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Catheter-induced coronary artery and aortic dissections. A study of mechanisms, risk factors and propagation causes

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Abstarct

Background: *Only the incidence, management, and prognosis of catheter-induced coronary artery and aortic dissections have been systematically studied until now. We sought to evaluate their mechanisms, risk factors, and propagation causes.*

Methods: *Electronic databases containing 76,104 procedures and complication registries from 2000–2020 were searched and relevant cineangiographic studies adjudicated.*

Results: *Ninety-six dissections were identified. The overall incidence was 0.126%, and 0.021% for aortic injuries. The in-hospital mortality rate was 4.2%, and 6.25% for aortic dissections. Compared to the non-complicated population, patients with dissection were more often female (48% vs. 34%, $p = 0.004$), with a higher prevalence of comorbidities such as hypertension (56% vs. 25%, $p < 0.001$) or chronic kidney disease (10% vs. 4%, $p = 0.002$). They more frequently presented with acute myocardial infarction (72% vs. 43%, $p < 0.001$), underwent percutaneous coronary intervention (85% vs. 39%, $p < 0.001$), and were examined with a radial approach (77% vs. 65%, $p = 0.011$). The most prevalent predisposing factor was small ostium diameter and/or atheroma. Deep intubation for support, catheter malalignment, and vessel prodding were the most frequent precipitating factors. Of the three dissection mechanisms, 'wedged contrast injection' was the commonest (the exclusive mechanism of aortic dissections). The propagation rate was 30.2% and led to doubling of coronary occlusions and aortic extensions. The most frequent progression triggers were repeat injections and unchanged catheter. In 94% of cases, dissections were inflicted by high-volume operators, with ≥ 5-year experience in 84% of procedures. The annual dissection rate increased over a 21-year timespan.*

Conclusions: *Catheter-induced dissection rarely came unheralded and typically occurred during urgent interventions performed in high-risk patients by experienced operators.* (Cardiol J 2024; 31, 3: 398–408)

Keywords: catheter-induced coronary dissection, iatrogenic aortocoronary dissection, percutaneous coronary intervention complications

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Introduction

Catheter-induced coronary artery dissection is an infrequent but potentially life-threatening complication of percutaneous procedures. We have recently studied its patterns in electrophysiological procedures; however, the incidence is much greater for coronary interventions [1, 2]. Involving proximal segments of major coronary vessels, it usually jeopardizes a large myocardial area and may propagate into the aorta more easily than guidewire-related dissections. It is also more dangerous than injuries induced by a balloon or stent because it may occur prior to coronary artery wiring or during diagnostic angiography.

Traditionally, studies of iatrogenic dissections focused on one dissected vessel or structure, be it the left main coronary artery (LMCA) or the sinus of Valsalva (SoV), thereby presenting only selective data [2–6]. Still others exclusively included dissections complicating specific procedures, such as chronic total occlusion interventions [7]. Registries of iatrogenic injuries of the aorta are also available, and although catheter-induced aortocoronary dissection is a distinct entity with its specific risks and treatment, coronary artery dissection and its retrograde extension into the aorta represent not only an anatomic but also a pathophysiologic continuum [8]. At the same time, various dissection-triggering devices (e.g. wires, stents) were usually taken into account, which precluded the analysis of mechanisms and factors specific to the catheter-related trauma. We sought to identify all catheter-induced injuries complicating coronary procedures to analyze the mechanisms of dissection, predisposing and precipitating factors, and propagation causes. Iatrogenic complications are avoidable by their nature; it is thus crucial to search for patterns and risk factors.

Methods

We performed a retrospective study of catheter-induced coronary artery and aortic dissections (CICAAD) at two high-volume centers. CICAAD cases (including coronary graft dissections) were retrieved from prospectively collected registries of procedural complications. They were double-checked by a query of electronic catheterization databases, utilizing keywords such as dissection, iatrogenic, spiral, or extraluminal. Recorded images of the right coronary artery (RCA) intervention with multiple stents as well as *ad hoc* angioplasty of LMCA were additionally reviewed to check for underreported cases. All CICAAD angiograms were assessed by two interventionists to confirm and classify a dissection, its mechanism, predisposing and precipitating factors, and propagation causes. If there was a discrepancy, a third reviewer was included in the adjudication.

Definitions

Dissections were graded according to the National Heart, Lung, and Blood Institute criteria, Eshtehardi and Dunning classification (**Suppl. Table 1**) [9, 10].

After the review of the first 60 cases, a consensus was reached to discern three main mechanisms of dissection. 'Wedged contrast injection' was defined as a primarily hydraulic injury caused by contrast agent delivery with damped outflow, i.e., catheter tip embedded in the artery wall. Initial vessel wall filling was usually recorded *in statu nascendi* with dye persistence afterwards (**Suppl. Video 1**). 'Forceful catheter engagement' (or vigorous pecking motion) described mechanical trauma caused by a catheter tip impact with a spot damage of a coronary artery or the aortic wall, typically inflicted at first contact with forceful intubation and immediately visible, or after vigorous pecking at a vessel wall (**Suppl. Video 2**). 'Deep catheter insertion' indicated a long-segment mechanical injury caused by deep intubation for support, or by inadvertent dragging of the catheter into a vessel during difficult device retrieval (**Suppl. Video 3**).

Multiple predisposing and precipitating factors suggestive of their role in the injury have been identified. They were assigned after cineangiography review. Since the project launch in 2017, operators provided internal reports detailing problems encountered during CICAAD procedures, with presumable triggers pointed out. Predisposing factors were defined as unmodifiable anatomic conditions, both normal (e.g., shepherd's crook take-off) and anomalous or pathological (e.g., arteria lusoria, ostial atheroma), involving the arterial access route or a coronary artery origin, making it difficult to advance and/or manipulate a catheter. Precipitating factors included modifiable conditions that could have been reversed or corrected, such as catheter behavior (e.g., respiratory instability), vessel response (e.g., concertina effect), or procedural conditions (excessive blending) (**Suppl. Video 4–6**).

Hemodynamic collapse was defined as new signs and symptoms of organ hypoperfusion with systolic blood pressure drop < 90 mmHg, fre-

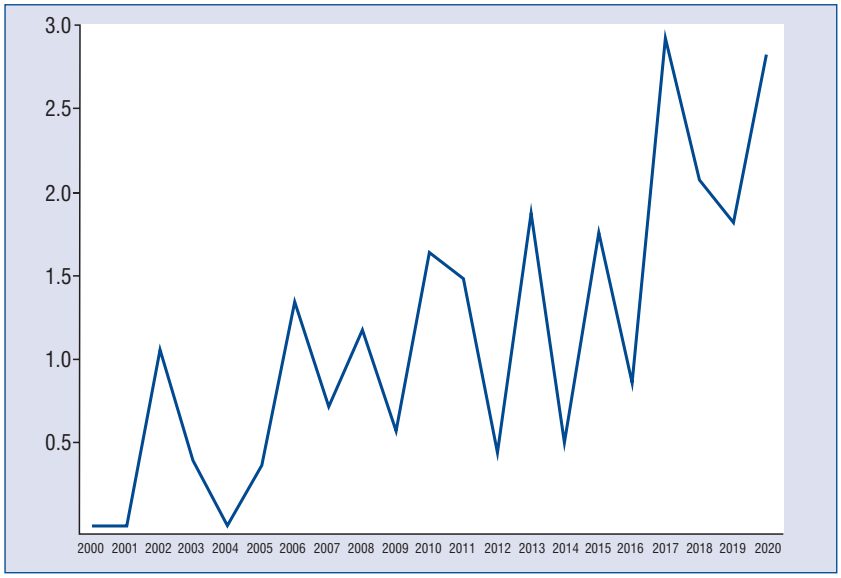

Figure 1. Annual number of catheter-induced coronary artery and aortic dissections per 1000 procedures.

quently requiring inotrope infusion and intra-aortic balloon pump insertion. Periprocedural infarction was defined according to the 2018 Fourth Universal Definition of Myocardial Infarction [11].

Low-, medium-, and high-volume operators were defined as per ACCF/AHA/SCAI 2013 [12] update of the competence statement on coronary interventions and the paper by Fanaroff et al. [13]. As complicated cases are usually taken over by senior interventionists, for the purpose of volume and experience estimation, the initial, culprit operator data was taken into account.

To account for each curve's dissection potential, the catheter culpability index (CCI) was calculated with the following formula (aortic CCI was calculated in the same way): $CCI = n/N \times 1000$, where n = number of dissections induced by a particular curve, and N = number of procedures in which the curve was used.

Statistical analysis

Categorical variables are presented as numbers and percentages, and compared by the Fisher exact test. Continuous variables were checked for normal distribution and expressed as means ± standard deviation, with comparisons made using Student's t-test. Due to unequal sample sizes of the CICAAD vs. non-CICAAD cohorts, the results were verified and confirmed with Mann-Whitney U test. P-values were two-sided with a significance level of 0.05. Analyses were performed using SPSS version 24.0 (IBM Corp., Armonk, NY, USA).

Results

CICAAD incidence

Overall, 76,104 diagnostic and therapeutic coronary procedures were performed between June 2000 and September 2020, and 96 cases of coronary artery (including 2 saphenous grafts) and aortic dissection were identified. The total incidence of CICAAD was 0.126%, with aortic involvement in 0.021% (16 cases). During the 21-year timespan, dissection occurred annually in 0–3 cases per 1000 procedures (Fig. 1). No dissection caused by sheathless and guide extension catheters was found.

CICAAD vs. non-CICAAD cohort

Compared to the non-CICAAD population, patients with dissection were significantly more often female (48% vs. 34%, $p = 0.004$), with a higher prevalence of comorbidities such as hypertension (56% vs. 25%, $p < 0.001$), chronic kidney disease (10% vs. 4%, $p = 0.002$), and prior stroke (8.3% vs. 2.8%, $p = 0.006$) (Table 1). They were more likely to present with acute myocardial infarction (72% vs. 43%, $p < 0.001$), and undergo percutaneous coronary intervention (PCI) (85% vs. 39%, $p < 0.001$), with transradial access also more frequently utilized in this group (77% vs. 65%, $p = 0.011$). Six French catheters were significantly more often used in the procedures complicated by dissection (81% vs. 44%, $p = 0.012$), as opposed to 5 French, which were more commonly utilized in the non-CICAAD group (55% vs. 16%, $p < 0.001$).

Table 1. Baseline characteristics.

	Non-CICAAD (n = 76008)	CICAAD (n = 96)	P
Age [years]	65.5 (11.5)	67.8 (11.6)	0.061
Female	25619 (33.7)	46 (47.9)	0.004
Body mass index	27.9 (4.5)	27.5 (4.4)	0.399
Diabetes mellitus	11503 (15.1)	21 (21.9)	0.085
Hypertension	18759 (24.7)	54 (56.3)	< 0.001
Chronic kidney disease	2719 (3.6)	10 (10.4)	0.002
Peripheral arterial disease	1417 (1.9)	5 (5.2)	0.034
Stroke	2119 (2.8)	8 (8.3)	0.006
COPD	1189 (1.6)	5 (5.2)	0.018
Prior MI	15101 (19.9)	22 (22.9)	0.449
Prior PCI	15950 (20.9)	29 (30.2)	0.029
Prior CABG	4479 (5.9)	6 (6.3)	0.827
CCS/CHF/valvular disease/other	35282 (46.4)	24 (25.0)	< 0.001
Acute coronary syndrome	40726 (53.6)	72 (75.0)	< 0.001
Unstable angina	7876 (10.4)	3 (3.1)	0.017
Acute MI	32850 (43.2)	69 (71.9)	< 0.001
NSTEMI	12176 (16.0)	29 (30.2)	< 0.001
STEMI	20674 (27.2)	40 (41.7)	0.002
Prehospital cardiac arrest	1103 (1.5)	4 (4.2)	0.052
Shock at admission	822 (1.1)	3 (3.1)	0.087
Femoral access	26615 (35.0)	22 (22.9)	0.015
Arm access (any arm artery)	49393 (65.0)	74 (77.1)	0.011
Diagnostic coronary angiography	46467 (61.1)	14 (14.6)	< 0.001
PCI	29541 (38.9)	82 (85.4)	< 0.001
CTO PCI	3104 (4.1)	7 (7.3)	0.117
Catheter size:			
4 French	371 (0.3)	1 (1.0)	0.375
5 French	65498 (55.2)	15 (15.6)	< 0.001
6 French	52451 (44.2)	78 (81.3)	0.012
7 French	247 (0.2)	2 (2.1)	0.040

Values are numbers (%) or means (± standard deviation); CABG — coronary artery bypass grafting; CCS — chronic coronary syndrome; CHF — congestive heart failure; CICAAD — catheter-induced coronary artery and aortic dissection; COPD — chronic obstructive pulmonary disease; CTO — chronic total occlusion; MI — myocardial infarction; NSTEMI — non–ST-segment elevation myocardial infarction; PCI — percutaneous coronary intervention; STEMI — ST-segment elevation myocardial infarction

Dissection and procedure characteristics

The right (RCA) and left (LCA) coronary artery were almost equally affected, in 47% vs. 46%, respectively (Table 2). The right SoV was dissected in twice as many cases as the left (8 vs. 4, respectively). The ascending aorta dissection extending above SoV occurred in 8 (8%) cases, in 6 involving the right cusp.

Reassuringly, all dissections manifested acutely during index procedure, and no cases of urgent reintervention due to delayed occlusion caused by unrecognized or conservatively treated dissection were found. In 30%, dissection occurred during an off-hours procedure (Table 2). Automatic injection of contrast was used in 35% of all CICAAD, and in 46% of aortic dissections. Dissections were managed conservatively in 28%, with stent in 71%, and surgically in 2%.

Hemodynamic collapse and mortality

The in-hospital mortality rate for all dissections was 4.2%, and 6.25% for dissections involving the aorta. Four patients with CICAAD died during index hospitalization, and in 3 of them, death could be attributed to the consequences of dissection or its management.

Table 2. Characteristics of procedures complicated by catheter-induced dissection.

Parameters	N = 96
Dissected characteristics	
Dissected vessel:	
LMCA	32 (33.3)
LAD	6 (6.3)
LCX	6 (6.3)
Anterolateral branch	1 (1.0)
All LCA dissections	44 (45.8)
RCA	45 (46.9)
SVG	2 (2.1)
Aortic involvement:	16 (16.7)
Sinus of Valsalva	12 (12.5)
Isolated SoV dissection	5 (5.2)
Right SoV	8 (8.3)
Left SoV	4 (4.2)
Ascending aorta above SoV:	8 (8.3)
Inflicted during RCA cannulation	6 (6.3)
Inflicted during LCA cannulation	2 (2.1)
Aortic involvement due to antegrade coronary propagation blocked by stent, CTO, or tight/calcified lesion	10 (10.4)
Dissection NHLBI type:	
Localized A-B	36 (37.5)
Extensive C-F	55 (57.3)
LMCA dissection Eshtehardi type I:	25 (26.0)
Type II	5 (5.2)
Type III	2 (2.1)
Aortic dissection Dunning type I:	8 (8.3)
Type II	5 (5.2)
Type III	3 (3.2)
Procedural characteristics	
Automatic contrast injection	34 (35.4)
Acute presentation of dissection	96 (100)
Dissection preceded by another complication (e.g., lost stent, broken wire)	8 (8.3)
Dissection followed by another intraprocedural complication (e.g., stent thrombosis)	5 (5.2)
Flow deterioration (by at least 1 TIMI grade)	24 (25.0)
Acute occlusion	18 (18.8)
Hemodynamic collapse due to dissection	10 (10.4)
Troponin rise indicative of periprocedural MI (or re-increase in acute MI)	18 (18.8)
Off-hours catheterization/PCI	29 (30.2)
Management:	
Conservative	27 (28.1)
Stenting (or balloon inflation)	68 (70.8)
Surgery	2 (2.1)
Operator's individual experience:	
Cath lab practice [years]	10.7 (5.5)
Mean procedures performed 12 months prior to CICAAD, n	750 (307)
Mean PCI performed 12 months prior to CICAAD, n	351 (168)
In-hospital adverse events:	
Death	4 (4.2)
Death in aortic dissection	1 (1.0)
Stroke	2 (2.1)
Urgent target vessel revascularization due to stent thrombosis or stent-related dissection	3 (3.1)
Shock	12 (12.5)
CA/VF/CPR during procedure	7 (7.3)
Stent intraprocedural/acute/in-hospital thrombosis	3 (3.1)

Values are numbers (%) or means (± standard deviation); CA — cardiac arrest; CICAAD — catheter-induced coronary artery and aortic dissection; CPR — cardiopulmonary resuscitation; CTO — chronic total occlusion; LAD — left anterior descending artery; LCA — left coronary artery; LCX — left circumflex artery; LMCA — left main coronary artery; MI — myocardial infarction; NHLBI — National Heart, Lung, and Blood Institute; PCI — percutaneous coronary intervention; RCA — right coronary artery; SoV — sinus of Valsalva; SVG — saphenous vein graft; TIMI — thrombolysis in myocardial infarction; VF — ventricular fibrillation

In 15.6% of cases, hemodynamic compromise was observed (Table 2). Five patients were either admitted in shock or manifested circulatory instability due to slow flow during primary angioplasty, while in the remaining 10, the collapse could be attributed to the dissection. Of the 10 dissection-related hemodynamic collapses (6 spiral RCA occlusions, 2 zipper and 1 spiral LMCA dissection, and 1 left anterior descending artery injury), in 8 cases vessel occlusion occurred (with cardiac arrest in 5). Eight of these patients had been admitted with acute coronary syndrome.

Predisposing and precipitating factors

In 52% of CICAAD, at least one predisposing factor was identified (Table 3). Cases of unfavorable origin of a coronary artery were more often found than unfavorable arterial access route (49% vs. 7%, respectively). Among the former, small proximal diameter and/or ostial atheroma were the most commonly identified (35%), whereas the most frequent access route obstacle was mediastinal arteries tortuosity.

The most commonly observed phenomena precipitating dissection were catheter deep seating or deep insertion during device delivery/removal (51%), catheter malalignment (34%), vessel prodding (32%), catheter systolic-diastolic mobility (27%), and respiratory instability (14%).

Mechanisms of dissection and its propagation

'Wedged contrast injection' proved to be the most common mechanism of CICAAD (46%) (Table 3). It was also responsible for all cases of primary aortic or aortocoronary dissection (11 patients). 'Forceful catheter engagement' and 'deep catheter insertion for device delivery/retrieval' were implicated in 28% and 26% of cases, respectively.

The overall propagation rate was as high as 30.2%. In 13.5% of CICAAD patients, the propagation was minor without flow impairment, while in the remaining 16.7%, it was serious, leading to spiral dissection, vessel occlusion, or retrograde extension into the aorta. The main causes of dissection expansion (multiple factors were often found in 1 patient) were repeat injections (62%), unchanged catheter (48%), and not immediately stenting a dissection (35%). Four out of 8 aortic dissections extending above the SoV occurred due to the progression of the original coronary artery injury. The main aortic propagation triggers (found in all such cases) were repeat injections and unchanged catheter. In 10 out of 16 aortic dissection cases, the retrograde propagation to the aorta was the consequence of downstream expansion having been blocked by a stent, chronic total occlusion, or a tight and/or calcified lesion.

Individual and institutional expertise

Most dissections (94%) were caused by high-volume interventionists (> 100 PCI/year), whose invasive experience in 84% of cases was at least 5 years (Fig. 2A, B). Only in 6 cases, an operator had performed < 100 PCI in the preceding 12 months and had < 3 years of experience. Dissections during angioplasty were caused by high-volume operators in 98% of cases (Fig. 2C).

Catheter culpability index

As per CCI, the most offending curves were HS III, ART4.5, IL3.5, and HS II (for catheter names see Table 4); however, being overall rarely used, they all caused only 6% of all dissections (Fig. 3; Table 4). The lowest CCI values (< 10) were calculated for EBU4.0, BLK4.0, and Judkins Left and Right curves, with Judkins curves being the most commonly used. The highest aortic CCI was calculated for HS II, AL1, and TIG4.0 curves, with AL1 implicated in 50% of aortic injuries.

Discussion

Catheter-induced coronary and aortic dissections have not been systematically studied as far as mechanisms of injury and its propagation, risk factors, and operator's competence are concerned. There is only anecdotal evidence on triggers and predictors of catheter-related trauma, and even the seminal papers by Dunning and Eshtehardi were based on small series of 9 and 28 patients, respectively [2, 9, 14].

Incidence

The largest study of catheter-induced coronary dissections so far reported 0.09% incidence and 5.9% mortality; findings similar to our population, with 80 isolated coronary injuries (0.105%) and 3.75% mortality [10]. A retrospective analysis of complications of diagnostic cardiac catheterization found coronary dissection in 0.003% of patients, corresponding to 0.004% in our sample (3 diagnostic angiography-related dissections) [15]. In the analyses of iatrogenic aortic dissections, triggers other than a catheter were also included, with the calculated catheter-induced injuries rate ranging from 0.017% to 0.067%, as compared to 0.021% in our material [4, 5]. The cited mortality rate for

Table 3. Risk factors and mechanism of dissection and its propagation.

Parameters	
Predisposing factors	**N = 49 (52.1)**
Unfavorable origin of coronary artery (n = 94, excluding SVG dissections n = 2), multiple choice:	46 (48.9)
Ostial-proximal atheroma and/or small ostial-proximal diameter (< 3.0 mm)	39 (41.5)
Ectopic coronary artery	3 (3.2)
Coronary ostium ectasia	2 (2.1)
Shepherd's crook	2 (4.3)
Unfavorable arterial access route (multiple factors in one patient not encountered):	7 (7.3)
Tortuosity of mediastinal arteries or low aortic origin of innominate artery	3 (3.1)
Dilated ascending aorta (aneurysm)	1 (1.0)
Arteria lusoria	1 (1.0)
Brachial loop	1 (1.0)
Difficult radial artery passage due to small diameter (or spasm)	1 (1.0)
Tortuous/stenosed iliac arteries	0
Abdominal aorta aneurysm	0
Precipitating factors (multiple choice)	**N = 82 (85.4)**
Catheter deep seating or deep insertion during device delivery/removal (> 10 mm)	49 (51.0)
Non-coaxial catheter alignment	33 (34.4)
Repeated vessel wall prodding with catheter tip	31 (32.3)
Catheter systolic-diastolic mobility (> 5 mm or dislodgement)	26 (27.1)
Too aggressive catheter	20 (20.8)
Too aggressive catheter — left coronary artery	16 (16.7)
Too aggressive catheter — right coronary artery	4 (4.2)
Catheter respiratory instability (> 10 mm or disengagement)	13 (13.5)
Excessive blending (catheter tip obscured)	11 (11.5)
Another complication preceding dissection (lost stent, broken wire, etc.)	7 (7.3)
Accordion effect	2 (2.1)
Coronary spasm	1 (1.0)
Mechanisms of dissection and its propagation	**N = 96**
Dissection mechanism (primary):	
Wedged contrast injection	44 (45.8)
Forceful catheter engagement (or vigorous pecking motion)	27 (28.1)
Deep catheter insertion (for device delivery or retrieval)	25 (26.0)
Aortocoronary dissection mechanism (aortic dissections secondary to propagation excluded):	N = 11
Wedged contrast injection	11 (100)
Propagation	**N = 29 (30.2)**
Propagation type:	
Minor	13 (13.5)
Vessel occlusion	9 (9.4)
Retrograde to SoV and into the ascending aorta above SoV	5 (5.2)
Non-occlusive spiral dissection	2 (2.1)
Propagation causes (multiple choice):	
Repeated injections	18 (62.1)
Unchanged catheter (or its position)	14 (48.3)
Dissection not stented immediately or incompletely covered	10 (34.5)
Dissection unnoticed or misinterpreted	8 (27.6)
Catheter inserted through dissected segment	3 (10.3)
Excessive blending	2 (6.9)
False lumen wired or stented	2 (6.9)
Wire removal	1 (3.4)

Values are numbers (%); SoV — sinus of Valsalva; SVG — saphenous vein graft

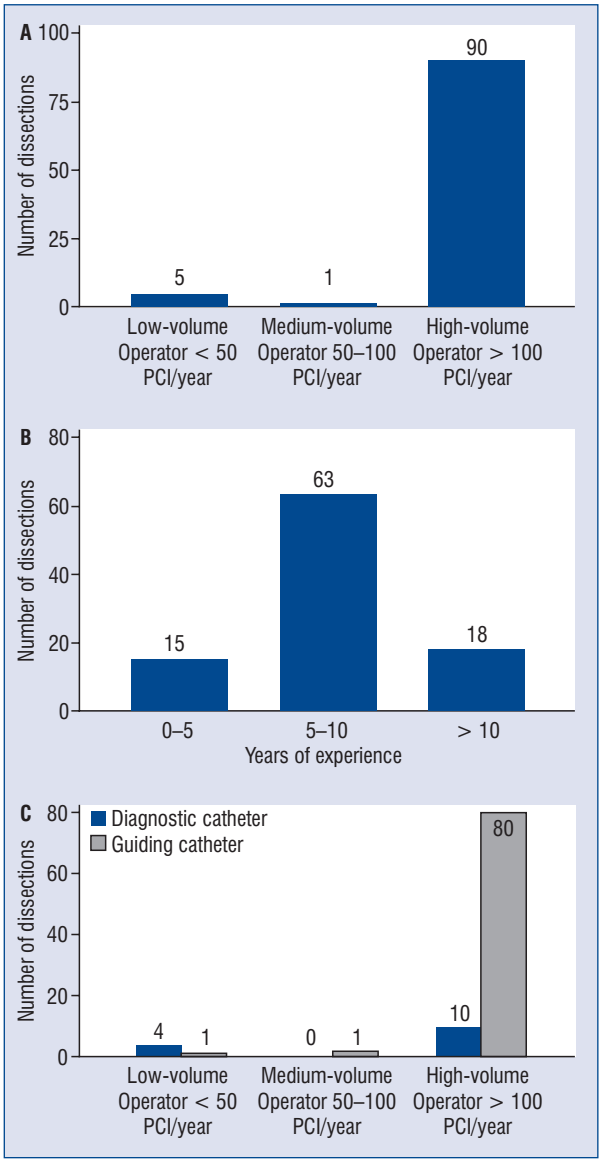

Figure 2. Operator's percutaneous coronary intervention (PCI) volume **(A)** and experience 12 months prior to dissection (**B**); **C.** Operator's PCI volume 12 months prior to dissection induced by a diagnostic vs. guiding catheter.

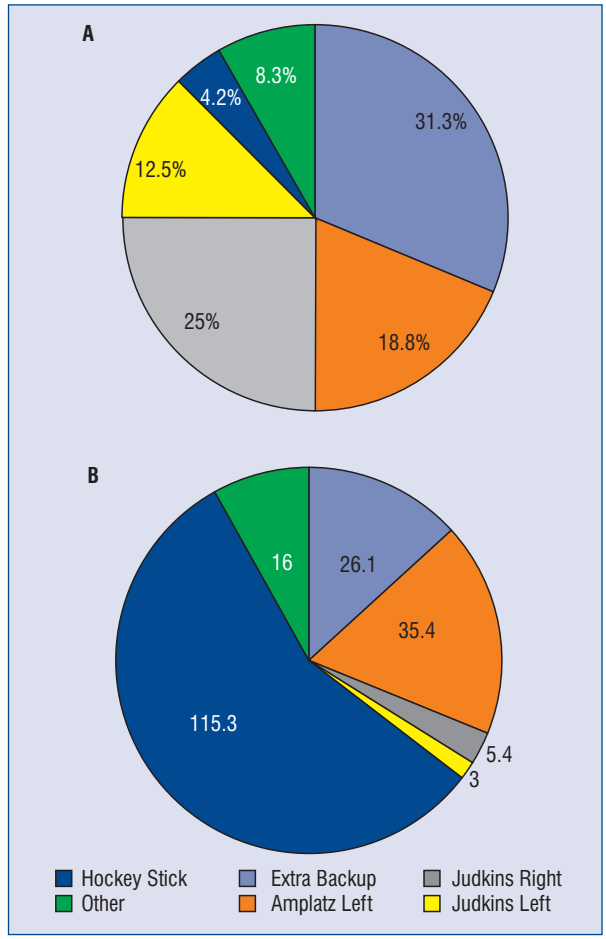

Figure 3. A. Catheter curves involved in dissections (%); **B.** Catheter Culpability Index as calculated for major catheter curves.

the aortic dissections was 2.7%, and 6.25% in our cohort. Dunning et al. [9] reported a 0.02% rate of iatrogenic aortocoronary injuries, which in our population amounted to 0.014% — 11 cases. In keeping with its reported vulnerability, the right SoV was dissected twice as often as the left, and the dissection propagated above the aortic root three times more often during RCA cannulation [5, 16]. A novel finding of the study is that in 62.5% of aortic dissections, the retrograde extension into the aorta occurred when the downstream progression was blocked by a stent, chronic occlusion, or a tight, calcified lesion. In the presence of such antegrade obstacles, catheter wedging is particularly dangerous because intramural contrast injection is redirected into the aortic root wall.

CICAAD vs. non-CICAAD cohort

We did not find any significant differences between CICAAD and non-CICAAD populations regarding age and body mass index. Not unexpectedly for an adverse event cohort, CICAAD patients had more comorbidities and more often presented with acute myocardial infarction. As in previous reports, we also observed a much higher rate of dissections induced by guiding as compared to diagnostic catheters (85% vs. 15%, respectively, $p < 0.001$), with a diagnostic catheter being responsible for only 1 out of the 16 cases of aortic involvement [2, 4, 5, 7, 10]. The relation between the access route and catheter-induced injury has

Table 4. Catheter culpability index (CCI) and aortic catheter culpability index (AoCCI).

Curve	Guiding/ /diagnostic catheter	4/5/6/7 French size	Percent of all catheters used	Number of dissections	CCI	Dissected vessel	Number of aortic dissections	AoCCI	Dissected aortic structure
HS III	2/0	0/0/1/1	0.10	2	181.8	2 RCA	0	0	0
ART 4.5	1/0	0/0/1/0	0.06	1	156.3	1 RCA	0	0	0
IL 3.5	1/0	0/0/1/0	0.06	1	149.3	1 LMCA	0	0	0
HS II	2/0	0/0/2/0	0.20	2	84.4	1 RCA	1	42.2	1 R-SoV + 1 AA
CLS 4.0	1/0	0/0/1/0	0.13	1	66.7	1 LMCA	0	0	0
LCB	1/0	0/0/1/0	0.13	1	66.2	1 SVG Ao-LAD	0	0	0
AL 1	18/0	0/1/17/0	4.39	18	35.4	16 RCA, 1 SVG Ao-RCA	8	15.7	6 R-SoV + 4 AA
EBU 3.75	21/0	0/0/20/1	5.38	21	33.8	16 LMCA, 3 LAD, 2 LCX	2	3.2	2 L-SoV + 1 AA
JCL 3.5	1/0	0/0/1/0	0.34	1	25.1	1 IM	0	0	0
EBU 3.5	8/0	0/2/6/0	2.77	8	25.0	5 LMCA, 1 LAD, 1 RCA	1	3.1	1 L-SoV
Q 3.5	1/0	0/0/1/0	0.40	1	21.5	LMCA	0	0	0
TIG 4.0	0/1	0/1/0/0	0.75	1	11.5	0	1	11.5	1 L-SoV
JL 3.5	2/4	1/2/3/0	8.98	6	5.8	3 LMCA, 1 LAD, 2 LCX	0	0	0
JR 4.0	19/4	0/5/18/0	36.03	23	5.5	21 RCA, 1 ectopic LCX	2	0.5	1 R-SoV + 1 AA
EBU 4.0	1/0	0/0/1/0	1.79	1	4.8	1 LMCA	0	0	0
JR 3.5	1/0	0/1/0/0	2.40	1	3.6	1 RCA	0	0	0
BLK 4.0	0/1	0/1/0/0	2.45	1	3.5	1 LMCA	0	0	0
JL 4.0	2/4	0/2/4/0	25.61	6	2.0	4 LMCA, 1 LAD, 1 LCX	1	0.3	1 AA
Catheter family									
Hockey Stick	4/0	0/0/3/1	0.30	4	115.3		1	28.8	
Extra BackUp	30/0	0/2/27/1	9.93	30	26.1		3	2.6	
Judkins Right	20/4	0/6/18/0	38.43	24	5.4		2	0.5	
Judkins Left	4/8	1/4/7/0	34.60	12	3.0		1	0.3	

Catheter name abbreviations: AL — Amplatz Left; ART — all Right; CLS — Contralateral Left Support; EB (or XB) — Extra BackUp; HS — Hockey Stick; IL — Ikari Left; JCL — Judkins Curve Left; JL — Judkins Left; JR — Judkins Right; LCB — Left Coronary Bypass; Q — Q curve; TIG — Tiger
Anatomical abbreviations: AA — ascending aorta; Ao — aorta; IM — intermediate branch; LAD — left anterior descending artery; LCX — left circumflex artery; LMCA — left main coronary artery; RCA — right coronary artery; SoV — sinus of Valsalva (R — right or L — left); SVG — saphenous vein graft
CCI (catheter culpability index) = the number of catheter-induced coronary artery and aortic dissections (CICAAD) events induced by a given curve divided by the number of procedures in which the curve was used ×10,000. Aortic CCI (AoCCI) was calculated in the same way.

not been evaluated so far, although 2 studies did observe a higher prevalence of transradial approach in procedures complicated by dissection [10, 17].

Predisposing and precipitating factors

Several types of ominous situations and dangerous catheter behavior portending dissection have been identified in the CICAAD cohort, suggesting that catheter-induced injuries rarely come unheralded. Many of them have been previously mentioned in case reports/series [2, 3, 14, 18]. Despite all the disadvantages of a retrospective analysis, only in 48% of patients were no predisposing factors found, and in just 14.6%, no precipitating factors were present, while in the rest, multiple triggers typically contributed to a dissection.

Data on the incidence of coronary artery origin variants in the general population are not available in the literature; however, with 14% and 13% rates, respectively, both the superior and inferior take-offs are likely to be overrepresented in the CICAAD group. Although not considered as

predisposing to dissection *per se*, they may impede coaxial catheter alignment and its stable positioning. Similarly, Shepherd's crook type RCA origin (4.3%) and coronary artery ectopy rate (3.2%), with cited incidence for the latter of 0.05–0.1%, were relatively high in our cohort.

Mechanisms of dissection and its propagation

Wedged contrast injection appears to be the commonest and most dangerous dissection mechanism. It was implicated in 46% of coronary dissections, 75% of spiral dissections, and was exclusively responsible for both primary and secondary (i.e. caused by propagation) aortic injuries. Although not always the principal mechanism of injury, intentional or inadvertent deep catheter insertion was observed in nearly half of CICAAD, suggesting that deep penetration of the vessel may facilitate subsequent dissection by its wall disruption. The mechanisms identified in our analysis, although never systematically studied before, agree with the observations of previous case series [2, 3, 14, 18].

The iatrogenic propagation rate of 30% is a disturbing finding, and although local expansion cases were also included, repeated injections or sticking to the culprit catheter led to the doubling of occlusive dissections. Moreover, out of 8 aortic dissections extending above SoV, 4 could have been avoided because they were caused by a retrograde progression. In a 2010 study of iatrogenic LMCA dissections, the reported propagation rate was also alarmingly high, reaching 32% [2].

Operator's and center's expertise

Both individual and institutional learning curves have been described for coronary invasive procedures, and an inverse relationship between operator's angioplasty volume and in-hospital mortality was reported [13]. However, in most of the study cases, dissections were inflicted by high-volume operators with extensive experience. Despite equipment sophistication, the annual rate of dissections did not decrease with growing individual and institutional experience, showing an upward trend (Fig. 1). This fact, however disappointing, may at least partially be explained by more complex procedures having been performed over time, with older and sicker patients qualified to intervention.

Catheter culpability index

The most harmful catheter curves are reported in our study not just in absolute numbers but in relation to their overall use. CCI was calculated to compensate for the fact that the most frequently utilized catheters typically cause more dissections (Fig. 3). Non-conventional guides (e.g., HS or ART) are more challenging to handle and thus less commonly used, which accounts for an explosive combination of operator's low familiarity and catheter's high aggressiveness. Even with their infrequent usage, their CCI value was disproportionately high. Uncommonly employed curves (< 1% usage) accounted for 12% of dissections, although they comprised only 2.2% of all catheters used. Regular extra support guides like AL1 and EBU (both accounting for 14% of used catheters), despite their reported notoriety, had relatively low CCI, even though they were responsible for almost half of dissections (49%) [4, 9, 10]. Amplatz Left, however, had the second-largest aortic CCI and was implicated in nearly half of aortic dissections (7/16), as also suggested by previous studies [4, 8]. This correlation may be due to Amplatz's frequent usage in RCA interventions and RCA's susceptibility to retrograde dissection (Table 4) [16]. In fact, of all 18 dissections induced by Amplatz, 17 involved RCA and/or the right SoV. All culprit Amplatz Left catheters were guiding ones, and all of them were 6 French except one 5 French. Equally surprising was a good track record of bilateral catheters, infamous for popping into coronary ostia and dissection potential [3, 18]. On the other hand, despite being implicated in 37.5% of dissections, Judkins catheters had the lowest CCI thanks to their frequent utilization (73% of all used catheters).

Limitations of the study

Evaluation of uncommon complications is necessarily based on retrospective analyses. This is the largest series presented so far; however, 96 is too small a number of patients to draw definite conclusions. Despite the relative uniformity of techniques, our study may not reflect practices and equipment used elsewhere. Over the 21-year period, catheters from several manufacturers have been used, differing in material, and thus in stiffness of the shaft and tip, with slight differences also in their shape despite the same curve name, all of which may have additionally affected the dissection potential of a given catheter type. To limit the observer bias, all cases were reviewed by 2 operators, with a third interventionist called in whenever a discrepancy arose. Risk factors such as prohibitive chest vasculature, or radial artery spasm, were probably more prevalent than could be deduced *a posteriori*. As in any *post hoc* analysis, the causative role of the identified precipitating

factors is speculative; however, they were repeatedly observed in the studied cohort and regularly preceded the injury.

Conclusions

It was the complexity and urgency of a procedure that decided the fate of a dissection rather than the operator's inexperience. The significant number of serious propagations underscores how many severe complications could potentially be avoided.

Deeper understanding of the mechanisms and risk factors of catheter-induced dissections may help their prevention and enable prompt recognition and management.

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