Dissection coronaire spontanée: état des lieux

Sabine ZIGHOUD
cardiologue, BLIDA - ALGERIE

DÉCLARATION DE LIENS D'INTÉRÊT POTENTIELS

Intervenant : Sabine ZIGHOUD, Blida

☑ Je n'ai pas de lien d'intérêt potentiel à déclarer

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Cause de syndrome de coronarien aigu

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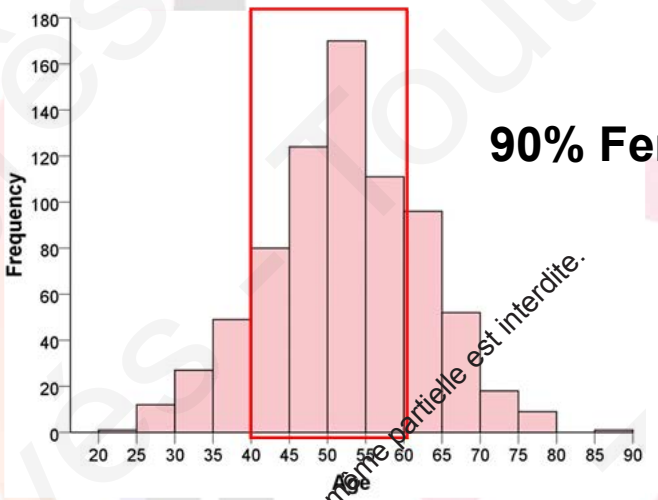
Table 1. Angiographic Prevalence of SCAD in ACS Cohorts

Reference	Year	Patients With SCAD, n	SCAD Prevalence as a Proportion of All ACS Cases, %	Women Among SCAD Cases, %	PA-SCAD, %	SCAD Prevalence in Subgroups With ACS	Methods, Population, Inclusion Criteria
Vanzetto et al[31]	2009	23	0.2 (0.6 women, 0.07 men)	74	0	8.7% SCAD among ACS in women ≤50 y	Systematic retrospective review of 11 605 angiograms Included type 1 SCAD only Atherosclerosis-related coronary dissection not excluded
Mortensen et al[30]	2009	22	2.0	77	2	NR	Retrospective search for coded diagnoses in database of 32 969 angiograms; reviewed only those with prior SCAD diagnosis
Alfonso and Bastante[14]	2014	27	0.16	85	3.7	NR	Retrospective search for coded diagnoses among 16 813 first angiograms (2004–2010)
Saw et al[15]	2014	16	NR	100	NR	24.2% SCAD among ACS in women ≤50 y	Retrospective review of 177 angiograms in women ≤50 y representing 9% of angiograms (n=7605) performed during the study period (2009–2011)
Rashid et al[16]	2016	21	1.7	95.2	NR	22.5% SCAD among ACS in women ≤60 y	Retrospective search for coded diagnoses among 1332 angiograms (2012–2013)
Nakashima et al[11]	2016	63	0.31	94	8.1	35% SCAD among ACS in women ≤50 y	Retrospective review of 20 195 angiograms (2000–2013) Excluded atherosclerosis-related coronary dissection Included type 2 SCAD Separate analysis for women ≤50 y with ACS (n=45)
Nishiguchi et al[32]	2016	13	4	53.8	NR	NR	326 Selected ACS patients undergoing OCT (2008–2012) Atherosclerosis-related coronary dissection not excluded

ACS indicates acute coronary syndrome; NR, not reported; OCT, optical coherence tomography; PA-SCAD, pregnancy-associated spontaneous coronary artery dissection; and SCAD, spontaneous coronary artery dissection.

Circulation
Volume 137, Issue 19, 8 May 2018; Pages e523-e557
https://doi.org/10.1161/CIR.0000000000000564

Figure 2 Histogram of age distribution.



Eur Heart J, Volume 40, Issue 15, 14 April 2019, Pages 1188–1197, https://doi.org/10.1093/eurheartj/ehz007

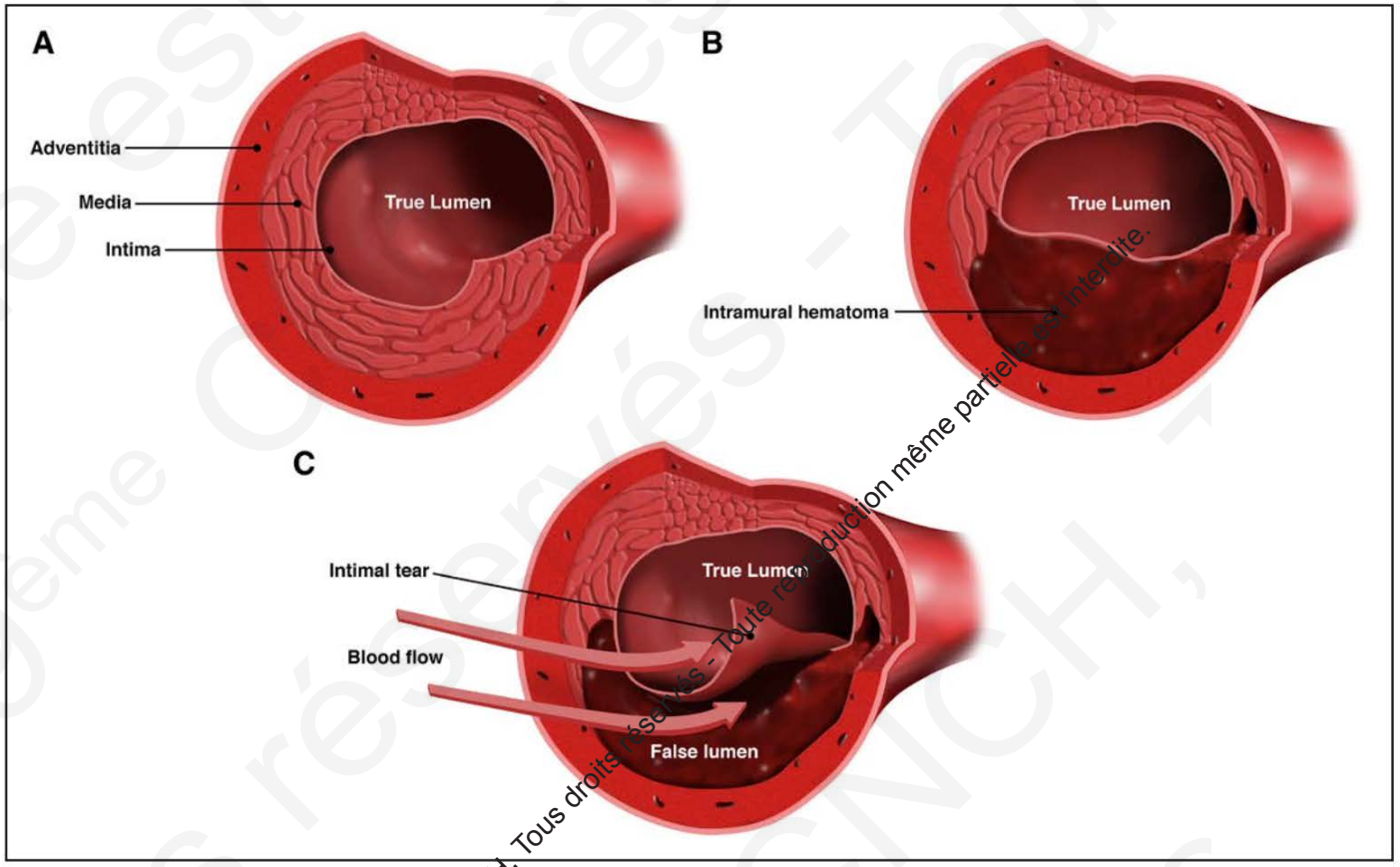
90% Femmes

- **1 - 4 % des SCA**
- **35% des SCA de la femme de moins de 50 ans**

Mécanisme

- **Inside - out**

- **Outside – in**



Acute development of a false lumen within the coronary artery wall which may compromise coronary flow by external compression of the true lumen. that is not associated with atherosclerosis or trauma and not iatrogenic.

ESC European Society of Cardiology
European Heart Journal (2018) **39**, 3353–3368
doi:10.1093/eurheartj/ehy080

Table I Demographics and risk factors of patients with spontaneous coronary artery dissection (SCAD) in contemporary case series (studies with n > 20)

	Max N	Age (years)	Gender (female, %)	HTN (%)	Chol (%)	Smoking (%)	DM (%)	FH (%)	P-SCAD (%)
Mayo Clinic[3]	189	44 ± 9	92	31	22	15	2	NA	15
Saw[4]	168	52 ± 9	92	39	24	13	5	29	2
Lettieri[5]	134	52 ± 11	81	51	33	34	2	25	NA
Faden[6]	79	33 ± 5	100	17	18	17	NA	NA	100
Rogowski[7]	64	53 ± 11	94	45	53	8	0	19	5
Nakashima[8]	63	46 ± 10	94	33	3	32	0	8	8
Motreff[3]	55	50	00	27	11	22	4	22	4
McGrath-Cadell[9]	40	45 ± 10	95	18	10	8	5	28	8
Rabou[10]	3	47 ± 12	94	NA	NA	NA	NA		15
Alfonso[11]	27	52 ± 10	85	37	33	52	4	NA	4
Ito[12]	23	45 ± 11	100	57	22	30	4	NA	30
Vanzetto[14]	23	46 ± 9	74	26	39	43	13	13	0
Mortensen[15]	22	49 ± 9	81	38	NA	57	0	40	10
Rashid[16]	21	53 ± 9	95	48	48	43	5	24	0

Data are given as mean ± standard deviation or percentages.
HTN, hypertension; Chol, dyslipidaemia; DM, diabetes mellitus; FH, family history of coronary artery disease; NA, not available; P-SCAD, pregnancy-associated coronary artery dissection.

Peu ou pas de FDR cardio vasculaires

Table 2. Conditions and Factors Associated With SCAD

Associated Condition or Factor	Reported Prevalence in Cohort Studies, %
Fibromuscular dysplasia	25–86[9,29,33,34]
Pregnancy	2–8[8,9,13,33]
Multiparity (≥4 births)	8.9–10[13,33]
Inherited arteriopathy and connective tissue disorder (see Table 4)	1.2–3.0[8,13]
Marfan syndrome, Loeys-Dietz syndrome, vascular Ehlers-Danlos syndrome, α1-antitrypsin deficiency, polycystic kidney disease	
Exogenous hormones	10.7–12.6[8,13]
Oral contraceptives, postmenopausal therapy, infertility treatments, testosterone, corticosteroids	
Systemic inflammatory disease	<1–8.9[9,13]
Systemic lupus erythematosus, Crohn disease, ulcerative colitis, polyarteritis nodosa, sarcoidosis, Churg-Strauss syndrome, Wegener granulomatosis, rheumatoid arthritis, Kawasaki disease, celiac disease	
Migraine headache	NR
Coronary artery spasm	NR

Circulation
Volume 137, Issue 19, 8 May 2018, Pages e523-e557

AHA SCIENTIFIC STATEMENT

Spontaneous Coronary Artery Dissection: Current State of the Science: A Scientific Statement From the American Heart Association

Circulation: Cardiovascular Interventions
Volume 7, Issue 5, October 2014; Pages 645-655
https://doi.org/10.1161/CIRCINTERVENTIONS.114.001760

CARDIAC CATHETERIZATION

Spontaneous Coronary Artery Dissection

Association With Predisposing Arteriopathies and Precipitating Stressors and Cardiovascular Outcomes

Precipitating factors	>50% Patients recall a precipitating factor
Intense exercise (isometric or aerobic)	
Intense Valsalva	
Retching, vomiting, bowel movement, coughing, lifting heavy objects	
Intense emotional stress	
Labor and delivery	
Recreational drugs (cocaine, methamphetamines)	
Exogenous hormones/hormone modulators	
β-hCG injections, corticosteroid injections, clomiphene	

hCG indicates human chorionic gonadotropin; NR, not reported; and SCAD, spontaneous coronary artery dissection.

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CURRENT OPINION
Coronary artery disease

La dysplasie fibromusculaire

Une maladie non athérosclérotique et non inflammatoire des parois artérielles,

Touche les femmes d'âge moyen, peu de facteurs de risque cardiovasculaire.

La dysplasie fibromusculaire peut entraîner une **sténose, des dissections et des anévrismes** des **artères de taille moyenne**, y compris, mais sans s'y limiter, les **artères rénales, cervicocéphaliques et viscérales.**

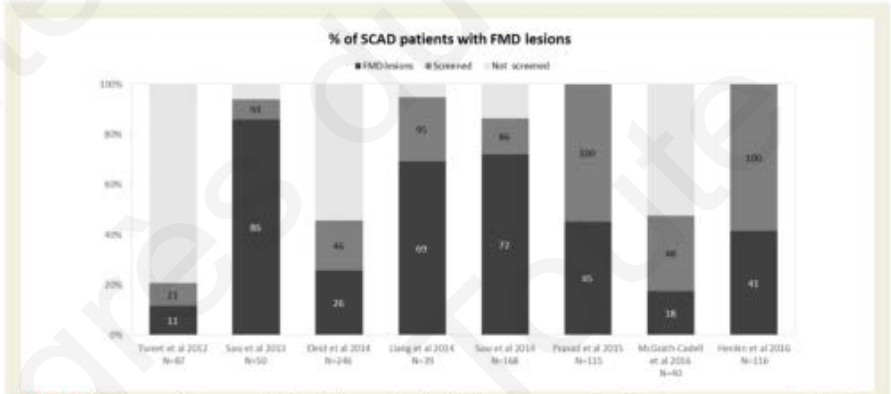

Figure 4 Prevalence of extracoronary lesions of fibromuscular dysplasia in contemporary series of spontaneous coronary artery dissection patients.

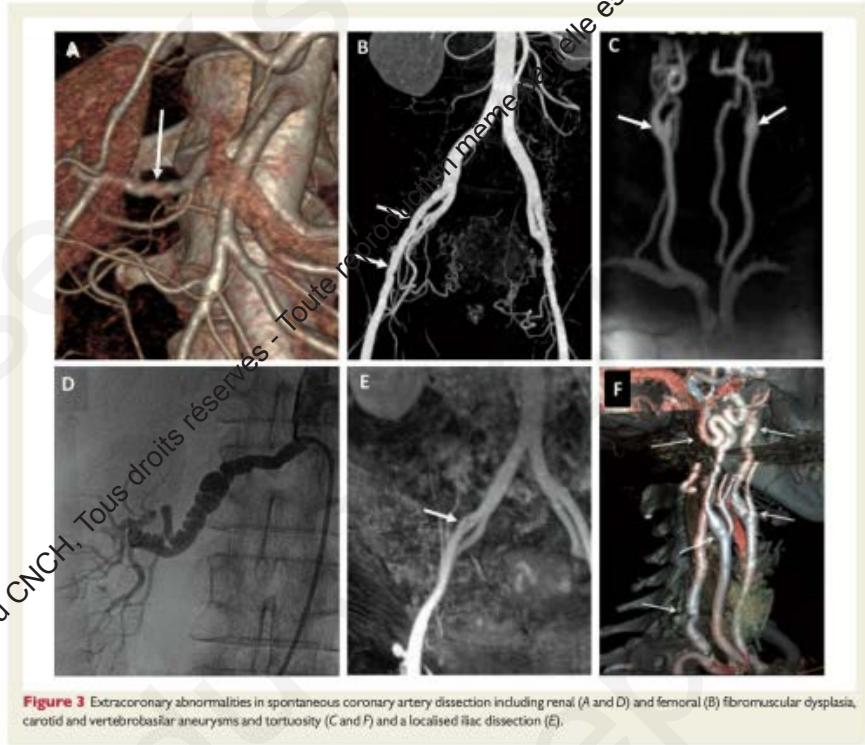

Figure 3 Extracoronary abnormalities in spontaneous coronary artery dissection including renal (*A* and *D*) and femoral (*B*) fibromuscular dysplasia, carotid and vertebrobasilar aneurysms and tortuosity (*C* and *F*) and a localised iliac dissection (*E*).

Grossesse / post partum et Dissection coronaire spontanée

- Le P-SCAD représente **5% à 17%** de l'ensemble des cas de SCAD
- **Première cause** d'IDM associé à la grossesse (14,5% à 43%)
- La majorité (>70%) survient dans le post-partum, le plus souvent au cours de la première semaine
- Facteurs prédisposons: multiparité , traitements antérieurs de la stérilité , pré-éclampsie

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- Des **présentations cliniques plus sévères** avec une altération de la fonction ventriculaire gauche, un choc, une dissection principale gauche et une dissection multi-vaisseaux

TABLE 2 Pregnancy-Associated Spontaneous Coronary Artery Dissection

First Author, Year (Ref. #)	N	Design	Mean ± Age at SCAD (yrs)	Mean Gravidity	Left Main SCAD (%)	Multivessel SCAD (%)	Cardiac Function
Higgins et al., 2013 (96)	47	Case series	33.5 ± 5.3	2.7	36	34	Hemodynamically unstable*: 21%
Havakuk et al., 2017 (32)	120	Case series	34 ± 4	—	36	40	LVEF <40%: 44%
Koller et al., 1998 (69)	43	Case series	33.5 ± 5.3	3.1	18	18	—
Koul et al., 2001 (31)	58	Case series	33	2.1	24	40	—
Tweet et al., 2017 (30)	54	Registry cohort	35 ± 4	3.2	24	33	LVEF <35%: 26%
Faden et al., 2016 (38)	79	Population-based cohort†	33 ± 5.2	—	—		Cardiogenic shock: 20%

*3 patients underwent cardiac transplantation in this series. †National Inpatient Sample administrative database

LVEF = left ventricular ejection fraction; SCAD = spontaneous coronary artery dissection.

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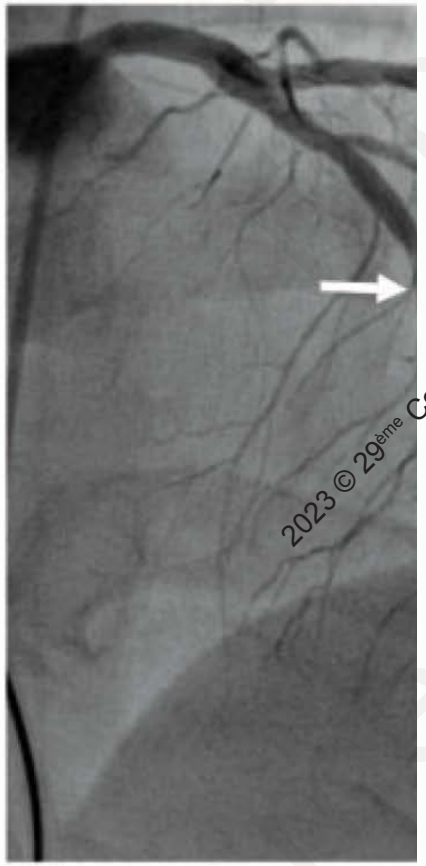
European Heart Journal (2019) **40**, 1188–1197
doi:10.1093/eurheartj/ehz007

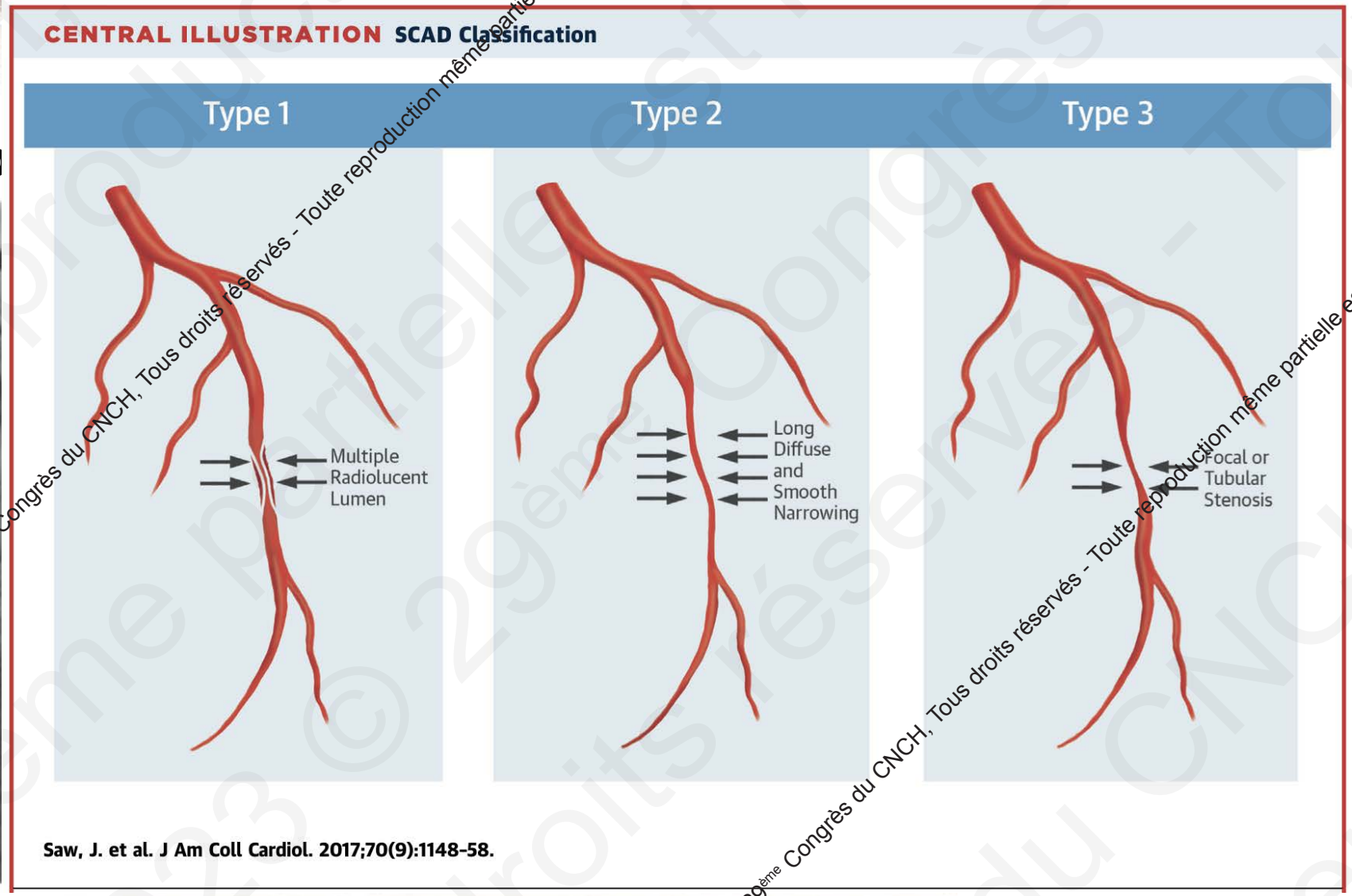
Table 2 Hospital presenting characteristics

Median (Q1–Q3) or *n* (%)	*N* = 750
Acute coronary syndrome	
STEMI	223 (29.7)
NSTEMI	524 (69.9)
Unstable angina	3 (0.4)
Presenting main symptom	
Chest discomfort	686 (91.5)
Back discomfort	15 (2.0)
Shoulder or arm discomfort	10 (1.3)
Dyspnoea	7 (0.9)
Arrhythmia	8 (1.1)
Other	24 (3.2)
Ventricular tachycardia or fibrillation	61 (8.1)
Left ventricular function assessment	
Ejection fraction assessed	737 (98.2)
Angiogram	491 (65.5)
Echocardiogram	243 (32.4)
Initial ejection fraction (%)	55 (50–60)
Ejection fraction <50%	188/734 (25.6)
Ejection fraction <35%	28/734 (3.8)

Classification des Dissections coronaires spontanées

Typ





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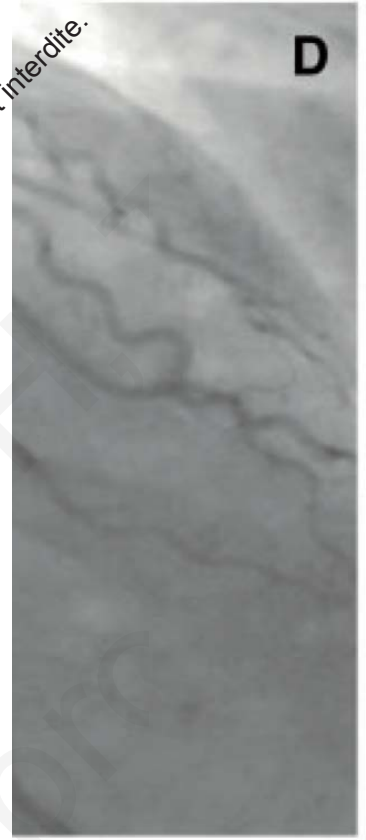

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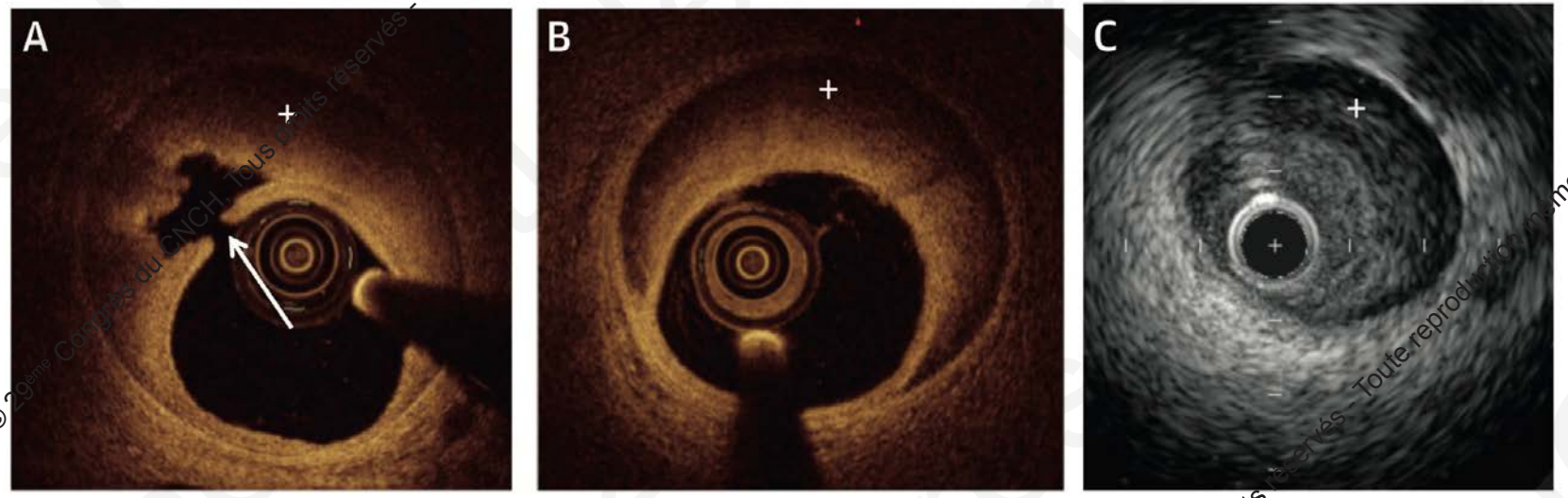
Imagerie endo coronaire

FIGURE 5 Intracoronary Imaging of SCAD

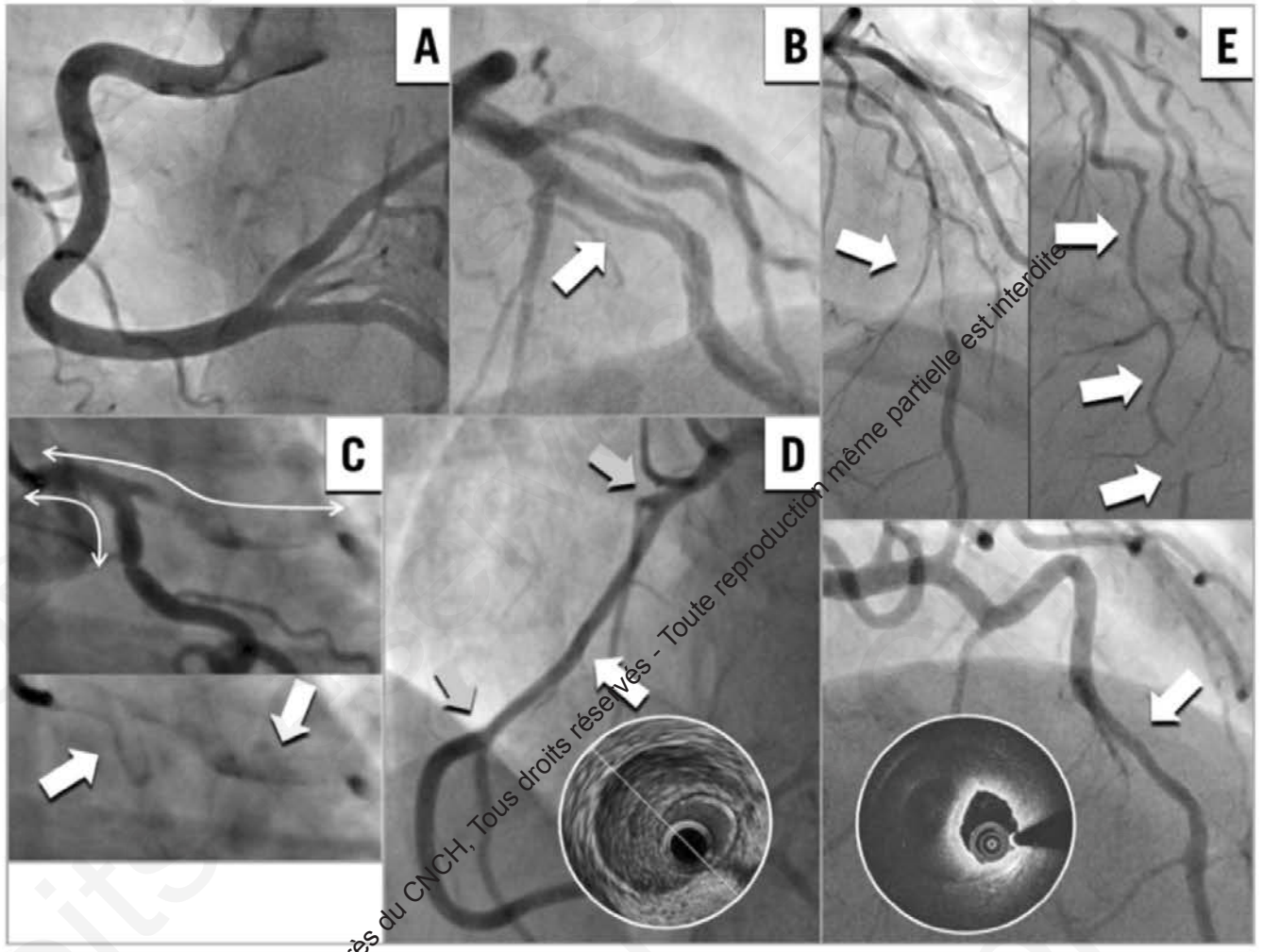


(A) OCT image showing false lumen with intramural hematoma (IMH) **(plus sign)** and intimal rupture **(arrow)**. **(B)** OCT image showing false lumen with IMH **(plus sign)**. **(C)** IVUS image showing false lumen with IMH **(plus sign)**. Abbreviations as in Figures 1 and 4.

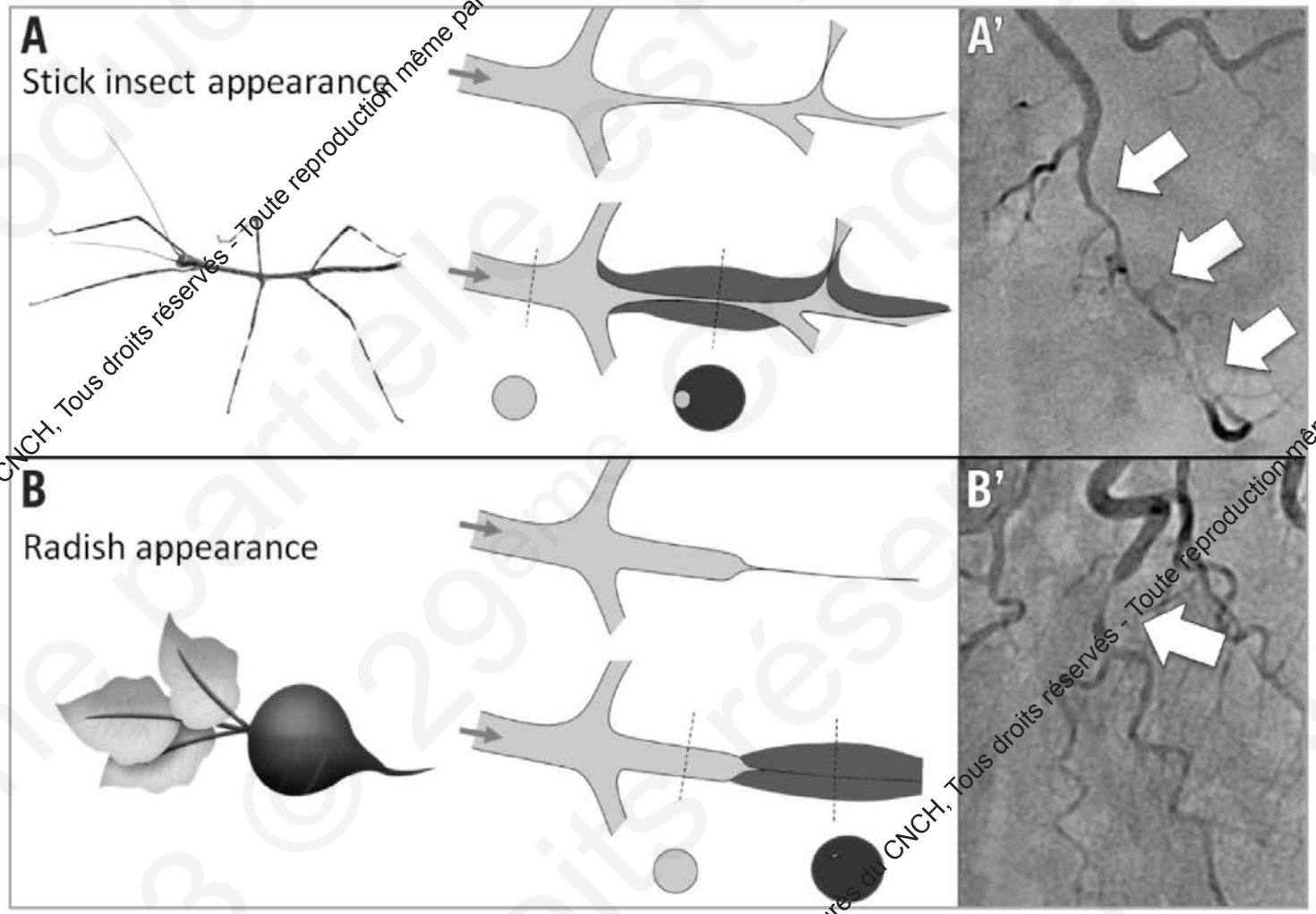
How and when to suspect spontaneous coronary artery dissection: novel insights from a single-centre series on prevalence and angiographic appearance

5 signes angiographiques

A. Absence d'athérome
B. Flap intimal
C. Tatouage du contraste
D. Début et/ou fin sur collatérales
E. Réduction longue et lisse du calibre



Motreff P, Eurointervention 2017



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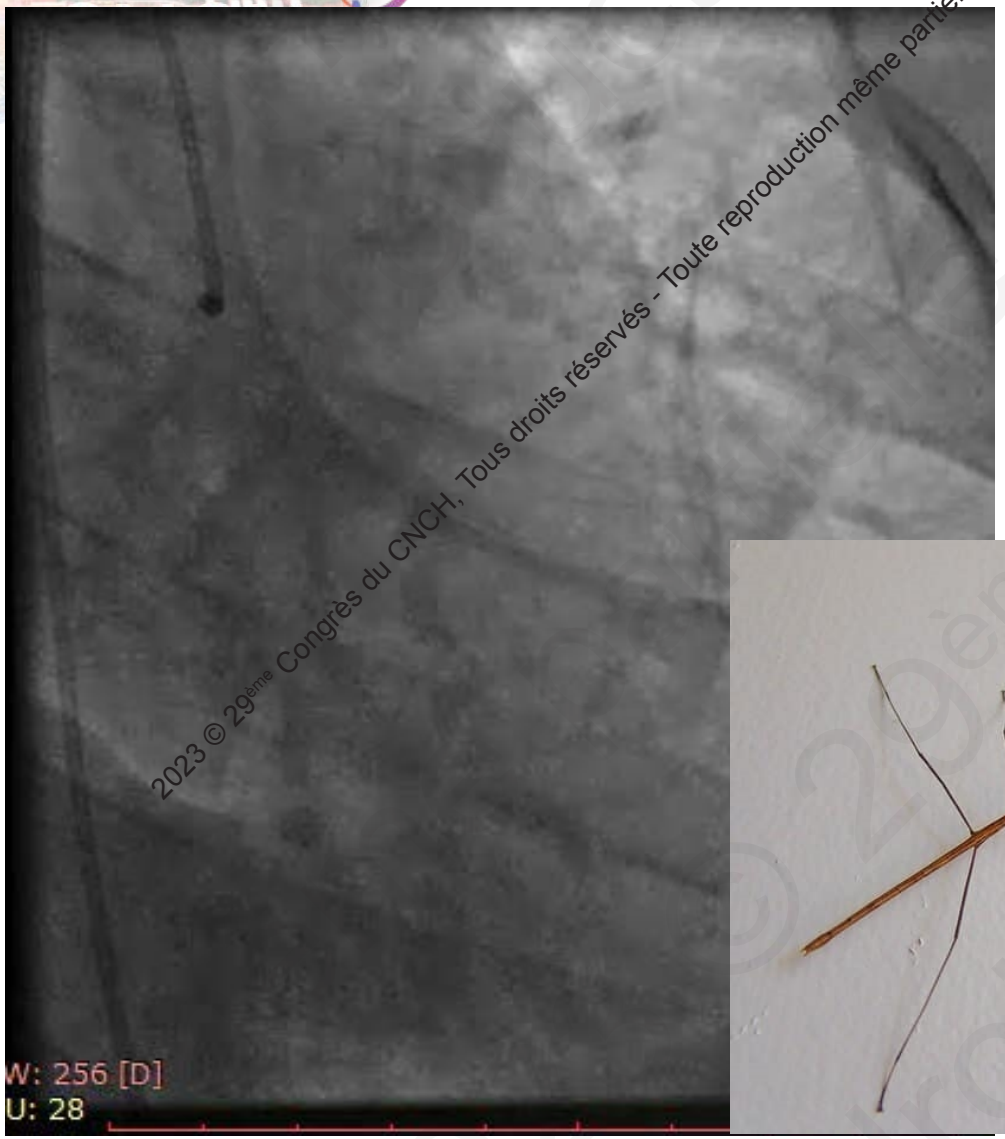

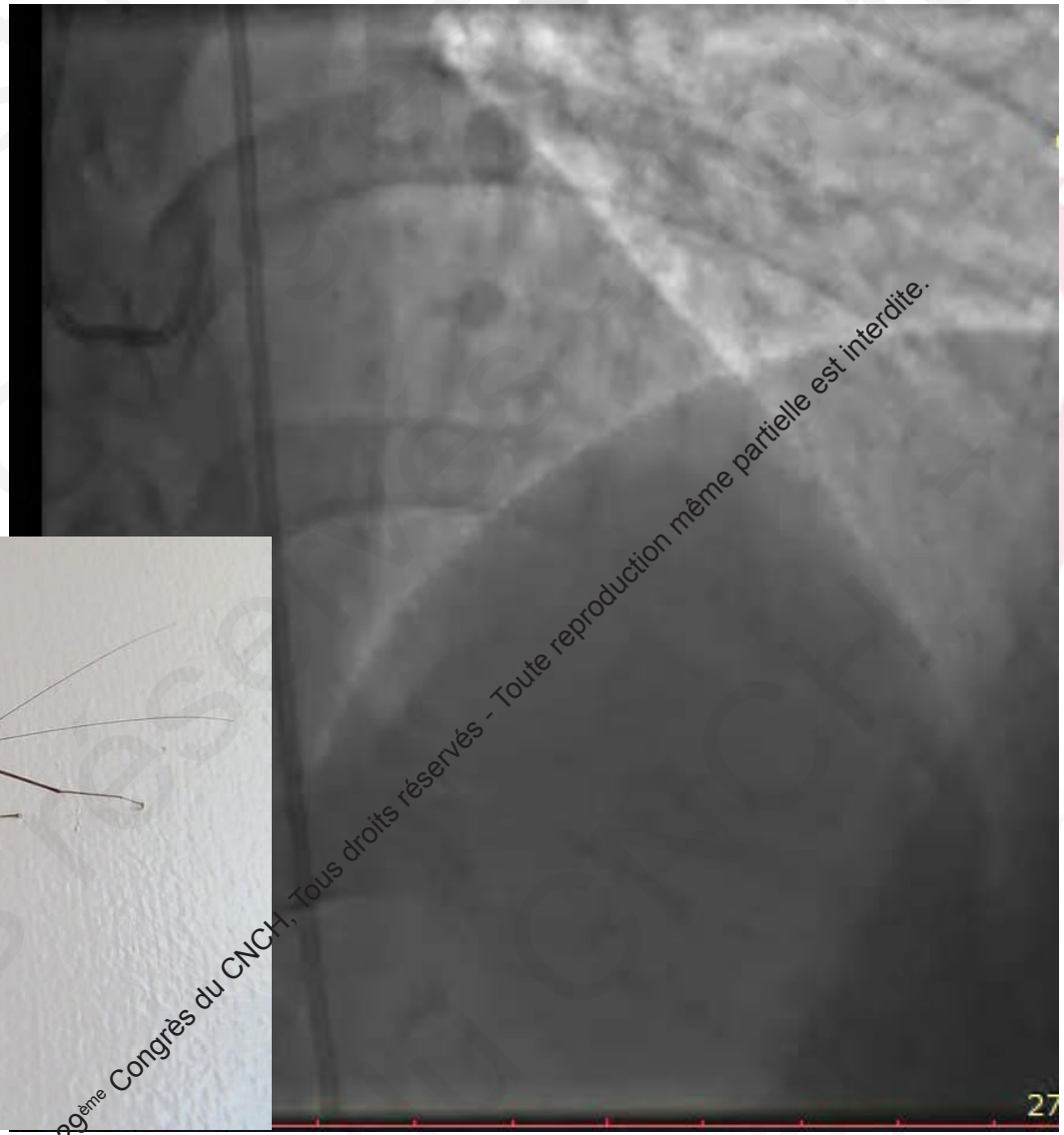

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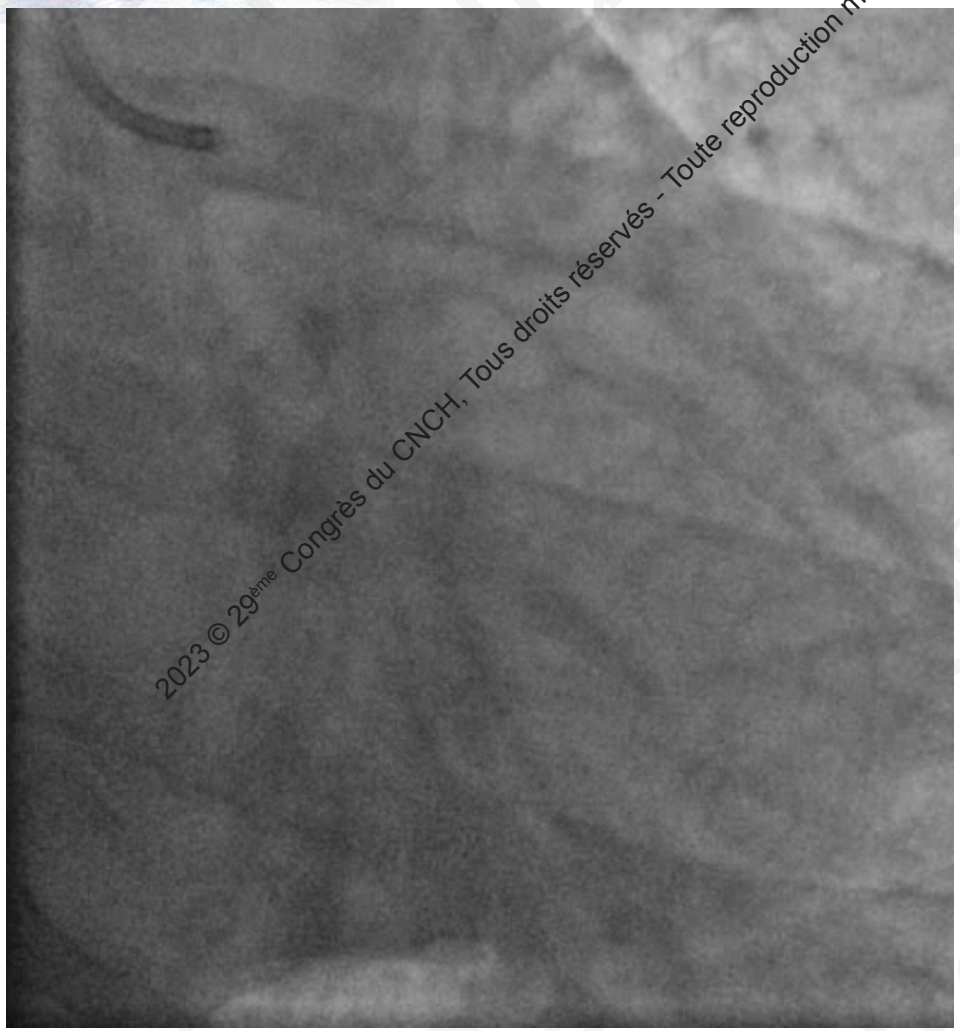

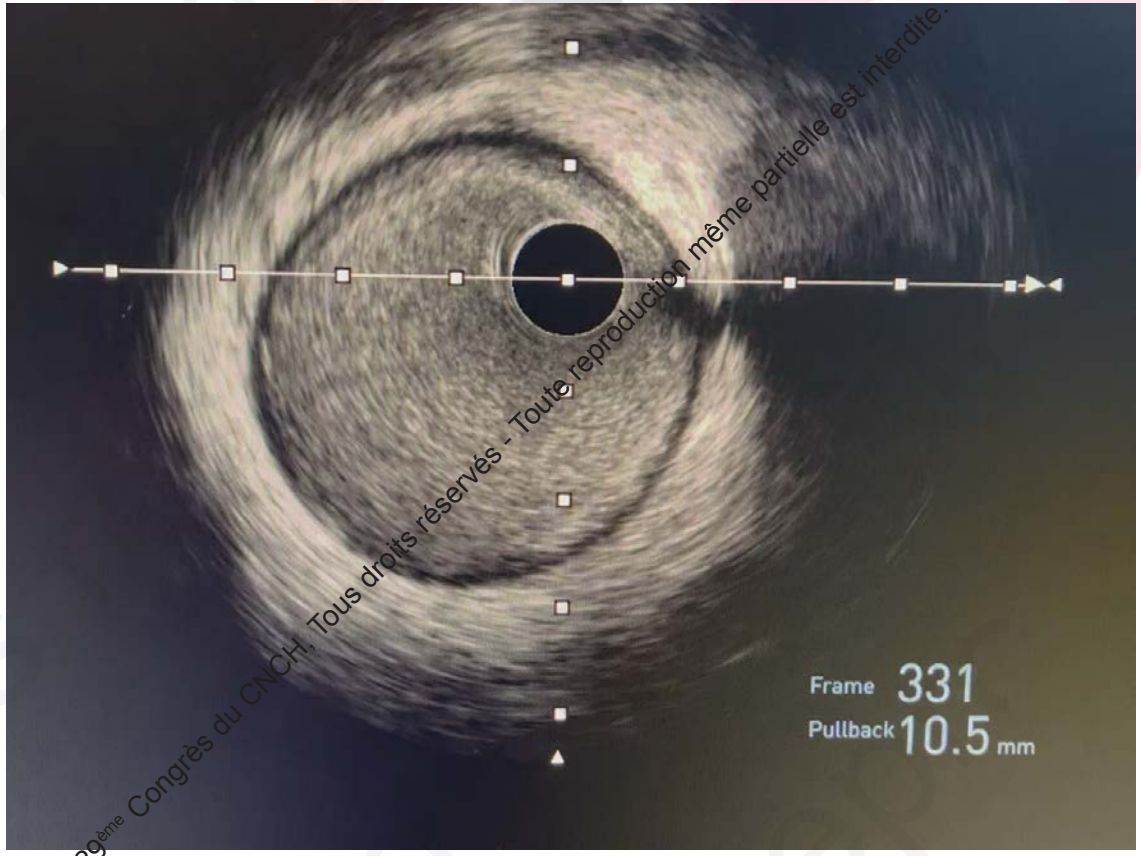

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Quelle prise en charge ?

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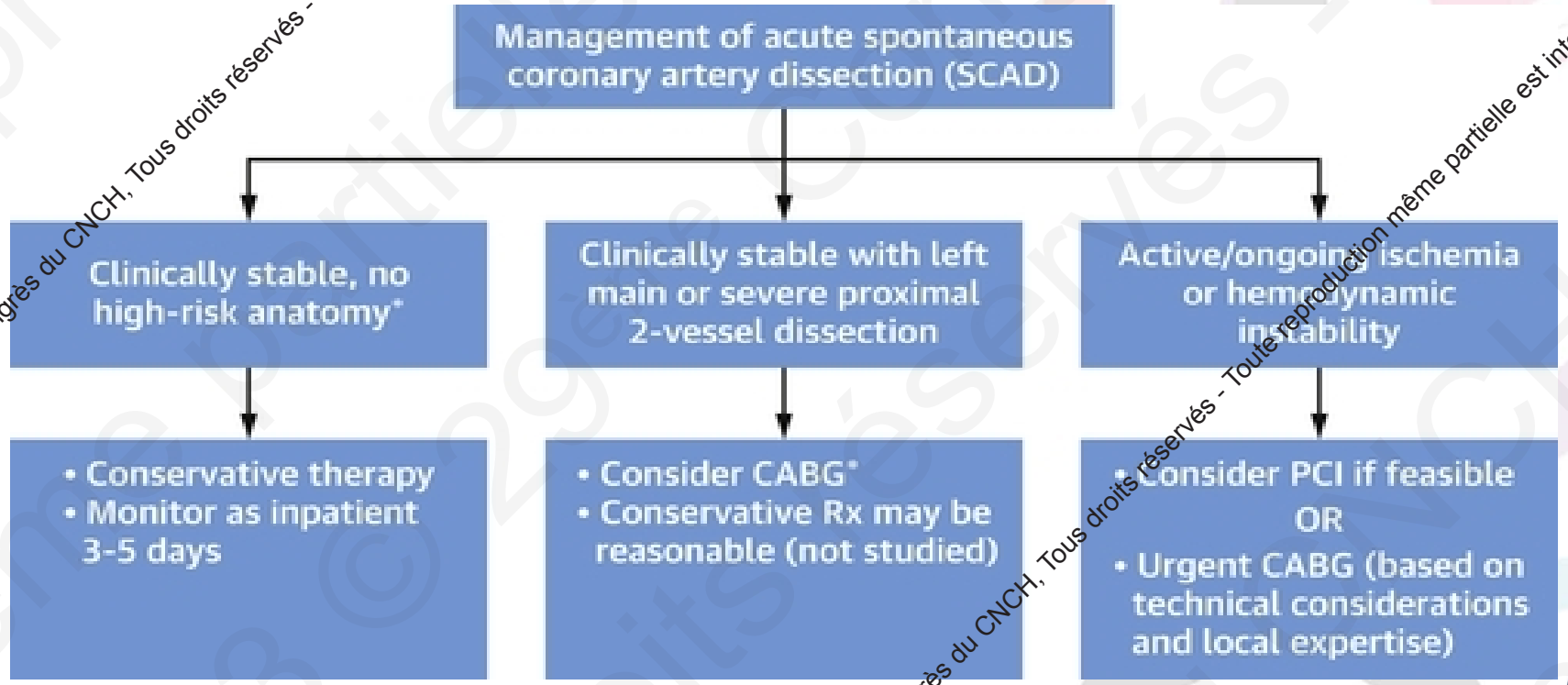

Sharonne N. Hayes et al. *J Am Coll Cardiol* 2020; 76:961-984.

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Natural History of Spontaneous Coronary Artery Dissection With Spontaneous Angiographic Healing

Saber Hassan, MD, Roshan Prakash, MD, Andrew Starovoytov, MD, Jacqueline Saw, MD

ABSTRACT

OBJECTIVES Given the uncertainty regarding the degree and prevalence of spontaneous healing following spontaneous coronary artery dissection (SCAD), the aim of this study was to assess the angiographic characteristics of the dissected segments in a large cohort of patients with SCAD who underwent subsequent repeat coronary angiography.

BACKGROUND SCAD is an uncommon yet important cause of myocardial infarction in women. Very little is known about the characteristics of healing of dissected arteries.

METHODS Patients with nonatherosclerotic SCAD followed prospectively at Vancouver General Hospital who underwent repeat angiography were included in this study. Those who underwent percutaneous coronary intervention for SCAD were excluded. Baseline patient demographics and in-hospital and long-term cardiovascular events were recorded. Angiographic characteristics of the SCAD artery at index and repeat angiography were assessed by 2 experienced angiographers. Criteria for angiographic healing were as follows: 1) improvement of stenosis severity from index event; 2) residual stenosis <50%; and 3) TIMI (Thrombolysis In Myocardial Infarction) flow grade 3.

RESULTS One hundred fifty-six patients with 182 noncontiguous SCAD lesions were included. The mean age was 51.5 ± 8.7 years, 88.5% were women, 83.3% were Caucasian, and 75.6% had fibromuscular dysplasia. All patients presented with myocardial infarction. At index angiography, type 2 SCAD was most commonly observed, in 126 of 182 lesions (69.2%); TIMI flow grade <3 was present in 85 of 182 (46.7%); and median lesion stenosis was 79.0% (interquartile range: 56.0% to 100%). Median time to repeat angiography was 154 days (interquartile range: 70 to 604 days), with median residual lesion stenosis improving to 25.5% (interquartile range: 12.0 to 38.8 days), and TIMI flow grade <3 observed in 10 of 182 lesions (5.5%). Angiographic healing occurred in 157 of 182 lesions (86.3%). Of repeat angiography performed ≥30 days post-SCAD, 152 of 160 (95%) showed spontaneous angiographic healing.

CONCLUSIONS The majority of coronary arteries affected by SCAD heal spontaneously on repeat angiography, with apparent time dependency, with the vast majority having complete healing after 30 days from the SCAD event. (J Am Coll Cardiol Intv 2019;12:518–27) © 2019 Published by Elsevier on behalf of the American College of Cardiology Foundation.

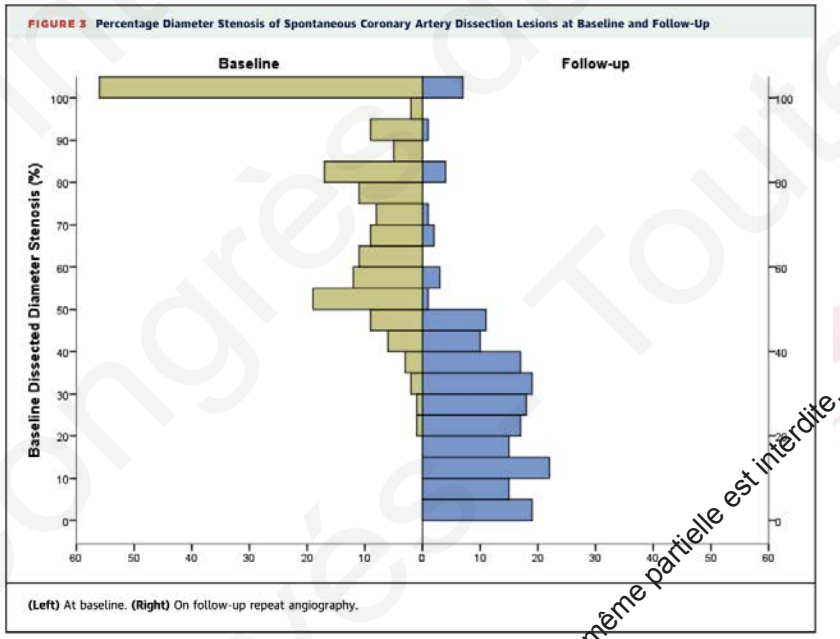


FIGURE 3 Percentage Diameter Stenosis of Spontaneous Coronary Artery Dissection Lesions at Baseline and Follow-Up

(Left) At baseline. **(Right)** On follow-up repeat angiography.

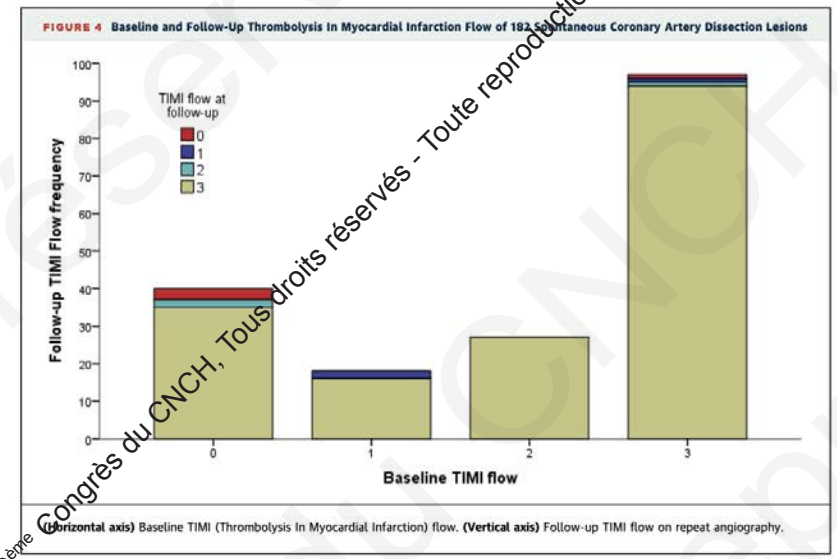
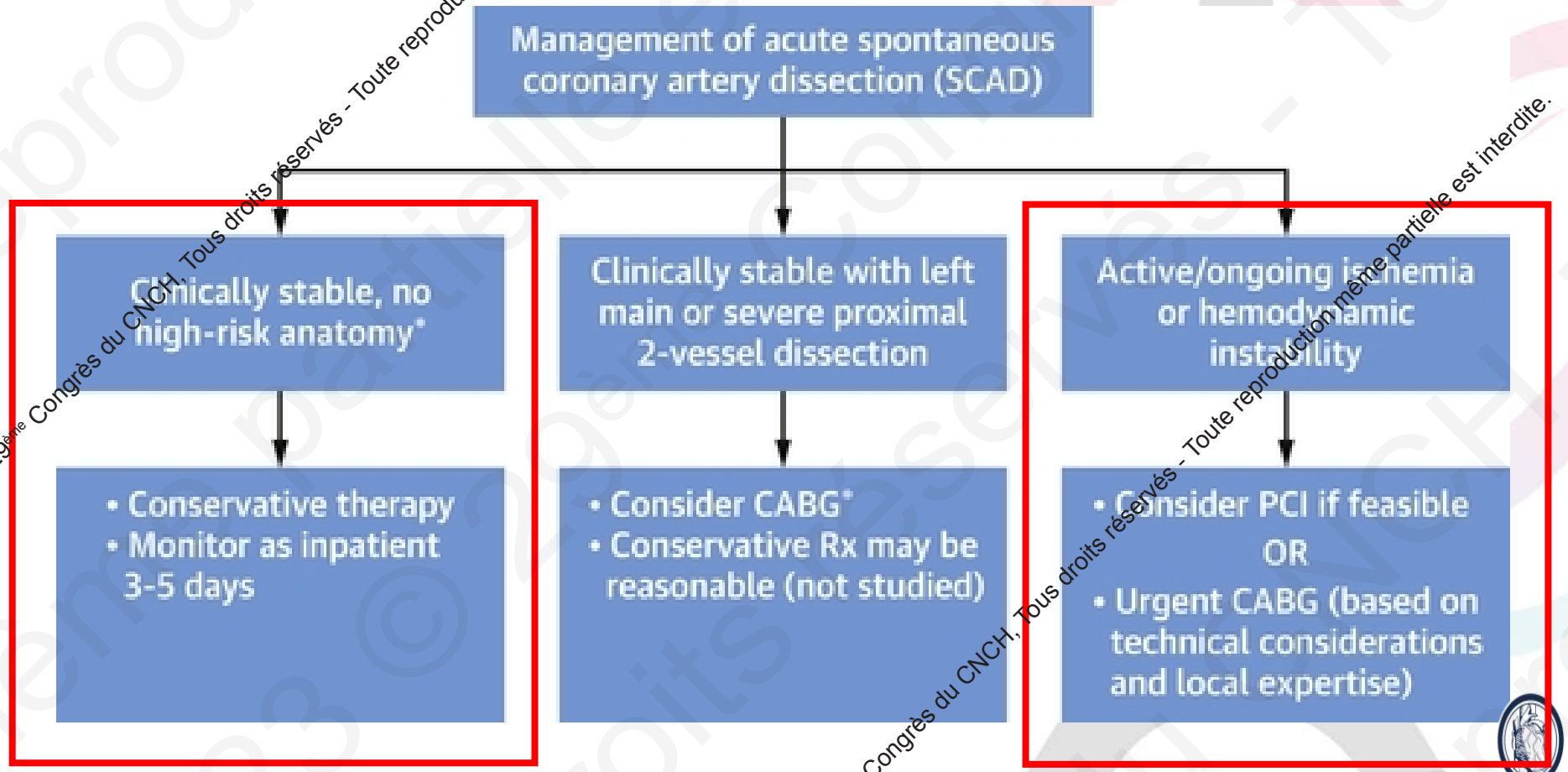


FIGURE 4 Baseline and Follow-Up Thrombolysis In Myocardial Infarction Flow of 182 Spontaneous Coronary Artery Dissection Lesions

(Horizontal axis) Baseline TIMI (Thrombolysis In Myocardial Infarction) flow. **(Vertical axis)** Follow-up TIMI flow on repeat angiography.

Quelle prise en charge ?



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Circulation: Cardiovascular Interventions
Volume 7, Issue 6, December 2014; Pages 777-786
https://doi.org/10.1161/CIRCINTERVENTIONS.114.001659

CORONARY INTERVENTIONS

Spontaneous Coronary Artery D

Revascularization Versus Conservative Therapy

Marysia S. Tweet, MD, Mackram F. Eleid, MD, Pa
MS, Amir Lerman, MD, Charanjit S. Rihal, MD, M
N. Hayes, MD, and Rajiv Gulati, MD, PhD

Background— Spontaneous coronary artery dissection (S
coronary syndrome for which optimal management remains

Methods and Results— We performed a retrospective st
a first SCAD episode. We evaluated outcomes acco
revascularization versus conservative therapy and (2) p
(PCI) versus conservative therapy stratified by vessel fl
were similar in revascularization versus conservative (me
both groups), but vessel occlusion was more frequent
18/94). There was 1 in-hospital death (revascularization
Procedural failure rate was 53% in those managed with
presenting with preserved vessel flow, rates of PCI failur
(13%) required emergency coronary artery bypass graftin
94 (90%) had an uneventful in-hospital course, but 9
progression requiring revascularization. Kaplan–Meier esti
revascularization and recurrent SCAD were no diffe
conservative therapy (30% versus 19%; P=0.06 and 23% v

Conclusions— PCI for SCAD is associated with high rate
presenting with preserved vessel flow and does n
revascularization or recurrent SCAD. A strategy of conser
observation may be preferable.

Given the increased risk of adverse outcomes with PCI in SCAD, a number of less conventional interventional approaches have been reported. These include:

- Minimal plain old balloon angioplasty (POBA) to restore flow followed by a conservative strategy[178]
- Extended stent lengths to reduce the chances of proximal or distal haematoma propagation
- Sealing the proximal and distal extremes of the affected segments with short stents to restrict the haematoma before stenting the intermediate segments.[179,180]
- Targeting an intimal tear or 'flap' for focal stenting or stenting just the proximal extent of the dissection to prevent proximal propagation.[8,181]
- Cutting balloon inflation to fenestrate the intimal-medial membrane and depressurize the false lumen as a stand-alone strategy or prior to stenting.[182–185]
- Use of bioresorbable coronary scaffolds.[184,186–188]

These anecdotal case reports are subject to publication bias. Additionally, the role of bioresorbable coronary scaffolds more generally has been the subject of recent scrutiny.[189] In the absence of randomized data, no specific alternative PCI strategy can at present be specifically recommended.

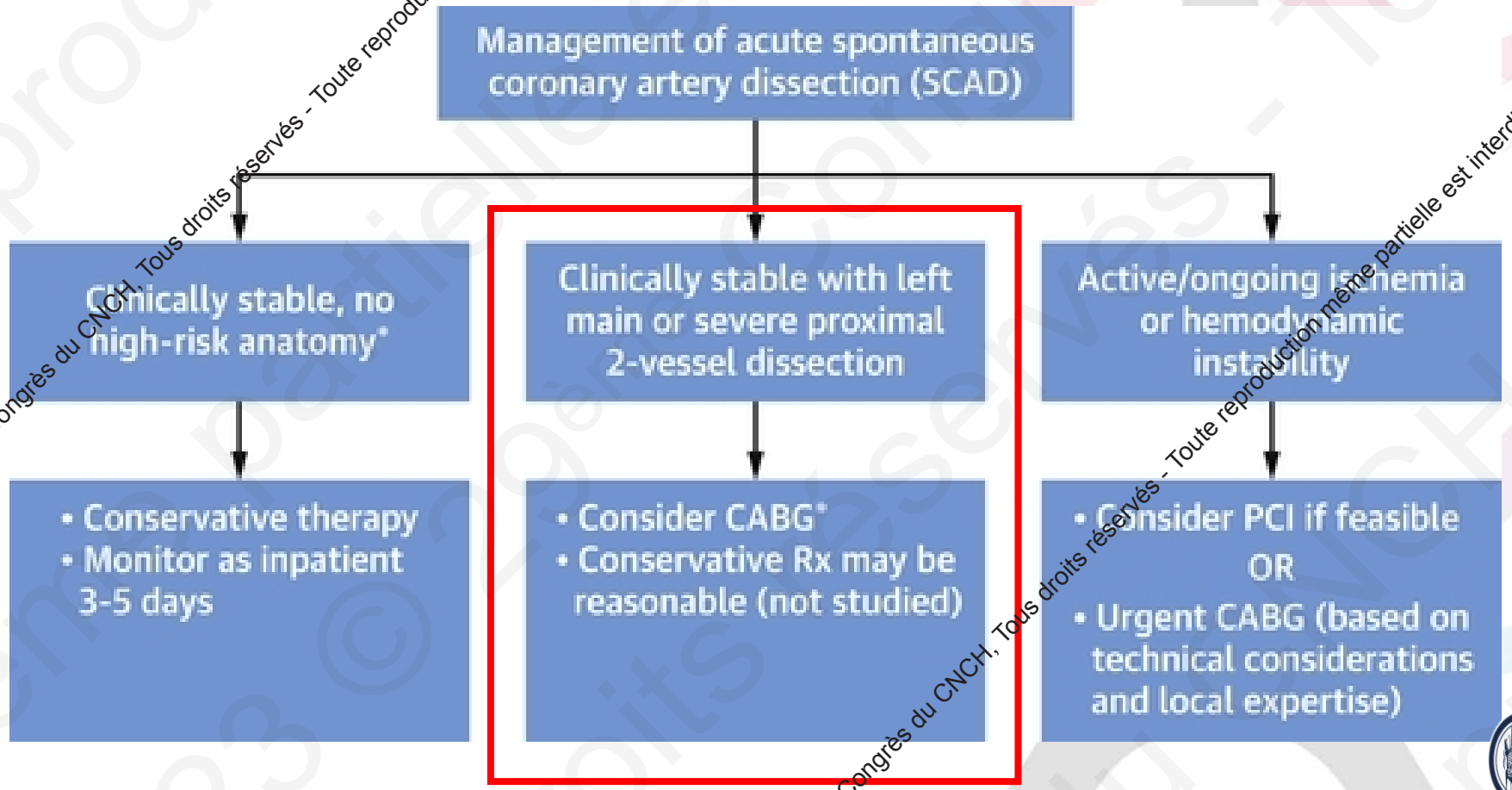
ary intervention

ow an increased risk of coronary
e Canadian series, revascularization
eved in 64% of patients and, in add-
maintained durable results at long-
es from the Mayo Clinic, most pa-
derwent coronary revascularization
nary interventions. However, were
rates. Procedural success was only
ermore revascularization was not
erm risk of repeated revasculariza-
re ongoing ischaemia or infarction
nal cardiologists should be mindful
ciated with SCAD interventions.

genic dissection
e lumen[3,168–170] (*Figure 8*)
men propagation during stent de-

or occlusion by propagation of

Quelle prise en charge ?



Sharonne N. Hayes et al. *J Am Coll Cardiol* 2020; 76:961-984.

Circulation: Cardiovascular Interventions
Volume 7, Issue 6, December 2014; Pages 777-786
https://doi.org/10.1161/CIRCINTERVENTIONS.114.001659

American Heart Association.

CORONARY INTERVENTIONS

Spontaneous Coronary Artery Dissection

Revascularization Versus Conservative Therapy

Marysia S. Tweet, MD, Mackram F. Eleid, MD, Patricia J.M. Best, MD, Ryan J. Lennon, MS, Amir Lerman, MD, Charanjit S. Rihal, MD, MBA, David R. Holmes, Jr, MD, Sharonne N. Hayes, MD, and Rajiv Gulati, MD, PhD

Table 5. Early and 5-Year Outcomes of Patients With SCAD Treated With In-Hospital CABG (Table view)

	In-Hospital CABG (n=20)
Early outcomes, n	
Death	1
SCAD vessels not bypassed	2/34
Five-year outcomes, n (Kaplan–Meier % estimates)	
Death	0
Recurrent SCAD	1 (10)
Heart failure	3 (19)
Target vessel revascularization	6 (36)
Graft details	
Left internal mammary artery	9
Saphenous vein	22
Radial artery	1
F-U patent grafts, n (11 angiograms, median F-U, 3.5 y; Q1–Q3, 0.7–3.7)	5/16

CABG indicates coronary artery bypass grafting; F-U, follow-up; and SCAD, spontaneous coronary artery dissection.

Table 3. Early and 5-Year Outcomes With Comparison According to Treatment Strategy: Revasc by CABG or PCI vs Con therapy (Table view)

	All (n=189)	Revasc (n=95)	Con (n=94)	*P* Value (Revasc vs Con)
Early outcomes, %				
Death	0.5	1	0	>0.99
Urgent/emergent CABG*	7	13	2	0.01
Progression Rx PCI	6	4†	7†	0.37
Progression Rx consv	1	0	2	0.25
PCI procedural outcomes				
% Conventional PCI failure (residual stenosis ≥30%)	...	53	NA	NA
% SCAD-specific PCI failure	...	30	NA	NA
Mean no. of stents, n ±SD		2.4±1.4	NA	NA
Mean stented length, mm ±SD	...	44±29	NA	NA
Mean residual stenosis, % ±SD	...	51±40	NA	NA
Five-year outcomes (median, F-U, 2.3 y; Q1–Q3, 0.7–5.6), n (Kaplan–Meier % estimates)				
Death	2 (2)	1 (1)	1 (4)	0.92
Recurrent SCAD	29 (27)	14 (23)	15 (31)	0.70
Heart failure	13 (13)	7 (12)	6 (16)	0.47
Target vessel revascularization	38 (25)	24 (30)	14 (19)	0.06
F-U LVEF %, mean±SD‡	57±10	55±11	58±9	0.09

Oui à une attitude conservatrice, …

Circulation

Volume 137, Issue 19, 8 May 2018; Pages e523-e557

http://doi.org/10.1161/CIR.0000000000000564

American Heart Association.

AHA SCIENTIFIC STATEMENT

Spontaneous Coronary Artery Dissection: Current … the Science: A Scientific Statement From the Ameri… Heart Association

Sharonne N. Hayes, MD, FAHA, Chair, Esther S.H. Kim, MD, MPH, FAHA, Co-… Jacqueline Saw, MD, FAHA, Co-Chair, David Adlam, BA, BM, BCh, DPhil, Cyr… Arslanian-Engoren, PhD, RN, FAHA, Katherine E. Economy, MD, MPH, Santhi… Ganesh, MD, FAHA, Rajiv Gulati, MD, PhD, Mark E. Lindsay, MD, PhD, FAHA, … Mieres, MD, FAHA, Sahar Naderi, MD, MHS, Svati Shah, MD, MHS, FAHA, Dav… Thaler, MD, PhD, FAHA, Marysia S. Tweet, MD, Malissa J. Wood, MD, and On… the American Heart Association Council on Peripheral Vascular Disease; Co… Clinical Cardiology; Council on Cardiov… and Stroke Nursing; Co… Genomic and Precision Medicine; and …

Abstract: Spontaneous coronary artery dissection (SCAD) has emerged as a… cause of acute coronary syndrome, myocardial infarction, and sudden death, … among young women and individuals with few conventional atherosclerotic … Patient-initiated research has spurred increased awareness of SCAD, an… diagnostic capabilities and findings from large case series have led to changes in… to initial and long-term management and increasing evidence that SCAD not … common than previously believed but also must be evaluated and treated diff… atherosclerotic myocardial infarction. High rates of recurrent SCAD; its associati… sex, pregnancy, and physical and emotional stress triggers; and concurre… arteriopathies, particularly fibromuscular dysplasia, highlight the differences…

Avis d'experts

ESC European Society of Cardiology

European Heart Journal (2018) **39**, 3353–3368
doi:10.1093/eurheartj/ehy080

CURRENT OPINION
Coronary artery disease

European Society of Cardiology, acute cardiovascular care association, SCAD study group: a position paper on spontaneous coronary artery dissection

ESC-ACCA Position Paper on spontaneous coronary artery dissection

Da… Adlam, Cha… Olivecrona, … Fernandez-Hidalgo (Section Editor Spain)[2], An… Ia… (Section Editor Netherlands)[3], Christiaan Vrints (Co-Chairperson; Section Editor Belgium)[4]
Writing Committee: Abtehale al-Hussaini (UK)[1], Hector Bueno (Spain)[5,6], Piera Capranzano (EAPCI Women, Italy)[7†], Sofie Gevaert (Belgium)[8], Stephen G. Hoole (UK)[9], Tom Johnson (UK)[10], Corrado Lettieri (Italy)[11], Michael Maeder (Switzerland)[12], Pascal Motreff (France)[13], Peter Ong (Germany)[14], Alexandre Persu (European FMD initiative, Belgium)[15,16], Hans Rickli (Switzerland)[12], Francois Schiele (France)[17], Mary N. Sheppard (UK)[18,19], and Eva Swahn (Sweden)[20]

[1]Department of Cardiovascular Sciences, University of Leicester and NIHR Leicester Biomedical Research Centre, Glenfield Hospital, Leicester, UK; [2]Department of Cardiology, Hospital Universitario de La Princesa, Madrid, Spain; [3]Department of Cardiology, Women's Cardiac Health, Radboud University Medical Center, Nijmegen, The Netherlands; [4]University of Antwerp - Antwerp University Hospital, Wilrijkstraat 10, B-2650 Edegem, Belgium; [5]Centro Nacional de Investigaciones Cardiovasculares (CNIC), Melchor Fernandez Almagro, 3, 28029 - Madrid, Spain; [6]Cardiovascular Research Area, imas12 Research Institute, CCU, Department of Cardiology, Hospital Universitario 12 de Octubre, Spain; [7]Division of Cardiology, Cardio-Thoracic-Vascular Department, Azienda Ospedaliero-Universitaria "Policlinico-Vittorio Emanuele", University of Catania, Catania, Italy; [8]Department of Cardiology, Ghent University Hospital, Ghent, Belgium; [9]Department of Interventional Cardiology, Papworth Hospital, Cambridge, UK; [10]Bristol Heart Institute, University Hospitals Bristol NHS Foundation Trust, UK; [11]Department of Cardiology, ASST Mantova-Ospedale Carlo Poma, Mantova, Italy; [12]Cardiology Department, Kantonsspital St. Gallen, Rorschacherstrasse 95, 9007, St. Gallen, Switzerland; [13]Cardiology Department, CHU Clermont-Ferrand, CNRS, University of Clermont Auvergne (UCA) and ISIT-CAVITI, UMR 6284, University of Clermont Auvergne (UCA), France; [14]Department of Cardiology, Robert-Bosch-Krankenhaus, Stuttgart, Germany; [15]Pole of Cardiovascular Research, Institut de Recherche Expérimentale et Clinique, Université Catholique de Louvain, Brussels, Belgium; [16]Division of Cardiology, Cliniques Universitaires Saint-Luc, Université Catholique de Louvain, Brussels, Belgium; [17]Department of Cardiology, University Hospital Besancon, and EA3920, University of Burgundy-Franche-Comté, Besancon, France; [18]Cardiac Risk in the Young Department of Cardiovascular Pathology, Cardiovascular Sciences Research Centre, St George's University of London, London, UK; [19]St George's Hospital NHS Trust, London, UK; and [20]Department of Cardiology and Department of Medical and Health Sciences, Linköping University, Sweden.

Received 31 July 2017; revised 19 October 2017; editorial decision 31 January 2018; accepted 7 February 2018; online publish-ahead-of-print 22 February 2018

- **Anticoagulants**
- **Antiagrégants plaquettaires : mono ou DAPT**
- **Bétabloquants**
- **Statines**

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Circulation
Volume 137, Issue 19, 8 May 2018; Pages e523-e557
https://doi.org/10.1161/CIR.0000000000000564

AHA SCIENTIFIC STATEMENT

Spontaneous Coronary Artery Dissection: Current State of the Science: A Scientific Statement From the American Heart Association

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CURRENT OPINION
Coronary artery disease

European Society of Cardiology, acute cardiovascular care association, SCAD study group: a position paper on spontaneous coronary artery dissection

ESC-ACCA Position Paper on spontaneous coronary artery dissection

Anticoagulation and Antiplatelet Therapy

Because the pathophysiology, mechanisms of ischemia, PCI outcomes, and residua of SCAD are distinct from those associated with atherosclerotic ACS, many investigators have questioned the rationale and potential risks of using standard ACS therapies in patients with SCAD. For instance, early heparin use may provide benefit by reducing thrombus burden, but there are theoretical concerns about its use in the setting of acute SCAD presentation related to accentuating the risk of bleeding into the IMH or extension of dissection. Therefore, if systemic anticoagulation is started at hospital presentation, in the absence of other indications for systemic anticoagulation, consideration of discontinuation is appropriate once SCAD is diagnosed.[190]

- Anticoagulants
- **Antiagrégants plaquettaires : mono ou DAPT**
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Aspirine

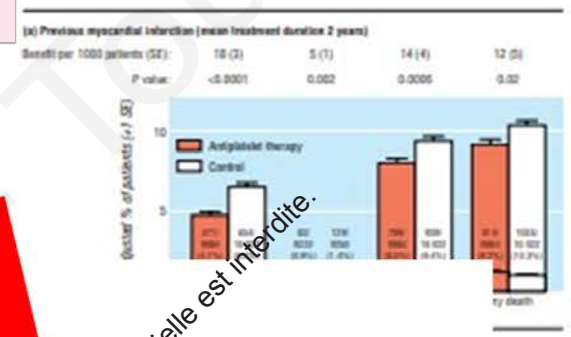
Collaborative meta-analysis of randomised trials of antiplatelet therapy for prevention of death, myocardial infa

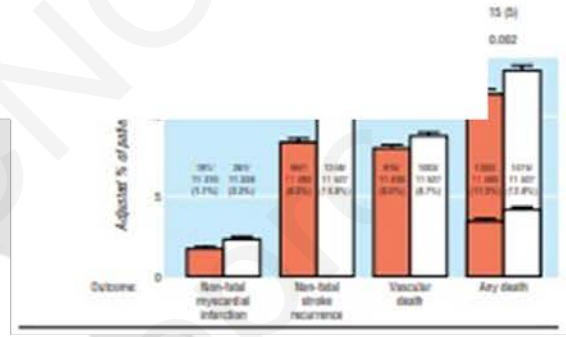
Antiplatelet

- Profil d'effet
- Ava

Avis d'experts

et en prévention secondaire





(10.7) (13.2)

Heterogeneity of odds reductions between:
5 categories of trial: χ^2=21.4, df=4; P=0.0003
Acute stroke v other: χ^2=18.0, df=1; P<0.00002

Fig 1 Proportional effects of antiplatelet therapy on vascular events (myocardial infarction, stroke, or vascular death) in five main high risk categories. Stratified ratio of odds of an event in treatment groups to that in control groups is plotted for each group of trials (black square) along with its 99% confidence interval (horizontal line). Meta-analysis of results for all trials (and 95% confidence interval) is represented by an open diamond. Adjusted control totals have been calculated after converting any unevenly randomised trials to even ones by counting control groups more than once, but other statistical calculations are based on actual numbers from individual trials

https:/ / doi.org/ 10.1136/ bmj.324.7329.71

Ajout d'une deuxième anti agrégant plaquettaire,

Circulation

AHA SCIENTIFIC STATEMENT

Spontaneous Coronary Artery Dissection: Current State of the Science

A Scientific Statement From the Americar

MEDICAL THERAPY

The ultimate goals of short- and long-term m
therapy of SCAD are to alleviate symptoms, to im
short- and long-term outcomes, and to preven
rent SCAD. Unfortunately, there is a substantial
evidence to guide clinicians in this regard because
relatively recent recognition of SCAD as an imp
clinical entity and the absence of identified cellul
molecular targets or randomized controlled trials t
port an evidence-based approach. Given the pau
evidence currently available, the following appro
are based largely on expert opinions derived from the
clinical experience of members of the writing group.

Anticoagulation and Antiplatelet Therapy

Because the pathophysiology, mechanisms of ischemia, PCI outcomes, and residua of SCAD are distinct from

based antiplatelet therapy after PCI.[156] Clear evidence supporting the use of dual-antiplatelet therapy in patients with SCAD who do not undergo coronary intervention is lacking. Although theoretical benefits of early dual-antiplatelet therapy in SCAD include protection

tively. Bleeding complications were not reported, but long-term outcomes in both groups were similar.[10]

Most experts recommend aspirin use for at least 1 year and frequently indefinitely after SCAD in patients who receive medical treatment, in the absence of contraindications.[10,31,190,191] In light of increased bleeding risks with antiplatelet agents, especially menorrhagia in premenopausal women, and uncertain benefits and risks, individual selection of suitability for dual-antiplatelet therapy and aspirin therapy in conservatively managed survivors of SCAD is indicated.

ESC European Society of Cardiology European Heart Journal (2018) 39, 3353–3368 doi:10.1093/eurheartj/ehy080

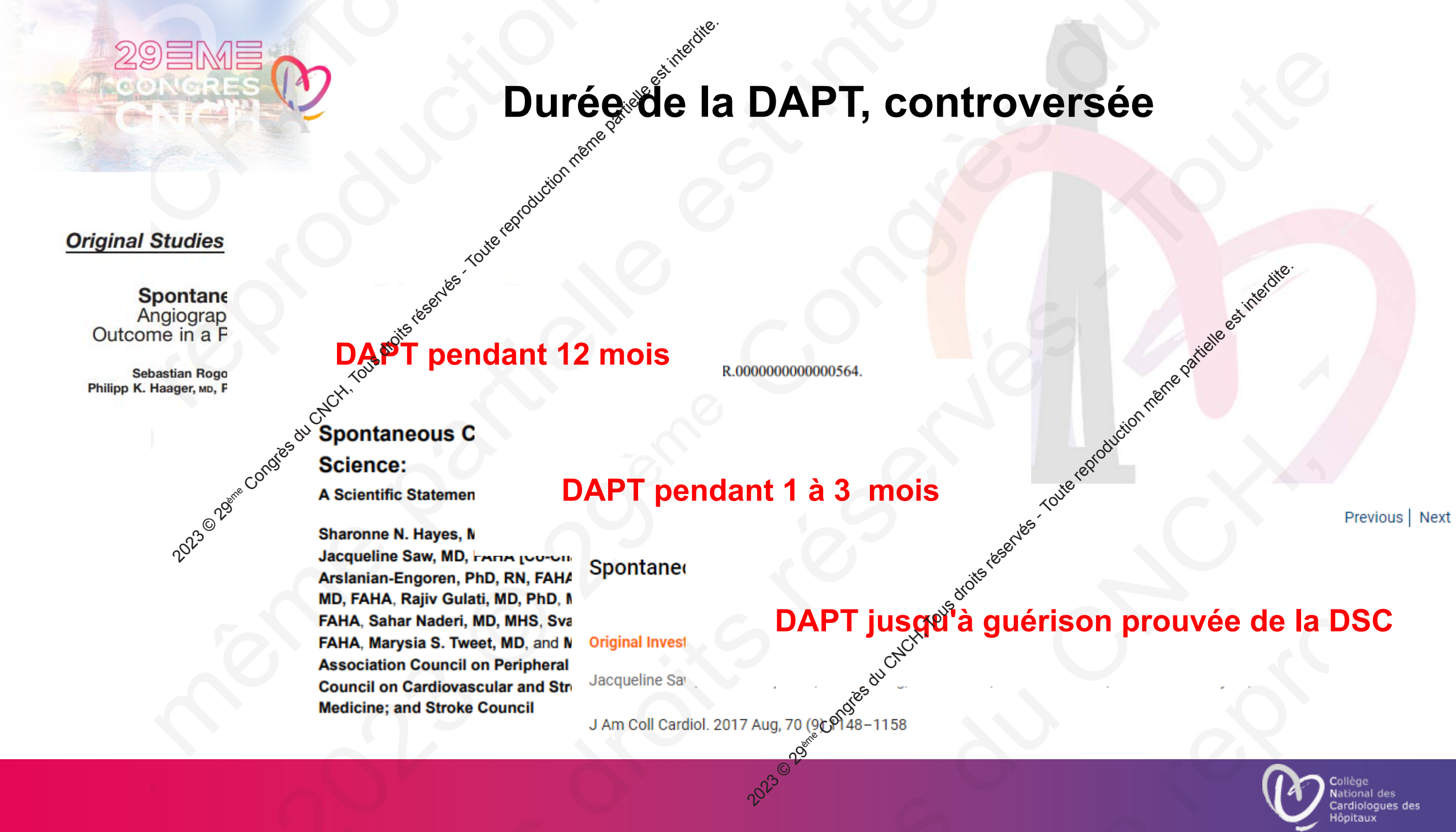
CURRENT OPINION
Coronary artery disease

European Society of Cardiology, acute cardiovascular care association, SCAD study group: a position paper on spontaneous coronary artery dissection

duration of treatment re-
practice in SCAD. This re-
strength of existing data
erent concern (albeit un-
ng bleeding time for a con-
be an intramural bleed.[145]
matic menorrhagia which
rual age taking antiplatelet
should receive dual anti-
ged or lifelong monother-
nce with current ACS
guidelines.[206,] In patients managed conservatively, there is evidence from OCT studies of high grade stenosis sometimes with true luminal thrombus in association with SCAD.[30,166] This provides justification for antiplatelet therapy in the acute phase and most authors advocate acute dual antiplatelet therapy (usually with aspirin and clopidogrel rather than the newer P2Y12 inhibitors and avoiding intravenous antiplatelet therapies).[19,106,108] The optimal duration of dual and subsequent monotherapy remains unknown with some authors advocating lifelong aspirin[19,108] and others questioning this approach.[106]

Pas de recommandations formelles

Durée de la DAPT, controversée



DAPT pendant 12 mois

DAPT pendant 1 à 3 mois

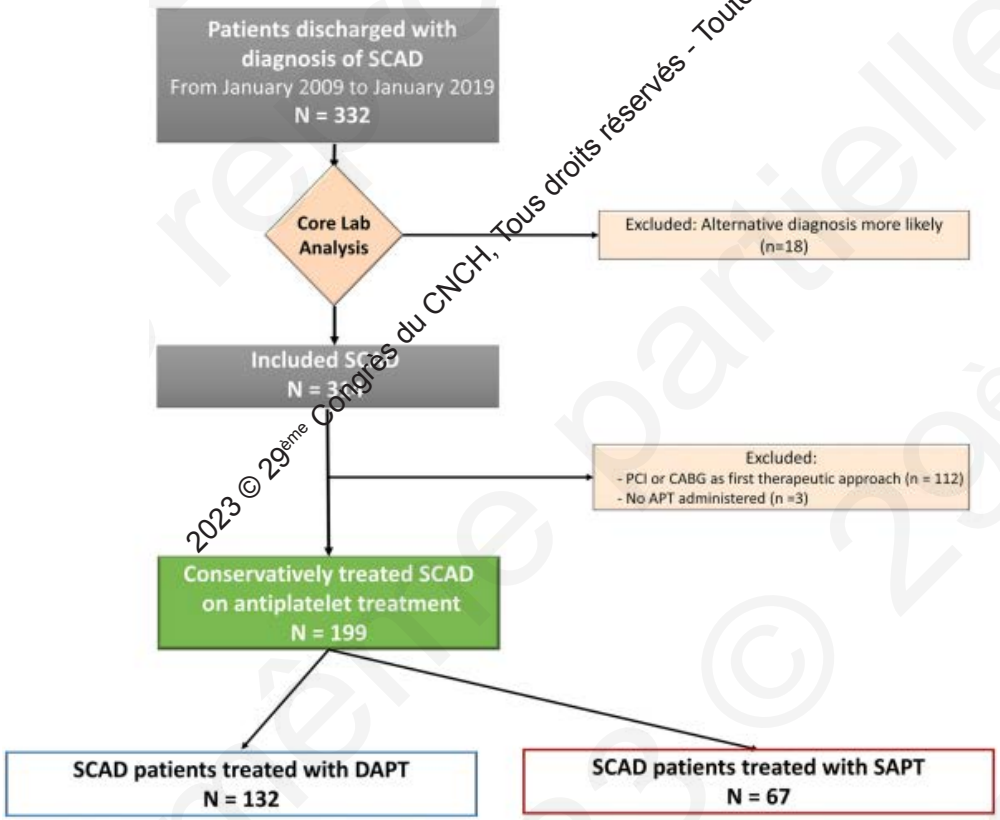
DAPT jusqu'à guérison prouvée de la DSC

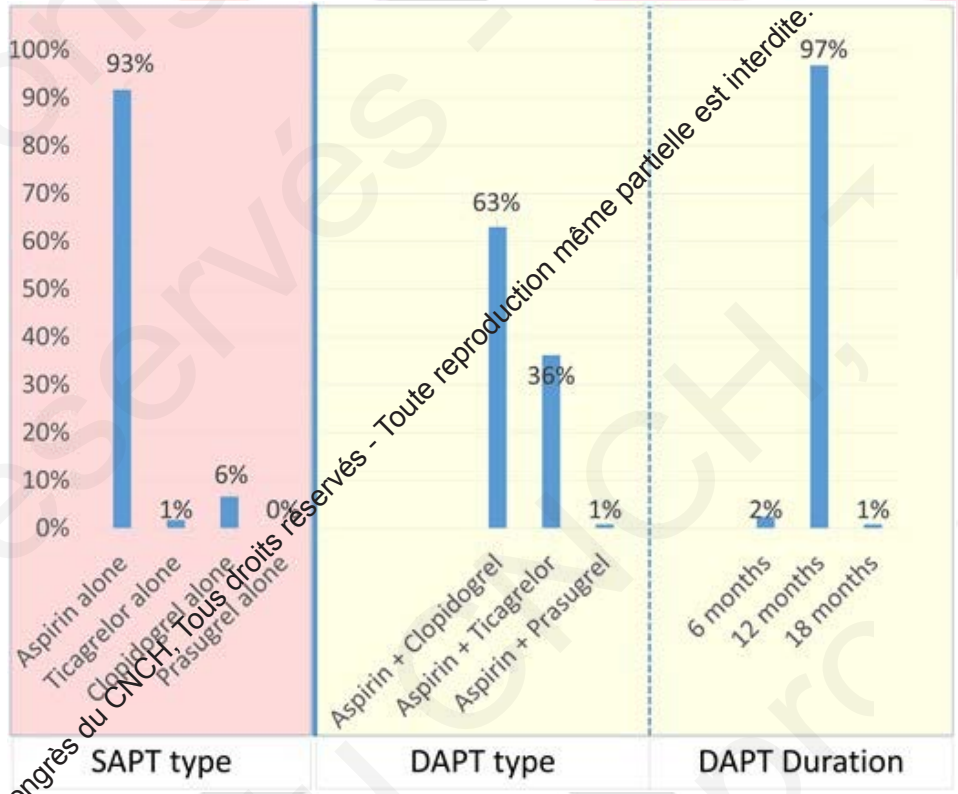
International DISCO SCAD Registry

SCAD conservately treated* (n=199)

Antiplatelet regimen according to physician's choice

DAPT	SAPT
N = 132 (66%)	N = 67 (34%)





doi.org/10.1093/eurheartj/ehab372

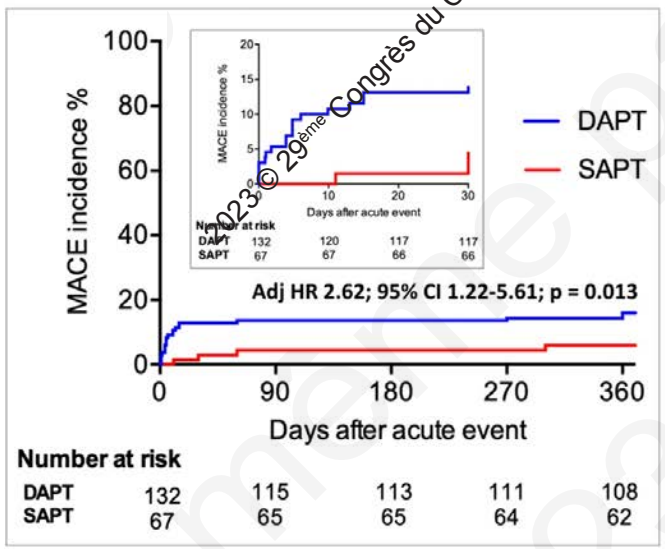
Une preuve pour un bénéfice incertain

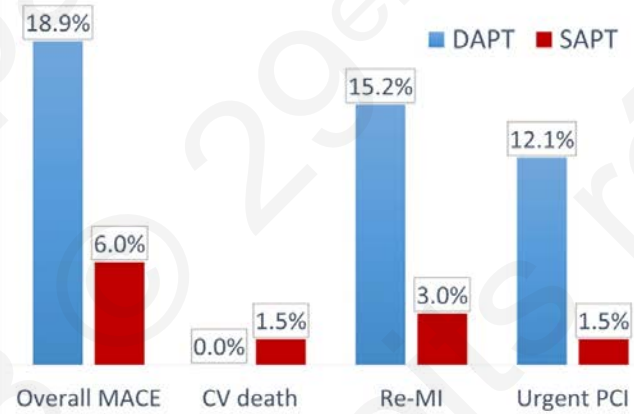
International DISCO SCAD Registry

SCAD conservately treated* (n=199)

Antiplatelet regimen according to physician's choice

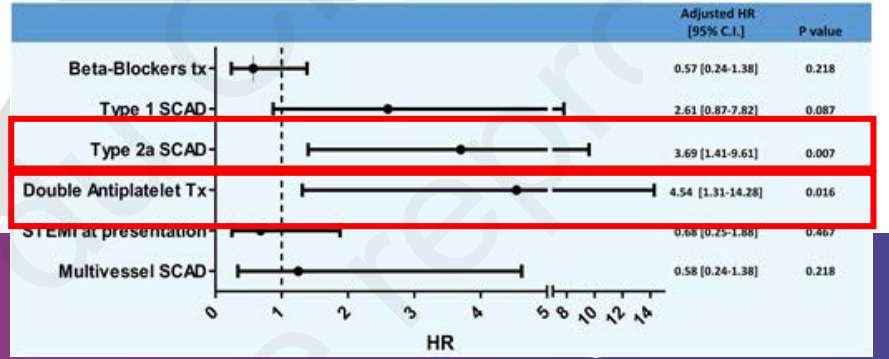
DAPT	SAPT
N = 132 (66%)	N = 67 (34%)





	Overall (*N*=199)	DAPT (*N*=132)	SAPT (*N*=67)	*P*-value
In-hospital events				
Overall MACE	16 (8.0)	15 (11.4)	1 (1.5)	0.016
All-cause death	1 (0.5)	0	1 (1.5)	—
Non-fatal MI	11 (5.5)	11 (8.3)	0	—
Any unplanned PCI	14 (7.0)	14 (10.6)	0	—
Bleeding	2 (1.0)	2 (1.5)	0	—
BARC 1	1 (0.5)	1 (0.7)	0	—
BARC 2	1 (0.5)	1 (0.7)	0	—
12-month events				
Overall MACE	29 (14.6)	25 (18.9)	4 (6.0)	0.013
All-cause death	1 (0.5)	0	1 (1.5)	—
Non-fatal MI	22 (11.1)	20 (15.2)	2 (3.0)	0.009
Any unplanned PCI	17 (8.5)	16 (12.1)	1 (1.5)	0.010
Bleeding	15 (7.5)	12 (9.1)	3 (4.5)	0.24
BARC 1	10 (5.0)	7 (5.3)	3 (4.5)	1.00
BARC 2	5 (2.5)	5 (3.8)	0	—

In-hospital and 1-year major adverse cardiovascular events



doi.org/10.1093/eurheartj/ehab372

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Spontaneous Coronary Artery Dissection

Clinical Outcomes and Risk of Recurrence

Jacqueline Saw, MD,[a] Karin Humphries, DSc,[b] Eve Aymong, MD,[c] Tara Sedlak, MD,[a] Roshan Prakash, MBBS,[a] Andrew Starovoytov, MD,[a] G.B. John Mancini, MD[a]

ABSTRACT

BACKGROUND Spontaneous coronary artery dissection (SCAD) is underdiagnosed and an important cause of myocardial infarction (MI), especially in young women. Long-term cardiovascular outcomes, including recurrent SCAD, are inadequately reported.

OBJECTIVES This study sought to describe the acute and long-term cardiovascular outcomes and assess the predictors of recurrent SCAD.

METHODS Nonatherosclerotic SCAD patients were prospectively followed at Vancouver General Hospital systematically to ascertain baseline, predisposing and precipitating stressors, angiographic features, revascularization, use of medication, and in-hospital and long-term cardiovascular events. Clinical predictors for recurrent de novo SCAD were tested using univariate and multivariate Cox regression models.

RESULTS The authors prospectively followed 327 SCAD patients. Average age was 52.5 ± 9.6 years, and 90.5% were women (56.9% postmenopausal). All presented with MI; 25.7% had ST-segment elevation MI, 74.3% had non-ST-segment elevation MI, and 8.9% had ventricular tachycardia/ventricular fibrillation. Precipitating emotional stressors were reported in 48.3% and physical stressors in 28.1%. Fibromuscular dysplasia was present in 62.7%, connective tissue disorder in 4.9%, and systemic inflammatory disease in 11.9%. The majority (83.1%) were initially treated medically, with only 16.5% or 2.2% undergoing in-hospital percutaneous coronary intervention or coronary artery bypass graft surgery, respectively. The majority of SCAD patients were taking aspirin and beta-blocker therapy at discharge and at follow-up. Median hospital stay was 3.0 days, and the overall major adverse event rate was 7.3%. Median long-term follow-up was 3.1 years, and overall major adverse cardiac event rate was 19.9% (death rate: 1.2%; recurrent MI: 16.8%; stroke/transient ischemic attack: 1.2%; revascularization: 5.8%). Recurrent SCAD occurred in 10.4% of patients. In multivariate modeling, only hypertension increased (hazard ratio: 2.46; p = 0.011) and beta-blocker use diminished (hazard ratio: 0.36; p = 0.004) recurrent SCAD.

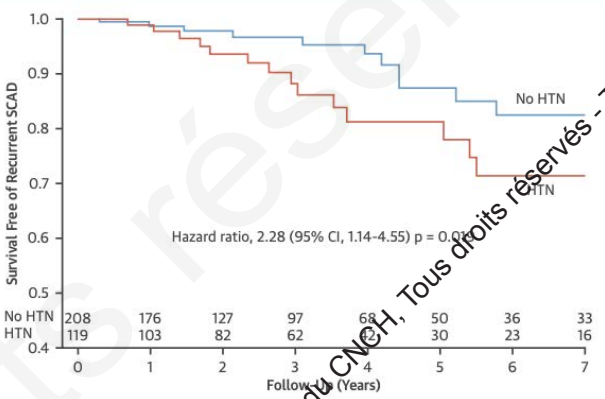
CONCLUSIONS In our large prospectively followed SCAD cohort, long-term cardiovascular events were common. Hypertension increased the risk of recurrent SCAD, whereas beta-blocker therapy appeared to be protective.
(J Am Coll Cardiol 2017;70:1148–58)

TABLE 8 Univariate and Multivariate Predictors of Recurrent SCAD

	Univariate Model		Multivariate Model	
Predictor	Hazard Ratio (95% CI)	p Value	Hazard Ratio (95% CI)	p Value
Hypertension	2.28 (1.14–4.55)	0.019	2.46 (1.23–4.93)	0.011
Beta-blocker	0.39 (0.19–0.78)	0.008	0.36 (0.18–0.73)	0.004
Calcium-channel blocker	2.57 (1.25–5.31)	0.011		
Aspirin	0.36 (0.18–0.73)	0.004		

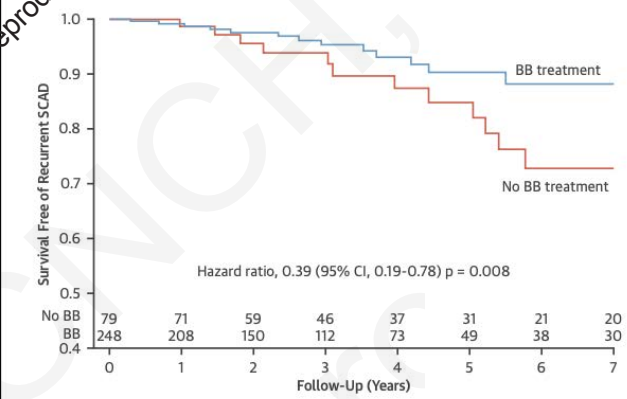
CI = confidence interval; SCAD = spontaneous coronary artery dissection.

FIGURE 3 Survival Free of Recurrent SCAD: Hypertension



Presence of HTN increased the risk of recurrent SCAD. CI = confidence interval; HTN = hypertension; SCAD = spontaneous coronary artery dissection.

FIGURE 4 Survival Free of Recurrent SCAD: Beta-Blocker Treatment



BB treatment reduced the risk of recurrent SCAD. BB = beta-blocker; other abbreviations as in Figure 3.

- **Anticoagulants**
- **Antiagrégants plaquettaires : mono ou DAPT**
- **Bétabloquants**
- **Statines**

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Clinical Features, Management, and Prognosis of Spontaneous Coronary Artery Dissection

Marysia S. Tweet, MD; Sharonne N. Hayes, MD; Sridevi R. [illegible], MBBS; Robert D. Simari, MD; Amir Lerman, MD; Ryan J. Lennon, MS; Bernard J. Gersh, MB, ChB, DPhil; Sherezade Khambatta, DO; Patricia J.M. Best, MD; Charanjit S. Rihal, MD, MBA; Rajiv Gulati, MD, PhD

Long-Term Outcomes

SCAD Recurrence

During a median follow-up of 47 months (interquartile range, 18–106), 15 patients (17%) experienced a recurrent SCAD episode. Figure 4A illustrates the Kaplan-Meier–estimated rate of SCAD recurrence. After a primary SCAD event, the 10-year SCAD recurrence rate was 29.4%. Of those with

cardiac medications begun during the index admission on subsequent risk of SCAD recurrence (Table II in the online-only Data Supplement), although sample size remains a limitation for analysis. An exception appeared to be statin use, which was higher in the SCAD recurrence group. However, because the median index event year was 2007 for those prescribed statins versus 2002 for those not prescribed statins, the date of event is a potential confounding factor. More so, it is certainly possible that those not prescribed statins initially were prescribed them at a later date. Lack of information on statin use is a limitation of the analysis.

Spontaneous Coronary Artery Dissection

Clinical Outcomes and Risk of Recurrence

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spital systematically
tion, use of
e novo SCAD were

rs, and 90.5% were
3% had non-ST-
motional stressors
%, connective tissue
treated medically,
tery bypass
apy at discharge and

at follow-up. Median hospital stay was 3.0 days, and the overall major adverse event rate was 7.3%. Median long-term follow-up was 3.1 years, and overall major adverse cardiac event rate was 19.9% (death rate: 1.2%; recurrent MI: 16.8%; stroke/transient ischemic attack: 1.2%; revascularization: 5.8%). Recurrent SCAD occurred in 10.4% of patients.
In multivariate modeling, only hypertension increased (hazard ratio: 2.46; p = 0.011) and beta-blocker use diminished (hazard ratio: 0.36; p = 0.004) recurrent SCAD.

CONCLUSIONS In our large prospectively followed SCAD cohort, long-term cardiovascular events were common. Hypertension increased the risk of recurrent SCAD, whereas beta-blocker therapy appeared to be protective.
(J Am Coll Cardiol 2017;70:1148–58)

statines

Pas de statines systématiques

Pronostic

TABLE 3 Demographics, Presentation, and Cardiovascular Outcomes in Contemporary SCAD Series

First Author (Ref. #)	Year	N	Age (yrs)	Women	ACS	STEMI	NSTEMI	Revasc, PCI*	PCI Success	In-hosp Death	In-hosp MI	In-hosp Urgent Revasc	Median F/U Time	F/U Death	F/U MI	F/U SCAD	F/U Revasc	F/U HF
Alfonso (8)	2012	27	53.0 ± 11.0	85.0	85.0	52.0	33.0	55.6, 100.0	80.0	0.0	0.0	7.4	730 days	0.0	0.0	NR	3.7	3.7
Saw (9)	2014	168	52.1 ± 9.2	92.3	100.0	26.1	73.9	20.2, 82.3	63.6	0.0	4.8	4.8	6.9 yrs	2.4	15.5	13.1	6.5	0.0
Tweet† (10)	2014	189	54.0 ± 9.0	92.0	100.0	37.0	63.0	50.3, 93.7	47.0	0.5	0.0	7.0	2.3 yrs	2.0	19.6	27.0	25.0	13.0
Lettieri (11)	2015	136	52.0 ± 11.0	81.0	93.0	49.2	40.3	42.0, 91.1	72.5	2.2	5.2	5.8	22 days	3.1	1.6	4.7	4.6	3.9
Rogowski (12)	2015	64	53.0 ± 11.2	94.0	100.0	69.0	30.0	12.5, 87.5	66.7	1.5	0.0	0.0	4.5 yrs	0.0	6.3	6.3	0.0	0.0
Roura (14)	2015	34	47.0 ± 12.0	94.1	100.0	55.0	45.0	23.5, 100.0	75.0	0.0	0.0	0.0	321 days	0.0	5.9	2.9	0.0	0.0
Rashid (13)	2016	21	53.3 ± 8.8	95.2	100.0	34.8	56.5	28.6, 100.0	66.7	0.0	0.0	NR	NR	NR	NR	NR	NR	NR
Nakashimi (15)	2016	63	46.0 ± 10.0	94.0	100.0	87.0	13.0	55.6, 97.1	91.0	NR	NR	NR	2.8 yrs	1.6	28.6	22.0	NR	NR

Values are % or mean ± SD. *The first percentage is those who had revascularization, and the second percentage is the proportion of those who underwent revascularization that was PCI. †Follow-up events for this study are Kaplan-Meier estimates.

ACS = acute coronary syndrome; F/U = follow-up; HF = heart failure; In-hosp = in-hospital; MI = myocardial infarction; NR = not reported; NSTEMI = non–ST-segment elevation myocardial infarction; Revasc = revascularization; STEMI = ST-segment elevation myocardial infarction; other abbreviations as in Tables 1 to 3.

Take Home Messages

- Dissection coronaire spontanée ➡ pathologie sous diagnostiquée
- Le diagnostic purement angiographique est possible, parfois trompeur, grand apport de l'**imagerie endocoronaire.**
- Pas de guidelines.
- **Une stratégie conservatrice** est souvent préconisée, sauf ischémie persistante, instabilité hémodynamique
- la récurrence n'est pas rare ➡ Follow up
- Les données des registres ➡ une meilleure compréhension et prise en charge des dissections coronaires spontanée .

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