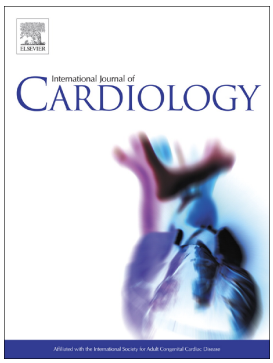

Acute coronary syndromes in young patients: Phenotypes, causes and clinical outcomes following percutaneous coronary interventions.

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Acute coronary syndromes in young patients: phenotypes, causes and clinical outcomes following percutaneous coronary interventions.

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Abstract

Background: The prevalence of acute coronary syndromes (ACS) among young individuals is increasing, but the phenotypic characteristics, causes and clinical outcomes in this group have not been well described.

Methods: Between 2009-2017, 8712 ACS patients underwent percutaneous coronary intervention (PCI) and were prospectively enrolled. We defined a young patient as female <50 years and male <45 years. The causes of ACS were defined by an adjudication committee. The primary endpoint was the patient-oriented composite endpoint (POCE) of all-cause mortality, myocardial infarction or any revascularization at 12 months.

Results: Among 8712 ACS patients, 472 (5.4%) patients were young (26% female). The main cause of ACS in young patients was atherosclerosis (85.5%), followed by coronary artery embolism (9%), and spontaneous coronary artery dissection (SCAD) (4.5%). POCE occurred less frequently in young compared to old patients (8.5% vs. 16.7%, hazard ratio 0.48 (95% confidence interval 0.35-0.66), p<0.001). The rates of the individual components of the POCE were lower in young including all-cause mortality (3.2% versus 9.5%, 0.32 (0.19-0.54), p<0.001), myocardial infarction (1.9% versus 3.7%, 0.49 (0.25-0.95), p=0.035) and any revascularization (5.1% versus 7.4%, 0.65 (0.43-0.97), p=0.037). Young patients with SCAD had a higher rate of death as compared to those with atherosclerosis, mainly attributed to cardiac deaths.

Conclusions: One out of 20 ACS patients undergoing PCI was young and the principal cause was atherosclerosis. Young carry a lower risk for future events compared to older ACS patients. The underlying cause leading to ACS should be considered in appropriate risk stratification of young patients.

Clinical Trial Registration: Clinicaltrials.gov. NCT02241291

Keywords: coronary artery disease, acute coronary syndromes, young, coronary artery embolism, spontaneous coronary artery dissection

INTRODUCTION

The reduction of coronary artery disease (CAD) burden and its related condition, namely acute coronary syndromes (ACS), are a global aim [1,2]. While the incidence of ACS in elderly individuals steadily decreased during the last decades, ACS has been more frequently diagnosed among young individuals in recent years [3–7]. These observations related to the young patient population merit particular attention and investigation due to the potential impact in long-term prognosis and quality of life [8]. Sparse evidence suggests differences in risk factor patterns, clinical presentation and angiographic disease severity of coronary atherosclerosis in young as compared to older patients presenting with an ACS [9,10]. While in older patients the principal mechanism of ACS is atherosclerosis, non-atherosclerotic mechanisms have been described in young patients. The two most important non-atherosclerotic causes include coronary artery embolism (CAE) and spontaneous coronary artery dissection (SCAD) [11]. The determination of the underlying mechanism of ACS plays an important role with regard to diagnostic work-up, acute therapy, prognosis, and optimal secondary prevention therapy [11–13]. Nevertheless, an in-depth contemporary evaluation of this group of young individuals presented with ACS is lacking. In light of this background, we investigated the phenotypes, underlying causes and clinical outcomes among young unselected patients undergoing PCI due to ACS in a large population at a tertiary care center.

METHODS

Definition of study population

All patients undergoing PCI at high-volume tertiary care center (Bern University Hospital, Switzerland), between January 2009 and December 2017 were prospectively and consecutively enrolled into the CARDIOBASE Bern PCI Registry (ClinicalTrials.gov, NCT02241291). This prospective PCI registry complied with the Declaration of Helsinki and was approved by the institutional ethics committee. All patients provided written informed consent for participation in the study. All patients were consecutively enrolled in the registry. For the purpose of the present analyses, we included patients who underwent PCI for ACS (unstable angina, non-ST elevation myocardial infarction, or ST-elevation myocardial infarction). We did not apply any other specific patient or lesion exclusion criteria. We defined a young patient as female <50 years and male <45 years; older individuals with ACS were considered as the control group. Even though there is no consensus for the specific cut-offs, the specific have been also previously applied in different studies to define young individuals with ACS [6,10,14]. The diagnosis of ACS was based on clinical signs/symptoms, cardiac biomarkers, and electrocardiographic changes following established diagnostic approaches.

Procedures

PCI were performed according to locally applied standards and the available clinical practice guidelines at the time of intervention. Unfractionated heparin at a dose of at least 5,000 IU or 70-100 IU/kg was administered during the procedure with the aim to achieve and maintain an activating clotting time during the procedure between 200-300 seconds. All patients received a loading dose of aspirin before PCI if not on a maintenance dose. A loading dose of P2Y12 inhibitor was administered before, at the time of, or immediately after the procedure, unless a maintenance

dose of P2Y12 inhibitor had been prescribed previously. A potent P2Y12 inhibitor (prasugrel or ticagrelor) was preferred over clopidogrel in discretion of the operator, while considering the individual bleeding and ischemic risk. The duration of dual antiplatelet therapy (DAPT) was differed according to individuals' risk of bleeding.

Definition of underlying ACS mechanisms in young patients

The causes of ACS in young patients were retrospectively adjudicated by a committee of two experienced interventional cardiologists based on clinical and angiographic criteria and according to previously validated methods [12,15]. Each ACS case among young patients was categorized into one of the following 3 groups:

Atherosclerosis

ACS was adjudicated due to an atherosclerotic cause in the presence of the following criteria: (1) angiographic evidence of a culprit lesion with lumen narrowing >90% with or without thrombus or ≥25% and ≤90% with angiographic evidence of thrombus, or plaque disruption or coronary erosion detected by intravascular imaging AND (2) either coronary artery ectasia or coronary artery stenosis outside the culprit lesion ≥25% on coronary angiography, and (5) absence of nonatherosclerotic causes (i.e. CAE, SCAD) [15].

Coronary artery embolism (CAE)

CAE was adjudicated according to three major and three minor criteria as previously reported by Shibata et al.[15] The 3 major criteria were (1) angiographic evidence of CAE and thrombosis without atherosclerotic components, (2) concomitant multisite CAE, and (3) concomitant systemic embolization excluding left ventricular thrombus attributable to ACS. The 3 minor criteria included (1) coronary angiography showing <25% stenosis, except for the culprit lesion; (2) evidence of an embolic source detected by any imaging modality; and (3) coexistence of a potential for

thromboembolic disease, including atrial fibrillation, cardiomyopathy, rheumatic valvular disease, infective myocarditis, prosthetic left-sided valve implantation, recent cardiac surgery, hypercoagulable state, patent foramen ovale, or atrial septal defect. The diagnosis of definite CAE was based on the presence of at least ≥2 major criteria, 1 major criterion plus ≥2 minor criteria, or 3 minor criteria.

Spontaneous coronary artery dissection (SCAD)

SCAD was adjudicated according to the following angiographic criteria proposed by the European Society of Cardiology position paper on SCAD [16]: *Type 1* represents the classical angiographic radiolucent "flap" and linear double lumen often associated with contrast hold-up; *Type 2* is characterized by a long diffuse and smooth stenosis predominantly located in mid-to-distal segments (*Type 2a* where there is recrudescence of a normal caliber distal vessel and *Type 2b* where the stenosis extends angiographically to the end of the vessel); *Type 3* is defined as angiographically indistinguishable from a focal atherosclerotic stenosis requiring diagnostic confirmation by intracoronary imaging; *Type 4* is defined as a total occlusion, usually of a distal vessel.

Follow-up strategies

Patients were systematically followed throughout 12 months for major adverse cardiac events (including death, cardiac death, myocardial infarction, target vessel revascularization, target lesion revascularization, definite or probable stent thrombosis) and for changes in medical treatment. Survival data were obtained from hospital records and municipal civil registries. A health questionnaire was sent to all living patients with questions on rehospitalization and adverse events, followed by telephone contact in case of missing responses. General practitioners, referring cardiologists, and patients were contacted as necessary for additional information. For patients who underwent treatment for adverse events at other medical institutions, external medical records,

discharge letters, and coronary angiography documentation were all systematically collected and reviewed.

Definition of clinical endpoints

A clinical event committee consisting of two cardiologists adjudicated all events by using original source documents. Any disagreements were resolved by consensus with a third cardiologist. The primary composite endpoint was the patient-oriented cardiovascular endpoint (POCE) – a composite of all-cause mortality, any myocardial infarction and any repeat revascularization. Secondary endpoint was the device-oriented composite endpoint (DOCE) – a composite of cardiac death, target vessel myocardial infarction, and clinically indicated target lesion revascularization. Cardiac death was defined as any death due to an immediate cardiac cause, procedure-related mortality, and death of unknown cause. Myocardial infarction was defined according to the modified historical definition [17]. The diagnosis of Q-wave myocardial infarction required ischemic signs or symptoms and new pathological Q waves in ≥2 contiguous electrocardiogram leads. Target vessel myocardial infarction was defined as a myocardial infarction not clearly attributable to a non-target vessel. Stent thrombosis was adjudicated based on the Academic Research Consortium (ARC) criteria [18] and was categorized on the basis of the timing as acute (<24 hours after PCI), subacute (between >24 hours and 1 month after PCI), or late (between 1 month and 1 year after PCI). Target lesion revascularization was defined as any clinically indicated revascularization for a coronary stenosis >50% within the stent or the 5-mm borders adjacent to the stent. Target vessel revascularization was defined as repeat revascularization of any segment within the entire major coronary vessel, proximal and distal to a target lesion.

Statistical analysis

Categorical variables are presented as counts with percentages and were compared among groups using Chi-squared or Fischer's exact tests. Continuous variables are presented as mean ± standard deviation (SD) after having evaluated the values' distribution and were compared among groups using Student's t-tests or ANOVAs. Lesion characteristics were compared between groups by using mixed-effect models accounting for the multiple lesions per patient. Cumulative event rates were compared between groups using Cox proportional hazard models. Kaplan-Meier curves are presented together with hazard ratios (HR) and the corresponding 95% confidence intervals (95%CI). All patients were analyzed up to the last valid contact date (i.e., at 12 months) and censored at the time point of the last contact if they were lost to follow-up before 12 months. We also calculated the SCORE (10-year risk score to suffer a fatal CVD event) according to the available guidelines [19,20]. Statistical analyses were performed using Stata 15 (StataCorp, College Station, Texas). Significance tests were two-tailed with a significance test set to 0.05.

RESULTS

Study population and procedural characteristics

Between January 2009 and December 2017, 15374 patients were consecutively and prospectively enrolled into the CARDIOBASE Bern PCI Registry (NCT02241291). Of those, 8712 patients underwent PCI due to an ACS with 472 (5.4%) qualifying as young patients according to the criteria mentioned above. **Supplementary Figure 1** shows a detailed patient selection flowchart. **Table** and **Supplementary Table 1 and 2** show baseline clinical, angiographic, and procedural characteristics. The mean (SD) age of the young ACS population was 41 (5) years of age and 26% of young patients were female. The phenotypic characteristics differed considerably between young and old patients undergoing PCI in the setting of ACS. The following three cardiovascular risk factors were more frequently encountered in young patients: obesity, smoking, and family history of CAD. Left ventricular ejection fraction did not differ between the two groups. While older patients presented more frequently with NSTE-ACS, young patients presented more frequently with STEMI and single vessel disease, but with longer lesions. The estimated 10-year risk score to suffer a future fatal CVD event based on SCORE was overall low for the young ACS patients (0.5%) (**Table**), but differed considerably across the three groups of different underlying mechanisms (p<0.001) (**Supplementary Table 3**), being higher for the group of young ACS patients with atherosclerosis. Adherence to the prescribed lipid lowering therapy was in favor to young patients at 1 year following the index ACS event **(Supplementary Table 2).**

Causes of ACS in young individuals

We were able to define the underlying cause of ACS in all 472 young patients (**Supplementary Table 3**). **Supplementary Figure 2** shows representative cases of each mechanism of ACS in young individuals. The principal mechanism of ACS was atherosclerotic (408 young patients;

86.5%), followed by CAE (43 young patients; 9%), and SCAD (21 young patients; 4.5%). **Supplementary Table 3** summarizes clinical and angiographic characteristics of the young patients stratified according to the underlying mechanism. Young patients with atherosclerotic cause of ACS were relative older and suffered more commonly from known cardiovascular risk factors including smoking, hypercholesterolemia, hypertension and family history of CAD. The vast majority of patients with SCAD were female (81%) and presented more frequently with symptoms of congestive heart failure. The most frequent SCAD type was type 2 (62%; type 2A 14%; type 2B 48%), followed by type 4 (24%), and type 1 (14%). The SCORE was higher in young patients with atherosclerosis as compared to those with CAE or SCAD (0.3% vs. 0.3 and 0.05% respectively; p<0.001).

Clinical outcomes

Information related to clinical events at 12 months of follow-up was available in 409 (86%) and 7639 (93%) young and old patients respectively. The primary composite endpoint POCE occurred in 40 (8.5%) young patients and in 1376 (16.7%) old patients (HR (95% CI) of 0.48 (0.35-0.66), p<0.001) **(Figure 1)**. The rates of the individual components of the primary endpoint were lower in young patients including all-cause mortality (3.2% vs. 9.5%, 0.32 (0.19-0.54), p<0.001), any myocardial infarction (1.9% vs. 3.7%, 0.49 (0.25-0.95), p=0.035) and any revascularization (5.1% vs. 7.4%, 0.65 (0.43-0.97), p=0.037). Detailed event rates of the clinical outcomes and the corresponding ratios of the hazard rates are presented in **Supplementary Table 4** and **Supplementary Table 5**. **Supplementary Figure 3** shows stratified analyses for the primary endpoint POCE for young patients across different subgroups. As shown in **Figure 2** and **Supplementary Table 5** POCE at 12 months of follow-up for young patients did not significantly differ between the three subgroups. There was no difference in the risk of DOCE or any individual

component of the primary outcome between the young individuals with atherosclerosis and those with CAE. However, young patients with SCAD who underwent clinically indicated PCI had a higher rate of all-cause mortality at 12 months as compared to young patients with atherosclerotic disease (SCAD vs. atherosclerotic: 14.3% vs. 2.2%, 2.6 (1.3-4.9), $p<0.01$). The higher death rate was driven by a higher rate of cardiac deaths in this high-risk group.

DISCUSSION

In this prospective registry of an all-comer patient population who underwent PCI for ACS, we observed that:

(1) Approximately one out of 20 ACS patients undergoing PCI for ACS was young (female <50 years, male <45 years).

(2) The principal mechanism of ACS in young patients was atherosclerosis (86.5%), followed by CAE (9%), and SCAD (4.5%).

(3) As compared to older patients, young patients had a lower risk for the primary endpoint POCE and for its individual components of all-cause mortality, any myocardial infarction and any revascularization at 12 months.

Our reported frequency of ACS among young patients (5%) is in line with previous studies [21]. However, we may have observed a lower prevalence of young patients suffering from ACS at our tertiary care center, since in the CARDIOBASE Bern PCI registry only patients undergoing PCI are enrolled and patients undergoing invasive diagnostic coronary angiography without stent implantation are not included. Therefore, conservatively treated non-atherosclerotic causes such as coronary vasospasm, myocardial bridging, stress-induced cardiomyopathy (Takotsubo syndrome), and conservatively treated SCAD are not captured in this dataset. For this reason, we explicitly focused on CAE and SCAD with clinical indications for PCI, two known and important mechanisms of non-atherosclerotic causes of ACS. Moreover, while the patient and lesion characteristics and overall outcomes of young patients have been previously described [21], the frequency of each cause in clinical practice and the clinical outcomes in this young patient population is poorly understood.

The most prevalent mechanism for ACS in this young ACS cohort was atherosclerosis, followed by CAE, and SCAD. In line with previous studies we showed that young patients suffering ACS are characterized by the traditional well established cardiovascular risk factors [22]. However, our findings provide additional insights, since we validated this observation only for those young patients with atherosclerotic cause of ACS, while among those young ACS patients with CAE or SCAD typical cardiovascular risk factors were lacking. Although the SCORE [23] for predicting the 10-year risk of future fatal cardiovascular events was higher in the atherosclerotic group, the overall risk was very low in this population. Nevertheless, the presence of "premature" atherosclerosis may not be correlated with the traditional risk factors [24,25]. Along these lines, novel biomarkers and/or intravascular imaging in addition to established risk prediction tools may provide enriched information to appropriate risk stratify those vulnerable young individuals presented with ACS.

SCAD used to be underdiagnosed among ACS patients and it was previously described as a rare cause of ACS with a prevalence of <1% [11]. However, with growing awareness of this new entity, broader use of new intravascular diagnostic imaging modalities and high sensitivity troponin assays, its prevalence has increased to 2-4% [16]. Previously reported figures are in line with our reported prevalence of SCAD of 4% and the predominance of women in our study sample. As has been previously reported [26], we also report the highest prevalence of SCAD type 2 with approximately 62%. Moreover, our study showed that patients with SCAD experienced a higher event rate of death, which was mainly driven by cardiac deaths, as compared to young patients with atherosclerotic disease. Previous studies reported that PCI in SCAD-related ACS has been associated with a higher rate of complications and worse outcomes as compared to patients who suffered from ACS due to atherosclerosis [27][28]. The longest follow-up of SCAD cohort was published by Saw et al. [26] and reports an overall major cardiac event rate of 19.9% (death rate: 1.2%; recurrent MI: 16.8%; stroke/transient ischemic attack: 1.2%; revascularization: 5.8%) during

a median long-term follow-up of 3.1 years. The much lower death rate reported by Saw et al. may be explained due to the inclusion of patients treated conservatively and the inclusion of "less severe" cases of SCAD. The current European Society of Cardiology guidelines [16] recommend a conservative management for hemodynamically stable patients with maintained distal flow in the culprit coronary artery and without demonstrable ongoing ischemia. Hence, we assume that our SCAD patient cohort represents a high-risk subgroup which had to undergo clinically indicated PCI to restore normal blood flow in the affected coronary arteries. The revascularization by PCI in the setting of atherosclerotic ACS passivates the culprit lesion and has been shown to "salvage" myocardium and therefore is also critically time-dependent. Conversely, PCI for SCAD is controversial and eliminates the "culprit" only in localized setting.

Limitations

This study has to be interpreted in light of some limitations. First, only the mechanisms of ACS in young patients were adjudicated, and therefore a matched comparison of the same underlying causes between young and older patients was not possible. Second, there is no commonly accepted age threshold to define "young". Previous literature used cut offs ranging between 35 and 65 [6,10,14]. Third, the definition of underlying mechanism was done retrospectively and therefore may be subjected to relevant biases. Nevertheless, the adjudication was performed by two experienced interventional cardiologists following previously well validated approaches for clinical trials. Fourth, in our prospective CARDIOBASE Bern PCI registry, we recruit only patients undergoing PCI. As a result, the specific ACS population of patients with coronary embolism and those with SCAD who were treated predominantly conservatively, has not be included and therefore the current findings may underestimate the true epidemiological burden of these conditions in young individuals presented with ACS. However, we included in our analysis those "high-risk"

individuals with SCAD required PCI due to ongoing ischemia or hemodynamic instability, which allowed us to derive comparative conclusions among the three main young ACS groups. Last, 63 young patients presented with ACS (46 atherosclerotic, 5 coronary embolisms, and 2 SCAD lesions) were not included in the final analysis. Of note, of those 63 patients 16% were lost to follow-up and 84% withdrew consent. However, we did not detect any relevant baseline differences between those included and not in the final analysis (Supplementary Table 6).

Conclusions

One out of 20 ACS patients undergoing PCI was young and the principal cause was atherosclerosis. Young carry a lower risk for future events compared to older ACS patients. The underlying cause leading to ACS should be considered in appropriate risk stratification of young patients.

Conflict of interests: Prof. Pilgrim reports research grants to his institution from Edwards Lifesciences, Boston Scientific, and Biotronik; personal fees from Biotronik and Boston Scientific as a speaker and HighLife SAS for clinical event committee participation; and proctoring with Medtronic outside of this study. Prof. Stortecky reports research grants to the institution from Edwards Lifesciences, Medtronic, Abbott Vascular, and Boston Scientific and personal fees from Boston Scientific, BTG, and Teleflex outside the submitted work. Prof. Windecker reports research and educational grants to his institution from Abbott, Amgen, AstraZeneca, BMS, Bayer, Boston Scientific, Biotronik, Cardinal Health, CardioValve, CSL Behring, Daiichi Sankyo, Edwards Lifesciences, Guerbet, InfraRedx, Johnson & Johnson, Medicure, Medtronic, Novartis, Pfizer, Polares, OrPha Suisse, Regeneron, Sanofi Aventis, Terumo, Sinomed, and V-Wave. Prof. Windecker serves as an unpaid advisory board member and/or unpaid member of the steering or

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REFERENCES

[1] A. Timmis, N. Townsend, C. Gale, R. Grobbee, N. Maniadakis, M. Flather, E. Wilkins, L. Wright, R. Vos, J. Bax, M. Blum, F. Pinto, P. Vardas, ESC Scientific Document Group, A. Goda, A.F. Demiraj, F. Weidinger, B. Metzler, F. Ibrahimov, A.A. Pasquet, M. Claeys, Y. Thorton, Z. Kusljugic, E. Omajic, V. Velchev, N. Ivanov, L. Antoniades, P. Agathangelou, M. Táborský, C. Gordon, M. Viigima, P.M. Juhani, Y. Juilliere, S. Cattan, A. Aladashvili, C. Hamm, K.-H. Kuck, K. Papoutsis, K. Bestehorn, S. Foussas, G. Giannoulidou, C. Varounis, I. Kallikazaros, R.G. Kiss, T. Czétényi, D. Becker, T. Gudnason, P. Kearney, K. McDonald, Y. Rozenman, B. Ziv, L. Bolognese, P. Luciolli, G. Boriani, S. Berkinbayev, A. Rakisheva, E. Mirrakhimov, A. Erglis, S. Jegere, G. Marinskis, J. Beissel, N. Marchal, S. Kedev, R.G. Xuereb, T. Tilney, T. Felice, M. Popovici, J. Bax, B. Mulder, M. Simoons, M. Elsendoorn, T.K. Steigen, D. Atar, Z. Kalarus, M. Tendera, J.S. Cardoso, J. Ribeiro, C. Mateus, G. Tatu-Chitoiu, P. Seferovic, B. Beleslin, I. Simkova, P. Durcikova, V. Belicova, Z. Fras, S. Radelj, J.R. Gonzalez Juanatey, S. Legendre, F. Braunschweig, U.P. Kaufmann, M. Rudiger-Sturchler, L. Tokgozoglu, A. Unver, V. Kovalenko, E. Nesukay, A. Naum, P.T. de Courtelary, S. Martin, D. Sebastiao, D. Ghislain, I. Bardinet, S. Logstrup, European Society of Cardiology: Cardiovascular Disease Statistics 2017, European Heart Journal. 39 (2018) 508–579. https://doi.org/10.1093/eurheartj/ehx628.

[2] J.-P. Collet, H. Thiele, E. Barbato, O. Barthélémy, J. Bauersachs, D.L. Bhatt, P. Dendale, M. Dorobantu, T. Edvardsen, T. Folliguet, C.P. Gale, M. Gilard, A. Jobs, P. Jüni, E. Lambrinou,

B.S. Lewis, J. Mehilli, E. Meliga, B. Merkely, C. Mueller, M. Roffi, F.H. Rutten, D. Sibbing, G.C.M. Siontis, ESC Scientific Document Group, 2020 ESC Guidelines for the management of acute coronary syndromes in patients presenting without persistent ST-segment elevation, Eur. Heart J. (2020). https://doi.org/10.1093/eurheartj/ehaa575.

[3] R.W. Yeh, J.V. Selby, Population Trends in the Incidence and Outcomes of Acute Myocardial Infarction, N Engl j Med. (2010) 11.

[4] J.H. Cole, J.I. Miller, L.S. Sperling, W.S. Weintraub, Long-term follow-up of coronary artery disease presenting in young adults, Journal of the American College of Cardiology. 41 (2003) 521–528. https://doi.org/10.1016/S0735-1097(02)02862-0.

[5] A. Gupta, Y. Wang, J.A. Spertus, M. Geda, N. Lorenze, C. Nkonde-Price, G. D'Onofrio, J.H. Lichtman, H.M. Krumholz, Trends in Acute Myocardial Infarction in Young Patients and Differences by Sex and Race, 2001 to 2010, Journal of the American College of Cardiology. 64 (2014) 337–345. https://doi.org/10.1016/j.jacc.2014.04.034.

[6] R. Gulati, A. Behfar, J. Narula, A. Kanwar, A. Lerman, J. Cooper, M. Singh, Acute Myocardial Infarction in Young Individuals, Mayo Clinic Proceedings. 95 (2020) 136–156. https://doi.org/10.1016/j.mayocp.2019.05.001.

[7] B. Ibanez, S. James, S. Agewall, M.J. Antunes, C. Bucciarelli-Ducci, H. Bueno, A.L.P. Caforio, F. Crea, J.A. Goudevenos, S. Halvorsen, G. Hindricks, A. Kastrati, M.J. Lenzen, E. Prescott, M. Roffi, M. Valgimigli, C. Varenhorst, P. Vranckx, P. Widimský, ESC Scientific Document Group, J.-P. Collet, S.D. Kristensen, V. Aboyans, A. Baumbach, R. Bugiardini, I.M. Coman, V. Delgado, D. Fitzsimons, O. Gaemperli, A.H. Gershlick, S. Gielen, V.-P. Harjola, H.A. Katus, J. Knuuti, P. Kolh, C. Leclercq, G.Y.H. Lip, J. Morais, A.N. Neskovic, F.-J. Neumann, A. Niessner, M.F. Piepoli, D.J. Richter, E. Shlyakhto, I.A. Simpson, P.G. Steg, C.J. Terkelsen, K. Thygesen, S. Windecker, J.L. Zamorano, U. Zeymer, S. Windecker, V. Aboyans, S. Agewall, E. Barbato, H. Bueno, A. Coca, J.-P. Collet, I.M. Coman, V. Dean, V. Delgado, D. Fitzsimons, O. Gaemperli, C. Hindricks, B. Iung, P. Jüni, H.A. Katus, J. Knuuti, P. Lancellotti, C. Leclercq, T. McDonagh, M.F. Piepoli, P. Ponikowski, D.J. Richter, M. Roffi, E. Shlyakhto, I.A. Simpson, J.L. Zamorano, M. Chettibi, H.G. Hayrapetyan, B. Metzler, F. Ibrahimov, V. Sujayeva, C. Beaulove, L. Dizdarevic-Hudic, K. Karamfiloff, B. Skoric, L. Antoniades, P. Tousek, P.J. Terkelsen, S.M. Shaheen, T. Marandi, M. Niemelä, S. Kedev, M. Gilard, A. Aladashvili, A. Elsaesser, I.G. Kanakakis, B. Merkely, T. Gudnason, Z. Iakobishvili, L. Bolognese, S. Berkinbayev, G. Bajraktari, M. Beishenkulov, I. Zake, H.B. Lamin, O. Gustiene, B. Pereira, R.G. Xuereb, S. Ztot, V. Juliebø, J. Legutko, A.T. Timóteo, G. Tatu-Chiţoiu, A. Yakovlev, L. Bertelli, M. Nedeljkovic, M. Studenčan, M. Bunc, A.M. García de Castro, P. Petursson, R. Jeger, M.S. Mourali, A. Yildirir, A. Parkhomenko, C.P. Gale, 2017 ESC Guidelines for the management of acute myocardial infarction in patients presenting with ST-segment elevation, European Heart Journal. 39 (2018) 119–177. https://doi.org/10.1093/eurheartj/ehx393.

[8] K.A. Wilmot, M. O'Flaherty, S. Capewell, E.S. Ford, V. Vaccarino, Coronary Heart Disease Mortality Declines in the United States From 1979 Through 2011, (n.d.) 6.

[9] T. Iragavarapu, T. Radhakrishna, Kj. Babu, R. Sanghamitra, Acute coronary syndrome in young - A tertiary care centre experience with reference to coronary angiogram, Journal of the Practice of Cardiovascular Sciences. 5 (2019) 18. https://doi.org/10.4103/jpcs.jpcs_74_18.

[10]B. Ricci, E. Cenko, Z. Vasiljevic, G. Stankovic, S. Kedev, O. Kalpak, M. Vavlukis, M. Zdravkovic, S. Hinic, D. Milicic, O. Manfrini, L. Badimon, R. Bugiardini, Acute Coronary Syndrome: The Risk to Young Women, Journal of the American Heart Association. 6 (2017). https://doi.org/10.1161/JAHA.117.007519.

[11]T.M. Waterbury, G. Tarantini, B. Vogel, R. Mehran, B.J. Gersh, R. Gulati, Non-atherosclerotic causes of acute coronary syndromes, Nature Reviews Cardiology. (2019). https://doi.org/10.1038/s41569-019-0273-3.

[12]S.N. Hayes, E.S.H. Kim, J. Saw, D. Adlam, C. Arslanian-Engoren, K.E. Economy, S.K. Ganesh, R. Gulati, M.E. Lindsay, J.H. Mieres, S. Naderi, S. Shah, D.E. Thaler, M.S. Tweet, M.J. Wood, Spontaneous Coronary Artery Dissection: Current State of the Science: A Scientific Statement From the American Heart Association, Circulation. 137 (2018). https://doi.org/10.1161/CIR.0000000000000564.

[13]J.D. Häner, Impact of intracoronary optical coherence tomography in routine clinical practice: A contemporary cohort study, (n.d.) 8.

[14]Z. Vasiljevic- Pokrajcic, N. Mickovski, G. Davidovic, M. Aranin, B. Stefanovic, G. Krljanac, M. Radosavljevic- Radovanovic, N. Radovanovic, R. Lačica, S. Milanović, J. Bjekić, M. Majstorovic- Stakic, D. Trifunovic, A. Karadzic, D. Rajic, A. Milosevic, M. Zdravkovic, J. Saric, R. Bugiardini, Sex and age differences and outcomes in acute coronary syndromes, International Journal of Cardiology. 217 (2016) S27–S31. https://doi.org/10.1016/j.ijcard.2016.06.217.

[15]T. Shibata, S. Kawakami, T. Noguchi, T. Tanaka, Y. Asaumi, T. Kanaya, T. Nagai, K. Nakao, M. Fujino, K. Nagatsuka, H. Ishibashi-Ueda, K. Nishimura, Y. Miyamoto, K. Kusano, T. Anzai, Y. Goto, H. Ogawa, S. Yasuda, Prevalence, Clinical Features, and Prognosis of Acute Myocardial Infarction Attributable to Coronary Artery Embolism, Circulation. 132 (2015) 241–250. https://doi.org/10.1161/CIRCULATIONAHA.114.015134.

[16]D. Adlam, F. Alfonso, A. Maas, C. Vrints, Writing Committee, A. al-Hussaini, H. Bueno, P. Capranzano, S. Gevaert, S.R. Hoole, T. Johnson, C. Lettieri, M.T. Maeder, P. Motreff, P. Ong, A. Persu, H. Rickli, F. Schiele, M.N. Sheppard, E. Swahn, European Society of Cardiology, acute cardiovascular care association, SCAD study group: a position paper on spontaneous coronary artery dissection, European Heart Journal. 39 (2018) 3353–3368. https://doi.org/10.1093/eurheartj/ehy080.

[17]P. Vranckx, D.E. Cutlip, R. Mehran, P.-P. Kint, S. Silber, S. Windecker, P.W. Serruys, Myocardial infarction adjudication in contemporary all-comer stent trials: balancing sensitivity and specificity. Addendum to the historical MI definitions used in stent studies., EuroIntervention : Journal of EuroPCR in Collaboration with the Working Group on Interventional Cardiology of the European Society of Cardiology. 5 (2010) 871–4. https://doi.org/10.4244/EIJV5I7A146.

[18]D.E. Cutlip, S. Windecker, R. Mehran, A. Boam, D.J. Cohen, G. Van Es, P.G. Steg, M. Morel, L. Mauri, P. Vranckx, E. Mcfadden, A. Lansky, M. Hamon, M.W. Krucoff, P.W. Serruys, Clinical End Points in Coronary Stent Trials A Case for Standardized Definitions, (2007). https://doi.org/10.1161/CIRCULATIONAHA.106.685313.

[19]M.F. Piepoli, A.W. Hoes, S. Agewall, C. Albus, C. Brotons, A.L. Catapano, M.-T. Cooney, U. Corrà, B. Cosyns, C. Deaton, I. Graham, M.S. Hall, F.D.R. Hobbs, M.-L. Løchen, H. Löllgen,

P. Marques-Vidal, J. Perk, E. Prescott, J. Redon, D.J. Richter, N. Sattar, Y. Smulders, M. Tiberi, H.B. van der Worp, I. van Dis, W.M.M. Verschuren, 2016 European Guidelines on cardiovascular disease prevention in clinical practice: The Sixth Joint Task Force of the European Society of Cardiology and Other Societies on Cardiovascular Disease Prevention in Clinical Practice (constituted by representatives of 10 societies and by invited experts)Developed with the special contribution of the European Association for Cardiovascular Prevention & Rehabilitation (EACPR), European Heart Journal. 37 (2016) 2315–2381. https://doi.org/10.1093/eurheartj/ehw106.

[20]F.L.J. Visseren, F. Mach, Y.M. Smulders, D. Carballo, K.C. Koskinas, M. Bäck, A. Benetos, A. Biffi, J.-M. Boavida, D. Capodanno, B. Cosyns, C. Crawford, C.H. Davos, I. Desormais, E. Di Angelantonio, O.H. Franco, S. Halvorsen, F.D.R. Hobbs, M. Hollander, E.A. Jankowska, M. Michal, S. Sacco, N. Sattar, L. Tokgozoglu, S. Tonstad, K.P. Tsioufis, I. van Dis, I.C. van Gelder, C. Wanner, B. Williams, ESC Scientific Document Group, G. De Backer, V. Regitz-Zagrosek, A.H. Aamodt, M. Abdelhamid, V. Aboyans, C. Arbus, R. Asteggiano, M. Bäck, M.A. Borger, C. Brotons, J. Čelutkienė, R. Cifkova, M. Cikes, F. Cosentino, N. Dagres, T. De Backer, D. De Bacquer, V. Delgado, H. Den Ruijter, P. Dendale, H. Drexel, V. Falk, L. Fauchier, B.A. Ference, J. Ferrières, M. Ferrini, M. Fisher, D. Fliser, Z. Fras, D. Gaita, S. Giampaoli, S. Gielen, I. Graham, C. Jennings, T. Jorgensen, A. Kautzky-Willer, M. Kavousi, W. Koenig, A. Konradi, D. Kotecha, U. Landmesser, M. Lettino, B.S. Lewis, A. Linhart, M.-L. Løchen, K. Makrilakis, G. Mancia, P. Marques-Vidal, J.W. McEvoy, P. McGreavy, B. Merkely, L. Neubeck, J.C. Nielsen, J. Perk, S.E. Petersen, A.S. Petronio, M. Piepoli, N.G. Pogosova, E.I.B. Prescott, K.K. Ray, Z. Reiner, D.J. Richter, L. Rydén, E. Shlyakhto, M. Sitges, M. Sousa-Uva, I. Sudano, M. Tiberi, R.M. Touyz, A. Ungar, W.M.M. Verschuren, O. Wiklund, D. Wood, J.L. Zamorano, Y.M. Smulders, D. Carballo, K.C. Koskinas, M. Bäck, A. Benetos, A. Biffi, J.-M. Boavida, D. Capodanno, B. Cosyns, C.A. Crawford, C.H. Davos, I. Desormais, E. Di Angelantonio, O.H. Franco Duran, S. Halvorsen, F.D. Richard Hobbs, M. Hollander, E.A. Jankowska, M. Michal, S. Sacco, N. Sattar, L. Tokgozoglu, S. Tonstad, K.P. Tsioufis, I. van Dis, I.C. van Gelder, C. Wanner, B. Williams, 2021 ESC Guidelines on cardiovascular disease prevention in clinical practice, European Heart Journal. 42 (2021) 3227–3337. https://doi.org/10.1093/eurheartj/ehab484.

[21]A. Avezum, M. Makdisse, P. Spencer, J.M. Gore, K.A.A. Fox, G. Montalescot, K.A. Eagle, K. White, R.H. Mehta, E. Knobel, J.- Philippe Collet, Impact of age on management and outcome of acute coronary syndrome: Observations from the global registry of acute coronary events (GRACE), American Heart Journal. 149 (2005) 67–73. https://doi.org/10.1016/j.ahj.2004.06.003.

[22]A.W. Schoenenberger, D. Radovanovic, J.-C. Stauffer, S. Windecker, P. Urban, G. Niedermaier, P.-F. Keller, F. Gutzwiller, P. Erne, Acute coronary syndromes in young patients: Presentation, treatment and outcome, International Journal of Cardiology. 148 (2011) 300–304. https://doi.org/10.1016/j.ijcard.2009.11.009.

[23]C. Baigent, K.C. Koskinas, M. Casula, L. Badimon, M.J. Chapman, G.G.D. Backer, V. Delgado, B.A. Ference, I.M. Graham, A. Halliday, B. Mihaylova, G. Riccardi, D.J. Richter, M.S. Sabatine, 2019 ESC/EAS Guidelines for the management of dyslipidaemias: lipid modification to reduce cardiovascular risk, (n.d.) 78.

[24]M. Nielsen, C. Andersson, T.A. Gerds, P.K. Andersen, T.B. Jensen, L. Køber, G. Gislason, C. Torp-Pedersen, Familial clustering of myocardial infarction in first-degree relatives: a

nationwide study, European Heart Journal. 34 (2013) 1198–1203. https://doi.org/10.1093/eurheartj/ehs475.

[25]T.A. Mulders, S. Sivapalaratnam, E.S.G. Stroes, J.J.P. Kastelein, A.D. Guerci, S.-J. Pinto-Sietsma, Asymptomatic Individuals With a Positive Family History for Premature Coronary Artery Disease and Elevated Coronary Calcium Scores Benefit From Statin Treatment, JACC: Cardiovascular Imaging. 5 (2012) 252–260. https://doi.org/10.1016/j.jcmg.2011.11.014.

[26]J. Saw, K. Humphries, E. Aymong, T. Sedlak, R. Prakash, A. Starovoytov, G.B.J. Mancini, Spontaneous Coronary Artery Dissection, Journal of the American College of Cardiology. 70 (2017) 1148–1158. https://doi.org/10.1016/j.jacc.2017.06.053.

[27]M.S. Tweet, M.F. Eleid, P.J.M. Best, R.J. Lennon, A. Lerman, C.S. Rihal, D.R.H. Jr, S.N. Hayes, R. Gulati, Spontaneous Coronary Artery Dissection, (n.d.).

[28]J. Saw, E. Aymong, T. Sedlak, C.E. Buller, A. Starovoytov, D. Ricci, S. Robinson, T. Vuurmans, M. Gao, K. Humphries, G.B.J. Mancini, Spontaneous Coronary Artery Dissection: Association With Predisposing Arteriopathies and Precipitating Stressors and Cardiovascular Outcomes, Circulation: Cardiovascular Interventions. 7 (2014) 645–655. https://doi.org/10.1161/CIRCINTERVENTIONS.114.001760.

TABLE

Table. Baseline clinical characteristics.

FIGURES

Figure 1. Time-to-event curves for the primary endpoint POCE in young and old patients for the individual components of the primary endpoint at 12 months.

(A) Patient-oriented composite endpoint, (B) all-cause mortality, (C) myocardial infarction, (D) revascularization. Blue lines indicate young patients. Red lines indicate old patients. HR=hazard ratio. P=p for superiority. POCE=device-oriented composite endpoint (death, myocardial infarction, revascularization).

Figure 2. Time-to-event curves for the primary endpoint POCE in young patients for the individual components of the primary composite endpoint at 12 months according to the mechanism of ACS.

(A) Patient-oriented composite endpoint (POCE), (B) death, (C) myocardial infarction, (D) revascularization. Red lines indicate young patients with atherosclerosis as the mechanism of acute coronary syndrome. Blue lines indicate coronary embolism. Green lines indicate spontaneous coronary artery dissection. HR=hazard ratio. P=p for superiority. POCE=device-oriented composite endpoint (death, myocardial infarction, revascularization).

Figure 3. Time-to-event curves for the primary endpoint DOCE in young and old patients and the individual components of the primary endpoint at 12 months.

Depicted are first events (% Kaplan Meier estimates), hazard ratios from Cox regression survival models comparing BP-SES vs. DP-EES (with 95% confidence intervals CI) and p-values. DOCE refers to the primary outcome, defined as the composite of cardiac death, target vessel myocardial infarction, and target lesion revascularization. POCE is the patient-oriented outcome, defined as the

composite all-cause death, myocardial infarction including peri-procedural (CK >2ULN, if not available CK-MB >3ULN), and any revascularization.

Table. Baseline clinical characteristics.

	Overall (n = 8712)	Young (n = 472)	Old (n = 8240)	*p*-value
Age (years)	66.3 ± 12.9	40.9 ± 4.9	67.8 ± 11.6	<0.001
Gender (female)	2232 (25.6%)	124 (26.3%)	2108 (25.6%)	0.74
Body Mass Index (kg/m^2)	27.2 ± 4.6	27.6 ± 4.8	27.1 ± 4.6	0.020
Current smoker	2863 (32.9%)	316 (66.9%)	2547 (30.9%)	<0.001
Hypercholesterolemia*	4649 (53.4%)	189 (40.0%)	4460 (54.1%)	<0.001
Hypertension	5306 (60.9%)	151 (32.0%)	5155 (62.6%)	<0.001
Diabetes mellitus	1716 (19.7%)	46 (9.7%)	1670 (20.3%)	<0.001
Diet	234 (2.69%)	6 (1.27%)	228 (2.77%)	0.789
Oral treatment	1071 (12.3%)	21 (4.45%)	1050 (12.7%)	0.024
Insulin dependent	625 (7.17%)	22 (4.66%)	603 (7.32%)	0.073
Family history of CAD	1968 (22.6%)	168 (35.6%)	1800 (21.8%)	<0.001
Chronic kidney disease (<60 eGFR)	1885 (21.6%)	14 (3.0%)	1871 (28.9%)	<0.001
Chronic obstructive lung disease	495 (5.7%)	3 (0.6%)	492 (6.0%)	<0.001
Peripheral arterial disease	549 (6.3%)	7 (1.5%)	542 (6.6%)	<0.001
History of cerebrovascular accident	480 (5.5%)	10 (2.1%)	470 (5.7%)	<0.001
History of gastrointestinal bleeding	183 (2.1%)	3 (0.6%)	180 (2.2%)	0.022
History of malignancy	816 (9.4%)	7 (1.5%)	809 (9.8%)	<0.001
Previous myocardial infarction	1112 (12.8%)	26 (5.5%)	1086 (13.2%)	<0.001
Previous PCI	1268 (14.6%)	27 (5.7%)	1241 (15.1%)	<0.001
Previous CABG	566 (6.5%)	2 (0.4%)	564 (6.8%)	0.493
Killip III or IV	957 (11.0%)	37 (7.8%)	920 (11.2%)	0.026
IABP (prior or during PCI)	192 (2.2%)	12 (2.5%)	180 (2.2%)	0.61
Percutaneous LVAD	78 (0.9%)	5 (1.1%)	73 (0.9%)	0.71
Vasopressors	487 (5.6%)	24 (5.1%)	463 (5.6%)	0.63
LVEF (%)	49.2 ± 13.4	49.9 ± 12.6	49.2 ± 13.4	0.29
Indication for PCI				<0.001
Unstable Angina	733 (8.4%)	19 (4.0%)	714 (8.7%)	
NSTE-ACS	3852 (44.2%)	156 (33.1%)	3696 (44.9%)	
STEMI	4127 (47.4%)	297 (62.9%)	3830 (46.5%)	
Laboratory values				
Total cholesterol (mmol/l)	4.9 ± 1.3	5.2 ± 1.3	4.8 ± 1.3	<0.001
HDL cholesterol (mmol/l)	1.2 ± 0.4	1.1 ± 0.3	1.21 ± 0.4	<0.001
LDL cholesterol (mmol/l)	3.0 ± 1.1	3.3 ± 1.1	2.9 ± 1.1	<0.001

Triglyceride (mmol/l)	1.5 ± 1.1	1.9 ± 1.5	1.5 ± 1.1	<0.001
Anemia†	1741 (20.0%)	40 (8.5%)	1701 (20.6%)	<0.001
Thrombocytopenia‡	85 (1.0%)	3 (0.6%)	82 (1.0%)	0.41
SCORE (% of risk of fatal CVD at 10 years)	4.04 (2.99)	0.47 (0.57)	4.31 (2.93)	<0.001

Depicted are counts (%, p-values from Chi-square tests) and means (±standard deviation, p-values from unpaired t-tests). Young patients defined as female <50 years and male <45 years.

CAD = coronary artery disease. eGFR = creatinine estimated glomerular filtration rate calculated by using the Cockcroft-Gault formula. PCI = percutaneous coronary intervention. CABG = coronary artery bypass graft. IABP = intra-aortic balloon bump. LVAD = left ventricular assist device. LVEF = left ventricular ejection fraction. NSTE-ACS = non-ST-elevation acute coronary syndrome. STEMI = ST-elevation myocardial infarction. HDL = high-density lipoprotein. LDL = low-density lipoprotein.

*Hypercholesterolemia defined as cholesterol >5mmol/l or requiring treatment

†Anemia defined as female <120 hemoglobin g/l, male <130 hemoglobin g/l.

‡Thrombocytopenia defined as $<100.10^9$ g/l thrombocytes.

Figure 1. Time-to-event curves for the primary endpoint POCE in young and old patients for the individual components of the primary endpoint at 12 months.

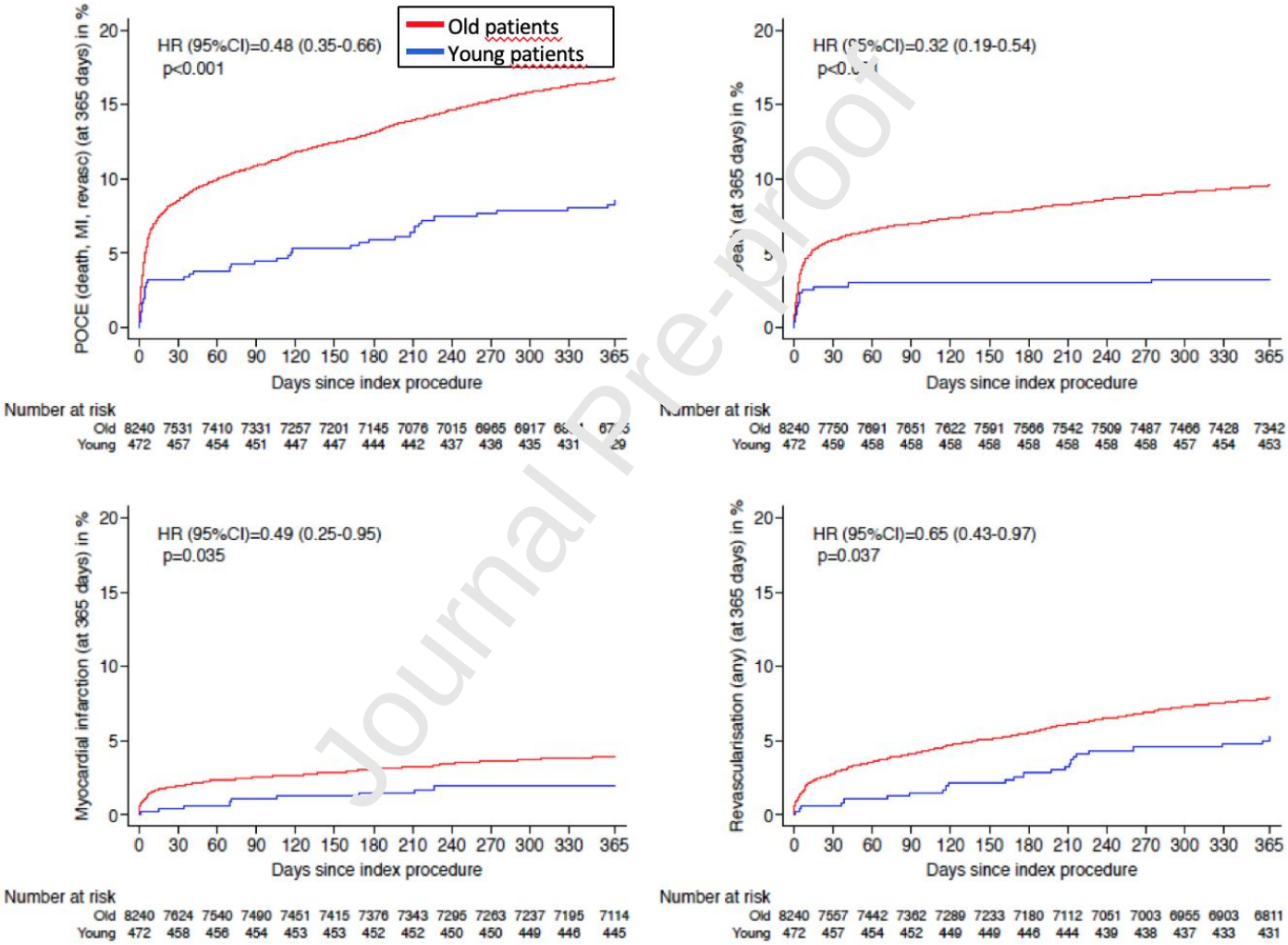


Figure 2. Time-to-event curves for the primary endpoint POCE in young patients for the individual components of the primary composite endpoint at 12 months according to the mechanism of ACS.

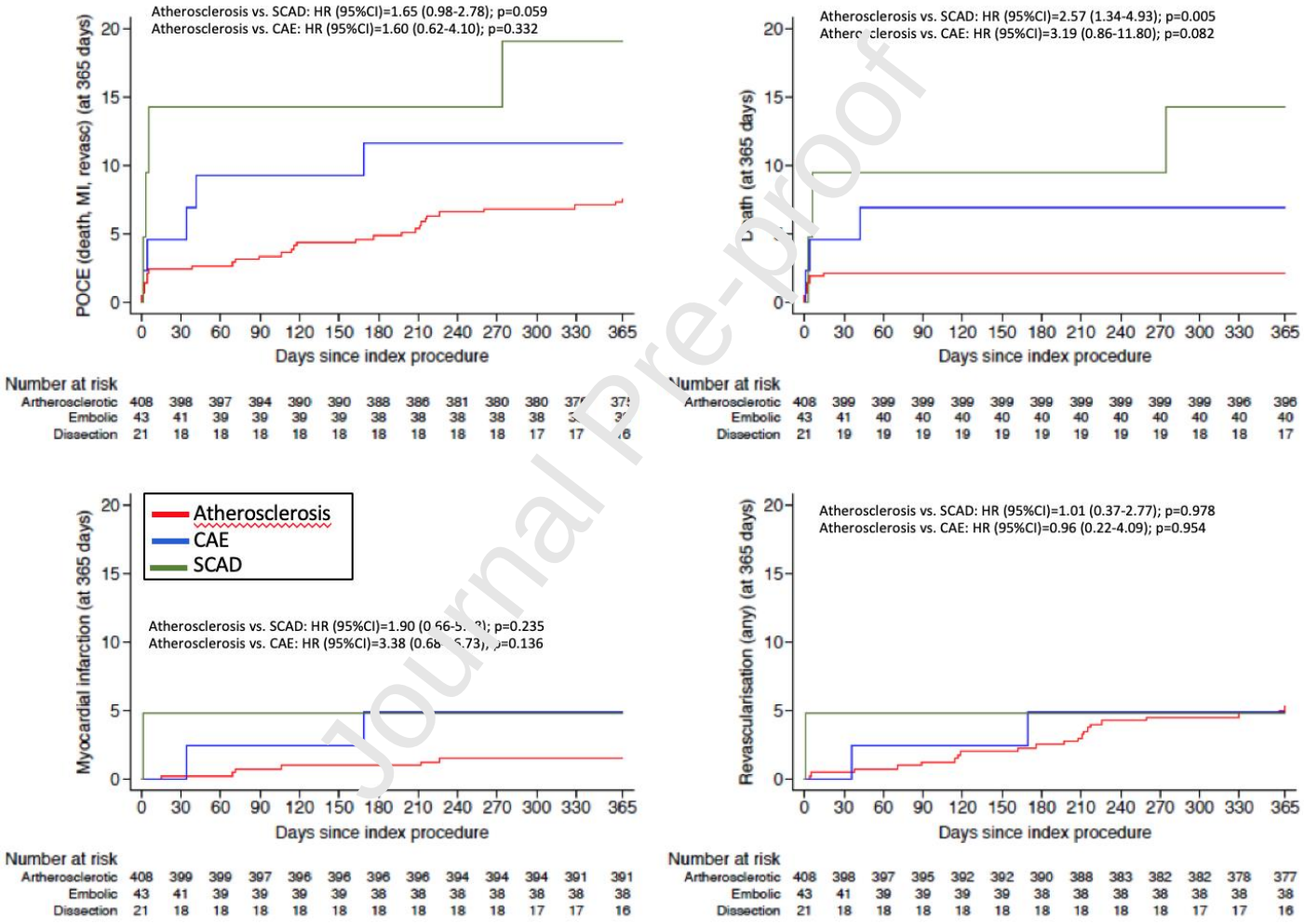
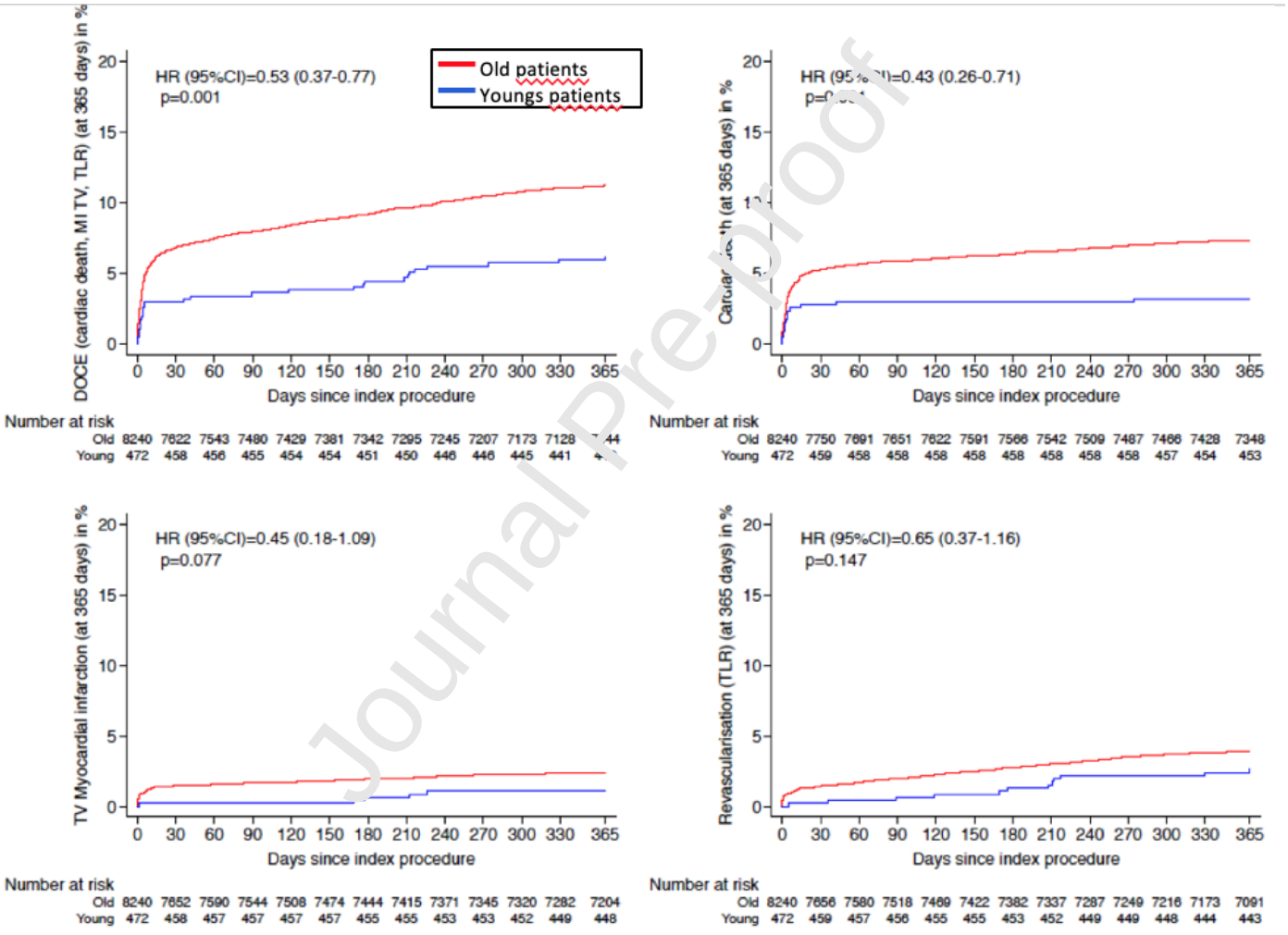


Figure 3. Time-to-event curves for the primary endpoint DOCE in young and old patients and the individual components of the primary endpoint at 12 months.



All listed authors fulfil the following 4 criteria:

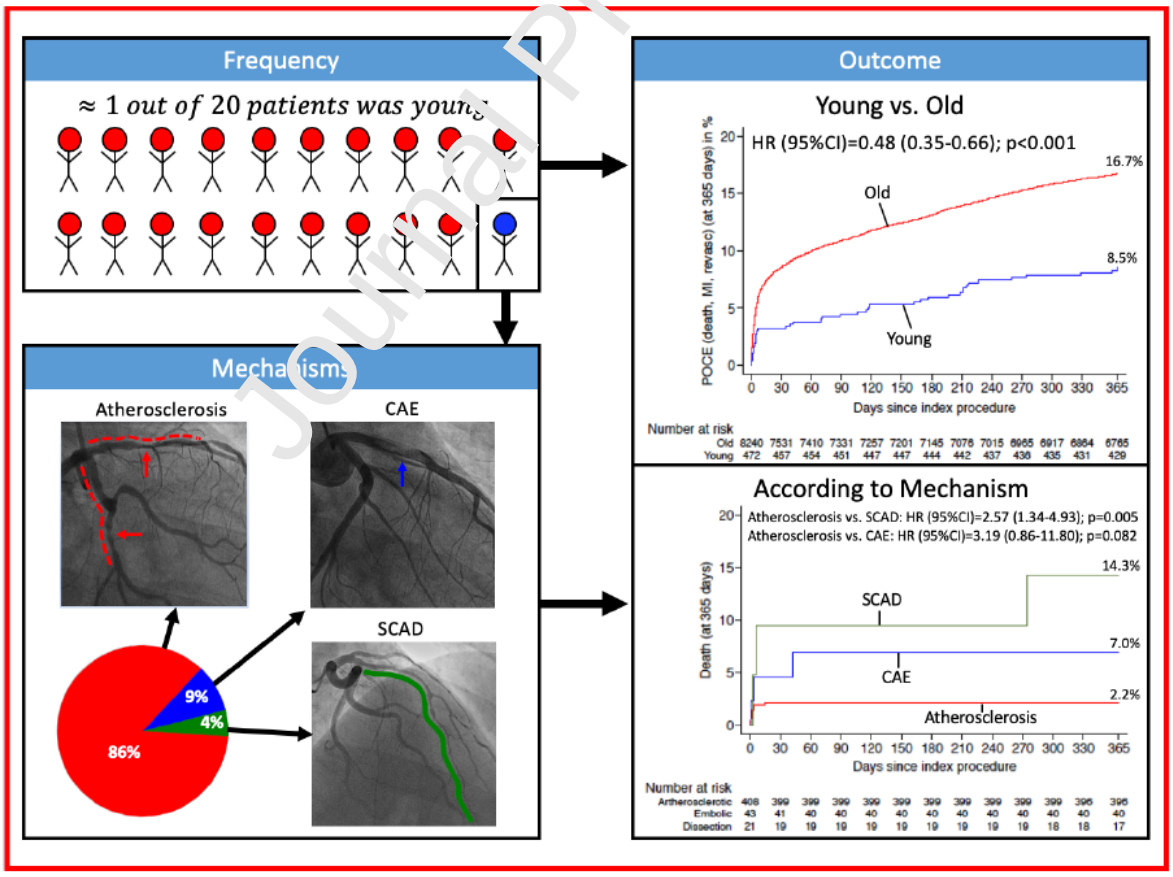
1) Substantial contributions to the conception or design of the work; or the acquisition, analysis, or interpretation of data for the work; AND

2) Drafting the work or revising it critically for important intellectual content; AND

3) Final approval of the version to be published; AND

4) Agreement to be accountable for all aspects of the work in ensuring that questions related to the accuracy or integrity of any part of the work are appropriately investigated and resolved.

Graphical abstract



Highlights

- Approximately one out of 20 ACS patients undergoing PCI for ACS was young (female <50 years, male <45 years).
- The principal mechanism of ACS in young patients was atherosclerosis (86.5%), followed by coronary artery embolism (CAE) (9%), and spontaneous coronary artery dissection (SCAD) (4.5%).
- As compared to older patients, young patients had a lower risk for the primary endpoint POCE and for its individual components at 12 months.