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**ÖZGÜN ARAŞTIRMA** **ORIGINAL ARTICLE**

# DETECTION OF MICROEMBOLIC SIGNALS BEFORE AND AFTER STENT TREATMENT OF PATIENTS WITH CAROTID STENOSIS AND DETERMINATION OF RELATED FACTORS

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**ABSTRACT**

INTRODUCTION: Ischemic stroke is among the primary causes of morbidity and mortality in the world. Carotid artery stenosis plays a critical role in the etiology of ischemic stroke. Carotid artery stenting (CAS) in severe carotid artery stenosis is an alternative treatment to endarterectomy in both symptomatic and asymptomatic patients and it is increasingly preferred. Microembolic signals (MES), which can be detected by transcranial doppler (TCD), can arise due to either carotid artery stenosis or CAS. In our study, MES count was performed before and after stent treatment to determine its associated condition.

METHODS: This prospective our study includes a total of 40 symptomatic and asymptomatic patients scheduled for carotid artery stenting. Demographic properties, anamnesia, risk factors, medical treatments, vital parameters and neuroimaging results of the patients were evaluated. National Institutes of Health Stroke Scale (NIHSS) and modified Rankin Scale (mRS) scores before and after CAS were calculated. MES counts were performed with TCD and DWI 12-24 hours before and 12-24 hours after CAS. Clinical findings and complications were monitored after the stent.

RESULTS: While 44,7% of the patients were ipsilateral MES before the stent, 13,2% were ipsilateral MES after the stent. 55% of the patients had new silent infarct after the stent. Before and after CAS, patients' NIHSS was 1,7 and mRS was 0,6. No significant correlation was detected between MES and DWI results. However, plaque morphology was found to have an effect on result of both MES and DWI ($p<0.05$)

DISCUSSION AND CONCLUSION: Microembolic signals are considered as a parameter that can lead to stroke recurrence and neuronal ischemic damage. Therefore, imaging cerebral microembolies is becoming increasingly important. Recent studies and developed methods significantly decreased the risk of CAS-related major complications and stroke. Nevertheless, in order to decrease the microembolic signals that arise in the meantime with no clinical symptoms, more comprehensive studies are required.

**Keywords:** Microembolic signal, transcranial Doppler, carotid artery stenting, plaque morphology, silent infarction.

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# KAROTİS STENOZU OLAN HASTALARIN STENT TEDAVİSİ ÖNCESİ VE SONRASINDA MİKROEMBOLİK SİNYALLERİNİN TESPİT EDİLEREK İLİŞKİLİ OLDUĞU FAKTÖRLERİN BELİRLENMESİ


## ÖZ

GİRİŞ ve AMAÇ: İskemik inme, dünyada mortalite ve morbiditenin önde gelen nedenlerindendir. İskemik inme etyolojisinde karotis arter stenozu önemli bir rol oynamaktadır. Ciddi karotis darlıklarında karotis arter stentleme (CAS), hem semptomatik hem de asemptomatik hastalarda endarterektomiye alternatif bir tedavidir ve CAS'ın tercih edilme sıklığı giderek artmaktadır. Transkraniyal doppler (TCD) ve difüzyon MR (DWI) saptanabilen mikroembolik sinyallerler (MES) hem karotis arter stenozuna bağlı hem de CAS'a bağlı olarak ortaya çıkabilir. Çalışmamızda hastalarda stent tedavisi öncesi ve sonrası MES sayımı yapılarak ilişkili olduğu durumlar saptanmak istenmiştir.

YÖNTEM ve GEREÇLER: Prospektif olarak yaptığımız bu çalışmaya karotis arter stentleme kararı verilmiş olan semptomatik ve asemptomatik 40 hasta dahil edildi. Hastaların demografik özellikleri, anamnezleri, risk faktörleri, medikal tedavileri, vital parametreleri ve nörogörüntülemeleri değerlendirildi. CAS öncesi ve sonrası National Institutes of Health Stroke Scale (NIHSS) ve modified Rankin Scale (mRS) hesaplandı. Hastalara CAS'dan 12-24 saat önce ve 12-24 saat sonra TCD ve DWI ile MES sayımı yapıldı. Stent sonrası klinik bulgular ve komplikasyonlar takip edildi.

BULGULAR: Hastaların %44,7'sinde stent öncesi, %13,2'sinde stent sonrası ipsilateral MES saptandı. %55 hastada stent sonrası yeni sessiz enfarkt olduğu görüldü. CAS öncesi ve sonrası hastaların NIHSS: 1,7 ve mRS: 0,6 olarak saptandı. MES ve DWI sonuçları arasında anlamlı bir ilişki saptanmadı. Ancak plak morfolojisinin hem MES hem de DWI sonuçlarına anlamlı etkisi olduğu tespit edildi ($p<0,05$).

TARTIŞMA ve SONUÇ: Mikroembolik sinyaller inme rekkurrensine ve nöronal iskemik hasara yol açabilecek bir parametre olarak kabul edilmektedir. Bu nedenle serebral mikroembolileri de gösterebilmek giderek önemli hale gelmektedir. Günümüzde yapılan çalışmalar ve geliştirilen yöntemler ile CAS'a bağlı majör komplikasyon ve inme riski oldukça azalmıştır. Ancak bu süreçte klinik bulgu vermeden ortaya çıkan mikroembolik sinyalleri de azaltabilmek için daha kapsamlı çalışmalara ihtiyaç olduğu düşünülmektedir.


**Anahtar Sözcükler:** Mikroembolik sinyal, transkraniyal Doppler, karotis arter stentleme, plak morfolojisi, sessiz enfarkt.

## INTRODUCTION

Cerebrovascular diseases are the second leading cause of death in the population over the age of sixty and the first cause of disability and labor loss (1,2). Approximately 80% of the strokes are ischemic stroke. Now, very successful results are obtained with thrombolytic and endovascular treatment options in acute stroke. However, primary prevention is still the strongest therapy in stroke treatment. Therefore, primary protection can be provided by identifying and reducing modifiable risk factors in patients. Carotid artery stenosis, one of the modifiable risk factors, has become more common with the widespread use of imaging methods.

Carotid stenosis is an atherosclerotic process that can be symptomatic or asymptomatic. The NASCET (North American Symptomatic Carotid Endarterectomy) study compared the risk of recurrent stroke in patients with symptomatic carotid stenosis between groups receiving medical treatment and endarterectomy. Absolute risk reduction was 17% in endarterectomy group.. Similar results were obtained in the ECST study. Consequently, surgical treatment of more than 50% of carotid stenosis has been shown to be important in reducing the risk of ipsilateral stroke. In the CREST (Carotid Revascularization Endarterectomy versus Stenting) trial, endarterectomy and carotid stenting were compared and the benefit rates of both treatments were similar. In asymptomatic carotid stenosis, the annual risk of ipsilateral stroke is between 1-2% (3). There is no standard approach to the treatment of these patients. In the Asymptomatic Carotid Atherosclerosis Study (ACAS) and Asymptomatic Carotid Surgery Trial (ACST) studies, it was found that the endarterectomy group in patients with asymptomatic carotid stenosis had a half-year decrease in stroke risk compared to the group receiving medical treatment. However, it was emphasized that in order to achieve these benefits while operational risks of endarterectomy patients should be below 3%. (4) Since there is no consensus in asymptomatic patients, additional parameters should be considered when choosing recanalization therapy. These include imaging and monitoring of cerebrovascular reserve, evaluation

of plaque characteristics (e.g. presence of ulceration), and detection of microembolic signal (MES) by transcranial doppler ultrasonography (TCD). It was found that the risk of stroke in patients with asymptomatic carotid stenosis with MES in TCD increased from 1% to 15% in one year (5). MES can only be detected by TCD. Ultrasound probes are fixed on both MCA for 30 minutes. High intensity (solid) and short duration signals are recorded. MES can be an early warning sign of stroke risk. (6) While diffusion MRI can show acute micro-infarcts, TCD can also capture active microembolic signals. Nowadays, silent ischemic strokes have been included in stroke classification with increasing neuroimaging methods. Therefore, more research is being done on the importance of silent ischemic areas. In our study, silent infarcts and microembolic signals were detected in patients who underwent carotid stenting and the related conditions were determined. In these patients, pre-stent and post-stent MES measurements and DWI imaging were used to determine the relationship between demographic characteristics, risk factors, plaque morphology, medical treatment and stent type.

## METHODS

The study was conducted in accordance with the Helsinki Declaration ethical standards and approved by the Uludağ University Faculty of Medicine Noninterventional Clinical Studies Ethics Committee (Number: 2016-2/4, Date: 02.02.2016) and informed consent was obtained from all participants. Carotid artery stenting (CAS), made of 40 patients were included in the study. Age, risk factors, examinations, cerebrovascular reserves, stenosis rate, localization and plaque morphology were evaluated. TCD and DWI imaging were performed 12-24 hours before and 12-24 hours after CAS.

DWL-mutlidop® T digital TCD was used in the study. From the transtemporal window, both MCAs were fixed at 40-60 mm depth with 2 MHz head probes. Monitored microembolic signals for 30 minutes.

AXIOM Artis, Siemens device was used for stenting stenotic carotid arteries. Procedures were performed with local anesthesia (2% prilocaine, Citanest). A vascular sheath (6-8F) was passed through the femoral artery. Intraarterial 50-70 U/kg heparin was administered. in patients with distal embolic protection devices (EPDs) it was used and PTA balloons (5x20 mm or 6x20 mm) was used. Protege (eV3, Minnesota) and Xact (Abbott Laboratories, Illinois) stents were used. 1000 U heparin was injected per hour in the first 12 hours to patients receiving dual antiplatelet treatment. Then, low molecular weight 4000 U heparin was injected twice daily.

MES ratios and DWI results in TCD were analyzed between themselves and the relationship between these two groups and other variables. Descriptive statistics were given as mean ± standard deviation for continuous variables conforming to normal distribution, median (min-max) for continuous variables not conforming to normal distribution, and frequency and percentage for categorical variables. Fisher exact and Fisher Freeman Halton tests were used to compare categorical variables between groups. One-way analysis of variance and Kruskal Wallis test were used to compare continuous variables between groups. The analysis was performed in IBM SPSS v.20. $p < 0.05$ was considered statistically significant.

## RESULTS

34 (85%) of the patients were male and 6 (15%) were female. The mean age was 65.75 ± 8.68 years. Hypertension was the most common risk factor. The number of symptomatic patients was 25 (62.5%) and the number of asymptomatic patients was 15 (37.5%). CAS was administered to 12 of the symptomatic patients in the first month. NIHSS (National Institutes of Health Stroke Scale) and mRS(modified Rankin's Score) did not change after CAS. Stenotic ICA revealed MES in 17 ipsilateral patients. After treatment, the number of patients with ipsilateral MES decreased to 5. MES could not be counted due to the closed transtemporal window of 2 patients. In the contralateral MES evaluation, MES was detected in 5 patients and regressed to 3 after treatment (Figure 1). Ipsilateral MES was detected in 17 patients before stenting. After stenting, MES was detected in 5 patients. Contralateral MES was detected in 5 patients before stenting. After stenting, MES was detected in 3 patients. MES could not be counted due to the closed transtemporal window of 2 patients.

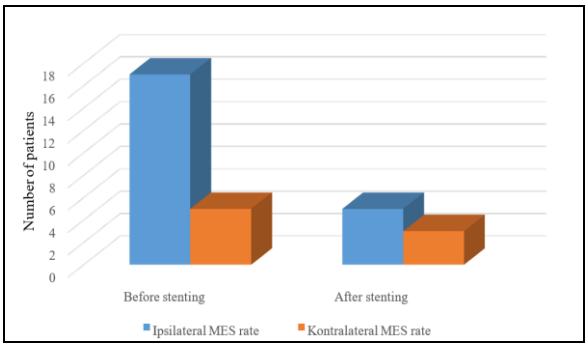


**Figure 1.** MES rate before and after treatment.

Although there was a decrease in the MES values from MCAs after ipsilateral stent treatment, early MES reduction was not statistically significant (p=0,793). Statistical analysis could not be made because the contralateral MES values were 5 or less. 18 (45%) patients had ulcerated stenosis and 7 (17.5%) patients had long segment stenosis. Open cell stent was used in 20 patients (50%) and closed cell stent was used in 20 patients (50%). After stenting, 22 (55%) patients had new infarct areas on control diffusion MR images. The diameter of these infarcts was between 2 mm and 10 mm and the number was between one and nine. In 14 (35%) patients, infarcts were in the stenting ICA irrigation area, while 8 (20%) patients were observed contralaterally or bilaterally (Figure 2). In addition, it was observed that 6 (15%) patients had aortic and tortiose structure, 10 (25%) patients had normal aorta, and 24 (60%) patients were not included in cerebral vascular examination. Other carotid lesions were performed in all patients and 15 (37.5%) patients had stenosis that did not require treatment.

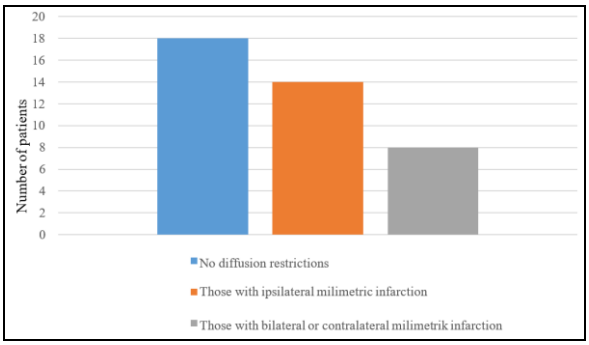


**Figure 2.** Diffusion MR results after treatment.

When the risk factors of patients with ipsilateral MES and those without MES were compared, there was no difference in terms of hypertension, diabetes mellitus, hyperlipidemia, smoking and coronary artery disease When stroke treatments were evaluated, it was found that only 3 of the patients with MES used statins, and 6 patients in the non-MES group used statins. There was no difference between the groups in terms of statin use. Statistical analysis could not be performed because there were only 5 people using antiplatelet except the acetylsalicylic acid (ASA) + clopidogrel combination. In 8 of 17 patients with MES, ipsilateral new ischemic lesion was detected in control DWI, while contralateral or bilateral diffusion restriction was observed in 5 patients (P=0.182). Plaque ulceration was detected in 13 of the MES positive patients. On the other hand, only 5 of the MES negatives had plaque formation.The effect of plaque ulceration on the detection of MES was found to be significant (p=0.006). When the effect of increase in stenosis rate on MES results was evaluated, no significant relationship was found between these two groups (P=0.721) (Table 1).

There was no difference in terms of hypertension, diabetes mellitus, hyperlipidemia, smoking, coronary artery disease in patients with ADC restriction in DWI after stent compared to those without diffusion restriction. There was no effect of being symptomatic or asymptomatic on post-procedural diffusion restriction (p=0.710). It was found that the use of open or closed stents did not affect DWI results after stenting (p=0.782). DWI results were found to be similar in patients with stroke duration less than 1 month and longer than 1 month (p=1). Hypotension developed in 11 postoperative patients. While 8 of these patients had negative DWI, only 3 had millimetric infarction in DWI. The effect of this complication on the detection of new lesion in ipsilateral DWI was not significant in patients who developed poststent hypotension.

When the effect of plaque ulceration on DWI results after stent was evaluated, the relationship between the presence of ipsilateral DWI (+) and ulcerated plaque was significant (p=0.001). There was no significant relationship between ipsilateral DWI and MES. However, contralateral MR (+) after stenting was found to be significant in patients

with contralateral MES (+) before stenting (p=0.028). When the degree of stenosis was compared with the presence of new lesion in DWI, p=0.032, the relationship between the two groups was significant. In particular, the effect of high stenosis on ipsilateral infarct was significantly higher than in the contralateral infarct group (p=0.031) (Table 2).

**Table 1.** Statistical results of variables compared with pre-stent ipsilateral MES.

| | **MES (-) (n:21, 52,5%)** | **MES 1-10(+) (n:13 ,32,5%)** | **MES 11-20(+) (n:4, 10%)** | **p value** |
|---|---|---|---|---|
| Age | 64,71 ± 7,72 | 64,30 ± 8,39 | 66,5 ± 5,91 | |
| Sex (F,M) | 4 (19%), 17 (81%) | 1 (7,7%), 12 (92,3%) | 0, 4 (100%) | p= 0,793 |
| Diabetes (n:22, 57,9%) | 12 (57,1%) | 7 (53,8%) | 3 (75%) | p=0,804 |
| Hypertension (n:17, 44,7%) | 10 (47,6%) | 6 (46,2%) | 1(25%) | p=0,800 |
| Coronary artery disease (n:14, 36, 8%) | 8 (38,1%) | 5 (38,5%) | 1 (25%) | p=1,00 |
| Smoker (n:14, 36,8%) | 6 (28,6%) | 7 (53,8%) | 1(25%) | p=0,620 |
| Statin (n:9, %23,7) | 6 (%28,6) | 2 (%15,4) | 1(%25 | p=0,752 |
| Other antiplatelet drugs | 1 (%4,8) | 3 (%23,1) | 1(%25) | P= inestimable |
| Antiplatelet resistance evaluated (n:25, 65,8%) | 15 (39,5%) | 10(30,3%) | P=0,934 | Antiplatelet resistance evaluated (n:25, 65,8%) |
| Symptomatic (n:24, 63,2%) | 11 (52,4%) | 9 (69,2%) | 4 (100%) | p=0,210 |
| Asymptomatic (n:14, 36,8%) | 10 (47,6%) | 4 (30,8%) | 0 (0%) | |
| Stenting in first month (n:12, 31,5%) | 4 (19%) | 6 (35,2%) | | p=1,00 |
| Plaque Ulceration (n:18, 47,4%) | 5 (23,8%) | 10 (76,9%) | 3 (75%) | p=0,006 |
| Long segment plaque | 5 (23,8%) | 2 (15,4%) | 0 (0%) | p=0,210 |
| Degree of stenosis 85% or more (n:20, 52,6%) | 10 (47,6%) | 7 (53,8%) | 3 (75%) | p=0,721 |

**Table 2.** Statistical results of variables compared with DWI.

| | **DWI (-) (n:18, 45%)** | **Ipsilateral DWI (+) (n:14, 35%)** | **Bilateral or contralateral DWI (+) (n:8, 20%)** | **p value** |
|---|---|---|---|---|
| Age | 65,77 ± 11,24 | 64,57 ± 6,12 | 67,75 ± 8,68 | |
| Sex (F,M) | 5 (27,8%), 13 (72,2%) | 1 (7,1%), 13 (92,9%) | 0, 8 (100%) | p= 0,180 |
| Hypertension (n:24, 60%) | 11 (61,1%) | 9 (64,3%) | 4 (50%) | p=0,837 |
| Diabetes (n:22, 55%) | 8 (44,4%) | 9 (64,3%) | 5 (62,5%) | p=0,551 |
| Hyperlipidemia (n:17, 42,5%) | 6 (33,3%) | 7 (50%) | 4(50%) | p=0,642 |
| Coronary artery disease (n:14, 35%) | 5 (27,8%) | 5 (35,7%) | 4 (50%) | p=0,518 |
| Smoker (n:14, 35%) | 7 (38,9%) | 4 (28,6%) | 3 (37,5%) | p=0,984 |
| Symptomatic (n:25, 62,5%) | 12 (66,7%) | 9 (64,3%) | 4 (50%) | p=0,710 |
| Asymptomatic (n:15, 37,5%) | 6 (33,3%) | 5 (35,7%) | 4 (50%) | |
| Stenting in first month (n: 12, 33,3%) | 6 (33,3%) | 6 (27,3%) | | p=1,00 |
| Open cell stent (n:20, 50%) | 10 (55,6%) | 7 (50%) | 5 (62,5'%) | p=0,782 |
| Infarct localization, cortical | 0 (0%) | 11 (78,6%) | 3 (21,4%) | p<0,001 |
| Post-op hypotension (n:11, 27,5%) | 8 (44%) | 3 (21,4%) | 0 (0%) | p=0,049 |
| Other ICA stenosis (n: 15, 37.5%) | 7 (38,9%) | 5 (35,7%) | 3 (37,5%) | p=1,00 |
| Plaque ulseration (n:18, 45%) | 5 (27,8%) | 8 (57,1%) | 5 (62,5%) | p=0,004 |
| Aortic arch evaluated patient (n: 16, 40%) | 5 (27,7%) | 11 (50%) | | p=0,154 |
| MES 1-10 (+), ipsilateral before stent | 3 (18,8%) | 7 (50%) | 3 (37,5%) | P=0,182 |
| MES 11-20 (+), ipsilateral before stent | 1 (6,3%) | 1 (7,1%) | 2 (25%) | |
| MES (+), ipsilateral after stent | 0 (0%) | 2 (14,3%) | 3 (37,5%) | p=0,027 |
| MES (+), contralateral before stent | 0 (0%) | 2 (14,3%) | 3 (37,5%) | p=0,027 |
| MES (+), contralateral after stent | 0 (0%) | 1 (7,1%) | 2 (25%) | p= incalculable |
| Degree of stenosis 85% or more (n:22, 55%) | 12 (66,7%) | 9 (64,3%) | 1 (12,5%) | p=0,032 |

**DWI:** Diffusion Weighted Magnetic ResonanceImaging, **MES:** Microembolic Signals.

## DISCUSSION AND CONCLUSION

There have been many studies evaluating the relationship between cerebral embolization and MES during endovascular treatment. Almekhlafi et al. performed 30 CAS patients with TCD and MES counting during stenting and DWI after the procedure; found a new ischemic lesion after stent in 76.9% of patients (7). However, they found no significant relationship between the number of MES detected in TCD during stenting and new ischemic lesions in DWI. They explained this situation as follows. MESs can reach penetrating arteries that cannot be seen on MRI. In addition, gas emboli may be involved in solid emboli in TCD performed during stenting. Lasek-Bal et all in both CEA and CAS patients performed pre-procedural and post-procedural TCD and MES counts, and found no significant relationship between the amount of MES and clinical findings (8). Similarly, in our study, no significant relationship was found between MESs detected in TCD and the detection of new ischemic lesions in DWI.

In our study, the effect of plaque morphology on both the presence of MES detected in TCD and DWI results was found to be significantIt is thought that plaque ulceration and recent intraplaque hemorrhage (IPH) increase cerebral embolism. In a study conducted in 2014, the effect of intraplaque hemorrhage and the presence of MES on stroke recurrence was compared with the ECST risk model (9). Of 123 patients with symptomatic carotid stenosis (50-99%), 46 (37.4%) had ipsilateral MES, 82 (66.7%) IPH, and these patients were prospectively followed up until CEA or recurrent cerebral disease. At the end of the study, it was reported that the risk of stroke recurrence was high in both MES and IPH patients, but higher in those with IPH (+), and the relationship between IPH and MES was also significant (9). In a meta-analysis, major and minor complications that occurred after stenting were compared in patients with and without IPH, and found that the rate of silent infarction after stent was significantly higher in those with IPH, even though there was no difference in terms of major complications (10). Both plaque ulceration and IPHare evaluated in the type 6 plaque category histopathologically with high probability of being symptomatic. Type 6 plaques are complex plaques with defects on their surfaces and hemorrhagic or thrombus inside. It was emphasized that MESs are a strong marker for these unstable plaques, but should be supported by luminal imaging methods (11). These luminal imaging methods can be MRI, CT, Ultrasonography, or even PET / CT. MRI is the gold standard for differentiating IPH, ulceration, a lipid-rich necrotic core (LRNC), and inflammation, but time and artifacts may limit this. CT also determines plaque morphology well, but may not distinguish LRNC from IPH. While the PET/CT shows the inflammation in the plaque well, it is insufficient in anatomy and ulceration. Ultrasonography, with the aid of contrast enhancement, is a cost-effective technique to assess plaque morphology and characteristics, but it is limited in sensitivity and specificity for detecting LRNC, plaque hemorrhage, and ulceration compared with MRI (11).

In the study conducted by Kuliha et al. in 2016, 81 patients underwent CAS, these patients were evaluated pre- and post-procedurally with DWI, and 46 (56.8%) patients detected new ischemic lesions. Clopidogrel resistance was detected in 6 of 81 patients and new ischemic lesion was seen in 3 of these patients, and the effect of anti-platelet resistance on ischemic lesions was not significant (12). In our study, no difference was found between the group with and without antiplatelet resistance in terms of cerebral embolization (p=0.934 for TCD, p=1 for DWI).

The stent to be used in endovascular treatment in carotid stenosis can be open-celled or closed-celled. In a study conducted by Schillinger et al. in 1684 (1010 asymptomatic, 674 symptomatic) CAS patients, 859 (51%) closed-cell and 825 (49%) open-cell stents were used; and TIA, stroke and mortality rates were compared for the first 30 days. The event rate was 2.4% in patients with closed cell stents used and 4.1% in patients with open cell stents used, and no difference was found between stent types in terms of acute / subacute complications (13). Timaran et al. used 40 open-cell stents in 20 patients and closed-cell stents in 20 patients, and microembolic signals screening with TCD during stenting and MES at 24 hours. They found that there was no difference between open cell and closed cell stents in terms of cerebral microembolization detected by TCD and DWI (14). Similarly, 20 closed cell and 20 open cell stents were used in our study, and no difference was observed between the two groups

in terms of DWI imaging (p=0.782). For this reason, stent should be chosen according to anatomical localization, length of stenotic segment and plaque character.

In meta-analysis performed by Schnaudigel et al. distal protection filters were found superior to stent than proximal protection filters (15). In our study, distal protection filter was used in all patients. Our new cerebral lesion rates (55%) detected in DWI after stenting are consistent with the literature. The most common early complication in our study was hypotension, which noted in 27.5% of the procedures.

If the blood pressure is below 90/60 mmHg and/or the heart beat rate is under 60 per minute for more than 6 hours, it is called "persistent hemodynamic depression (PHD)", and if it is not responding to volume replacement therapy and/or inotropic use, it is called resistant PHD. While all of the hypotensions after the CAS we detected were diagnosed as PHD, but there was no resistant PHD cases. In a study by Gupta et al. included 500 CAS patients, PHD was noted in 210 (42%) patients and resistant PHD in 84 (17%). In the study performed by Csobay et al., PHD was diagnosed in 216 (37%) of 542 CAS patients, but resistant PHD was detected in only 1.8% (16,17). In a study conducted by Nii et al. on 95 CAS patients, 32.6% post-procedural PHD appeared, and 30.6% post-procedural new cerebral embolism was observed, but no significant relationship was found between these two complications (18). In our study, there was no significant new ischemic lesion in DWI in patients with post-stent PHD. (p=0.049). In other words, it was seen that hypotension after stenting had no effect on cerebral embolism.

The post-stent ipsilateral new DWI lesion is described due to emboli that arise directly from the thrombus during stenting or by endothelial injury. However, this explanation is insufficient for embolic signals that appear contralaterally after stenting. Some studies have suggested that aortic pathologies can lead to this phenomenon. In a study by Bazan et al., aortic arch geometry and calcification in 94 patients were determined by thorax CT, and it was suggested that type 2 aortic arch and significant calcification may increase the risk of embolization after stenting (19).

In another study, the aortic arch anatomy, tortuosity index, and presence of aortic plaque were evaluated using TEE in 59 patients undergoing carotid stenting. Complex aortic arch / severe tortuosity, presence of complicated plaques (≥5 mm or mobile debris) were found to be related to the number and volume of cerebral lesions (20). In our study, no significant relationship was found between the aortic arch structure and the presence of MES or DWI lesions (p=0.154). However, aortic evaluation of 24 patients was not performed. Because some centers do not include aortic imaging within the scope of cerebral angio. In order to predict the complications of ipsilateral or contralateral cerebral embolization that may occur during stenting, it seems necessary that at least luminal imaging of the aortic arch may also be required.

Today, silent ischemic strokes are included in stroke classification albeit unclear importance of their highly increased detection rate with current neuroimaging methods. While evaluating patients for major stroke prevention treatment in this study; cerebral microembolisms were detected and associated factors were evaluated. According to our study, carotid plaque morphology is the most important predictor of microembolism. This important observation should be strengthened with more comprehensive studies focused on this subject. We believe that further studies will reduce cerebral damage by minimizing procedural microembolism even if they do not results in clinical findings.

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**Ethics**

**Ethics Committee Approval:** The study was approved by the Uludağ University Faculty of Medicine Noninterventional Clinical Studies Ethics Committee (Number: 2016-2/4, Date: 02.02.2016).

**Informed Consent:** Informed consent was signed by all included patients.

**Authorship Contributions:** Surgical and Medical Practices: PUU, MB, BH. Concept: PUU, MB, BH. Design: PUU, MB, BH. Data Collection or Processing: PUU, MB, BH. Analysis or Interpretation PUU, MB, BH. Literature Search: PUU, MB, BH. Writing: PUU, MB, BH.

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