Quantity, particle size, and histologic composition of embolic debris collected in a distal protection filter after carotid angioplasty and stenting: Correlation with patient characteristics, timing of carotid artery stenting, and procedural details

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The occurrence of distal embolization during carotid artery stenting (CAS) is a major complication. Determining the preoperative risk of embolization may lead to improved patient selection and outcome of CAS. This study examined the quantity, particle size, and histologic composition of embolic debris collected in a distal protection filter and its possible correlation with patient characteristics, timing of CAS, and procedural details.

CLINICAL SUMMARY

Subjects

Patients who underwent a CAS procedure during a 17-month period in which a SPIDER Embolic Protection Device (EPD) (ev3 Endovascular Inc, Plymouth, Minn) was used were included. Patient demographics, cardiovascular risk factors, neurologic symptoms, and time interval between symptoms and CAS were retrospectively recorded.

Procedure

CAS was performed according to the standardized protocol in our center as described previously.¹ This protocol also dictates the antiplatelet regimen.

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TABLE 1. Patient (n = 55) and lesion (n = 59) characteristics

Patients, n (%)	
Age, y	Mean, 67.2 (range, 47-89)
Male sex	36 (66)
Hypertension	43 (78)
Hypercholesterolemia	41 (75)
Diabetes	9 (16)
Cardiac disease	13 (24)
Chronic renal insufficiency	9 (16)
Smoking history	39 (71)

Lesions, n (%)	
Asymptomatic	5 (8.5)
Symptomatic	54 (91.5)
Amaurosis fugax	8 (14)
Retinal infarction	1 (2)
Transient ischemic attack	28 (48)
Stroke	17 (29)
Onset of symptoms before CAS	
0-28 d	35 (59)
>28 d	24 (41)
Contralateral occlusion	10 (17)
Percent stenosis	
50%-69%	4 (7)
70%-99%	55 (93)

CAS, Carotid artery stenting.

Histopathology and Filter Analysis

Debris was photographed before (Figure 1, A) and after (Figure 1, B) removal from the EPD. Clemex image analysis software (Clemex Technologies Inc, Longueuil, Canada) was used to analyze the number and size of the particles.

Consecutive series of paraffin sections were stained with hematoxylin-eosin as a routine stain, resorcin-fuchsin for

FIGURE 1. Macroscopic images of EPD containing embolic material (A, arrow) and debris as retrieved from EPD (B).

TABLE 2. Patient and procedural characteristics of 44 cases with embolic particles in the Embolic Protection Device and number and size of particles

	N = (%)	No. of particles, median (min-max)	P value	Length of particles, median (min-max)	P value
Age (y) mean, 67.6 (47-89)					
Less than or equal to mean	22 (50)	8 (1-29)	.51	670 (60-6045)	.09
Greater than mean	22 (50)	9 (1-47)		609 (79-9423)	
Sex					
Male	29 (65.9)	10 (1-47)	.98	615 (60-9423)	.31
Female	15 (34.1)	7 (1-29)		728 (79-6771)	
Hypertension					
Yes	38 (86.4)	9.5 (1-31)	.37	684 (60-9423)	.013
No	6 (13.6)	3.5 (1-47)		474 (129-3681)	
Hypercholesterolemia					
Yes	30 (68.2)	10 (1-24)	.31	653 (60-6771)	.43
No	14 (31.8)	5.5 (1-47)		621 (79-9423)	
Diabetes					
Yes	9 (20.5)	13 (3-31)	.5	782 (135-9423)	.018
No	35 (79.5)	8 (1-47)		615 (60-6045)	
Cardiac disease					
Yes	10 (22.7)	9.5 (3-24)	.83	707 (60-6771)	.17
No	34 (77.3)	8.5 (1-47)		629 (79-9423)	
Chronic renal insufficiency					
Yes	9 (20.5)	8 (1-24)	.77	706 (147-4676)	.38
No	35 (79.5)	9 (1-47)		636 (60-9423)	
Smoking history					
Yes	31 (70.5)	7 (1-47)	.029	643 (60-9423)	.42
No	13 (29.5)	14 (4-29)		643 (79-6771)	
Asymptomatic					
Yes	3 (6.8)	3 (1-14)	.23	546 (169-1728)	.76
No	41 (93.2)	9 (1-47)		645 (60-9423)	
Symptomatic					
Amaurosis fugax	4 (9.1)	4 (1-21)	.24	650 (213-4095)	.22
Transient ischemic attack	23 (52.3)	8 (1-31)		669 (79-9423)	
Stroke	14 (31.8)	11.5 (2-47)		606 (60-6771)	
Onset of symptoms before CAS					
0-28 d	28 (64)	10 (1-47)	.048	653 (60-9423)	
>28 d	16 (36)	4 (1-23)		615 (135-4676)	.51
Duration CAS					
<2 h	21 (48)	8 (1-31)	.46	695 (60-9423)	.25
≥2 h	23 (52)	10 (2-47)		603 (79-6045)	
Predilation					
Yes	40 (91)	8.5 (1-47)	.87	630 (60-9423)	.52
No	4 (9)	9 (1-19)		838 (125-4102)	
Postdilation					
Yes	41 (93)	9 (1-47)	.89	633 (60-9423)	.14
No	3 (7)	7 (5-13)		881 (160-4676)	
Stent type					
Acculink	30 (68)	8 (1-47)	.60	599 (79-6045)	.21
Protege	7 (16)	13 (3-31)		714 (60-9423)	
Other	7 (16)	10 (4-21)		615 (135-4676)	

CAS, Carotid artery stenting.

collagen and elastin, and von Kossa for collagen and calcified tissue to study the particles at different depths. Debris was classified as thrombus (containing platelets, erythrocytes, and fibrin), atheroma (fibrous matrix, cholesterol clefts, and foam cells), and calcified tissue on the basis of morphologic criteria.

Statistical Analysis

Data were analyzed using SPSS software (SPSS Statistics 17.0; SPSS Inc, Chicago, Ill). Continuous variables were expressed as median (minimum-maximum) in case normality could not be assumed and compared with nonparametric tests.

FIGURE 2. Histology of embolic debris contains both thrombus and atheromatous material characterized by the cholesterol clefts (A, arrows). Within the debris, polymeric material (B, arrows) and calcific nodules (C, arrows) were observed. A and C, Hematoxylin–eosin. B, von Kossa.

RESULTS

Patients and Procedures

Demographics of the 55 included patients and details of the 59 treated stenotic lesions in these patients are presented in Table 1.

Histopathology

Macroscopic evaluation detected particles in 44 of the 59 collected filters (75%) (Figure 1). Table 2 represents patient and procedural details of these cases and the number and size of the detected particles. The EPD in patients who smoked showed a significantly lower number of particles compared with nonsmokers (7 vs 14, $P = .029$). The number of particles was significantly higher when CAS was

performed in the first 4 weeks after the qualifying neurologic event (10 vs 4, $P = .048$).

Qualitative assessment of histologic sections showed determinable embolic debris in 36 (82%) of the processed filters. Embolic particles consisted of atheromatous debris in 13 cases (36%), thrombus in 16 cases (44%), and calcified tissue in 7 cases (19%) (Figure 2). Atheromatous plaques generated a significantly higher number of particles (median 14, $P = .01$).

DISCUSSION

Despite the use of protection devices, cerebral embolization has been shown by transcranial Doppler and diffusion-weighted magnetic resonance imaging.^{2,3} Although most of the clinical implications of detected microemboli remain

undefined, association with deterioration in cognitive function 6 months after CAS has been reported.⁴ Currently, no protection device can completely prevent distal embolization. Furthermore, dislodgement of embolic debris can occur during guidewire manipulation and protection device positioning or removal. Determining the risk factors for embolization could lead to a better patient selection, improving outcomes of CAS.

Unstable, echolucent atherosclerotic plaques have been associated with increased embolic potential and increased risk of stroke in CAS.⁵ This study confirms the increased embolic potential, because we found a significantly higher number of embolic particles when CAS was performed in the first weeks after symptoms and a significantly higher number of particles in atheromatous plaques.

Older age, hypertension, hypercholesterolemia, coronary artery disease, symptomatic lesions, number of balloon dilations, and stent diameter have unequivocally been associated with increased embolic potential. In this study, we found only smoking to be negatively correlated with the number of dislodged particles during CAS. A possible explanation for this finding is that plaque composition in smokers differs from nonsmokers because of different etiology, and these plaques could be more stable.

Because of the method of histologic processing and section staining, not all filters containing embolic debris

appeared in the qualitative analysis. In addition to the small sample size, this is a limitation of this study.

CONCLUSIONS

The detailed analysis of embolic debris and the findings that lesion characteristics and timing of intervention seem to be the most important factors influencing distal embolization during CAS and contribute to further understanding the mechanism of embolization and better patient selection.

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Is there any benefit in using awake anesthesia with thoracic epidural in thoracoscopic talc pleurodesis?

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Supplemental material is available online.

General anesthesia is commonly used for video-assisted thoracic surgery (VATS) with talc pleurodesis, although use of regional anesthesia in the awake patient might

improve outcomes.^{1,2} The purpose of this randomized study is to compare the hospital discharge of patients undergoing VATS with talc pleurodesis using thoracic epidural or general anesthesia.

CLINICAL SUMMARY

Forty patients with malignant pleural effusion were randomized to undergo VATS with talc pleurodesis by awake anesthesia with thoracic epidural (awake group, N = 20) or general anesthesia and 1-lung ventilation (control group, N = 20) (ClinicalTrials.gov registration No. NCT01469728). The study was approved by the Policlinico Tor Vergata University ethical committee, and written informed consent was obtained by the patients. Eligibility criteria are summarized in Table E1.

In the awake group, the thoracic epidural catheter was inserted at the T4 level for continuous infusion of ropivacaine

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The Awake Thoracic Surgery Research Group is listed in the Appendix.

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