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ORIGINAL RESEARCH

ISCHEMIC HEART DISEASE

30-Day and 1-Year Acute Myocardial Infarction Outcomes in Côte d'Ivoire

The REACTIV Study

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ABSTRACT

BACKGROUND Whereas the increasing burden of acute myocardial infarction (MI) has been reported in sub-Saharan Africa, little is known about short- and long-term prognosis following acute MI.

OBJECTIVES The purpose of this study was to assess in-hospital, 30-day, and 1-year all-cause mortality and adverse outcomes in patients with MI hospitalized at a cardiac center in Côte d'Ivoire.

METHODS This prospective cohort study used data from the REgistre des syndromes coronariens Aigus de CôTe d'IVoire (REACTIV). All consecutive patients admitted to the intensive care unit with acute MI were included. The primary endpoints included in-hospital, 30-day, and 1-year all-cause mortality and major adverse cardiovascular events. Multivariable Cox regression analyses were performed to identify factors associated with 30-day and 1-year all-cause mortality.

RESULTS A total of 272 participants were included (average age 56.93 ± 11.1 years, 76.8% men). The in-hospital mortality was 9.9%. Mortality rates and major adverse cardiovascular events were 11% and 2.8% at 30 days and 21.7% and 27.3% at 1 year, respectively. In the multivariable regression model, factors associated with 30-day all-cause mortality were Killip stage ≥2 at admission (relative risk [RR]: 3.65; 95% CI: 1.61-8.26) and impaired renal function (RR: 3.44; 95% CI: 1.63-7.26). One-year all-cause mortality was associated with Killip stage ≥2 at admission (RR: 2.74; 95% CI: 1.52-4.94), anterior MI (RR: 2.48; 95% CI: 1.37-4.48), impaired renal function (RR: 3.44; 95% CI: 1.63-7.26), and sustained ventricular tachycardia (RR: 5.24; 95% CI: 2.67-10.3). At both 30-day and 1-year follow-up, myocardial reperfusion therapies improved prognosis.

CONCLUSIONS Acute MI is associated with substantial mortality and morbidity in Côte d'Ivoire. These data can help local care providers identify those at highest risk and plan surveillance accordingly. (JACC Adv. 2024;3:101285)

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The authors attest they are in compliance with human studies committees and animal welfare regulations of the authors' institutions and Food and Drug Administration guidelines, including patient consent where appropriate. For more information, visit the Author Center.

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ABBREVIATIONS AND ACRONYMS

AIC = Akaike information criterion

BMI = body mass index

CAD = coronary artery disease

CV = cardiovascular

ECG = electrocardiogram

ICU = intensive care unit

eGFR = estimated glomerular filtration rate

LVEF = left ventricular ejection fraction

MACE = major adverse cardiovascular event

MI = myocardial infarction

NSTEMI = non-ST-segment elevation myocardial infarction

PCI = percutaneous coronary intervention

STEMI = ST-segment elevation myocardial infarction

VT = ventricular tachycardia

Myocardial infarction (MI) is the most critical manifestation of coronary artery disease (CAD), which is the leading cause of death worldwide.[1] In low- and middle-income countries, particularly in sub-Saharan Africa, the burden of MI has been increasingly recognized past years, resulting from changes in risk factor profiles and behaviors.[2,3] In Côte d'Ivoire, admissions for MI have dramatically increased over the past 2 decades, from 7.3% to 22.6%, with an estimated in-hospital mortality of 10.4%.[4] While Côte d'Ivoire is the largest economy in the West African Economic and Monetary Union according to the World Bank, few facilities are equipped to handle cardiovascular (CV) emergencies. The Abidjan Heart Institute, located in the capital of 5.6 million, is the only tertiary-level public hospital in Côte d'Ivoire dedicated to the management of CV disease and supports >7,000 emergency visits and nearly 250 cases of acute coronary syndromes annually.

Despite the increasing burden of CAD, there are limited data on outcomes and prognostic variables of patients with post-MI in sub-Saharan Africa, both over the short-term[5,6] and the long-term[7-12] Furthermore, a wide range of determinants of late mortality have been reported, including reduced left ventricular ejection fraction (LVEF) <40% at 30 days,[5] heart failure, older age, female sex, elevated serum creatinine, and diabetes[7-9] at 1 year. A study in Tanzania, among 152 consecutive patients with MI admitted to a tertiary care hospital,[11] found that only higher initial troponin I and older age were associated with mortality at 1 year.[11]

Among patients presenting with ST-segment elevation MI (STEMI) in Western countries, 1-year mortality has markedly declined in the recent decades, both related to the increased use of reperfusion therapies, especially percutaneous coronary intervention (PCI), and evidence-based therapies.[13,14] Nonetheless, identification of factors associated with mortality is a key issue in sub-Saharan Africa as there are still many unknowns given the scarcity of data. Thus, more comprehensive and reliable data are needed to target management strategies in vulnerable groups of patients in the acute phase and after discharge.

Therefore, the aim of this study is to assess in-hospital, 30-day, and 1-year all-cause mortality and major adverse cardiovascular events (MACE) in patients presenting with MI at a tertiary heart center in Abidjan, Ivory Coast. Proper assessment and optimal follow-up of these patients may help raise awareness of the national health systems in sub-Saharan Africa and to respond to the growing burden of MI, which has surpassed traditional tropical diseases in this part of the world.

METHODS

STUDY DESIGN AND SELECTION CRITERIA. This study was carried out at Abidjan Heart Institute, University Teaching Hospital and national referral center for the management of CV diseases in Côte d'Ivoire, capable of providing CV care 24/7. The center includes emergency department, intensive care unit (ICU), wards, department for noninvasive explorations, pediatric cardiology department, operating rooms for CV and thoracic surgery, interventional cardiology laboratory (with 2 cardiac catheterization rooms), and cardiac rehabilitation department.

For the present study, all patients aged 18 years old hospitalized in ICU from January 2019 to December 2020 for an acute MI were included. Patients were enrolled consecutively, provided they agreed to participate and signed a written consent form. MI was diagnosed according to the Fourth Universal Definition of Myocardial Infarction criteria.[15] STEMI was defined by the presence of ischemic symptoms with persistent ST-segment elevation in 2 contiguous leads on an electrocardiogram (ECG) or new bundle branch blocks with ischemic repolarization patterns suggestive for ST-segment elevation MI.[16,17] In contrast, patients with acute chest discomfort but no persistent ST-segment elevation were defined as having non-ST-segment elevation MI (NSTEMI). That include transient ST-segment elevation, persistent or transient ST-segment depression, T-wave inversion, flat T waves, or pseudo-normalization of T waves; or the ECG may be normal.[18,19]

These patients were consecutively enrolled in the REACTIV (REgistre des syndromes coronAriens aigus de CôTe d'Ivoire) registry and were followed up during in-hospital stay and after discharge (at 1 month, 3-6 months, and 12 months).

STUDY PROCEDURES AND DEFINITIONS. Follow-up was performed by trained cardiologists of emergency and interventional cardiology departments during their consultation and by analysis of the patients' digitized records (OLYMPE software). Data were collected consecutively over the study period, using a standardized survey form from the REACTIV

registry. For patients who did not attend appointments, data were also collected by telephone call to the patient or his family members. We considered as "lost to follow-up" patients who were enrolled in the REACTIV registry, but who did not attend appointments after discharge and who were unreachable (patient or relative) after 4 attempts within 1 month.

For each patient, the following variables were collected: sociodemographic and anthropometric data (age, gender, body mass index [BMI], CV risk factors) and history and clinical data (admission delay, hemodynamic parameters, Killip stage) and ECG data (infarct territory, supraventricular and ventricular arrhythmias, conduction disorders [left and right bundle branch block, high degree atrioventricular block]), echocardiographic data and information on reperfusion therapies.

Hypertension was defined as systolic blood pressure ≥140 mm Hg and/or a diastolic blood pressure ≥90 mm Hg twice during hospitalization, or treatment of previously diagnosed hypertension.[20] Diabetes mellitus was defined as one of the following: chronic fasting hyperglycemia ≥1.26 g/l (7 mmol/L), or blood glucose levels ≥2.0 g/l (11.1 mmol/L) at any time of day, or a glycated hemoglobin level ≥6.5% or treatment of previously diagnosed diabetes mellitus.[21] Dyslipidemia: defined as total cholesterol ≥2 g/L and/or high-density lipoprotein-cholesterol ≤0.4 g/L and/or low-density lipoprotein-cholesterol ≥1.6 g/L, or treatment of previously diagnosed hypercholesterolemia. Active smoking was current smoking at the time of the study or stopped within the last 3 years. Overweight was defined as BMI ≥25 kg/m^2. Family history of CAD was defined as the occurrence of MI, stroke, or sudden death in the patient's father or first-degree male relative before 55 years and in the patient's mother or first-degree female relative before 65 years. LVEF was determined within 48 hours after admission using transthoracic echocardiography and biplane Simpson's method. Blood tests were performed to measure: glycemia, glycated hemoglobin, complete blood count, renal function with estimated glomerular filtration rate (eGFR) calculated using Modification of Diet in Renal Disease formula, troponin, and lipid levels.

Coronary angiography was performed according to current international guidelines. Severity of CAD was established according to the number of epicardial coronary arteries with narrowing ≥50% (1-, 2-, or 3-vessel disease/left main). Revascularization procedures (PCI or fibrinolysis) and pharmacological treatment were also reported.

OUTCOMES. In-hospital outcomes (including complications and mortality) were recorded, as well as ICU stay duration. After hospital discharge, the following data were collected at 1 month, 3 to 6 months, and 12 months:

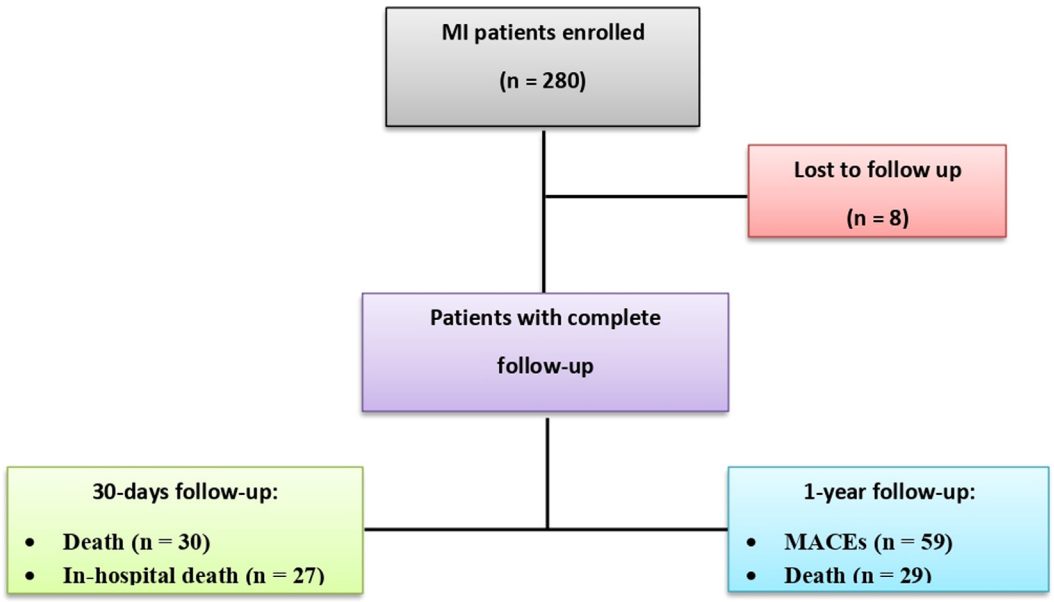
- Occurrence of death,
- Occurrence of MACE, defined as a composite parameter combining new nonfatal MI, nonfatal stroke, and CV death plus hospitalization for heart failure and unstable angina.

STATISTICAL ANALYSES. Categorical variables were presented as counts (proportions) and compared using chi-squared or Fisher's exact test. Continuous variables were summarized by median (IQR) and compared using Student's *t*-test or nonparametric Wilcoxon test. To identify factors associated with 30-day and 1-year all-cause mortality, univariable analyses were performed considering relevant variables from the literature: age, sex, BMI, risk factors, blood pressure, Killip stage ≥2, biological data (admission glycemia, eGFR, hemoglobin, hs troponin Ic peak, low-density lipoprotein-cholesterol level), electrocardiographic data (anterior wall MI, bundle branch blocks, sustained ventricular tachycardia [VT] or ventricular fibrillation), LVEF, severe CAD (3-vessel CAD or left main), and reperfusion therapy. Multivariable Cox regression models were created to assess factors associated with 30-day and 1-year mortality, respectively. Any variable demonstrating a significant association with the outcomes in the univariate analysis was included in the 30-day and 1-year mortality multivariable models (with a limit of 10 events per variable).[22] Age was also included in the model because of its clinical relevance in the literature. In addition, we carefully considered collinearity when selecting the best models. The final model retained was the one with the best Akaike information criterion (AIC) and lowest log likelihood. Survival curves were estimated by the Kaplan-Meier method and compared by the log-rank test. *P* value <0.05 was considered statistically significant. Due to the lack of multiplicity control, interval estimates should be interpreted with caution.

Data were analyzed using Epi info, version 7.2 2016 software (CDC). Medistica.*pvalue.io,* a Graphic User Interface to the R statistical analysis software for scientific medical publications, was also used for multivariable analysis as well as for obtaining survival curves.

ETHICAL APPROVAL. The present study complied with the Declaration of Helsinki and was approved by the Ethics Committee of Abidjan Heart Institute.

FIGURE 1 Study Flow Chart



MACE = major adverse cardiovascular event; MI = myocardial infarction.

RESULTS

Of the 1,718 patients admitted to the ICU, 280 MI cases were recorded. One-year follow-up data were available for only 272 patients, including 183 with STEMI and 89 with NSTEMI. The flow chart is described in Figure 1.

The baseline characteristics of the patients are presented in Table 1. The median age was 57.5 years (IQR: 49.0-64.0 years) and patients with NSTEMI were older. Most participants were men (sex ratio = 3.37), main risk factors were hypertension and overweight (63.24%). The median delay onset of symptoms–admission to ICU was 24 h (IQR: 9-72 h). At initial presentation, 72 participants (26.5%) reported a known history of diabetes mellitus and 17 (6.25%) reported previous MI. Twenty-five percent of patients presented with Killip class 2. Coronary angiography was performed in 224 patients (82%), and more than half received reperfusion therapy (PCI or fibrinolysis).

IN-HOSPITAL OUTCOMES. The in-hospital mortality rate during the initial hospitalization was 9.9% (27 of 272 participants; Table 2).

30-DAY AND 1-YEAR OUTCOMES. For participants who completed follow-up, the all-cause mortality rate was 11% (30 of 272 participants) at 1 month and 21.7% (59 of 272 participants) at 12 months (Figure 2). At 30-day follow-up, the majority of deaths occurred during in-hospital stay. MACE occurred in 2.8% at 30 days and 27.3% at 1 year. Among the MACE, CV death was observed in notable proportions, especially at 1 year (13.1%). Tables 3 and 4 present univariate analyses of 30-day and 1-year factors associated with all-cause mortality, respectively. In multivariable backward stepwise Cox analysis, existence of heart failure (relative risk [RR]: 3.65; 95% CI: 1.61-8.26; $P < 0.001$) and impaired renal function (RR: 3.44; 95% CI: 1.63-7.26; $P < 0.001$) were positively associated with all-cause mortality after 30-day follow-up (Figure 3) (AIC: 129.8; $P < 0.001$). Killip stage ≥2 (RR: 2.74; 95% CI: 1.52-4.94; $P < 0.001$), impaired renal function (RR: 3.17; 95% CI: 1.82-5.52, $P < 0.001$), sustained VT (RR: 5.24; 95% CI: 2.67-10.3; $P < 0.001$), and anterior wall MI (RR: 2.48; 95% CI: 1.37-4.48; $P < 0.001$) were associated with one-year mortality (Figure 4) (AIC: 105.5; $P < 0.001$). At 30-day and 1-year follow-up, acute reperfusion therapy was associated with a

TABLE 1 Baseline Characteristics

	All (N = 272)	STEMI (n = 183)	NSTEMI (n = 89)	*P* Value
Risk factors and comorbidities				
Age, y	57.5 (49.0-64.0)	57.0 (48.0-63.0)	60.0 (54.0-66.0)	**0.013**
Male	209 (76.84)	145 (79.23)	64 (71.91)	0.179
Hypertension	172 (63.24)	105 (57.38)	67 (75.28)	**0.002**
Diabetes	72 (26.47)	46 (25.14)	26 (29.21)	0.102
Dyslipidemia	122 (44.85)	86 (46.99)	36 (40.45)	0.156
Smoking	73 (26.84)	47 (25.68)	26 (29.21)	0.269
Overweight	172 (63.24)	119 (67.61)	53 (63.86)	0.192
BMI, kg/m^2	26.9 (24.2-30.4)	26.8 (24.4-29.7)	27.2 (23.8-30.9)	0.886
Cardiovascular history				
MI	17 (6.25)	9 (4.92)	8 (8.99)	0.193
PCI	6 (2.21)	2 (1.09)	4 (4.49)	0.091
Heart failure/LV dysfunction	13 (4.78)	7 (3.83)	6 (6.74)	0.290
Chronic renal failure	2 (0.74)	1 (0.55)	1 (1.12)	0.548
AF/AFL	1 (0,37)	1 (0.55)	0 (0.00)	1.000
Clinical data				
Onset of symptoms-admission, h	24 (9-72)	24 (8-72)	24 (12-72)	0.775
Killip admission ≥2	58 (25.00)	46 (25.14)	22 (24.72)	0.940
SBP, mm Hg	137 (122-160)	134 (122-157)	141 (124-168.5)	0.116
DBP, mm Hg	87 (77-100)	88 (78-101)	86.5 (76-100)	0.714
HR, beats/min	85 (72-101)	86 (72-100)	84.5 (71-105)	**0.004**
ECG at admission				
Anterior wall MI	138 (50.74)	108 (59.0)	30 (33.7)	**<0.001**
AF/AFL	2 (0.74)	2 (1.09)	0 (0.00)	1.000
RBBB	21 (7.75)	14 (7.69)	7 (7.87)	0.950
LBBB	7 (2.57)	3 (1.64)	4 (4.49)	0.161
Echocardiography				
LVEF, %	52.0 (42.0-60.0)	52.0 (42.0-58.0)	53.0 (42.0-62.0)	0.469
LVEF ≤40%	36 (14.34)	23 (13.61)	13 (15.85)	0.634
Coronary angiogram				
1-vessel disease	111 (49.6)	79 (51.6)	32 (45.1)	
2-vessel disease	55 (24.6)	37 (24.2)	18 (25.4)	0.473
3-vessel disease/Left main	42 (18.8)	28 (18.3)	14 (19.7)	
Laboratory values				
Hemoglobin, g/100 mL	13.5 (12.1-14.7)	13.7 (12.4-15.0)	13.05 (11.6-14.2)	**0.017**
Creatinine, mg/L	11.5 (9.4-15.0)	11.6 (9.3-15.0)	11.2 (9.55-15.1)	0.702
eGFR, mL/min/1,73 m^2	79.9 (59.7-102.4)	79.9 (59.7-102.7)	80.6 (59.4-99.8)	0.850
Glucose, g/L	1.37 (1.12-1.90)	1.42 (1.13-1.91)	1.27 (1.06-1.85)	0.838
LDL cholesterol, g/L	1.29 (0.91-1.61)	1.25 (0.92-1.58)	1.36 (0.84-1.63)	0.354
hs troponin Ic peak, ng/L	2,719 (891.3-8,926)	4,404 (1,023-10000)	2087 (658-4,059)	0.059
Therapeutics				
Aspirin	258 (94.8)	173 (94.5)	85 (95.1)	0.494
Clopidogrel	243 (89.3)	163 (89.7)	80 (89.9)	**0.041**
Prasugrel	10 (3.70)	7 (3.8)	3 (3.4)	0.576
LMWH	205 (75.4)	142 (77.6)	63 (70.8)	0.221
Beta-blockers	188 (69.1)	132 (72)	56 (62.9)	0.123
Statin	233 (85.7)	159 (86.9)	74 (83.1)	0.682
ACE inhibitors	216 (79.4)	145 (79.3)	71 (79.8)	**0.011**
IV inotropes	13 (4.8)	7 (3.8)	6 (6.8)	0.291
Antialdosterone	19 (6.9)	13 (7.1)	6 (6.7)	0.567
Reperfusion therapy				
All	148 (54.1)	110 (60.1)	38 (42.7)	**0.007**
Thrombolysis	30 (11.0)	30 (16.4)	-	-
PCI	140 (51.5)	103 (56.3)	37 (41.6)	**0.023**
CABG	2 (0.74)	-	2 (2.2)	-

Values are median (IQR) or n (%). **Bold** values indicate STEMI, NSTEMI, and RR.

ACE = angiotensin-converting enzyme; AF = atrial fibrillation; AFL = atrial flutter; BMI = body mass index; CABG = coronary artery bypass graft surgery; DBP = diastolic blood pressure; ECG = electrocardiogram; eGFR = estimated glomerular filtration rate; HR = heart rate; hs = high sensitivity; IV inotropes = intravenous inotropes; LBBB = left bundle branch block; LDL = low-density lipoprotein; LMWH = low-molecular-weight heparin; LVEF = left ventricular ejection fraction; MI = myocardial infarction; NSTEMI = non-ST-segment elevation myocardial infarction; PCI = percutaneous coronary intervention; RBBB = right bundle branch block; SBP = systolic blood pressure; STEMI = ST-segment elevation myocardial infarction.

TABLE 2 Adverse Outcomes In-Hospital and During Follow-Up

	All (N = 272)	STEMI (n = 183)	NSTEMI (n = 89)	*P* Value
In-hospital outcomes				
Killip stage ≥2	48 (17.65)	30 (16.39)	18 (20.22)	0.436
Sustained VT	18 (6.64)	9 (4.95)	9 (10.11)	0.108
AF/AFL	3 (1.11)	2 (1.10)	1 (1.12)	0.985
Transfusion or major bleeding	7 (2.57)	5 (2.73)	2 (2.24)	0.812
Stroke	4 (1.48)	4 (2.20)	0 (0.00)	-
Cardiovascular death	27 (9.93)	18 (9.84)	9 (10.11)	0.942
Follow-up outcomes (30 days)				
All MACEs	7 (2.85)	6 (3.03)	1 (1.25)	0;432
Nonfatal MI	4 (1.63)	3 (1.81)	1 (1.25)	0.604
Nonfatal stroke	0 (0.0)	0 (0.0)	0 (0.0)	-
Hospitalization for heart failure	0 (0.0)	0 (0.0)	0 (0.0)	-
Cardiovascular death	3 (1.22)	3 (3.93)	0 (0.0)	-
Follow-up outcomes (1 year)				
All MACEs	67 (27.34)	45 (27.27)	22 (27.50)	0.971
Nonfatal MI	23 (9.38)	17 (10.30)	6 (7.50)	0.481
Nonfatal stroke	1 (0.41)	0 (0.0)	1 (1.25)	-
Hospitalization for heart failure	28 (11.42)	17 (10.30)	11 (13.75)	0.426
Cardiovascular death	32 (13.06)	21 (12.72)	11 (13.75)	0.823

Values are n (%).
MACE = major adverse cardiovascular events; VT = ventricular tachycardia; other abbreviations as in Table 1.

better prognosis. All Kaplan-Meier survival curves showed a significant trend between the 2 groups at both 30-day and 1-year follow-up (**Central Illustration**, Supplemental Figures 1 and 2).

DISCUSSION

In a cohort of consecutive patients with MI hospitalized at Abidjan Heart Institute, we investigated 30-day and 1-year factors associated with all-cause mortality. In sub-Saharan Africa, to date, only few studies have identified the main determinants of prognosis in the follow-up after an acute coronary event.[5-11] **Table 5** lists the different studies.[5-12,25,26] Our study shows that in the first month following MI, majority of deaths occur in hospital phase. Mortality significantly increased by 2-fold from 30 days (11%) to 1 year (21%). During follow-up, MACE, such as nonfatal MI or CV death, is observed both early and over the long term.

Mortality after MI has been reported in other African countries. ACCESS (Acute Coronary Events—a Multinational Survey of Current Management Strategies) registry data in South Africa indicated a 30-day mortality of 2.4% and this one reaches 6.7% at 1 year.[8] Recent data from RACE (Registry for Acute Coronary Events in Nigeria) found a mortality rate of 8.1% at 30 days and 13.3% at 1 year,[12] similar to results found in a previous study in Kenya.[10] Differences could be explained by delays in management (24 hours-median in our series), late consultation, and suboptimal referral system. In contrast, highest mortality rates were found in a Tanzanian study, reaching 43% at 30 days and 59% at 1 year.[6,11] Thus,

FIGURE 2 All-Cause Mortality Following Acute Myocardial Infarction, Abidjan, 2019 to 2020 (N = 272)

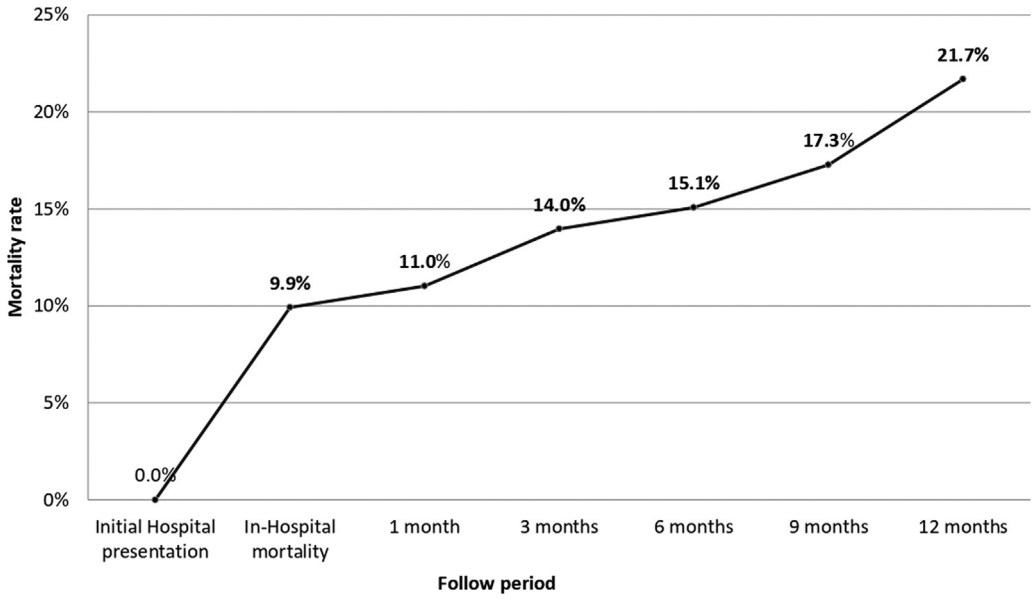


TABLE 3 Factors Associated With 30-Day All-Cause Mortality After Myocardial Infarction: Univariable Analysis

	Alive (n = 242)	Deceased (n = 10)	RR (95% CI)	*P* Value
Male	185 (76.47)	24 (80.00)	1.23 (0.48-3.16)	0.663
Age, y	57 (48-64)	60 (53-64)	-	0.162
Risk factors				
Hypertension	149 (66.81)	23 (76.67)	2.05 (0.85-4.97)	0.106
Diabetes mellitus	57 (23.55)	15 (50.00)	3.24 (1.49-7.04)	**0.002**
Dyslipidemia	67 (27.69)	6 (20.00)	0.65 (0.26-1.67)	0.370
Active smoking	115 (47.52)	7 (23.33)	0.34 (0.14-0.81)	**0.012**
Clinical data				
SBP, mm Hg	140 (124-161)	125 (107-143)	-	**<0.001**
HR, beats/min	84 (71-98)	89 (78-101)	-	**<0.001**
Killip stage ≥2	50 (20.66)	18 (60.00)	5.76 (2.60-12.74)	**<0.001**
In-hospital complications				
Sustained VT/VF	6 (2.49)	12 (40.00)	26.11 (8.77-77.74)	**<0.001**
ECG and echocardiography				
LBBB	6 (2.48)	1 (3.33)	1.35 (0.16-11.66)	0.563
RBBD	12 (4.98)	9 (30)	8.18 (3.09-21.64)	**<0.001**
Anterior wall MI	116 (47.93)	22 (73.33)	2.99 (1.28-6.97)	**0.009**
LVEF <40%	27 (12.22)	9 (30.00)	3.41 (1.41-8.20)	**0.009**
Laboratory values				
Glucose, g/L	1.33 (1.09-1.8)	1.83 (1.36-2.42)	-	**<0.001**
Creatinine, mg/L	11.1 (9.2-1.8)	15.8 (11.8-22.7)	-	**<0.001**
eGFR <60 ml/min/1,73 m^2	49 (21.21)	18 (60.00)	5.57 (2.51-12.35)	**<0.001**
Hemoglobin, g/100 mL	13.5 (12.2-14.8)	13.6 (9.4-14.3)	-	0.281
LDL-cholesterol, g/L	1.31 (0.93-1.63)	1.07 (0.71-1.39)	-	0.982
hs troponin Ic peak, ng/L	3,159 (899.6-9,417)	2,151 (560.7-5,231)	-	0.367
Coronary angiogram				
3-vessel disease/left main	37 (17.37)	5 (45.45)	3.96 (1.15-13.68)	**0.019**
Reperfusion therapy	142 (58.68)	6 (20.00)	0.17 (0.069-0.446)	**<0.001**

Values are n (%) or median (IQR) unless otherwise indicated. **Bold** values indicate STEMI, NSTEMI, and RR.
VF = ventricular fibrillation; other abbreviations as in Tables 1 and 2.

ongoing efforts to reduce this ever-increasing mortality must be made at all levels throughout the first year. It should be noted that 30-day mortality (11%) is almost similar to in-hospital mortality rate (9.9%). Several factors may explain poor in-hospital outcomes among patients with MI: late presentation to hospital after symptoms onset, resulting in a low percentage of patients undergoing revascularization procedures (PCI or fibrinolysis), unsafe transportation, unavailability of interhospital networks and protocols for the management of coronary emergencies. These shortcomings have been already identified in previous studies held in our center.[4,27]

Identifying predictors of long-term mortality is of particular interest and should be the cornerstone of post-MI phase management. In our study, occurrence of heart failure during hospitalization (Killip stage ≥2) and impaired renal function (GFR <60 ml/min/1.73 m^2) were associated with death at 30 days. After 1 year, in addition to the abovementioned predictive factors, anterior wall MI and occurrence of sustained VT were associated with death. Despite some variations between series, geographical regions, number of patients included, and methodology applied, some mortality factors are common in studies held in sub-Saharan Africa.[5,7,12] Most frequent are impaired renal function, heart failure (Killip stage ≥2 and/or ventricular dysfunction), early complications, and unrealized reperfusion procedure. These strong predictive factors have already been identified in the main western registries of patients with MI.[13,28,29] Consistently, reperfusion therapy appears to be a protective factor at all stages of the follow-up after discharge from hospital, although it is still rarely performed.

The identification of at-risk groups of patients at discharge after MI may provide several pathways to reduce the risk of cardiac events and mortality. First, these observations emphasize the urgent need for the widespread use of revascularization procedures in the acute phase (PCI and thrombolysis). In our study, as in Western registries including patients with MI, revascularization procedures were associated with a lower mortality rate, with maintained benefits at both

TABLE 4 Factors Associated With 1-Year All-Cause Mortality After Myocardial Infarction: Univariable Analysis

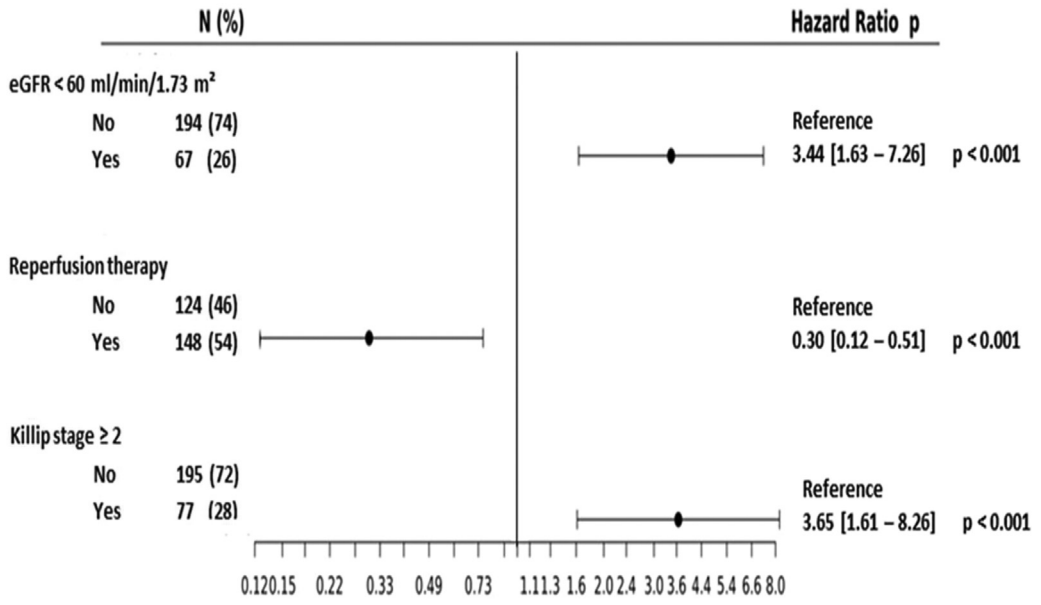
	1 Year Alive (n = 213)	1 Year Deceased (n = 59)	RR (95% CI)	*P* Value
Male	166 (77.93)	43 (72.88)	0.67 (0.34-1.33)	0.416
Age, y	57 (48-63)	60 (53-67)	-	**0.001**
Risk factors				
Hypertension	132 (61.97)	40 (67.79)	1.29 (0.70-2.38)	0.203
Diabetes mellitus	50 (23.47)	22 (37.30)	1.93 (1.04-3.58)	**0.033**
Dyslipidemia	61 (28.64)	12 (20.34)	0.63 (0.32-1.28)	0.204
Smoking	99 (46.48)	23 (38.89)	0.74 (0.41-1.36)	0.306
Clinical data				
SBP, mm Hg	140 (124-160)	128 (112-160)	-	**0.042**
HR, beats/min	82.5 (71-96.5)	103 (82-120)	-	**<0.001**
Killip stage ≥2	42 (19.71)	35 (59.32)	5.94 (3.20-11.03)	**<0.001**
In-hospital outcomes				
Sustained VT/VF	5 (2.36)	13 (22.03)	11.75 (3.99-39.6)	**<0.001**
ECG and echocardiography				
LBBB	3 (1.41)	4 (6.78)	5.09 (1.10-23.41)	**0.042**
RBBB	9 (4.25)	12 (20.34)	5.78 (2.30-14.53)	**<0.001**
Anterior wall MI	100 (46.95)	38 (64.41)	2.05 (1.10-3.71)	**0.017**
LVEF <40%	18 (9.33)	18 (31.03)	4.75 (2.28-9.91)	**<0.001**
Laboratory values				
Glucose, g/L	1.31 (1.09-1.73)	1.68 (1.31-4.09)	-	**<0.001**
Creatinine, mg/L	11.05 (9.2-13.5)	13.5 (10.2-20.6)	-	**<0.001**
eGFR <60 ml/min/1.73 m^2	37 (18.32)	30 (50.85)	4.61 (2.47-8.59)	**<0.001**
Hemoglobin, g/Dl	13.7 (12.4-15.0)	13.2 (11.1-14.0)	-	**0.007**
LDL-cholesterol, g/L	1.34 (0.97-1.70)	1.69 (1.31-2.23)	-	0.071
hs troponin Ic peak, ng/L	3,303 (916-9,606)	2024 (839-6,378)	-	0.559
Coronary angiogram				
3-vessel disease/left main	29 (15.18)	13 (39.39)	3.63 (1.62-8.10)	**0.001**
Reperfusion therapy	130 (61.03)	18 (30.51)	0.28 (0.15-0.52)	**<0.001**

Values are n (%) or median (IQR) unless otherwise indicated. **Bold** values indicate STEMI, NSTEMI, and RR.
Abbreviations as in Tables 1 to 3.

short (70% reduction) and long term (54% reduction) after MI. In the sub-Saharan African setting, PCI remains challenging, despite the recent implementation of cath labs in some countries, but major issues remain, particularly delays from symptoms to admission in heart centers and lack of interventional cardiologists.[2] Thus, pharmaco-invasive strategy (ie, early fibrinolysis combined with routine PCI within 24 hours or with rescue PCI in case of failed fibrinolysis) may be a good therapeutic alternative. There is long-term evidence of comparable benefits to primary PCI provided it is performed expeditiously.[30,31] Yet, no specifically randomized studies in sub-Saharan Africa has addressed this topic, given the low rate of PCI and easier access to fibrinolysis.[2] PCI or fibrinolysis both requires short admission delays that reflect the organization and performance of health care systems.[16] Second, the implementation of evidence-based therapies has led to significant benefits among patients with MI. In the majority of countries in sub-Saharan Africa, the most widely used antiplatelet agent remains clopidogrel. Potent anti-P2Y12 drugs such as prasugrel and ticagrelor[32,33] are still rarely used. Beyond P2Y12 inhibitors, full cardioprotective guideline-directed therapy with aspirin, β-blockers, angiotensin-2-converting enzyme inhibitors, and statins should also be assessed. In our study, we were not able to determine the rate of adherence after 1 year of follow-up. It seems clear that nonadherence to cardioprotective medications after MI increases mortality. Finally, CV rehabilitation should help reduce mortality and improve quality of life of patients after MI in the coming years, although is still in its early stages in sub-Saharan Africa. Implementation of cardiac rehabilitation results in 24% reduction of death at 5 years after adjustment for several confounders.[34] We did not evaluate whether it improved survival in our series.

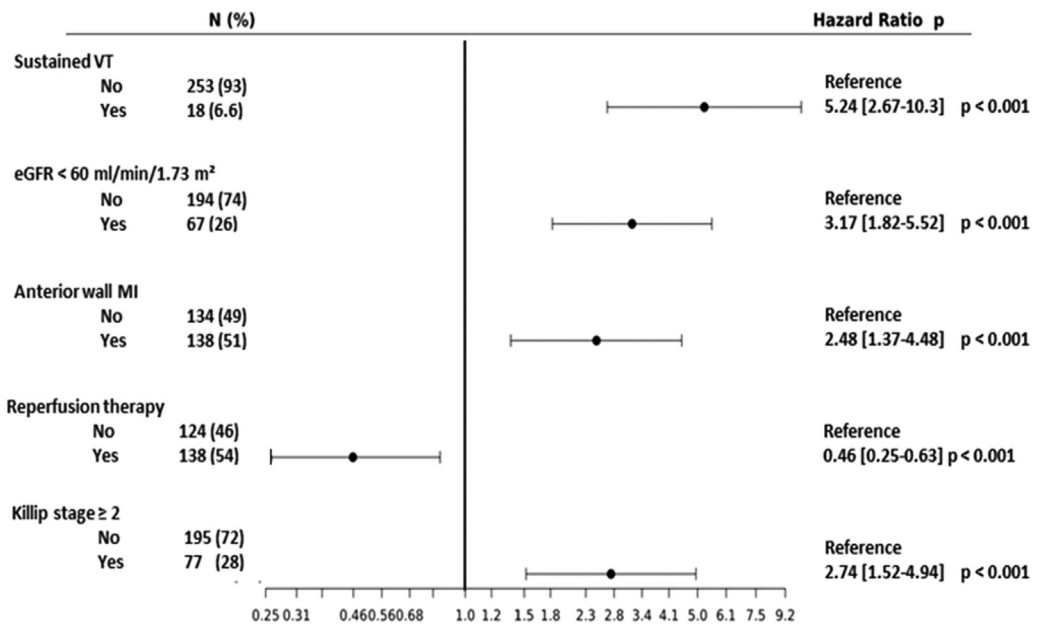
Reducing mortality after MI remains a difficult task in daily practice. Accurate identification of the determinants of mortality should help target management strategies in vulnerable subgroups to reduce the

FIGURE 3 Factors Associated With 30-Day Mortality After MI



Multivariable Cox analysis. eGFR = estimated glomerular filtration rate.

FIGURE 4 Factors Associated With 1-Year Mortality After MI



Multivariable Cox analysis. eGFR = estimated glomerular filtration rate; MI = myocardial infarction; VT = ventricular tachycardia.

CENTRAL ILLUSTRATION 30-Day and 1-Year Outcomes and Predictors of Mortality After Acute Myocardial Infarction in Côte d'Ivoire

Yao H, et al. JACC Adv. 2024;3(12):101285.

Factors associated with mortality at 30-day and 1-year follow-up. VT = ventricular tachycardia; other abbreviation as in Figure 2.

TABLE 5 Post-Myocardial Infarction Follow-Up Studies in Sub-Saharan Africa

First Author (Year)	Country	Study Participants	Age, y	Thrombolysis/PCI/CABG (Rates %)	In-Hospital Mortality (%)	Follow-Up Mortality	
						30 Days	1 Year
Cilliers (2023)[25]	South Africa	586 ACS	58.0	…/63.7/12.3	3.9	6.1	…
Isezuo (2022)[12]	Nigeria	1072 ACS (48.7% STEMI)	59.2	17.1/28.6/11.2	8.1	8.7	13.3
Hertz (2022)[11]	Tanzania	152 ACS (40.1% STEMI)	61.2	…/…/…	34.9	43.3	59.9
Fanta (2021)[5]	Ethiopia	181 ACS (61% STEMI)	55.8	…/7.2/…	20.4	25.4	…
Goli (2021)[6]	Tanzania	152 ACS (40.1% STEMI)	61.2	…/…/…	34.9	43.3	…
Kabore (2019)[26]	Burkina-Faso	111 ACS (88.3% STEMI)	57.6	7.2/…/…	8.1	16.2	…
Varwani (2019)[10]	Kenya	230 ACS (43.9% STEMI)	60.5	48.5/…/…	7.8	7.8	13.9
Yao (2019)[9]	Cote d'Ivoire	260 STEMI	57.0	3.5/31.9/0.77	14.3	…	10.41 (39 months)
Maurin (2012)[7]	Djibouti	35 STEMI	52.0	42/20/…	20	14.0	20.0
Schamroth (2012)[8]	South Africa	615 ACS (41.1% STEMI)	58.0	18.0/53.7/14.6	…	2.4 STEMI, 1.7 NSTEMI	5.7

ACS = acute coronary syndrome; N = no; Y = yes; other abbreviations as in Table 1.

high mortality in our regions compared to Western countries. Furthermore, several factors contribute to the high mortality rate of MI in this region. It is important to consider the socioeconomic, health, and cultural factors that are specific to the region. Limited access to health care facilities, especially in rural areas, frequently results in delayed presentation and diagnosis.[23,35] One issue is the limited health care infrastructure and low awareness of CV disease. Inadequate health care infrastructure, including a shortage of medical facilities, qualified cardiologists, and state-of-the-art equipment for reperfusion procedures, hampers the rapid and effective management of patients with MI.[2,23] Finally, high-prevalence infectious diseases, such as HIV/AIDS and malaria, could divert health care resources and attention away from chronic noncommunicable diseases, such as CV disease.[24,36] Therefore, a multifaceted approach is necessary, including improved health care infrastructure, increased awareness, better access to preventive measures, and a focus on managing CV risk factors. To reduce the growing mortality rate in sub-Saharan Africa, collaboration between governments and nongovernmental organizations is essential in implementing effective strategies. There is an urgent need for improved data collection and reporting on MI in sub-Saharan Africa, and for the development of local health policies and strategies to improve patient care and long-term outcomes. Local initiatives to reduce in-hospital and late mortality have begun to emerge. These include the organization of interventional cardiology fellowships in high-volume Western centers and the implementation of universal health care, which is now a reality in Côte d'Ivoire. In addition, the establishment of a national CV disease control program, and the involvement of nongovernmental organizations in the fight against risk factors such as diabetes, hypertension, and obesity are initiatives that are helping to reduce CV disease.

STUDY LIMITATION. There are some limitations. As we carried out a monocentric study, selection bias may be induced, and the generalization of the results to all MI population in Côte d'Ivoire should be considered with caution. We assumed many patients with MI died before referral to Abidjan Heart Institute, or in other medical centers. Some patients, particularly those with low-risk NSTEMI, were admitted to the ward rather than the ICU. They were therefore not included in the study, which introduced a selection bias. However, their small number mitigates the significance of this bias, as in our center, all patients with MI are systematically addressed in ICU, except in special circumstances. Incomplete medical records, data obtained by telephonic call, and lost to follow-up did not allow to make thorough analyses. The relatively small size of this study compared to Western MI registries, the short follow-up period (up to 1 year), and the observational type of study may affect validation of the results. The 5% threshold is arbitrary. Due to the absence of multiplicity control, interval estimates should be interpreted with caution. The management of patients with MI remains a key issue in our country, as in the majority of countries in sub-Saharan Africa. There is a wide practice gap between quality of care in sub-Saharan Africa and developed countries, mainly demonstrated by prolonged delay from symptoms to admission and limited access to PCI-capable heart centers and ICUs.

Despite all these limitations, this study, conducted in the national referral center for the management of MI, is among the first to address both the short- and long-term prognosis of MI in sub-Saharan Africa, with complete in-hospital and follow-up data. Our findings identified some strong factors associated with all-cause mortality, consistent with studies held in the West.

CONCLUSIONS

Mortality from MI is a major issue in sub-Saharan Africa, and factors associated with all-cause death in our study are consistent with Western MI registries. Thus, lessons learned from wealthy countries could help to address the high mortality after MI, mainly through the widespread use of acute revascularization procedures (particularly PCI) as the primary goal to be achieved in the acute phase in almost all patients. As in developed countries, prospective multicenter registries and randomized trials are needed to refine our findings, share tailored protocols adapted to our specificities, and propose feasible interventions. Precise identification of the determinants of mortality should lead to the implementation of aggressive interventions in these specific subgroups to reduce the high mortality in our regions compared to Western countries.

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PERSPECTIVES

COMPETENCY IN MEDICAL KNOWLEDGE: Findings from this study show that acute MI is responsible for high mortality in Côte d'Ivoire, as in most of sub-Saharan African countries, with strong predictive factors consistent with those in Western countries.

TRANSLATIONAL OUTLOOK: Future large-scale multicenter studies with longer follow-up are needed to accurately identify predictors of mortality in the sub-Saharan African population, and then to implement and evaluate proactive interventions to reduce the high burden of MI.

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APPENDIX For supplemental figures, please see the online version of this paper.