Comparison of Coronary Culprit Lesion Morphology Determined by Optical Coherence Tomography and Relation to Outcomes in Patients Diagnosed with Acute Coronary Syndrome During Winter −vs− Other Seasons

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Patients diagnosed with acute coronary syndrome (ACS) during winter have worse outcomes; however, mechanisms driving this trend are unclear. We examined coronary culprit lesion morphologies using optical coherence tomography (OCT). Features and outcomes were retrospectively compared between patients admitted with ACS in winter (W-ACS; n = 390) and in other seasons (O-ACS; n = 1,027). Angiography and OCT results were analyzed in patients who underwent OCT examination (173 patients in W-ACS and 450 in O-ACS). On initial angiography, minimum lumen diameter was smaller (median; 0.12 mm vs 0.25 mm, p = 0.021) and Thrombolysis in myocardial infarction flow grade was worse (Thrombolysis in myocardial infarction 0/1; 57% vs 44%, p = 0.005) in W-ACS. OCT performed before coronary interventions or just after intracoronary thrombectomy showed that plaque rupture (56% vs 46%) and calcified nodules (8% vs 5%) were more prevalent, and plaque erosion (37% vs 49%) was less prevalent in W-ACS (p = 0.039 for all 3 variables). At 2-year follow-up for all admitted ACS patients, Kaplan-Meier estimates showed higher cardiac mortality in W-ACS (11.8% vs 8.3%, p = 0.043). Multivariate Cox proportional hazard analysis showed that patients in W-ACS group had a 1.5-fold increased risk of cardiac death within 2 years after adjusting for traditional cardiovascular risk factors (hazard ratio, 1.54 [95% confidence interval, 1.06 to 2.23]; p = 0.024). In conclusion, patients diagnosed with ACS during winter had worse angiographic results and OCT revealed less plaque erosion (more plaque rupture or calcified nodules) at the culprit lesions, which may be partly associated with worse cardiac mortality within 2 years. **(Am J Cardiol 2019;124:31−38)**

Acute coronary syndrome (ACS) is the leading cause of mortality in developed countries, and the risk stratification of patients with ACS is very important. Although an increase in mortality rate in patients diagnosed with ACS during winter months is reported worldwide,[1−7] mechanisms driving this trend have not been clarified. In particular, studies investigating the association between seasonal variation of ACS and culprit lesion morphologies are lacking. Optical coherence tomography (OCT) is a useful method for examining underlying plaque morphologies at ACS culprit lesions owing to its high resolution.[8,9] The aims of the present study were (1) to compare culprit lesion plaques morphologies determined by OCT between patients diagnosed with ACS during winter and those diagnosed during other seasons and (2) to determine the long-term cardiac outcomes related to seasonal variation of ACS.

Methods

A total of 1,417 consecutive patients with ACS transferred to the emergency department (ED) at Nippon Medical School Chiba Hokusoh Hospital from July 2008 to June 2016 were enrolled into the present study. In them, patients admitted from December to February were defined as patients diagnosed with ACS during winter (Winter-ACS; W-ACS), and patients admitted with ACS in the other months were allocated to the group of Other-ACS (O-ACS).Winter months were designated using the average monthly maximum, mean, and minimum temperatures recorded from July 2008 to June 2016 in the local area (35°43.7' north latitude and 140°12.7' east longitude) (Figure 1). The data was obtained from the Japan Meteorological Agency website; http://www.data.jma.go.jp/obd/stats/etrn/index.php. For an ACS diagnosis, patients

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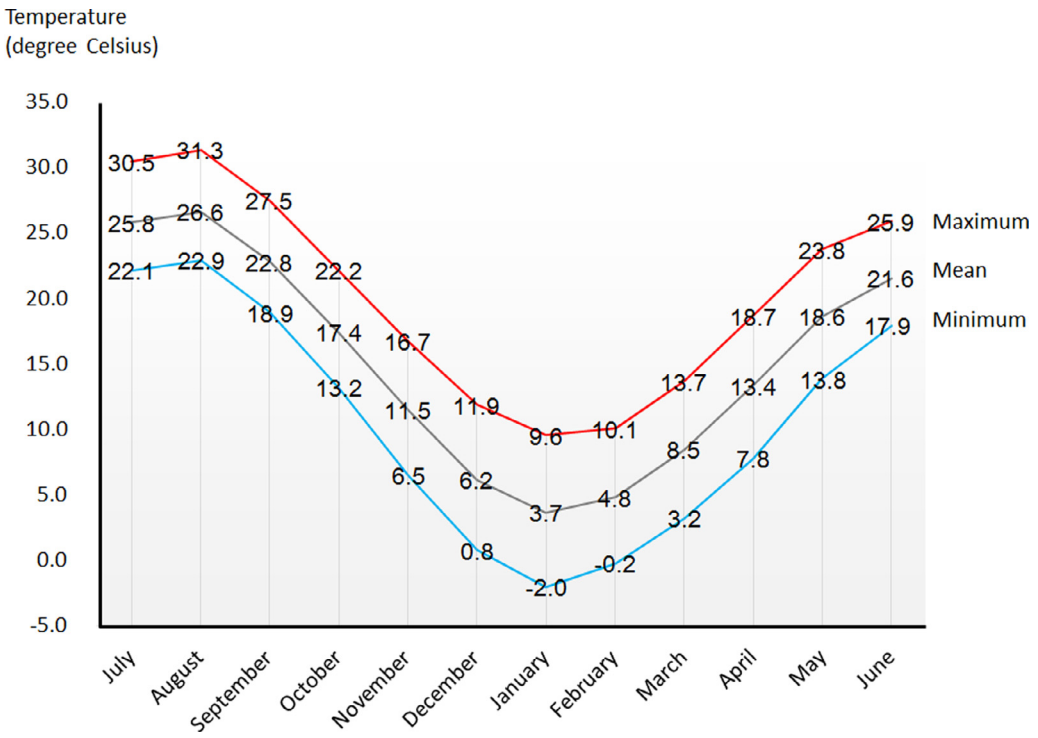

Figure 1. Monthly maximum, mean, and minimum temperature.
Average of monthly maximum (*red line*), mean (*gray line*), and minimum (*blue line*) temperatures at 35°43.7' north latitude and 140°12.7' east longitude from July 2008 to June 2016 are shown. (Color version of figure is available online.)

had to present at least 2 of the following criteria: (1) symptoms of cardiac ischemia, such as prolonged chest pain (≥20 minutes); (2) new or presumed new significant ST-segment-T wave changes or new left bundle branch block in electrocardiography; or (3) elevated cardiac troponin T levels. Patients diagnosed with ACS and with elevated cardiac troponin T (>99th percentile upper reference limit)[10] upon arrival or elevated peak creatine kinase MB (CK-MB; more than double the local laboratory upper limit of normal) were diagnosed with myocardial infarction (MI). Cases with neither elevated cardiac troponin T upon admission nor elevated peak CK-MB were diagnosed with unstable angina. ST-segment elevation ACS and non-ST-segment elevation ACS were defined as ACS with and without new (or presumably new) ST-segment elevation (≥0.1 mV) in 2 or more contiguous leads on electrocardiography, respectively. Baseline characteristics, laboratory data, and clinical outcomes over the 2-year follow-up period were compared between the groups. Peripheral blood samples were obtained upon arrival at the ED. To evaluate peak CK-MB values, blood samples were obtained serially every 6 hours after arrival at the ED until the CK-MB value began to decline. Time intervals from onset of chest pain to arrival at the ED were determined by medical interviews. The Ethics Committee of the hospital approved the study protocols.

Angiographic analyses were performed on patients that underwent an OCT examination before percutaneous coronary intervention or just after intracoronary thrombectomy (Figure 2). Initiation of OCT examination was per operator discretion. The culprit lesion was identified based on coronary angiography, electrocardiography, and echocardiography. Culprit lesions previously treated by balloon angioplasty, stents, or coronary bypass grafting were excluded from the current angiography and OCT analyses. Qualitative and quantitative coronary angiographic analyses were performed for the following variables: lesion location and length, pre- and postintervention minimal lumen diameter, % diameter stenosis, reference vessel diameter, Thrombolysis in myocardial infarction (TIMI) flow grade and Rentrop collateral circulation grade classification.

For cases with a TIMI grade <2 on the initial coronary angiography, intracoronary thrombectomy was performed before OCT examination. OCT imaging of culprit lesions was performed using either a commercially available time-domain OCT (Model M3 Cardiology Imaging System, St. Jude Medical, Westford, Massachusetts) or a frequency-domain OCT (C7XR system and the Dragon Fly catheter, Abbott Vascular, Santa Clara, California). The OCT procedure has been described previously.[11,12]

All OCT images were analyzed based on previously established criteria[13,14] and using agreeing interpretations from 2 independent cardiologists (N. Kobayashi and M. Tsurumi) blinded to the clinical presentation. Culprit lesion plaque morphology was classified as plaque rupture, plaque erosion, calcified nodules, or "others," according to previously published criteria.[15] Plaque rupture was identified by the presence of fibrous cap discontinuity with a clear cavity inside the lipidic plaque. Plaque erosion was identified by the absence of fibrous cap disruption with the presence of attached thrombi overlying an intact and visualized plaque, luminal surface irregularities at the culprit lesion in the absence of thrombi, or attenuation of underlying plaque by thrombus without superficial lipid or calcification immediately proximal or distal to the thrombus site. A calcified

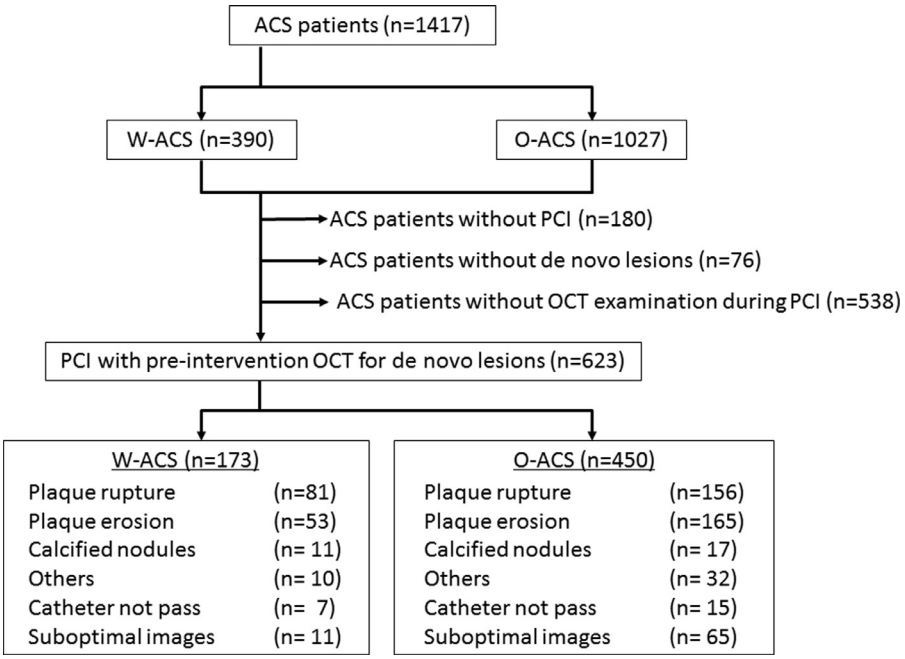

Figure 2. Patient enrollment.
A total of 1,417 consecutive patients with acute coronary syndrome (ACS) were divided into 2 groups: patients diagnosed during winter (W-ACS; n = 390) and those diagnosed during other seasons (O-ACS; n = 1,027). Of them, 623 underwent an optical coherence tomography (OCT) before percutaneous coronary intervention (PCI) (or just after intracoronary thrombectomy). Detailed analyses of coronary angiography and OCT were performed for patients with plaque rupture, plaque erosion, and calcified nodules (145 patients in the W-ACS and 338 patients in the O-ACS groups).

nodule was defined as a superficial calcified plaque with protruding nodular calcium and attached thrombus. Culprit plaques that did not meet the aforementioned criteria were classified as "others." Cases in which the OCT catheter could not pass the lesion before balloon angioplasty were excluded from the OCT analyses. OCT images with of suboptimal quality for analyses due to massive thrombi or catheter motion artifact were also excluded.

Further qualitative and quantitative OCT analyses were performed on patients with plaque rupture, plaque erosion, or calcified nodules on the culprit lesions, which were the main cause of ACS, as described previously.[15,16] Slices with minimum lumen cross-sectional areas on post-thrombectomy and poststenting OCT images were identified and assessed. Proximal and distal 5-mm long segments from each stent edge, but before a significant (>1.5 mm in diameter) side branch, were defined as reference segments. The slices with the smallest lumen cross-sectional areas within each reference segment were also identified. Tissue types of underlying atherosclerotic plaque were classified into fibrous plaque (a relatively homogeneous OCT signal region with high backscattering), lipidic plaque (a signal-poor OCT region within an atherosclerotic plaque with poorly delineated borders) or calcified plaque (a signal-poor or heterogeneous OCT region with a sharply delineated border). Intracoronary thrombi were defined as a mass with diameter >250 μm attached to the luminal surface or floating within the lumen. Red thrombi were defined as thrombi with high backscattering and high attenuation, and white thrombi as thrombi with less backscattering and low attenuation.

Clinical follow-up was performed over 2 years and the incidences of cardiac death were investigated. Cardiac death was defined as death from MI, cardiac perforation or pericardial tamponade, arrhythmia or conduction abnormality, procedural complications, or any death in which a cardiac cause could not be excluded.

Categorical variables were summarized using percentages and counts and were compared using chi-square statistics or the Fisher's exact test where appropriate. Continuous variables were compared using the nonparametric Mann-Whitney *U* test and are shown as median and interquartile range. Time-to-event data were summarized as Kaplan-Meier estimates and were compared between groups with the log-rank test. A Cox proportional hazards regression model was conducted to determine the independent predictors for cardiac death within 2 years whereas simultaneously controlling for potential confounders. In addition to admission during winter, age, male gender, and history of diabetes mellitus, hypertension, dyslipidemia and old MI, were initially selected as candidates for independent predictors in the model. Intra- and interobserver variability for diagnosing plaque rupture, plaque erosion, and calcified nodules were measured with the k test of concordance. Statistical analyses were conducted using the SPSS software package, version 22.0, with a p value of <0.05 considered statistically significant.

Results

In 1,417 patients with ACS, 390 were admitted during the winter months (16.3 per month) and 1,027 were admitted during the other months (14.3 per month) (Figure 2). Table 1 compares baseline clinical characteristics and laboratory data between W-ACS and O-ACS.

Table 1
Comparisons of baseline clinical characteristics and laboratory data

Variable	Winter (n = 390)	Other (n = 1,027)	p Value
Men	317/390 (81%)	814/1,027 (79%)	0.397
Age (years)	66 (57, 75)	67 (59, 75)	0.495
Diabetes mellitus	241/387 (38%)	383/1,021 (38%)	0.941
Hypertension*	123/387 (68%)	690/1,020 (68%)	0.838
Dyslipidemia†	233/387 (60%)	608/1,021 (60%)	0.822
Any smoking	247/385 (64%)	697/1,018 (68%)	0.125
Old myocardial infarction	29/387 (7%)	115/1,022 (15%)	<0.001
Body mass index (kg/m^2)	23.9 (21.9, 25.9)	23.9 (21.6, 26.4)	0.528
Systolic blood pressure (mm Hg)	136 (114, 158)	136 (112, 156)	0.882
Diastolic blood pressure (mm Hg)	80 (60, 90)	77 (62, 90)	0.272
Heart rate (beats/minute)	79 (66, 94)	76 (64, 90)	0.076
ST-segment elevation	263/390 (68%)	643/1,018 (63%)	0.072
Myocardial infarction	369/390 (95%)	942/1,027 (92%)	0.065
Killip class			
I	314 (81%)	783 (76%)	0.086
II, III, IV	76 (19%)	244 (24%)	
Time from symptom onset to arrival at the ED (minutes)	200 (87, 757)	234 (90, 805)	0.810
Left ventricular ejection fraction (%)‡	54 (45, 61)	52 (42, 62)	0.291
Laboratory data			
White blood cell (per μL)	9140 (7225, 11625)	9070 (6988, 11810)	0.882
Hemoglobin (g/dl)	14.1 (12.3, 15.3)	14.0 (12.6, 15.3)	0.205
Platelet ($10^4/\mu$L)	20.6 (17.6, 24.8)	19.9 (16.7, 24.4)	0.202
Creatinine (mg/dl)	0.85 (0.68, 1.07)	0.86 (0.71, 1.09)	0.625
Blood glucose (mg/dl)	156 (127, 222)	149 (120, 206)	0.128
Low-density lipoprotein cholesterol (mg/dl)	114 (91, 142)	110 (88, 139)	0.505
High-density lipoprotein cholesterol (mg/dl)	45 (37, 53)	43 (36, 53)	0.023
Triglyceride (mg/dl)	96 (63, 147)	106 (65, 164)	0.265
Hemoglobin A1c (%)	5.9 (5.5, 6.8)	5.9 (5.6, 6.6)	0.564
C-reactive protein, mg/dl	0.20 (0.07, 0.63)	0.16 (0.07, 0.54)	0.240
B-type natriuretic peptide (pg/ml)	96 (31, 268)	78 (29, 240)	0.954
Creatine kinase-MB on admission (U/L)	16 (10, 36)	16 (10, 38)	0.102
Peak creatine kinase-MB (U/L)	95 (37, 228)	90 (26, 237)	0.386
Revascularization procedure			
PCI with bare-metal stents	151 (43)	398 (42)	0.877
PCI with drug-eluting stents	144 (41)	407(43)	
PCI without stents	40 (11)	97 (10)	
Coronary artery bypass grafting	15 (4)	37 (4)	
Access site in PCI procedure			
Trans radial/brachial artery	160 (48)	437 (48)	0.830
Trans femoral artery	175 (52)	465 (52)	

ED = emergency department. PCI = percutaneous coronary intervention. Values are shown as median (interquartile range) or n (%).

* Defined as systolic blood pressure ≥140 mm Hg and/or diastolic blood pressure ≥85 mm Hg before admission, or the use of antihypertensive medication.

† Defined as low-density lipoprotein cholesterol ≥140 mg/dl, high-density lipoprotein cholesterol <40 mg/dl, and/or triglyceride ≥150 mg/dl, or treatment with lipid lowering drugs.

‡ Calculated by transthoracic echocardiography at emergency department.

Table 2 compares angiographic results between the W-ACS and the O-ACS groups. Minimum lumen diameter was smaller (median; 0.12 mm vs 0.25 mm, p = 0.021) and TIMI flow grade was worse (TIMI 0/1 57% vs 44%, p = 0.042) in W-ACS than in O-ACS. OCT findings are compared in Table 3. Plaque erosion was less prevalent in W-ACS than in O-ACS (37% vs 49%, p = 0.039). Inter- (N. Kobayashi and M. Tsurumi) and intraobserver (N. Kobayashi, 4 months apart) variability values for categorizing plaque rupture, plaque erosion, and calcified nodules were 0.89 and 0.91, respectively.

Comparisons of Kaplan-Meier estimates of cardiac death for the 2-year follow-up period between the W-ACS and O-ACS groups are shown in Figure 3. The incidence was significantly greater in W-ACS (11.8% vs 8.3%, p = 0.043). When comparing cardiac mortality in patients who underwent a preintervention OCT examination, this difference was not significant (2.5% vs 3.1%, p = 0.694). There was a large difference in the mortality between patients who received a preintervention OCT examination for a de novo lesion (n = 623) and those who did not (n = 538) (2.9% vs 13.3%, p < 0.001). Multivariate Cox proportional hazard analysis showed that a W-ACS diagnosis (hazard ratio 1.54 [95% confidence interval 1.06 to 2.23], p = 0.024) independently and significantly predicted risk of cardiac death within 2 years (Table 4).

Table 2
Comparisons of angiographic findings in patients with plaque rupture, plaque erosion, or calcified nodules identified by OCT

	Winter (n = 173)	Other (n = 450)	p Value
Preintervention angiographic results			
Number of diseased vessels*			
One	86 (49%)	233 (52%)	0.622
Two	52 (30%)	139 (31%)	
Three	36 (21%)	78 (17%)	
Culprit artery			
Right	60 (35%)	152 (34%)	0.974
Left anterior descending	87 (50%)	227 (51%)	
Left circumflex	26 (15%)	70 (16%)	
ACC/AHA classification type B_2/C	169 (98%)	419 (93%)	0.026
Lesion length (mm)	14.1 (10.6, 22.2)	13.7 (10.5, 19.8)	0.523
Minimum lumen diameter (mm)	0.12 (0.00, 0.38)	0.25 (0.00, 0.45)	0.021
Reference vessel diameter (mm)	2.58 (2.20, 3.04)	2.70 (2.27, 3.10)	0.246
Diameter stenosis (%)	94.9 (84.8, 100)	90.2 (83.2, 100)	0.034
TIMI flow grade			
0 or 1	98 (57%)	199 (44%)	0.005
2 or 3	75 (43%)	252 (56%)	
Collateral circulation			
Rentrop 0 or 1	117 (69%)	337 (76%)	0.067
Rentrop 2 or 3	53 (31%)	106 (24%)	
Postintervention angiographic results			
Minimum lumen diameter (mm)	2.64 (2.28, 2.99)	2.65 (2.34, 3.04)	0.637
Reference vessel diameter (mm)	2.87 (2.58, 3.26)	3.01 (2.63, 3.37)	0.162
Diameter stenosis (%)	8.7 (5.8, 11.6)	8.7 (6.0, 12.5)	0.619
TIMI flow grade			
0, 1 or 2	12 (7%)	23 (5%)	0.376
3	161 (93%)	427 (95%)	

ACC = American College of Cardiology; AHA = American Heart Association; OCT = optical coherence tomography; TIMI = Thrombolysis in Myocardial Infarction.

Values are shown as median (interquartile range) or n (%).

* Defined as diameter stenosis ≥50%.

Table 3
Comparisons of OCT findings in patients with plaque rupture, plaque erosion, or calcified nodules

	Winter (n = 145)	Other (n = 338)	p Value
Post-thrombectomy OCT findings			
Lesion length (mm)	15.0 (11.3, 19.0)	15.5 (12.6, 22.6)	0.949
Minimum lumen CSA (mm^2)	0.90 (0.62, 1.20)	0.97 (0.78, 1.24)	0.170
Distal reference lumen CSA (mm^2)	4.55 (3.53, 6.79)	5.14 (3.80, 6.66)	0.075
Proximal reference lumen CSA (mm^2)	6.28 (4.76, 8.49)	6.71 (5.29, 8.58)	0.091
Plaque morphology			
Rupture	81 (56%)	156 (46%)	0.039
Erosion	53 (37%)	165 (49%)	
Calcified nodule	11 (8%)	17 (5%)	
Lipidic plaque	127/145 (88%)	302/338 (89%)	0.573
Lipidic plaque arc (°)	262 (220, 295)	243 (199, 280)	0.105
Lipidic plaque length (mm)	13.4 (9.1, 17.9)	13.0 (9.4, 17.9)	0.878
Fibrous cap thickness (mm)	0.060 (0.057, 0.087)	0.063 (0.060, 0.090)	0.346
Calcium	71/145 (49%)	138/338 (41%)	0.098
Thrombus	138/145 (95%)	318/338 (94%)	0.633
Red thrombus	73/138 (53%)	147/318 (46%)	0.190
Poststenting OCT findings			
Minimum lumen CSA (mm^2)	4.86 (3.77, 6.78)	5.14 (4.03, 6.28)	0.142
Distal reference lumen CSA (mm^2)	5.10 (3.82, 6.61)	5.59 (4.16, 7.39)	0.055
Proximal reference lumen CSA (mm^2)	6.63 (5.17, 8.54)	7.01 (5.29, 8.89)	0.196

CSA = cross-sectional area; OCT = optical coherence tomography.

Values are shown as median (interquartile range) or n (%).

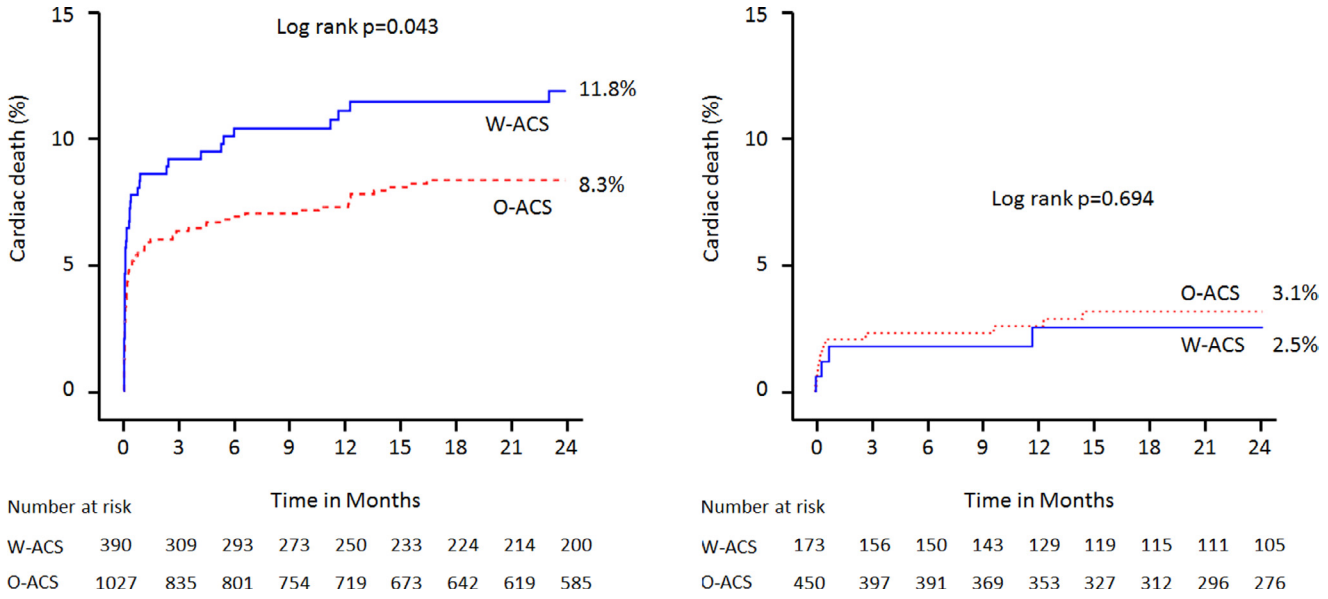

Figure 3. Kaplan-Meier estimates of cumulative incidences of cardiac death at 2 years.
Time-to-event curves of cardiac death throughout the 2-year follow-up period of patients diagnosed with acute coronary syndrome (ACS) during winter (W-ACS; *blue solid line*) and other seasons (O-ACS; *red dotted line*) in the total sample of patients (*left*) and those who underwent an optical coherence tomography examination (*right*). (Color version of figure is available online.)

Discussion

The present study sought to clarify the differences in culprit lesion morphologies between cases of ACS diagnosed during winter and other seasons, and examined then the impact of ACS presenting in winter on cardiac outcomes. The major findings of the present study were: (1) angiographic results before coronary intervention were worse if ACS occurred during winter; (2) preintervention OCT results showed that plaque erosion was less (i.e., plaque rupture and calcified nodules were more) prevalently observed by OCT in patients with ACS presenting during winter; and (3) 2-year cardiac mortality was worse in patients admitted with ACS during winter.

Increased cardiac mortality in patients diagnosed with ACS during winter has been observed in several countries including the United States,[1] Canada,[2] United Kingdom,[3] Portugal,[4] New Zealand,[5] Brazil,[6] and Japan.[7] However, the mechanisms driving this trend remain unclear. Multiple factors including increases in arterial blood pressure,[17,18] blood viscosity,[17] red cell and platelet counts,[17] activation of fibrinogen and factor VII,[19] cholesterol levels,[19] and respiratory infections[20,21] would play a role in the seasonal variation of ACS. In the present study, there were no significant differences in blood pressure, hemoglobin, platelet, low-density lipoprotein cholesterol, and triglyceride levels upon admission between the W-ACS and O-ACS groups, except that high-density lipoprotein cholesterol levels were higher in the W-ACS group than in the O-ACS group.

Seasonal variations of angiographic and intracoronary imaging findings in patients with ACS have not been investigated previously. We focused on plaque morphology identified by OCT in ACS culprit lesion to elucidate the seasonal variation of ACS occurrence. OCT is superior to other coronary imaging modalities for detecting plaque morphology in patients with ACS because of its high resolution.[8,9] Thus, OCT was used to clarify the associations between clinical presentations and coronary culprit lesion plaque morphologies in patients with ACS.[22,23] OCT revealed that plaque rupture and calcified nodules were

Table 4
Cox proportional hazards analysis for predictors of cardiac death within 2 years

	Univariable analysis			Multivariable analysis		
	Hazard Ratio	95% CI	p Value	Hazard Ratio	95% CI	p Value
W-ACS	1.46	1.01−2.12	0.045	1.54	1.06−2.23	0.024
Age, (10-year increase)	1.42	1.21−1.67	<0.001	1.43	1.21−1.69	<0.001
Male gender	1.28	0.79−2.06	0.319	1.52	0.93−2.49	0.097
Diabetes mellitus	1.34	0.94−1.91	0.111	1.38	0.96−1.99	0.083
Hypertension	0.98	0.67−1.43	0.914	0.90	0.61−1.32	0.573
Dyslipidemia	0.53	0.37−0.76	0.001	0.53	0.37−0.77	0.001
Old myocardial infarction	1.65	1.01−2.68	0.046	1.73	1.05−2.84	0.032

CI = confidence interval; W-ACS = acute coronary syndrome diagnosed during winter.

more prevalent in the W-ACS group, suggesting that patients diagnosed with ACS during winter had more advanced atherosclerotic lesions. Surprisingly, the lipidic plaque length and arc and fibrous cap thickness were comparable between the W-ACS and O-ACS groups, which might indicate that a lipidic plaque is more prone to rupture in winter than in other seasons. Culprit lesion plaque morphologies resulted in different angiographic results between patients diagnosed with ACS during winter and other seasons. Culprit lesion complexity was more severe (i.e., type B2/C lesions were more prevalent), initial minimum lumen diameter was smaller and initial TIMI flow grade was worse (i.e., TIMI 0/1 was more frequent) in patients with ACS during winter. These characteristics would be associated with a higher prevalence of plaque rupture in patients identified with ACS during winter. Finally, a higher prevalence of plaque rupture identified through OCT and a worse anterograde flow on initial angiography resulted in a higher prevalence of ST-segment elevation MI in patients diagnosed with ACS during winter.

Similar to previous findings,[1−7] patients diagnosed with ACS during winter had worse clinical outcomes, which may be partly associated with culprit lesion morphologies. A previous OCT study demonstrated that ACS patients with plaque rupture had more cardiac events.[24] There was no significant difference between the W-ACS and O-ACS groups subsampled for OCT, presumably because the number of patients examined using OCT was relatively small and the OCT subsample was not representative of patients with severe ACS. Cardiac mortality was much lower in ACS patients with OCT examination than in those without OCT examination in the present study cohort. To indicate that ACS diagnosed in winter season is a prognostic factor independent from established cardiovascular risk factors, multivariable analysis was conducted. After adjusting for traditional cardiovascular factors, diagnosis of ACS during winter was an independent predictor of cardiac death during the 2-year follow-up period. OCT results suggested that vulnerable plaque and advanced atherosclerosis might cause worse cardiac outcomes in patients diagnosed with ACS during winter.

Limitations of the study included, firstly, the fact that it was a retrospective, single center, observational study. Secondly, there was a selection bias because the indication for OCT examination was defined by operator discretion. In 1,161 patients with primary percutaneous coronary intervention for de novo narrowings, 538 (46%) did not undergo an OCT examination. The OCT subsample did not represent severe ACS patients; 2-year cardiac mortality was significantly different between patients with and without OCT examination (2.9% vs 13.3%). We failed to show the worse cardiac mortality in ACS patients with OCT examination admitted in winter. Thirdly, information for the medical treatment is unavailable. Fourthly, it is possible that some iatrogenic luminal injuries caused by intracoronary thrombectomies were incorrectly identified as plaque ruptures. Finally, the resolution and penetration depth of OCT imaging are inadequate for perfect detection of plaque rupture. When red thrombi are present, minor ruptured plaques located behind the thrombi may be missed by OCT imaging.

Disclosures

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