caused by a buildup of plaque with-

in a coronary artery. In CTO the occluding thrombus becomes organized and fibrotic. CTO refers to coronary luminal diameter stenosis with resultant TIMI grade flow 0 or 1 as evaluated by coronary angiography. In such, there is no or faint antegrade or retrograde flow due to collaterals **[5].**

The presence of CTO in patients presenting with ACS especially STEMI is associated with increased morbidity and mortality as showed in the HORIZONS AMI trial. The mechanisms underlying the increased mortality (especially late) in patients with MVD and a CTO are mostly multifactorial. Patients with a CTO in a non-IRA had a higher prevalence of cardiovascular risk factors and co-morbidities compared with SVD patients and MVD patients without a CTO. To the best of our knowledge there is no national data evaluating the trend and outcomes of AMI patients who have a CTO of the non-IRA. In the present study we are going to address whether the presence of CTO in non-IRA augments the in-hospital risk of MACE in STEMI patients treated with successful PCI **[6].**

Patients and Methods

This prospective observational study was conducted in the Cardiology Department, Ain Shams University Hospitals during the period from November 2022 to August 2023 to study the incidence of In-hospital MACE in STEMI patients presented with CTO in non-IRA. Our study included 111 divided into 2 Groups: Group A: 52 consecutive Patients undergoing primary PCI for Acute MI with concurrent CTO of a non-IRA as the study group and Group B: 59 consecutive patients undergoing primary PCI for Acute MI without concurrent CTO of any vessel and well matching baseline clinical characteristics to the study group as the control group.

The inclusion criteria were: Patients who are above 18 years old and below 80 years old, who fulfilled STEMI diagnosis, and have CTO in non-IRA. While the Exclusion Criteria were: Patients with acute MI in extremes of ages (below 18 or above 80 years of age), patients with failed primary PCI for the IRA, orpatients undergoing PCI for CTO of a non-IRA during the same setting.

The following data was collected from files for all the enrolled patients: Patient demographics:Age, sex, cardiovascular risk factors (Diabetes mellitus, Hypertension, Dyslipidemia), family history, history of ischemic heart disease, ECG at presentation and as well as before discharge. Predischarge Echocardiography: To assess overall functions and RSWMA and Clinical in-hospital course: Patients were followed-up during their hospital stay for the development of MACE which we defined in our study as the development of heart failure, Acute pulmonary edema, arrhythmia, cardiogenic shock, and or death.

Results

Our study showed that the majority of patients were males, smokers, hypertensive, and that all the involved patients in the study were dyslipidemic. In the majority of the cases the culprit was LAD as they mostly presented with Anterior STEMI. It was also evident that in the CTO cases, more than 50% of them the CTO was in the RCA. In most of the cases there was ECG resolution post PCI and that most patients developed Heart Failure with reduced Ejection Fraction. Our study showed that only 40% of the cases developed In-hospital MACE, mostly in the form of acute pulmonary edema(APO) and Cardiogenic Shock. Baseline demographic data and in-hospital MACE are illustrated in Tables (1,2) respectively.

Our study revealed there was no statistically significant difference between cases with and without CTO regarding the demographic data except for DM. Diabetes was found higher in cases with CTO than cases without CTO with *p*-value=0.004. There was a statistically significant difference between both groups in pain to door duration, cardiac enzymes, heart rate, and number of affected vessels, all of whichwerefound higher in cases with CTO. Also, there was a statistically significant difference between both groups with respect to ECG resolution being more common in cases without CTO with a *p*-value=0.0. In addition, there was a statistically significant difference regarding the Ejection Fraction being significantly lower in cases with CTO with a *p*-value=0.001. Comparison between demographic criteria and post PCI ECG and echocardiographic data between the 2 study groups are illustrated in Tables (3,4) respectively.

There was no statistically significant difference between cases with and without CTO regarding occurrence of arrhythmias and mortality as most cases didn't develop either one. While there was a statistically significant difference between both groups regarding Acute pulmonary edema and Cardiogenic Shock which was found higher in cases with CTO than cases without CTO.

As a whole cases with in-hospital MACE were higher in the CTO group in comparison to the Non-CTO group including single vessel disease and multivessel disease patients with a percentage of 50% of the CTO group developed MACE while only around 30% of either the single vessel patients or the multivessel patients developed MACE; However, it was not found to be statistically significant. In-hospital MACE incidence in cases with CTO and without CTO (single vessel and multivessel) is illustrated in Table (5) and Figs. (1,2).

Table (1): Demographic data and characteristics of the studied patients.

	Total no. = 111
Sex:	
Female	22 (19.8%)
Male	89 (80.2%)
Age (years):	
Mean ± SD	54.83±12.03
Range	28 – 86
Smoking:	
No	34 (30.6%)
Yes	77 (69.4%)
Diabetes:	
No	65 (58.6%)
Yes	46 (41.4%)
Hypertension:	
No	54 (48.6%)
Yes	57 (51.4%)
Dyslipidemia:	
No	0 (0.0%)
Yes	111 (100.0%)
ISHD:	
No	83 (74.8%)
Yes	28 (25.2%)
Family history:	
No	67 (60.4%)
Yes	44 (39.6%)

Table (2): In-hospital MACE among the studied patients.

	Total no. = 111
Acute pulmonary edema:	
No	87 (78.4%)
Yes	24 (21.6%)
Cardiogenic shock:	
No	92 (82.9%)
Yes	19 (17.1%)
Arrhythmia:	
No	96 (86.5%)
AF/SVT	3 (2.7%)
VT/VF	11 (9.9%)
Heart block	1 (0.9%)
Mortality:	
No	108 (97.3%)
Yes	3 (2.7%)
In-hospital MACE:	
No	66 (59.5%)
Yes	45 (40.5%)

Table (3): Comparison between cases with and without CTO regarding demographic data and characteristics of the studied patients.

	Non CTO No. = 59	CTO No. = 52	Test value	*p*-value	Sig.
Sex:					
Female	14 (23.7%)	8 (15.4%)	1.211*	0.271	NS
Male	45 (76.3%)	44 (84.6%)			
Age (years):					
Mean ± SD	53.44±13.73	56.40±9.65	–1.299•	0.197	NS
Range	28 – 86	39 – 80			
Smoking:					
No	16 (27.1%)	18 (34.6%)	0.731*	0.393	NS
Yes	43 (72.9%)	34 (65.4%)			
Diabetes:					
No	42 (71.2%)	23 (44.2%)	8.276*	0.004	HS
Yes	17 (28.8%)	29 (55.8%)			
Hypertension:					
No	32 (54.2%)	22 (42.3%)	1.575*	0.210	NS
Yes	27 (45.8%)	30 (57.7%)			
Dyslipidemia:					
No	0 (0.0%)	0 (0.0%)	–	–	–
Yes	59 (100.0%)	52 (100.0%)			
ISHD:					
No	46 (78.0%)	37 (71.2%)	0.680*	0.410	NS
Yes	13 (22.0%)	15 (28.8%)			
Family history:					
No	38 (64.4%)	29 (55.8%)	0.862*	0.353	NS
Yes	21 (35.6%)	23 (44.2%)			

p-value >0.05: Non significant. *: Chi-square test.
p-value <0.05: Significant. •: Independent *t*-test.
p-value <0.01: Highly significant.

Table (4): Comparison between cases with and without CTO regarding ECG resolution after CA and ECHO.

	Non CTO No. = 59	CTO No. = 52	Test value	*p*-value	Sig.
ECG resolution after CA:					
No	7 (11.9%)	29 (55.8%)	24.313*	0.000	HS
Yes	52 (88.1%)	23 (44.2%)			
Echo: EF:					
Mean ± SD	42.37±7.29	36.04±10.15	3.809•	0.000	HS
Range	25 – 65	15 – 69			
Preserved	9 (15.3%)	7 (13.5%)	14.778*	0.001	HS
Moderate	22 (37.3%)	4 (7.7%)			
Reduced	28 (47.5%)	41 (78.8%)			
Echo RSWMA:					
Apex	29 (49.2%)	36 (69.2%)	4.592*	0.032	S
Inferior	31 (52.5%)	42 (80.8%)	9.781*	0.002	HS
Anterior	29 (49.2%)	26 (50.0%)	0.008*	0.929	NS
Lateral	31 (52.5%)	34 (65.4%)	1.878*	0.171	NS
Posterior	25 (42.4%)	34 (65.4%)	5.878*	0.015	S
Apical segments	29 (49.2%)	29 (55.8%)	0.485*	0.486	NS

p-value >0.05: Non significant. *: Chi-square test.
p-value <0.05: Significant. •: Independent *t*-test.
p-value <0.01: Highly significant.

Table (5): Comparison between cases with CTO and without CTO (single vessel and multivessel) regarding acute pulmonary edema, cardiogenic shock, arrhythmia, mortality and in-hospital MACE.

	Non CTO single vessel No. = 37	Non CTO multiple vessels No. = 22	CTO No. = 52	Test value	*p*-value	Sig.
Acute pulmonary edema:						
No	33 (89.2%)	20 (90.9%)	34 (65.4%)	9.771*	0.008	HS
Yes	4 (10.8%)	2 (9.1%)	18 (34.6%)			
Cardiogenic shock:						
No	33 (89.2%)	21 (95.5%)	38 (73.1%)	7.012*	0.030	S
Yes	4 (10.8%)	1 (4.5%)	14 (26.9%)			
Arrhythmia:						
No	32 (86.5%)	18 (81.8%)	46 (88.5%)	6.410*	0.379	NS
AF/SVT	0 (0.0%)	1 (4.5%)	2 (3.8%)			
VT/VF	5 (13.5%)	2 (9.1%)	4 (7.7%)			
Heart block	0 (0.0%)	1 (4.5%)	0 (0.0%)			
Mortality:						
No	37 (100.0%)	22 (100.0%)	49 (94.2%)	3.498*	0.174	NS
Yes	0 (0.0%)	0 (0.0%)	3 (5.8%)			
In-hospital MACE:						
No	25 (67.6%)	15 (68.2%)	26 (50.0%)	3.634*	0.163	NS
Yes	12 (32.4%)	7 (31.8%)	26 (50.0%)			

p-value >0.05: Non significant. *p*-value <0.01: Highly significant.
p-value <0.05: Significant. *: Chi-square test.

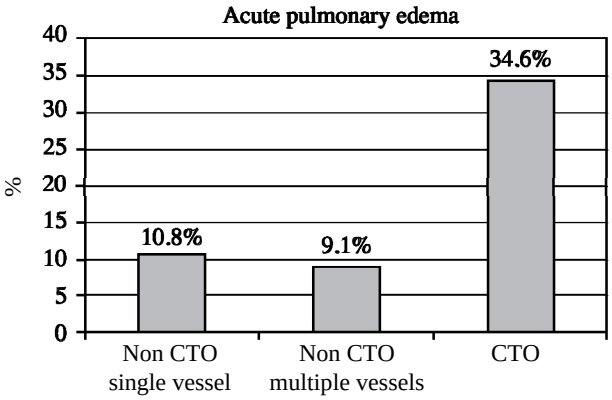

Fig. (1): Comparison between CTO cases and cases without CTO: Single vessel disease and multivessel disease regarding incidence of APO.

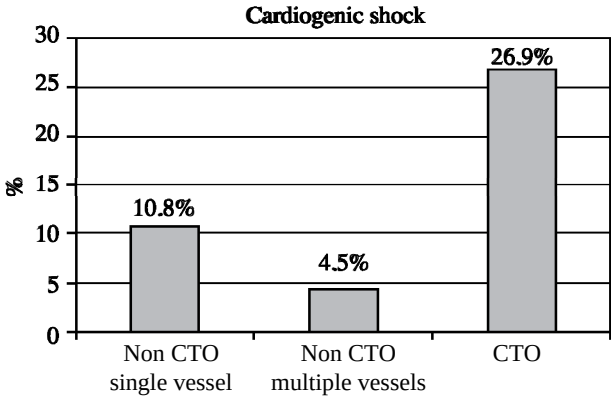

Fig. (2): Comparison between CTO cases and cases without CTO: Single vessel disease and multivessel disease regarding incidence of Cardiogenic Shock.

Discussion

Our study showed that patients presenting with STEMI, acute pulmonary oedema and cardiogenic shock incidence was significantly higher in cases undergoing primary PCI with CTO in non-IRA compared to cases without CTO in non-IRA (either single vessel disease patients and multi-vessel disease). In-hospital MACE incidence was still higher yet not statistically significant in patients with CTO in non-IRA.

Other studies have shown that the presence of CTO itself is a risk factor to develop in hospital MACE. Theories as to why the presence of CTO in non-IRA in STEMI patients is associated with adverse outcomes are multifactorial. One of the theories is that CTO patients tend to have more co-morbidities and more risk factors than patients without CTO leading to a higher ischemic burden **[6,7].** Another theory is the "double Jeopardy" theory which claims that if collaterals arise from the IRA supplying the myocardium of the CTO artery, abrupt occlusion of the IRA would lead to not only infarction of the distal myocardium but also the collaterally supplied myocardium of the CTO. Also, patients with CTO tend to present with more severe presentations causing them to be more likely to develop adverse outcomes. All these are possible explanations **[8].**

Our study has provided outcomes similar to those of other studies. It has shown that patients with Diabetes were more likely to have CTO. This may be due to the correlation between diabetes and the progression of atherosclerosis or the possibility of silent ischemia. Cases with CTO were found to have longer pain to door time, higher levels of enzymes, and number of vessels affected. They were less likely to experience ECG resolution suggesting suboptimal revascularization and were more likely to have impaired ejection fraction suggesting greater ischemic burden as seen in previous research done on the subject.

In cases without CTO in non-IRA, diabetics were three times more likely to develop in-Hospital MACE mostly due to having advanced coronary artery disease thus higher likelihood of MVD even in the absence of CTO. In-addition cases who didn't experience ECG resolution had a higher chance of developing in-hospital MACE.

In cases with CTO in non-IRA, cases with a blood pressure lower than 100/60 and heart rate higher than 80 were more likely to develop In-Hospital MACE and that 100% of the CTO cases who developed MACE had reduced Ejection Fraction suggesting larger areas of myocardial ischemia. Also, cases with segmental wall motion abnormalities mainly in the inferior wall were less likely to develop MACE suggesting that possibly cases with CTO RCA had less severe outcomes.

Finally, concerning the focal point of our study, a comparison between cases with CTO in non-IRA and cases without CTO in non-IRA regarding the development of in-hospital MACE. We found cases with CTO were significantly more likely to develop APO and Cardiogenic shock than cases without CTO. As a total,we noticed that the incidence of In-hospital MACE was higher in the CTO group in comparison to the Non-CTO group including single vessel disease and multivessel disease patients. Half of the CTO group developed MACE while only around 30% of either the single vessel disease patients or the multivessel disease patients developed MACE.

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معدل حدوث المضاعفات القلبية الخطيرة لمرضى الاحتشاء الحاد لعضلة القلب المصحوب بانسداد كلى مزمن فى شريان غير ذلك المرتبط بالاحتشاء خلال وجودهم بالمستشفى

يعانى حوالى ٤٠-٦٠٪ من المرضى المصابين باحتشاء عضلة القلب الحاد منضيق فى العديد من الشرايين التاجية، ومنهم حوالي ١٠٪ يعانون من انسداد كلى مزمن فى احد هذه الشرايين. كان الهدف من دراستنا هو تقييم حدوث المضاعفات القلبية الخطيرة اثناء العلاج فى المستشفى فى المرضى الذين يعانون من احتشاء عضلة القلب الحاد وانسداد كلى مزمن فى شريان غير ذلك المرتبط بالاحتشاء.

أظهرت دراسات عديدة ان وجود انسداد كلى مزمن فى شريان غير ذلك المرتبط بالاحتشاء في مرضى احتشاء عضلة القلب الحاد هو فى حد ذاته عامل خطورة منفرد يؤدى فى المستشفى لمضاعفات قلبية خطيرة أكثر من وجود ضيق فى العديد من الشرايين التاجية. كما ثبت أنه يرتبط بارتفاع معدل الوفيات على مدى فترة تصل إلى ١٠ سنوات.

يوجد الكثير من النظريات حول سبب ارتباط وجود انسداد كلى مزمن فى شريان غير ذلك المرتبط بالاحتشاء فى مرضى احتشاء عضلة القلب الحاد بنتائج سلبية. إحدى هذه النظريات هي أن مرضى الانسداد الكلى المزمن يميلون إلى الإصابة بعوامل خطورة أكثر من المرضى الذين لا يعانون من انسداد كلى مزمن مما يؤدى إلى عبء قصور في الشرايين أعلى. نظرية أخرى هى «الخطر المزدوج» التى تدعي أنه إذا نشأت الأوعية الدموية الجانبية الصغيرة التى تغذى عضلة القلب مكان الشريان الكلى المزمن من الشريان المرتبط بالاحتشاء، فإن الانسداد المفاجئ للشريان المرتبط بالاحتشاء لن يؤدى فقط إلى احتشاء عضلة القلب المغذاة منه ولكن أيضًا إلى عضلة القلب المغذاة بشكل ضمنى من الشريان المسدود انسداد كلي مزمن. أيضًا، يميل المرضى الذين يعانون من انسداد كلى مزمن إلى حدوث اعراض أكثر شدة مما يجعلهم أكثر عرضة لتطور نتائج سلبية.

قدمت دراستنا نتائج مماثلة لتلك التي توصلت إليها دراسات أخرى. وقد أظهرت أن مرضى السكرى، بشكل عام، كانوا أكثر عرضة للإصابة بالانسداد الكلي المزمن. قد يكون هذا بسبب الارتباط بين مرض السكرى وتطور تصلب الشرايين أو احتمال الإصابة بنوبات قلبية صامتة. كذلك وجدنا ان حالات الانسداد الكلى المزمن لديها ألام بالصدر لمدة أطول قبل الوصول إلى المستشفى، ومستويات أعلى من انزيمات القلب، وعدد أكبر من الأوعية المتضررة. كما كانوا أقل فى احتمالية رجوع رسم القلب كما كان قبل الجلطة مما يشير إلى أن إعادة فتح الشريان كانت دون المستوى الأمثل.

كان مرضى السكرى أكثر عرضة للإصابة بالمضاعفات القلبية الخطيرة فى المستشفى بثلاثة أضعاف فى الحالات التى لا يوجد فيها انسداد كلى مزمن فى شريان غير ذلك المرتبط بالاحتشاء، ويرجع ذلك في الغالب إلى الإصابة بتصلب الشريان التاجى المتقدم وبالتالى زيادة احتمالية الإصابة بأمراض قى العديد من الأوعية حتى فى غياب الانسداد الكلى المزمن. بالإضافة إلى ذلك، فإن الحالات التى لم يتم رجوع رسم القلب كما كان قبل الجلطة لديها فرصة أكبر للإصابة بالمضاعفات القلبية الخطيرة داخل المستشفى.

فى حالات الانسداد الكلى المزمن فى شريان غير ذلك المرتبط بالاحتشاء، كانت الحالات التي تم قياس ضغط الدم فيها أقل من ٦٠/١٠٠ ومعدل ضربات القلب أعلى من ٨٠ أكثر عرضة للإصابة بمضاعفات قلبية خطيرة داخل المستشفى وأن ١٠٠٪ من حالات الانسداد الكلى المزمن التى تم حدوث مضاعفات قلبية خطيرة لها كانت تعانى من ضعف فى عضلة القلب مما يشير إلى مناطق أكبر من نقص تروية عضلة القلب. أيضًا، كانت الحالات التى تعانى من تغيرات فى حركة الجدار القطاعى بشكل رئيسى فى الجدار السفلى أقل عرضة للإصابة بالمضاعفات القلبية الخطيرة التى تشير إلى احتمال أن وجود انسداد كلى مزمن للشريان التاجى الأيمن يؤدى الى نتائج أقل حدة.

أخيرًا، فيما يتعلق بالنقطة المحورية لدراستنا وهى المقارنة بين الحالات ذات الانسداد الكلى المزمن في شريان غير ذلك المرتبط بالاحتشاء والحالات بدون انسداد كلى مزمن فى شريان غير ذلك المرتبط بالاحتشاء فيما يتعلق بحدوث المضاعفات القلبية الخطيرة اثناء العلاج بالمستشفى. كانت الحالات المصابة بالانسداد الكلى المزمن أكثر عرضة للإصابة بهبوط حاد فى الدورة الدموية وارتشاح رئوى حاد أكثر من الحالات التى لا تحتوى على انسداد كلى مزمن. كإجمالى، فإن حدوث المضاعفات القلبية الخطيرة اثناء العلاج بالمستشفى أعلى فى مجموعة الانسداد الكلى المزمن مقارنة بالمجموعة التي ليس فيها انسداد كلى مزمن بما فى ذلك المرضى المصابين بقصور فى أحد الأوعية الدموية والمرضى المصابين بقصور فى العديد من الأوعية. نصف مجموعة الانسداد الكلى المزمن حدث لهم مضاعفات قلبية خطيرة بينما حوالى ٣٠٪ فقط من المجموعة الغير مصابة بانسداد كلى مزمن سواء من المرضى المصابين بقصور فى أحد الأوعية الدموية او المرضى المصابين بقصور فى العديد من الأوعية الدموية حدث لهم مضاعفات قلبية خطيرة.