

Edition 2025

CLINICAL DECISION MAKING TOOLKIT

Instant guidance for diagnosis, risk stratification and management







The Clinical Decision Making Toolkit

is produced by the Association for Acute CardioVascular Care (ACVC) of the European Society of Cardiology (ESC).

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The Association for Acute CardioVascular Care Clinical Decision-Making TOOLKIT

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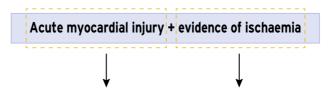
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ACUTE CORONARY SYNDROMES

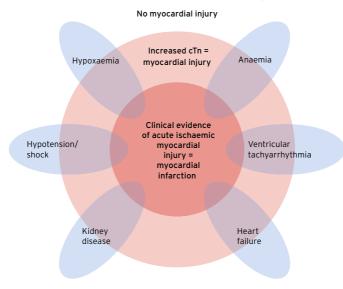
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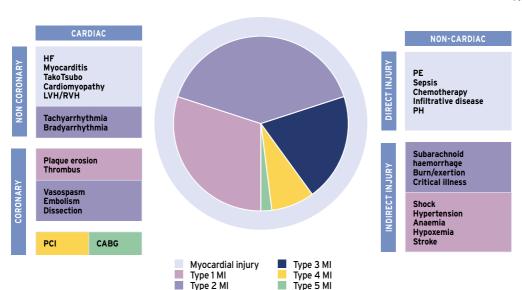
Rise and/or fall cTn (at least 1 value ≥ 99th% URL)

- Symptoms or
- ECG changes or
- Wall motion abnormalities (TTE)

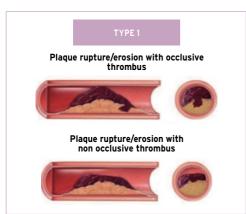
MI definition: pathophysiology

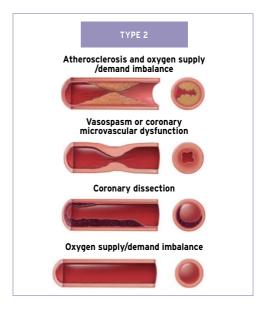


Myocardial injury: aetiology



MI definition: classification





TYPE 3

Cardiac death AND symptoms of ischaemia or MI detected by autopsy

Accompanied by new ischaemic ECG changes or ventricular fibrillation

TYPE 4

4a MI: PCI-related MI (≤48h after)

Normal pre-PCI cTn values: 1x5 p99th URL.

Elevated pre-PCI cTn: x5 99th% URL + rise >20%

4b MI: stent/scaffold thrombosis

4c MI: stent stenosis

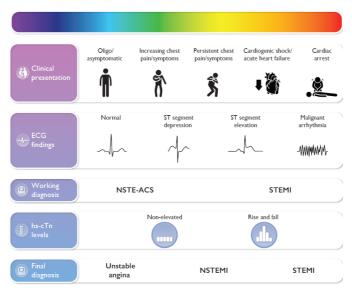
TYPE 5

MI associated with CABG (≤48h after)

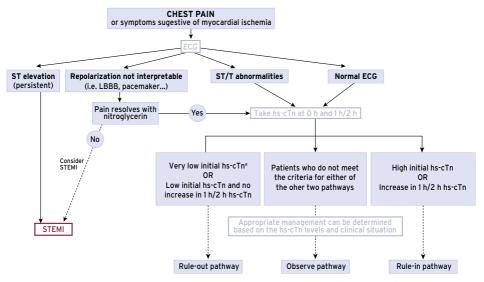
Normal pre- CABG cTn values: 1x10 p99th URL.

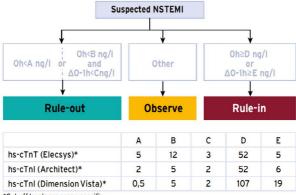
Elevated pre-CABG cTn: 1x10 p99th URL + 1 >20%

THE ACUTE CORONARY SYNDROMES SPECTRUM



2023 ESC Guidelines for the management of acute coronary syndromes European Heart Journal, Volume 44, Issue 38, 7 October 2023, Pages 3720-3826 Figure 2



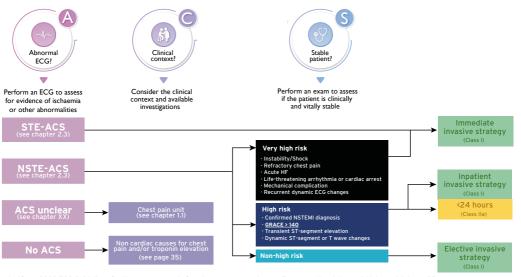


^{*}Cut-off levels are assay-specific.

- NSTEMI can be ruled-out at presentation, if hs-cTn concentration is very low
- NSTEMI can be ruled out by the combination of low baseline levels and the lack of a relevant increase within 1h
- NSTEMI is highly likely if initial hs-cTn concentration is at least moderately elevated or hs-cTn concentrations show a clear rise within the first hour

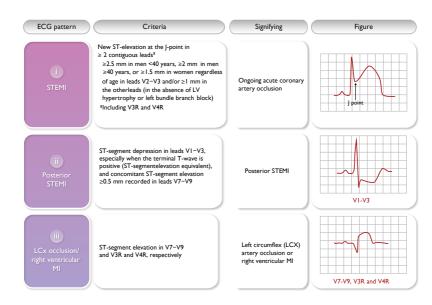
Causes of repolarisation abnormalities in the ECG not related to ACS

ST-segment elev	Negative T waves	
Fixed LV aneurysm LBBB, WPW, hypertrophic cardiomyopathy, Le Pacemaker stimulation Early repolarisation (elevated J-point) Dynamic Acute (myo)pericarditis Pulmonary embolism Electrolyte disturbances (hyperkalemia) Acute brain damage (stroke, subarachnoid he TakoTsubo syndrome	Normal variants, i.e. women (right precordial leads), children, teenagers Evolutive changes post myocardial infarction Chronic ischemic heart disease Acute (myo)pericarditis, cardiomyopathies BBB, LVH, WPW Post-tachycardia or pacemaker stimulation Metabolic or ionic disturbances	
ST-segment depre	Prominent T waves	
Fixed Abnormal QRS (LBBB, WPW, pacemaker stine LVH, hypertrophic cardiomyopathy Chronic ischemic heart disease Dynamic Acute (myo)pericarditis Acute pulmonary hypertension Electrolyte disturbances (hyperkalemia) Intermitent LBBB, WPW, pacing Post-tachycardia / cardioversion	Severe hypertensive crisis Drug effects (digoxin) Shock, pancreatitis Hyperventilation TakoTsubo syndrome	Normal variants, i.e. early repolarisation Metabolic or ionic disturbances (i.e. hyperkalemia) Acute neurological damage (stroke, subarachnoid haemorrhage)

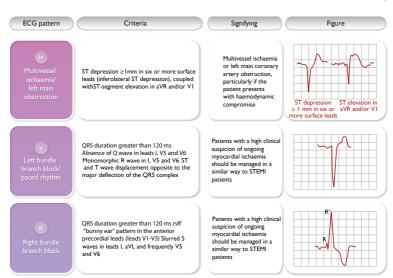


Adapted from 2023 ESC Guidelines for the management of acute coronary syndromes European Heart Journal, Volume 44, Issue 38, 7 October 2023, Pages 3720-3826 Figure 5

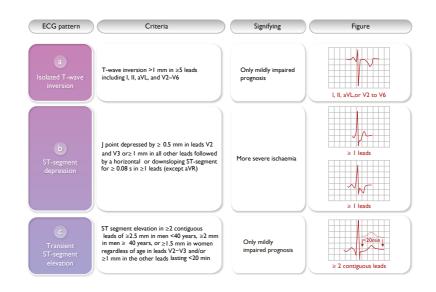
HIGH RISK FOR ACUTE CORONARY SYNDROME ECG PATTERNS(1)



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Adapted from 2023 ESC Guidelines for the management of acute coronary syndromes European Heart Journal, Volume 44, Issue 38, 7 October 2023, Pages 3720-3826 Figure S2



HIGH RISK FOR ACUTE CORONARY SYNDROME ECG PATTERNS(4)

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I-3 mm upsloping ST-segment depression at the J point in leads VI-V6 that continue into tall, positive, and symmetrical T waves

Proximal LAD occlusion/ severe stenosis



e Wellens sign

Isoelectric or minimally elevated J point (<1 mm)

biphasic T wave $% \left(1\right) =\left(1\right) \left(1\right) =\left(1\right) \left(1\right) \left(1\right)$ or $\left(1\right) \left(1\right) \left($

Proximal LAD occlusion/ severe stenosis

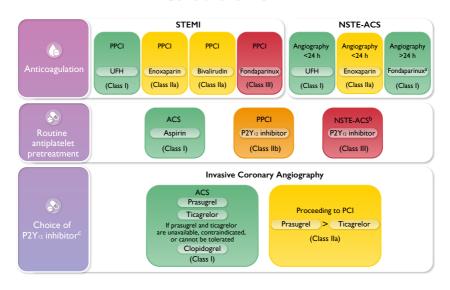


Adapted from 2023 ESC Guidelines for the management of acute coronary syndromes European Heart Journal, Volume 44, Issue 38, 7 October 2023, Pages 3720-3826 Figure S3

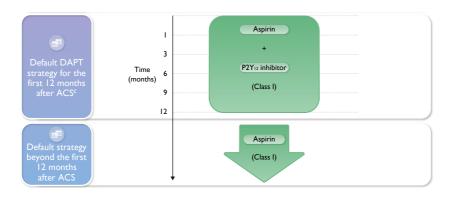
ACUTE CORONARY SYNDROMES: Differential Diagnosis

	1.12
Causes of chest pain Not related to ACS	Causes of troponin elevation Not related to ACS
Primary cardiovascular Acute pericarditis, pericardial effusion Acute myocarditis Severe hypertensive crisis Stress cardiomyopathy (TakoTsubo syndrome) Hypertrophic cardiomyopathy, aortic stenosis Severe acute heart failure Acute aortic syndrome (dissection, hematoma) Pulmonary embolism, pulmonary infarction Cardiac contusion	Primary cardiovascular Acute myo(peri)carditis Severe hypertensive crisis Pulmonary edema or severe congestive heart failure Stress cardiomyopathy (TakoTsubo syndrome) Post- tachy- or bradyarrhythmias Cardiac contusion or cardiac procedures (ablation, cardioversion, or endomyocardial biopsy) Aortic dissection, aortic valve disease or hypertrophic cardiomyopathy Pulmonary embolism, severe pulmonary hypertension
Primary non-cardiovascular Oesophageal spasm, oesophagitis, Gastro Esophageal Reflux (GER) Peptic ulcer disease, cholecystitis, pancreatitis Pneumonia, bronchitis, asthma attack Pleuritis, pleural effusion, pneumothorax Thoracic trauma Costochondritis, rib fracture Cervical / thoracic vertebral or discal damage Herpes Zoster	Primary non-cardiovascular Renal dysfunction (acute or chronic) Critical illness (sepsis, repiratory failure) Acute neurological damage (i.e. stroke, subarachnoid hemorrhage) Severe burns (affecting >30% of body surface area) Rhabdomyolysis Drug toxicity (chemotherapy with adriamycin, 5-fluorouracil, herceptin, snake venoms) Inflammatory or degenerative muscle diseases Hypothyroidism Infiltrative diseases (amyloidosis, hemochromatosis, sarcoidosis) Scleroderma

NSTE-ACS: Treatment (1) General overview

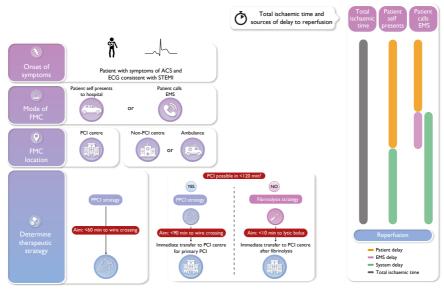


NSTE-ACS: Treatment (2) General overview



 $2023\,ESC\,Guidelines\,for\,the\,management\,of\,acute\,coronary\,syndromes\,European\,Heart\,Journal,\,Volume\,44,\,Issue\,38,\,7\,October\,2023,\,Pages\,3720-3826\,Figure\,10$

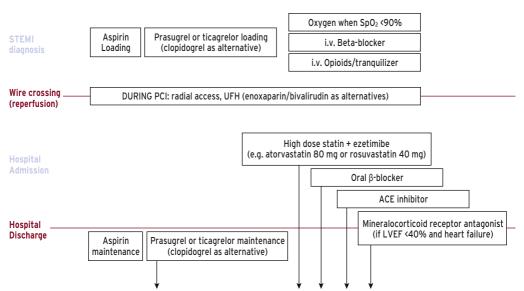
Timing and treatment of choice in STEMI



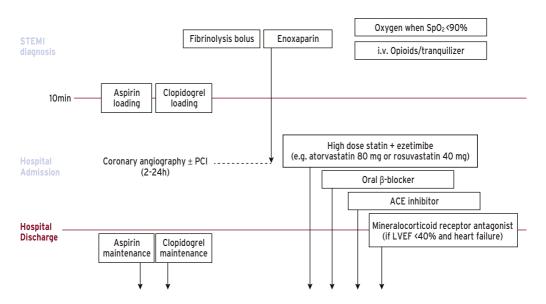
2023 ESC Guidelines for the management of acute coronary syndromes European Heart Journal, Volume 44, Issue 38, 7 October 2023, Pages 3720-3826 Figure 7

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STEMI Treatment (1): Medical management of patients treated with primary PCI



STEMI Treatment (2): Medical management of patients treated with fibrinolysis

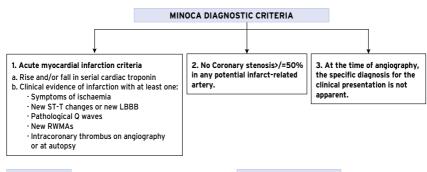


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MINOCA is a working (often transitionary) diagnosis that needs further evaluation in a patient presenting with symptoms suggestive of ACS.

It is an umbrella term encompassing a differential diagnosis of cardiac (coronary and non-coronary) and non-cardiac causes.



EPIDEMIOLOGY

MINOCA remains under-diagnosed and under-treated 1-14% of all patients presenting with an ACS that undergo angiography.

10-15% females and 3-4% males presenting with ACS.

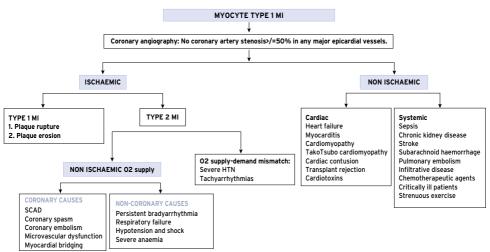
MORBIDITY & MORTALITY

When matched for age and sex with healthy individuals, MINOCA patients have worse survival and a substantial risk of repeated events.

5% 1-year mortality and 11% 5-year mortality

The 4-year rate of major adverse cardiac events after MINOCA is 25%.

MYOCYTE INJURY



The MINOCA diagnostic algorithm



- · Good history taking and physical examination is key
- · ECG assessment
- · Invasive coronary angiogram +/-LV gram (Takutsubo and other cardiomyopathies)
- · Intracoronary imaging with OCT (may be considered if plaque rupture/erosion or disection is suspected.)
- Functional assessment of coronary microvascular dysfunction +/-vasoreactivity may be considered if no cause identified.

Wards

- · Blood tests including inflammatory markers, serial troponins, thrombophilia screen (coronary embolus) and consider viral serology (myocarditis)
- · Echocardiography +/- bubble study
- · Early Cardiac MRI if diagnosis still uncertain
- \cdot If no OCT done acutely, consider CTCA to look for atherosclerotic disease
- · CTPA/CT brain
- · Consider Cardiac PET for the assessment of CMD
- Ambulatory ECG monitoring (screening of Paroxysmal atrial fibrillation or vasospasm)

Individualised care

- · Management as per final diagnosis
- · Consider repeat cardiac MRI
- · Cardiac Rehabilitation
- · Mental health well-being and psychosocial support
- · Long term care F/U clinic evaluation.

DEFINITION

- Acute non-iatrogenic and non-traumatic separation of the coronary artery wall creating a false lumen, causing external compression of the true lumen and compromising coronary flow.
- Presenting as an Acute Coronary Syndrome, Myocardial infarction, Ventricular Arrhythmias or Sudden Cardiac Death.

EPIDEMIOLOGY

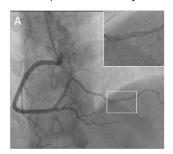
- · Up to ~ 90% of patients are women between 47-53 years of age.
- Up to ~ 35% of MIs in women </=50 years of age.
 Pregnancy related SCAD (P-SCAD): 43% of acute myocardial
- infarctions related to pregnancy (predominantly post partum).
- · Generally older women, at first childbirth and multigravidas.
- · During pregnancy: peak of events in the 3° trimester.
- \cdot After pregnancy: Peaks in the first 4 weeks after delivery.

RISK FACTORS AND PRECIPITATING TRIGGERS

- · Female sex
- · Pregnancy, multiparity
- · Hypertension
- · Migraine
- · Pulsatile tinnitus
- Fibromuscular dysplasia (both focal and multifocal) and EVAs*
- Connective tissue disorders and inherited arteriopathy (Vascular Ehlers-Danlos, Marfan's, Loeys-Dietz syndromes)
- Susceptibility genes associated with connective tissue function and tissue clotting.
- · Adult polycystic Kidney disease.
- · Perimenopausal
- · Increased emotional stress
- \cdot Extreme physical and isometric exercise.

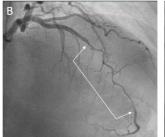
SACD: Angiographic classification (1)

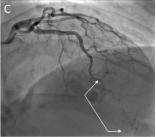
- · SCAD typically occurs in the mid-to-distal segments of coronary arteries.
- · The most affected vessel is the Left anterior descending artery.
- · 10 to 15% of patients with SCAD have multivessel involvement.
- · OCT helps confirm the diagnosis of SCAD.



Type 1 SCAD

- Multiple radiolucent lumens
- \cdot Contrast staining of the arterial wall
- Slow clearing of contrast or dye hang-up





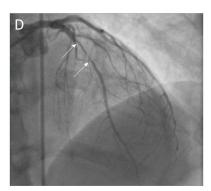
Type 2 SCAD: The most common type (60-75% of SCAD patients)
Diffuse (typically >20 mm) smooth narrowing which appears as a sudden change in caliber of the artery. No response to intracoronary nitroglycerin and absence of atherosclerosis in other vessels.

Type 2A: Dissected artery between proximal and distal segments of normal caliber.

Type 2B: dissection which extends to the tip of the artery.

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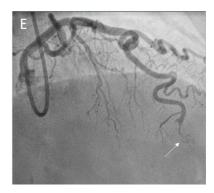
SACD: Angiographic classification (2)



Type 3 SCAD: Hazy, linear, focal or tubular stenosis which mimics atherosclerosis and therefore difficult to diagnose. OCT to show the presence of intramural hematoma when diagnosis uncertain.

To be suspected when:

- · Absence of atherosclerosis in other coronary arteries.
- · Coronary tortuosity.

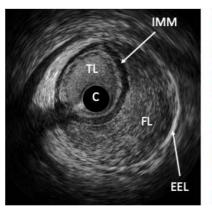


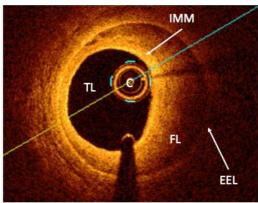
Type 4 SCAD: Abrupt complete occlusion of the vessel, usually involving the distal segment.

The dissection may be detected after the recanalization of the vessel or after a repeat coronary angiography shows healing of the vessel.

SCAD: Intracoronary imaging

Intracoronary imaging is essential when diagnosis is uncertain as diagnosis is critical in both the acute and long-term management of SCAD.





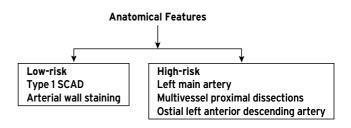
Classical Intracoronary imaging appearances in SCAD (left intravascular ultrasound, Right optical coherence tomography): C = imaging catheter, TL = true lumen, FL = False lumen IMM = intimal-medial membrane, EEL - external elastic lamina.

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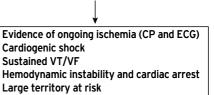
Management of SCAD

CONSERVATIVE MANAGEMENT

- Conservative therapy is the first line unless ongoing high-risk clinical features, as SCAD heals spontaneously and known poor outcome with PCI.
- Prolonged Inpatient stay >/= 5 days as there is a risk of extension.



High-Risk Clinical Features



Management of SCAD

REVASCULARIZATION

A very small proportion require revascularization in the presence of high-risk clinical features.

PCI

- · Challenging for several reasons: Rate of complications > 30%
- · Additional technical challenges due to increase tortuosity
- · Risk of secondary latrogenic dissection
- · Multiple stents for long dissections
- · Coronary wire positioning in the coronary false lumen
- Hematoma propagation (loss of distal-vessel patency or retrograde extension to more proximal vessels)
- Risk of malapposition in the long period as a result of intramural hematoma reabsorption. However, stent thrombosis seems rare.
- Extension of dissection into smaller distal vessels, which are difficult to stent.

CABG

- · Rarely needed (0.7% in CanSCAD study)
- Bail-out strategy in very high-risk scenarios and failed PCI.
- The goal is to achieve survival, so high-capacity SVG is safe in this setting.

Antiplatelet and anticoagulant therapy in SCAD

			LACK OF CONSENSUS ON THE USE AND DURATION OF DAPT-Clinical trials have now begun. CURRENT GUIDELINES: DAPT for 1 year and lifelong aspirin.	
CONSERVATIVE MANAGEMENT (no PCI)		ANTIPLATELET THERAPY	Some experts advise: Limit or avoid the use of early or prolonged DAPT (lack of evidence for beneficial effect, rare intracoronary thrombus, risk of bleeding within the intramural haematoma causing dissection extension, etc). Example: DAPT for 2-4 weeks after SCAD and then continue low-dose aspirin alone for a total of 3-12 months Aspirin and Clopidogrel rather than the other P2Y12 inhibitors and avoid to administer intravenous antiplatelet therapies In patients at higher risk of bleeding it is possible to consider aspirin alone or no antiplatelet therapy. Longer-duration of antiplatelet therapy depends on the presence of other comorbidities (FMD or other dissections where low-dose of aspirin is considered "reasonable"	
8		ANTICOAGULANTS	GENERAL APPROACH: To discontinue systemic anticoagulation when SCAD is diagnosed with the exception of situations when it is beneficial unequivocally (example: LV thrombus, thromboembolism).	
INTERVENTIONAL MANAGEMENT	PCI	ANTIPLATELET THERAPY	DAPT based on ACS guidelines.	
		ANTICOAGULANTS	Limit systemic anticoagulation to during revascularization procedures when SCAD is diagnosed with the exception of situations when it is beneficial unequivocally (examples: LV thrombus, thromboembolism). Risk-benefit is unknown with GP2b3a inhibitors, but luminal thrombus is uncommon.	
INTER/ MAN/	THROMBS	CONTRAINDICATED (reports of dissection extension and coronary rupture)		

SCAD: Long term management

SCAD recurrence prevention

- · Beta-blockers-Clinical trails have now started to look at the benefit of beta blockers in SCAD.
- · Well controlled blood pressure
- · Advice on physical activity post-SCAD
 - Pursue regular, moderate physical activity avoiding extreme endurance training, exercising to exhaustion, or exertion in extreme temperature conditions.
 - · To avoid heavy objects lifting (prolonged Valsalva).
- · Individualised advise on HRT and contraception.

Managing post-SCAD chest pain

- Counselling: Advise that this usually improves over 18 months-2 years, but can be worse and prolonged in patients with previous pain syndromes, psychological and psychiatric disorders.
- Trial of antianginal pharmacologic therapy (Ranolazine, Ca-antagonist, etc) to relieve ischaemic symptoms from presumed coronary vasospasm or coronary microvascular dysfunction.

Can be considered but variable and often limited response to treatment. Avoid multiple anti-anginals unless there is a definite treatment effect

Assessment of associated extra coronary vascular abnormalities

- CT angiogram or magnetic resonance angiogram from brain to pelvis.
- F/U coronary angiography is rarely indicated. CT coronary angiography may be considered in selected cases.

Quality of life

- · Cardiac rehabilitation
- · Heart failure therapy (in case of LV significant impairment)
- Migraine therapy
- Psychosocial/mental health treatment (drug therapy, psychological therapy, support groups)

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References and copyright acknowledgments

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Abbreviations

APTT = Activated partial thromboplastin time

AB = Airway and breathing

ABG = Arterial blood gas

AADs = Antiarrhythmic drugs

AAS = Acute aortic syndrome

ACEI = Angiotensin converting enzyme inhibitor

ACLS = Advanced cardiovascular life support

ACS = Acute coronary syndrome

ACT = Activated clotting time

AD = Aortic Dissection

AED = Automated external defibrillator

AF = Atrial fibrillation

ANA = Antinuclear antibodies

Ao = Aortic

aPTT = Activated partial thromboplastin time

ARB = Angiotensin receptor blockers

AS = Aortic stenosis

AV = Atrioventricular

AVB = Atrioventricular conduction block

AVN = Atrioventricular node

AVNRT = Atrioventricular nodal re-entrant

tachycardia

AVNT = Atrioventricular nodal tachycardia

BID = Twice a day

BBB = Bundle branch block

BLS = Basic life support

BNP = Brain natriuretic peptide

BP = Blood pressure

CABG = Coronary artery bypass grafting

CAD = Coronary artery disease

Cath Lab = Catheterisation laboratory

CCB = Calcium channel blockers

CCU = Coronary care unit

CHF = Congestive heart failure

CMR = Cardiovascular magnetic resonance

COPD = Chronic obstructive pulmonary disease

CPAP = Continuous positive airway pressure

 ${\bf CPR} = {\bf Cardiopulmonary} \ {\bf resuscitation}$

Cr = Creatinine blood level (mg/dL)

CrCl = Creatinine clearance

CRP = C-reactive protein

CS = Cardiogenic shock

CSM = Carotid sinus massage

CSNRT = Corrected sinus node recovery time

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Abbreviations (Cont.)

CSS = Carotid sinus syndrome
CT = Computed tomography

CT-angio = Computed tomography angiography

cTn = Cardiac troponin

CUS = Compression venous ultrasound

CV = Cardiovascular

CVA = Cerebrovascular accident

CXR = Chest X-ray

DAPT = Dual antiplatelet therapy

DD = Dyastolic dysfunction **DM** = Diabetes mellitus

dTT = Diluted thrombin time

DVT = Deep vein thrombosis

ECG = Electrocardiogram

Echo = Echocardiogram

ECMO = Extracorporeal membrane oxygenation

ECT = Ecarin clotting time

ED = Emergency department

EF = Ejection fraction **EG** = Electrograms

eGFR = Estimated glomerular filtration rate

 $(mI/min/1.73 m^2)$

EMB = Endomyocardial biopsy

EMS = Emergency medical services

EPS = Electrophysiological study

ERC = European Resuscitation Council ESR = Erythrocyte sedimentation rate

ETT = Exercice treadmill testing

FFP = Fresh frozen plasma

FMC = First medical contact

GER = Gastroesophageal reflux

GFR = Glomerular flow rate

GI = Gastrointestinal

GP = Glycoprotein **Hb** = Haemoglobin

HF = Heart failure

HIT = Heparin-induced thrombocytopenia

HOCM = Hypertrophic obstructive cardiomyopathy

HTN = Hypertension

HR = Heart rate

hsTn = High-sensitive troponin

IABP = Intra-aortic balloon pump

ICC = Intensive cardiac care

ICCU = Intensive cardiac care unit

Abbreviations (Cont.)

ICD = Implantable cardioverter defibrillator

ICI = Immune checkpoint inhibitors

IHD = Ischemic heart disease

IMH = Intramural hematoma

IRF = Immediate-release formulation

ISFC = International Society and Federation

of Cardiology

i.o. = Intraosseous

IV = Invasive ventilation

i.v. = Intravenous

KD = Kidney disease

LBBB = Left bundle branch block

LD = Loading dose

LGE = Late gadolinium enhancement LMWH = Low-molecular weight heparin

LOC = Loss of consciousness

LV = Left ventricular

LVAD/Bi-AD = left ventricular, bi-ventricular assist

LVD = Left ventricular dysfunction

LVEF = Left ventricular ejection fraction

LVH = Left ventricular hypertrophy

LVSD = Left ventricular systolic dysfunction

MCS = Mechanical circulatory support

MD = Maintenance dose

MDCT = Computed tomography with >4 elements

MI = Myocardial infarction

MRA = Mineralocorticoid receptor antagonist

MRI = Magnetic resonance imaging
Mvo = Microvascular obstruction

NIV = Non-invasive ventilation

NOAC = New oral anticoagulants

NSAID = Non-steroidal anti-inflammatory drugs NSVT = Non-sustained ventricular tachycardia

or recurrent

NSTE-ACS = Non ST-segment elevation

acute coronary syndrome

NSTEMI = Non ST-segment elevation myocardial

infarction

NTG = Nitroglycerin

NT-proBNP = N-terminal pro brain natriuretic

peptide

NVAF = Non-valvular atrial fibrillation

NYHA = New York Heart Association

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OH = Orthostatic hypotension

PAP = Pulmonary arterial pressure

PAU = Penetrating aortic ulcer

PCI = Percutaneous coronary intervention

PCM = Physical counter-measures

PCP = Pulmonary capillary pressure

PE = Pulmonary embolism

PEA = Pulmonary endarterectomy

PEEP = Positive end expiratory pressure

PPC = Prothrombin complex concentrate

PR = Pulmonary regurgitation

PRECISE-DAPT = PREdicting bleeding

Complications In patients undergoing Stent implantation and subsEquent Dual Anti Platelet

Therapy

PRF = Prolonged-release formulation

ProCT = Procalcitonin

PRN = Pro re nata

PS-PEEP = Pressure support-positive endexpiratory pressure

PSVT = Paroxysmal supraventricular tachycardia

QD = Once a day

QPM = Every evening

rFVIIa = Recombinant factor VIIa

rtPA = Recombinant tissue plasminogen activator

RV = Right ventricular

RVOT-VT = Right ventricular outflow tract

ventricular tachycardia

SBP = Systemic blood pressure

s.c = Subcutaneous

SIRS = Systemic inflammatory response syndrome

SLE = Systemic lupus erythematosus

SMU = Syncope management units

STE-ACS = ST-segment elevation acute

coronary syndrome

STEMI = ST-segment elevation myocardial infarction

SVT = Supraventricular tachycardia

Spo₂ = Oxygen saturation

TEE = Transesophageal echocardiography

TEVAR = Thoracic endovascular aortic repair

TIA = Transient ischemic attack

TID = Three times a day

TLOC = Transient loss of consciousness

TOE = Transoesopageal echocardiography

Abbreviations (Cont.)

TSH = Thyroid-stimulating hormone

TTE = Transthoracic echocardiography

UA = Unstable angina

UFH = Unfractionated heparin

ULN = Upper limit of normal

VBGA = venous blood gas analysis

VF = Ventricular fibrillation

VR = Vascular resistance

VT = Ventricular tachycardia

VTE = Venous thromboembolism

VVS = Vasovagal syncope

WBC = white blood cell count

WHO = World Health Organization

WPW = Wolff-Parkinson-White

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