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Incidence and 1-Year Prognostic of Unstable Angina After High-Sensitivity Troponin Assessment

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ABSTRACT

Background: Incidence and prognostic of unstable angina after high-sensitivity troponin assesment is controversial.

Aims: This study evaluated prognostic of a contemporary population of patients with UA defined using high sensitive T troponin (T hs-cTn) measurements and with significant coronary artery disease (CAD).

Methods: Consecutive patients admitted in 2 French university centres with UA defined as clinical ischemic symptoms and T hs-cTn dosages undetectable (< 5 ng/L), non-elevated (> 14 ng/L) or mildly elevated (14−50 ng/L) were included. The primary end-point included major events at 1-year follow-up.

Results: Among 1752 patients admitted for ACS between December 2021 and February 2023, 210 (12.0%) have UA and significant CAD. Mean age was 66 ± 12 years, with predominantly men ($n = 143$; 68.1%). Patients had undetectable ($n = 4$), non-elevated ($n = 80$) or mildly elevated and stable T hs-cTn ($n = 126$). History of CAD was found in 98 patients (46.6%). Percutaneous intervention was required in main patients ($n = 186$; 88.6%). Adverse non-fatal in-hospital event occurred in one patient. The primary outcome was achieved in 55 patients (26.2%; CI 95% [20.2−32.1]) mainly related to new ACS ($n = 34$, 16.2%). The level of troponin at admission ($p = 0.639$) was not associated with the primary outcome. In multivariate analysis, multiple risk factors (OR 1.93, [1.01−3.69], $p = 0.0194$), history of CAD (3.09; CI [1.63; 5.87], $p = 0.0005$), and tritroncular disease (OR 2.66; CI [1.24; 5.69], $p = 0.0118$) were significantly associated with major events at 1-year.

Conclusion: Contemporary incidence of UA with significant CAD is low with few in-hospital events, but with a 1-year incidence of cardiac events high (26%), mainly related to new ACS. Improving secondary prevention may be crucial for these patients. (ID: NCT06378333).

1 | Introduction

Unstable angina (UA) is defined as myocardial ischaemia at rest or with minimal effort in the absence of acute cardiomyocyte injury and necrosis. UA is usually considered as a separate entity with distinct characteristics from non-ST segment elevation myocardial infarction (NSTEMI) and a different pathophysiology, with platelet thrombus predominating and no detectable myonecrosis [1–3]. UA is diagnosed when there are new or worsening symptoms of ischaemia or when there is a change in symptoms, with or without ischaemic changes on the ECG and a non-elevated (below the 99th percentile) high-sensitive troponin (hs-cTn) concentrations with cut-off concentrations which are assay specifics [4, 5]. When hs-cTn is negative, a third measurement at 3 h and an echocardiogram are proposed if there is a strong clinical suspicion of coronary pathology [5–7]. UA may also be considered when hs-cTn ≥ 99th percentile but mildly elevated and without

significant rise between 2 dosages performed within 1 and 3 h after the first one [5, 6, 8]. Among unselected patients presenting to the emergency department with suspected acute coronary syndrome (ACS), the introduction of ultrasensitive troponin measurement in place of standard troponin assays resulted in an increase in the sensitivity of detection of myocardial necrosis and a consequent decrease in the diagnosis of UA in favour to NSTEMI [3, 6–9]. The 2017 and the updated 2023 European guidelines for the management of NSTE-ACS recommend an invasive strategy during hospital admission for NSTEMI whereas individual assessment is proposed for UA including delayed coronary angiography or a non-invasive functional test depending on the clinical suspicion, without a precise strategy proposed [4, 5, 10]. In terms of prognosis, numerous studies or registries showed lower 1-year mortality, but a similar rate of non-fatal infarction compared with NSTEMI, whereas numerous studies were performed before the use of hs-cTn as a screening test and/or with absence of objective criteria for the diagnosis of UA [9, 11, 12]. Indeed, previous studies showed heterogeneity of patients with the diagnostic of UA and those with normal angiography or non-significant coronary stenosis may include patients with misdiagnosis [13, 14].

This study aimed to prospectively evaluate clinical characteristics and 1 year prognostic of a contemporary population of patients with UA defined according to guidelines and associated with significant coronary artery disease (CAD) within a non-selected population of patients admitted in the emergency department of cardiology for ACS.

2 | Methods

All consecutive patients with UA admitted to Montpellier and Nîmes (France) intensive care units (ICU) between November 2021 and February 2023 were included in the study. UA was defined according to guidelines in patients presenting with ischaemic symptoms at rest or minor exercise that do not fulfill the criteria of myocardial infarction (MI) according to the universal definition of MI [2–4]. Ischaemic symptoms may include new chest pain discomfort or equivalent symptoms or worsening/deterioration of previously stable angina with or without electrical signs of ischaemia on the electrocardiogram [2, 4]. We further subdivided UA in three groups by hs-cTn T levels (*Elecsys Roche*) including undetectable hs-cTn T (<5 ng/L), non-elevated hs-Tn T, (5–14 ng/L), or hsTnT levels above the 99th percentile value (15–51 ng/L), but without a significant concentration change in serial samples [2, 4, 6]. Coronary angiography was performed in all patients, without minimal delay required in opposite to NSTEMI guidelines [4, 5].

To exclude false diagnosis of UA, the study evaluated only patients with significant CAD defined when coronary epicardial artery stenosis > 70% reduction in the diameter or coronary artery stenosis > 50% reduction in the diameter of the left main coronary artery. Vessel spasm induced by ergonivine or acetylcholine were included. Patients with lower degree of stenosis or myocardial bridging were excluded. PCI was performed when considered as required according to guidelines [10]. All patients had transthoracic echocardiography (TTE) during the hospital stay and evaluation of left ventricular ejection fraction. ICU admission after coronary angiography evaluation and/or PCI was left at the operator discretion. Patients were admitted in the general cardiology floor or in ICU depending of symptoms, results of PCI or hemodynamic or rhythmic state. According to ACS guidelines, an optimal duration of dual antiplatelet therapy of 12 months was recommended for all patients, excepted those with high bleeding risk with a minimal duration of 1 month. Patients with previous anticoagulant therapy and PCI, have triple therapy duration between a few days and 1 month.

The primary endpoint of the study was the incidence of major clinical events at 1-year follow-up using a composite criterion including overall mortality, new ACS or re-hospitalization for cardiac reasons. The secondary endpoints included evaluation of all components of the primary end-point, comparison of prognostic of patients according to clinical and angiographic characteristics and between non-elevated or minor troponin elevation, major in-hospital events, hospitalization length of stay.

All patients included in the study were evaluated at 1 year (12 ± 2 months), in the Montpellier and Nîmes University hospitals to assess major events. Medical data were collected in the patient's medical file and during the medical consultation at follow-up. Biological and clinical parameters and events during the hospital phase were also collected from the patient's medical file. The usual management of the patient remains unchanged. Our study was validated by an ethical and scientific committee at Montpellier University Hospital in February 2024 (number 2024-01-023). The study was registered on ClinicalTrials.gov (ID: NCT06378333).

The statistical analysis was performed to obtain a precision (half the width of a 95% confidence interval) of ±5% around and with an expected prevalence of our primary outcome of 15% [7–9, 11], and inclusion of 210 patients was required. The qualitative variables of the population were described by their numbers, frequencies and percentages and quantitative variables by their means and standard deviation or median and quartiles depending on their distribution. For the primary outcome, a percentage and its 95% confidence interval was calculated using the exact method. The others prevalences were described in the same way. To compare the two sub-groups with and without troponin rise, a Chi^2 test was used if the conditions of application permit, a Fisher's otherwise. To analyse risk factors of major clinical events at 1 year, we used a multivariate logistic regression including as independent variables some clinical variables with a $p < 0.05$ in univariate. All tests were two-tailed with an alpha set at 5%. All analyses were performed using *EasyMedStat*.

3 | Results

Among 1752 consecutive patients admitted with ACS and included in our regional database, 280 patients were diagnosed to have UA (16%), and 210 of them (75%) with significant CAD were enrolled, representing a prevalence of 12% of all ACS during the study period (Figure 1; flow chart). Description of the population is shown in Table 1. The mean age of patients was 66 ± 11 years, with a majority of men (63.1%). Main patients had multiple cardiovascular risk factors. Almost half of the patients had a history of CAD, mainly with percutaneous

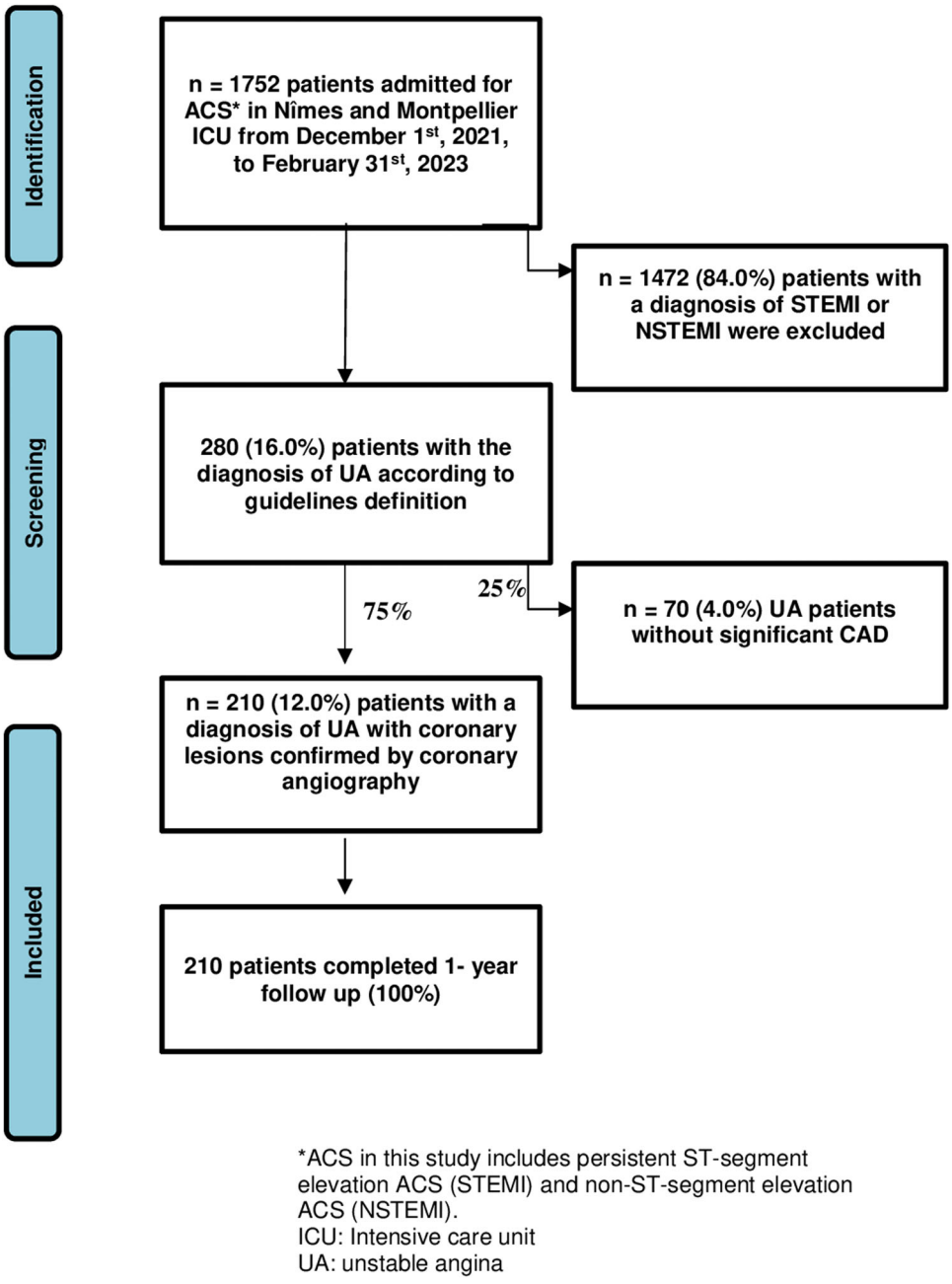


FIGURE 1 | Study flow chart. [Color figure can be viewed at wileyonlinelibrary.com]

coronary intervention (PCI). The study population had few co morbidities. Numerous patients had previous anti-platelet, antihypertensive and statins therapy. The ECG on admission was almost always in sinus rhythm, with signs of ischemia in half of patients (Central illustration 1).

Regarding troponin measurements, 84 patients (40%) had undetectable ($n = 4$) or non-elevated ($n = 80$) T hs-cTn (<99th percentile) while 126 patients (60%) had moderately elevated T hs-cTn (>99th percentile) with no kinetics ($n = 126$). All biological parameters at admission were reported in Table 2.

The mean time from admission to coronary angiography was 19.26 ± 16.45 h. The main culprit considered coronary artery was the left anterior descending artery (LAD) ($n = 105$, 50%), while the left main was involved in 18 patients (8.6%). The mean SYNTAX I score was (8.27 ± 5.06), and 57.6% of patients had multitroncular disease. Regarding therapeutic strategy, coronary revascularisation was proposed for the majority of patients ($n = 201$, 95.7%), mainly with PCI (Table 3).

During the hospital phase, there was only one adverse event related to a documented stroke. It was an ischaemic stroke related to a cerebral lacune with favourable evolution without sequela in a 51-year-old patient with a history of CAD. The average length of hospitalisation of the study population was 2.91 ± 1.52 days, with 128 (60.9%) patients being admitted to ICU after coronary angiography or PCI.

We have no missing patients at 1-year follow-up. The primary endpoint at 1-year follow- up was achieved in 55 patients (26.2%, CI 95% [20.2–32.1]). Only one patient died of a cardiac cause (0.5%). It

TABLE 1 | Clinical characteristics of patients at admission.

Variable	Whole sample ($n = 210$) n (%) or mean ± SD
Sex, Female	67 (31.9%)
Age (years)	66.42 (±11.58)
Cardiovascular risk factors	
At least 2 cardiovascular risk factors	128 (60.9%)
At least 3 cardiovascular risk factors	73 (34.8%)
Dyslipidaemia	113 (53.8%)
Arterial hypertension	111 (52.8%)
Smoking	81 (38.6%)
Diabetes mellitus	56 (26,7%)
Heredity	37 (17.6%)
BMI (kg/m^2)	26.13 ± 4.77
History of CAD	93 (44.3%)
History of PCI	86 (41.0%)
History of CABG	15 (7.1%)
History of heart failure	3 (1.4%)
Pacemaker	6 (2.8%)
ICD	3 (1.4%)
Chronic renal failure*	26 (12.3%)
LEAD	20 (9.5%)
COPD	12 (5.7%)
ECG modifications	110 (52.3%)
ST segment depression	48 (22.8%)
Negative T waves	62 (29.5%)
Conductive disorders	48 (22.8%)
LVEF (TTE) (%)	57.12 ± 7.13
Anti-platelet therapy	112 (53.3%)
Statins	100 (47.6%)
Anti-hypertensive drugs[#]	138 (65.7%)
Anticoagulant therapy	17 (8.1%)

Note: *Defined by a glomerular filtration rate of less than 60 mL/min according to CKD EPI; [#]Including beta blockers, angiotensin converting inhibitors, angiotensin receptors blockers, calcium channel blockers and thiazide diuretic.
Abbreviations: CABG, coronary artery bypass surgery; CAD, coronary artery disease; COPD, chronic obstructive pulmonary disease; ICD, implantable cardioverter-defibrillator; LVEF, left ventricular ejection fraction; PCI, percutaneous coronary intervention; SD, standard deviation; TTE, transthoracic echocardiogram.

was an 85-year-old patient with end-stage heart failure. The majority of events were recurrences of ACS ($n = 34$) or re-hospitalizations for cardiac causes (Table 4). Regarding new coronary events after PCI, we observed 32 ACS related to new coronary lesions (17.2%) and 10 patients with restenosis and UA (5.3%).

All recurrences of ACS were related to non-ST-segment elevation ACS, requiring PCI in all cases.

The indications of re-hospitalisation for others cardiac reasons were mainly related to heart failure (Table 4). We observed only two bleeding events related to gastric bleeding and requiring to stop prematurally DAPT (respectively 3 and 6 months after discharge)

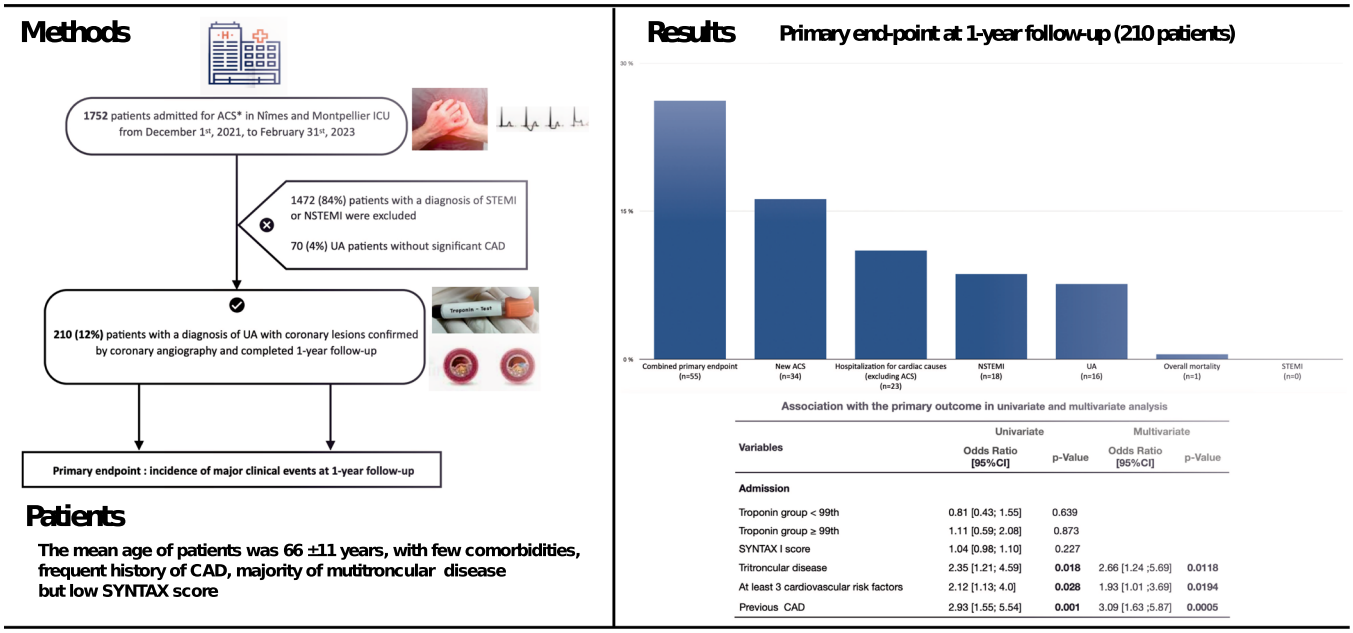
Regarding secondary objectives, we found no significant association between occurrence of the primary endpoint and troponin subgroups of patients or SYNTAX score. These variables were therefore not included in the multivariate analysis. In multivariate analysis, we found an association between the primary outcome and the group of patients with ≥ 3 cardiovascular risk factors, history of CAD, tritroncular disease, and anti-platelet drugs at admission (mono- or bi-therapy) (Table 5).

4 | Discussion

This prospective study aimed to describe 1-year prognostic of a contemporary population of patients with UA and significant CAD, and the main results were: (1) a low but still prevalence of UA with CAD among all ACS is observed (12%); (2) the population of patients is of relatively young age, with numerous cardiovascular risk factors, and frequent history of CAD, and most often mild troponin elevation at admission; (3) A high rate of major clinical events is observed at 1-year follow-up (26.2%), mainly related to new ACS, but with low mortality rate (0.5%); (4) a significant association is highlighted between the prognostic at 1 year and history of CAD, multiple risk factors, multitroncular disease but with not the troponin level at presentation

The use of high sensitivity cardiac troponin (hs-cTn) for the diagnostic strategy of ACS resulted in decrease of UA to the benefit of NSTEMI [3, 6–9]. A major obstacle is related to the diagnosis of UA itself, challenging in the absence of universally accepted definition [1, 2, 7, 8]. The risk of subjective diagnosis of UA was pointed out in several studies, with very poor concordance between investigator-reported and adjudicated UA [13, 14]. We considered only in our study patients admitted in ICU with suspicion of ACS with systematic coronary angiography performed, while patients with lower probability of CAD and with low-risk features were usually evaluated with non-invasive stress tests and were not included in our study [2, 4, 5]. We also focused only in our study on patients with significant coronary stenosis to include only patients with proved CAD and to exclude patients with misdiagnosis [13–15]. With this selective screening strategy, only 25% of patients with UA in our study had normal or non-significant coronary stenosis, slightly lower that previously described [6, 15, 16]. These results are probably related to selection of high risk patients admitted in ICU as well as inclusion of patients with hsTn levels above the 99th percentile which were usually considered with higher risk profile [6–8, 16]. Accordingly, patients with unstable symptoms and hsTn levels either below the 99th percentile or hsTn levels above the 99th percentile but without a significant change in serial samples could be regarded as having UA [6].

Our study confirmed that UA is still a prevalent entity corresponding to 4%−15% of patients with ACS in contemporary settings, depending to the threshold of troponin selected, that is, undetectable, non-elevated, mildly elevated [6–8]. Our study patients with UA and significant CAD have mainly (60%) troponin in the grey zone while only 84/210 patients (40%) have



CENTRAL ILLUSTRATION 1. | Incidence and prognostic of unstable angina: a prospective study in the current era. [Color figure can be viewed at wileyonlinelibrary.com]

TABLE 2 | Biological parameters at admission.

Variable	Whole sample ($n = 210$) n (%) or mean ± SD
Undetectable troponin hs-cTn (n)	4 (1.9%)
Not elevated troponin hs-cTn (n)	80 (38.1%)
Slightly elevated troponin hs-cTn (n)	126 (60.0%)
Mean hs-cTn T (ng/L)	22.04 ± 17.64
Mean NT pro BNP (ng/L)	471.82 ± 926.76
CRP (mg/L	7.4 ± 33.68
Fibrinogen (g/L)	3.78 ± 0.96
LDL level (g/L)	1.02 ± 0.46
HDL (g/L)	0.5 ± 0.19
Triglycerides (g/L)	1.48 ± 0.74

Abbreviations: BNP, brain natriuretic peptide; CRP, C-reactive protein; HDL, high density lipoprotein; hs-cTn T, high sensitivity T troponin; LDL, low density lipoprotein.

TABLE 3 | Coronary angiography results and revascularisation strategy in the study population.

Variable	(Whole sample: $n = 210$) n (%) or mean ± SD
SYNTAX score	8.27 ± 5.06
Mono-troncular disease	89 (42.4%)
Bi-troncular disease	66 (31.4%)
Tri-troncular disease	55 (26.2%)
Intra-stent restenosis	27 (12.9%)
Culprit considered coronary artery	
Left anterior descending artery	105 (50.0%)
Right coronary artery	52 (24.8%)
Circumflex artery	35 (16.6%)
Left main disease	18 (8.6%)
PCI	186 (88.6%)
Coronary bypass surgery	15 (7.1%)
Medical therapy alone	11 (5.2%)

Abbreviation: PCI, percutaneous coronary intervention.

troponin level < 99 percentile, representing 4.8% of all ACS during the study period, concordant with previous reports [6–9].

Compared to NSTEMI, a younger population with many cardiovascular risk factors is usually described [7, 9, 11]. Our cohort can also be described with relatively low mean age (66 year old), many risk factors but mainly treated (low LDL, high prevalence of anti-hypertensive, statin or antiplatelet therapy), frequent history of CAD, preserved LVEF. The low mean SYNTAX score but more than half patients with multitroncular disease depicted a population with no diffuse disease [17, 18]. More common ECG signs of ischemia and lower history of CAD or previous PCI were also highlighted in some studies in NSTEMI patients compared to UA patients [7, 9, 11].

Our primary endpoint was achieved in 26.2% of patients at 1-year follow-up and was mainly related to new ACS, with low mortality rate (0.5%) highlighting the risk of recurrence of coronary events despite revascularisation in majority of

patients. Interestingly, 94% of new ACS (32/34) were related to new coronary lesions. Our results are comparable to those reported in few studies related to UA, in terms of a high number of recurrences of ACS, repeat hospitalisations for cardiac reasons and a low overall mortality rate [6, 9, 11, 15, 19]. A recent study regrouping 2 large prospective multicentre trials and including 8892 patients showed a 1-year all-cause mortality rate significantly lower in UA compared to NSTEMI (3.3% vs. 10.4%). However, the rate of recurrence of ACS at 1 year was comparable between the two groups (11.2% vs. 7.9%, NS) and close to our study results regarding patients with UA [9].

TABLE 4 | Primary end-point at 1-year follow-up (210 patients).

Variable	*n*	%	CI 95%
Combined primary endpoint	55	26.2	20.2−32.1
Overall mortality	1	0.5	0.0−1.4
New ACS	34	16.2	11.2−21.2
STEMI	0	0	
NSTEMI	18	8.6	
UA	16	7.6	
Hospitalization for cardiac causes (excluding ACS)	23	11.0	6.7−15.2
Heart failure	10	4.7	
Valvulopathy	4	1.9	
Vascular events	4	1.9	
Supra ventricular arrhythmia	3	1.4	
Pace maker	2	0.9	
Bleeding	2	0.9	

Abbreviations: ACS, acute coronary syndrome; CI, confidence interval; NSTEMI, non-ST segment elevation myocardial infarction; STEMI, ST segment elevation myocardial infarction; UA, unstable angina.

In the SWEDEHEART registry, of 22,375 ACS patients from 2009 to 2013, 3204 (14.3%) were diagnosed with UA. Hazard ratios (95% CI) for all-cause mortality for UA was 2.06 (0.79–5.39, $p < 0.01$) and 8.14 (3.63–18.23, $p < 0.01$) for the NSTEMI group [7]. This low-1 year mortality compared to NSTEMI, was considered as indicating a less severe pathology in terms of atherosclerosis or plaque burden [9, 11, 19]. Similarly, our population can be described with high incidence of multitroncular CAD but low mean SYNTAX score, correlated with favorable long-term prognostic and low mortality rate [17, 19].

The rate of events remained high despite revascularisation (PCI or surgery) in majority of patients, probably reflecting the patients included in our study, selected with significant CAD and included those with mild elevation of troponin, both have been related to prognostic in UA [6, 15, 16, 19, 20]

Interestingly, near half of the patients in our study had a history of ischaemic heart disease (44%), rather higher that depicted in previous studies [9, 11, 12, 16]. Whether history of CAD or revascularisation, usually associated with chronic medical therapy including antiplatelet therapy, may influence expression of ACS in favour of UA, is not known.

In-hospital events were low, as previously described in others studies and lower than described in NSTEMI [9, 11, 19, 21]. Similarly, length of stay was short, reflecting the low risk profile of patients at admission (particularly young age, preserved LVEF). Interestingly, only 40% of them stay in ICU after

TABLE 5 | Univariate and multivariate analysis of 1-year prognostic (primary end-point).

	Univariate		Multivariate	
Variables	**Odds ratio [95% CI]**	***p* value**	**Odds ratio [95%CI]**	***p* value**
Admission				
Troponin group < 99th	0.81 [0.43; 1.55]	0.639		
Troponin group ≥ 99th	1.11 [0.59; 2.08]	0.873		
ECG signs of ischaemia	0,69 [0.37; 1.29]	0.313		
SYNTAX I score	1.04 [0.98; 1.10]	0.227		
Tritroncular disease	2.35 [1.21; 4.59]	110.018	2.66 [1.24;5.69]	0.0118
Age ≥ 65 years	1.05 [0.56; 1.98]	0.994		
Male sex	2.03 [0.99; 4.16]	0.075		
Medical treatment alone	2.53 [0.52; 12.44]	0.252		
Medical history				
At least 2 cardiovascular risk factors	2.03 [1.04; 3.98]	0.055		
At least 3 cardiovascular risk factors	2.12 [1.13; 4.0]	110.028	1.93 [1.01; 3.69]	0.0194
Previous CAD	2.93 [1.55; 5.54]	0.001	3.09 [1.63; 5.87]	0.0005
High blood pressure	1.30 [0.7; 2.43]	0.495		
Dyslipidemia	1.41; [0.75; 2.63]	0.364		
Previous anti-platelet therapy	3.46 [1.75; 6; 87]	0.001	2.54 [1.11; 5.84]	0.0279

Abbreviations: CAD, coronary artery disease; CI, confidence interval.

coronary angiography or PCI while recommendations state that rhythm monitoring in UA is unnecessary unless vasospastic angina is suspected, and that patients may be discharged in the absence of recurring symptoms or high-risk features [2, 4, 5].

The rate of revascularisation in our study, mainly with PCI, was high, related to selection of the patient after coronary angiography and only when significant CAD is proven. The study excluded patients with chest pain but with lower probability of CAD, which were usually referred for functional testing with rate of revascularization which can vary considerably between 10% and 78% [9, 11, 12, 15, 19].

As expected, cumulative risks factors, history of CAD or multitroncular disease were significantly associated with the primary outcome in multivariate analysis in our study. Previous antiplatelet therapy was not protective and even associated with higher risk of events, probably related to history of CAD or vascular disease. Although statistically non-significant, patients who undergone PCI tend to have less events than those undergoing medical treatment alone. We can also observe that severe bleeding events were rare in our study ($n = 2$).

A prospective study published in 2005 identified ST-segment depression, refractory angina and multivessel involvement as risk factors for 1-year cardiac events in UA (death, MI or coronary revascularization) but this study performed before the use of routinely hsTn dosage and included probably NSTEMI patients as currently defined [22].

Xu et al. in a single centre retrospective study identified a relation between a high SYNTAX score and long-term MACE for UA patients in the medical therapy group but not in the PCI group [23]. This relation was not found in our study but the population had a low mean SYNTAX score and a high rate of PCI.

We found no association between sub groups of patients with hs -cTn< or ≥99 th percentile and 1-year events. These results were different compared to previous reports as prognostic undetectable or low concentration of troponin correlated favourably with prognostic [6, 16, 21, 24]. Our study included however patients with significant CAD and main patients (60%) have stable elevated troponin > 99th percentile while only four patients (1.9%) have undetectable troponin level. While troponin level remains an important prognostic factor in non-selected population ACS and UA, it may probably no longer be correlated to events when coronary anatomy is considered and significant CAD proved [16].

Limitation of our study was first related to the follow-up which was obtained only in patients with UA and significant CAD, so we cannot compare events with the total ACS population, particularly NSTEMI or patients with suspected UA with no significant CAD. However, cohorts of NSTEMI in the literature allowed comparison of prognostic with our population. Second, it is not possible to know influence of control of risk factors during follow-up or to define the impact of any pharmacological treatment on MACE. Finally, all patients had coronary angiography in our study and a high rate of revascularisation. If functional tests have been performed, the rate of revascularisation could be different, probably lower, and may probably influence events (i.e., restenosis).

In conclusion, this study described a contemporary cohort of patients with UA and significant CAD on coronary angiography. Whereas very low rate of events was observed during the hospital phase, UA appears currently as a "false benign" pathology. The patients are young, with often numerous cardiovascular risk factors, mainly pluritroncular lesions and have a high rate of new events, mainly ACS at 1 year follow-up (26.2%), similar or even higher that observed in NSTEMI contemporary studies. Secondary prevention may probably be reinforced in this population to decrease new coronary events and reducing the health costs associated with repeat hospitalisations.

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Conflicts of Interest

The authors declare no conflicts of interest.

Data Availability Statement

The data that support the findings of this study are available on request from the corresponding author.

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