Outcomes Of Carotid Artery Stenting In Patients With Unstable Carotid Plaque And Patients With Carotid Occlusive Disease

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KEYWORDS

CAS, Unstable Plaques, Carotid Artery Stenting, CEA.

ABSTRACT

Background: Carotid atherosclerotic plaques exist as two distinct types, a majority is those that remain quiescent, and a smaller proportion is unstable and vulnerable to disruption. Increasing degrees of stenosis, however, have not been consistently accompanied by a correspondingly increased risk for stroke in asymptomatic patients. Not all plaques with unstable histologic features cause symptoms.
Aim: To report the follow-up of patients with unstable and stable carotid plaque in two different study groups after carotid stenting with embolic protection device.
Methods: This prospective interventional study included 20 patients with nine significant unstable plaque lesions and eleven stable plaques. All patients underwent carotid stenting with embolic protection device. Investigations included carotid duplex ultrasound, CTA and MRI to study the content of the plaque. The intervention was performed under local anesthesia in all patients. Follow-up included the neurological assessment and review of any post-procedure complication. Imaging follow-up included duplex ultrasound at 3 months post-procedure then every 6 months.
Results: Statistically significant relation between degree of stenosis (50-69 % or > 70 % stenosis) and age was observed. Statistically significant relation between occurrence of death and age. Neither comorbidities (DM, HTN, Hyperthyroid, Smoker and IHD) nor the diagnosis (Asymptomatic, Stroke and TIA) were statistically related to death (P>0.05). Meanwhile, there was a significant relation between the unstable plaque and the occurrence of death (p=0.038). There was non-statistically significant relation between occurrence of death among the studied cases and postoperative femoral hematoma or the brain edema that lasted less than 24 hours (P>0.05). However, there was statistically significant relation between occurrence of death among the studied cases and postoperative Ischemic stroke and Hemorrhagic stroke.
Conclusion: The findings of this study demonstrate favorable outcomes with carotid artery stent under filter protection, and carotid artery stenting is a safe alternative to carotid endarterectomy (CEA).

Introduction

Carotid atherosclerotic plaques exist as two distinct types, a majority are those that remain quiescent, and a smaller proportion are unstable and vulnerable to disruption, leading to athero-embolization and brain infarction. The percent diameter reduction in the carotid artery lumen is the most readily measured feature of the disease and currently used as a marker for identifying plaques that may be vulnerable to disruption [1].

Increasing degrees of stenosis, however, have not been consistently accompanied by a correspondingly increased risk for stroke in asymptomatic cases. Neither Asymptomatic Carotid Atherosclerosis Study (ACAS) nor Asymptomatic Carotid Surgery Trial (ACST) displayed an association between the degree of stenosis and stroke-risk. The degree of stenosis is as a result not an adequately sensitive or specific marker for determining plaque instability and stroke-risk [2, 3].

Histologic evaluation of explanted human arteries indicates that atherosclerotic lesions originate as "early" forms (types I, II, and III). Plaques start as fatty streaks, found early at 6 months of life. Over time, they coalesce into lipid cores. A fibroatheroma develops next as fibrous tissue accumulates over the core and forms a fibrous cap. Some plaques then continue to progress to "later" or more advanced forms (types IV, V, and VI), with a growing lipid rich necrotic core (LRNC), developing intraplaque hemorrhage (IPH) and demonstrating thinning of their fibrous cap [4].

Not all plaques with unstable histologic features cause symptoms. Approximately 50% of asymptomatic patients with carotid artery stenosis (CAS) have identifiable IPH or fibrous cap rupture, yet only approximately 2% of asymptomatic patients develop a neurologic event at 1 year. As a result, morphology alone has limited ability to identify unstable carotid plaques and predict future strokes the histomorphology of plaques is not static, and plaques may transition from one morphologic type to another, due to biological influences such as inflammation, generation of free radicals and wall shear stress (WSS) [5]. So, we did this study to report the follow-up of patients with unstable carotid plaque underwent carotid stenting with embolic protection device.

PATIENT AND METHODS

This prospective interventional study was conducted at Cardio-Vascular Surgery Center (CVSC) at Mansoura University Hospitals. This study started in December 2021 and included 20 patients with significant carotid lesion (9 unstable and 11 stable plaques). This study included patients with unstable carotid plaque despite the degree of stenosis, asymptomatic patients with CAS more than 70%, symptomatic patients with carotid stenosis more than 50%, patients with previous neck surgery or radiation with significant CAS or unstable plaque needs intervention when CEA is not an option, patients with abnormal anatomy like high bifurcation of the common carotid artery. (exposure of high bifurcation increase the risk for stroke and cranial nerve injury). This study excluded asymptomatic patients with CAS less than 70%. Symptomatic patients with non-significant CAS less than 50% were also excluded in addition to patients with absolute contraindications to CAS (patients with visible thrombus and hypersensitivity to contrast), or with relative contraindications to CAS (circumferential plaque with sever calcification, sever carotid tortuosity).

Methods

Every subject was subjected to full history taking including demographic details (full name, age, gender, marital Status, occupation, physical activity, smoking and socioeconomic status), medical history (Hypertension (HTN), diabetes mellitus (DM), hyperlipidemia and any other relevant conditions, history of any prior surgeries (CEA or carotid artery stenting), neurological history (history of transient ischemic attacks (TIAs), history of strokes) and family history of vascular diseases, stroke, or neurological conditions.

Clinical examination included motor power evaluation using the Medical Research Council (MRC) scale (0 to 5) by comparing bilateral muscle strength, sensory function assessment, to identify level of sensory loss, speech & language examination to check for dysarthria or aphasia, and cranial nerve examination to assess signs of neurological deficits such as facial drop or asymmetry in tongue movements.

Patients were evaluated for reflex changes, such as hyperreflexia or hyporeflexia, for coordination and balance to identify ataxia, and for gait asymmetry or signs of hemiparesis. vital Signs and Cardiovascular (CVS). Assessment included measuring blood pressure (BP), auscultation for carotid bruits or other vascular sounds and examination for signs of peripheral vascular disease (e.g., diminished pulses, ischemic changes in extremities).

Investigations

Laboratory investigations included basic blood tests (complete blood count (CBC), renal function tests (Urea, Creatinine, Electrolytes) and liver function test), coagulation profile (international normalized ratio (INR) and activated partial thromboplastin time (APTT)), lipid profile (total cholesterol, LDL, HDL, and triglycerides), blood glucose (fasting blood glucose & HbA1c), inflammatory markers (C-reactive protein (CRP) or erythrocyte sedimentation rate (ESR)), and thrombophilia screening.

Radiological investigations included carotid duplex ultrasound to assess plaque characteristics (plaque morphology, surface characteristics, plaque composition), stenosis grading (degree of stenosis Quantify stenosis as mild (<50%), moderate (50–69%), or severe (≥70%) using the NASCET method, flow dynamics (hemodynamics & waveform patterns), complications & risk factors (presence of emboli, contralateral side stenosis, and vertebral artery flow).

Computed tomography angiography (CTA) is the gold standard for adequate planning and sizing, and it provided detailed imaging of the carotid arteries, circle of wills and aortic arch. CTA assessed vessel anatomy, degree and location of stenosis.

Magnetic Resonance Imaging (MRI): Before the procedure, MRI was requested for all patients with inconclusive Doppler studies determine whether the plaque was stable or unstable. On black blood T1-weighted, black blood T2-weighted, and time-of-flight images, the signal intensity of the plaque within the carotid artery was compared with that of the ipsilateral sternocleidomastoid muscle. Plaques with intraplaque hemorrhage and a lipid-rich necrotic core were defined as vulnerable and those with fibrous tissue and calcification were defined as stable [6].

Steps of Intervention

The intervention was performed under local anesthesia in all patients. Pre-operative low dose of beta-blocker (continued for 30 days) plus 300 mg clopidogrel 4 hours before intervention (if the patient is already on clopidogrel, loading dose was not given). Puncture of CFA was done, and a 6-F sheath was introduced into the CFA followed by full heparinization, 0.035 wire over a vertebral catheter was directed to the ECA and kept it in the ECA, 6-F sheath was replaced by a long sheath on the 0.035 wire, and 0.014 Nitrex wire was introduced carefully through the ICA lesion. The EPD (embolic protection device) was pushed over the primary Nitrex wire more than 3 cm distal to the lesion. SpiderFX Filter (Medtronic, Minneapolis, MN, USA) was used with Protégé Stent (Medtronic, Minneapolis, MN, USA) meanwhile FilterWire EZ Protection Device (Boston Scientific, Marlborough, MA, USA) was utilized for Carotid Wallstent (Boston Scientific, Marlborough, MA, USA). The suitable size stent was deployed across the carotid bifurcation covering the lesion. Eighteen Self-expandable Protégé tapered stents were delivered while only two Carotid Wall stents were used in our study. Post-stenting dilatation was performed in some cases with strict monitoring of the hemodynamics. The EPD was removed on its own sheath. Reversal of the half dose of heparin to prevent ICH from reperfusion, 200 mg of Mannitol was injected through a peripheral line and another dose was given on the next day. All patients were admitted to the ICU for 24-48 hours post-operatively and for vital signs monitoring, neurological assessment and puncture site inspection.

Follow Up

Immediate post-operative care in the ICU (first 24–48 hours) involved vital signs assessment, neurological assessment, puncture site inspection (check for hematoma, bleeding, or pseudoaneurysm at the groin puncture site), and antiplatelet therapy adherence, and the blood tests included lipid profile, blood glucose, and renal function.

Short-term follow-up (first 1–3 months) included, in the 1–2 weeks post-discharge, assessment of patient's symptoms (headache, TIA/stroke symptoms, or groin discomfort), BP, heart rate, compliance with the dual antiplatelet & statin therapy and ultrasound duplex scan. After 4 to 6 weeks up to 3 Months, we assessed the neurological assessment and review of any post-procedure complications. Doppler ultrasound may be repeated at 3 months if there were concerns about stent patency.

Long-term follow-up included assessment every 6 months for the first year, then annually if no complications arise, considering shifting from dual APT to clopidogrel or aspirin only after 6 months and monitoring for signs of ISR (In-Stent Restenosis), recurrent neurological symptoms, or other vascular complications.

Imaging follow-up included duplex ultrasound at 3 months post-procedure then every 6 months for the first year then annually thereafter unless symptoms warrant earlier imaging. CT angiography was done if ultrasound findings were inconclusive or suspected complications.

Red flags requiring immediate attention included neurological symptoms (TIA/stroke), severe headaches or vision changes (suggestive of hyperperfusion syndrome), swelling, pain, or bleeding at the groin puncture site and unexplained neck pain or signs of infection.

Ethics Considerations

The institutional research board of Mansoura University's Faculty of Medicine reviewed and approved the study protocol. The Declaration of Helsinki's guiding principles were followed when conducting the research methods. After ensuring confidentiality, each study subject gave their informed consent. At any moment during the trial, patients were free to leave without incurring any penalty.

Statistical Analysis

Data analysis was conducted by SPSS software, version 25 (SPSS Inc., PASW statistics Chicago). Qualitative data were described using number and percentage. Quantitative data were described using median for non-normally distributed data and mean±SD for normally distributed data after testing normality using Kolmogrov-Smirnov test. Chi-Square, Fisher exact test was used to compare qualitative data between groups as appropriate. Student t test was used to compare 2 independent groups for normally distributed data. Significance of the results was set at the (≤0.05) level.

RESULTS

The mean age of the studied cases was 65.8±5.818/years, 70% of the cases were males and 30% were females. The majority of the studied cases (16 cases) had DM, fifteen cases had HTN, four cases were smokers and two case had ischemic heart disease. 45% of the cases were diagnosed by TIA, 40% by stroke and 15% were asymptomatic. Eleven patients had stable plaque sided lesion and nine patients had unstable plaque sided lesion by CEUS: Carotid Enhanced Ultra-sound and MRI. The mean Peak systolic velocity (PSV) was 248.75±82.26 and mean Degree of stenosis was 73.3±14.305 % among the studied cases. Majority of the studied cases (75%) had no postoperative complications, 10% had hemorrhagic stroke, 5% ischemic stroke, 5% had femoral hematoma and 5% had brain edema for 24 hours. 15% of the studied cases died. As shown in table (1)

Table (1): Demographic characteristics, Comorbidities, diagnosis, Plaque stability, Degree of stenosis, PSV, Postoperative complications and death among studied cases.

	N=20	%
Age / years		
Mean ±SD	65.8±5.818	
(min-max)	(56-76)	
Sex		
Male	14	70.0
Female	6	30.0
Comorbidities		
DM	16	80.0
Hypertension	15	75.0
Smoker	4	20.0
IHD	2	10.0
Diagnosis		
Asymptomatic	3	15.0
Stroke	8	40.0
TIA	9	45.0
Plaque stability		
Unstable	9	45.0

Stable	11	55.0
Degree of stenosis Mean ±SD (min-max)	73.3±14.305 % (50-90)	
PSV Mean ±SD (min-max)	248.75±82.26 (140-340)	
Postoperative complications Negative Ischemic stroke Hemorrhagic stroke femoral hematoma brain edema for 24 hours	15 1 2 1 1	75.0 5.0 10.0 5.0 5.0
Death	3	15.0

One patients developed dissection and stent induced dissection and subsequent ischemic stroke with the Carotid Wall stent.

Two patients experienced Hemorrhagic stroke and death with protégé stent and death in eighteen interventions (11%). Meanwhile, one mortality case was reported with the Wallstent as a sequalae of carotid dissection and ischemic stroke (50%). As shown in table (2)

Table (2) Relation between the types of stents used, complications and mortality.

		Protégé stent	Carotid Wall stent	P
Complications	Hemorrhagic stroke	2 (11.1%)	0 (0.0%)	0.289
	Ischemic stroke	0 (0.0%)	1 (50.0%)	
	Femoral hematoma	1 (5.6%)	0 (0.0%)	
	Brain edema	1 (5.6%)	0 (0.0%)	
Mortality		2 (11.1%)	1 (50.0%)	0.284

A statistically significant relation between degree of stenosis (50-69 % or > 70 % stenosis) and age was observed. No statistically significant relation between degree of stenosis (50-69 % or > 70 % stenosis) and Comorbidities (DM, HTN, Smoking and IHD), Diagnosis (Asymptomatic, Stroke and TIA) and stability of the plaque (P>0.05). There was insignificant relation between degree of stenosis (50-69 % or > 70 % stenosis) and postoperative complications or occurrence of death (P=0.57 and 1.0, respectively). As shown in table (3)

Table (3): Relation between stenosis and sociodemographic characteristics, comorbidities, diagnosis, complications and death of studied cases.

	Stenosis		**Test of significance**
	50-69 % stenosis	> 70 % stenosis	
Age / years Mean ±SD	62.22±4.994	68.73±4.839	t=2.948 p=0.009*
Sex			

Male Female	8(88.9) 1(11.1)	6(54.5) 5(45.5)	χ^2=2.78 p=0.095
Comorbidities DM Hypertension Smoking IHD	 8(88.9) 5(55.6) 1(11.1) 1(11.1)	 8(72.7) 10(90.9) 3(27.3) 1(9.1)	 P=0.369 P=0.127 P=0.591 P=1.0
Diagnosis Asymptomatic Stroke TIA	 1(11.1) 6(66.7) 2(22.2)	 2(18.2) 2(18.2) 7(63.6)	χ^2=4.96 p=0.084
Plaque stability Unstable Stable	 3(33.3) 6(66.7)	 6(54.5) 5(45.5)	χ^2=0.900 p=0.343
Postoperative complications Negative Ischemic stroke Hemorrhagic stroke femoral hematoma brain edema for 24 hours	8(88.9) 0 1(11.1) 0 0	7(63.6) 1(9.1) 1(9.1) 1(9.1) 1(9.1)	χ^2=2.89 p=0.575
Death	1(11.1)	2(18.2)	FET=0.194 P=1.0

t: Student t test, χ2=Chi-Square test, FET: Fisher exact test

Table (4) In our study there was statistically significant relation between occurrence of death and age. However, there was no significant relation between the sex of the studied cases and the occurrence of death (P=0.007 and 1.0, respectively). Comorbidities (DM, HTN, Hyperthyroid, Smoker and IHD) and Diagnosis (Asymptomatic, Stroke and TIA) were not statistically related to death (P>0.05). Meanwhile, there was a significant relation between the unstable plaque and the occurrence of death (p=0.038).

There was non-statistically significant relation between occurrence of death among the studied cases and postoperative femoral hematoma or the brain edema that lasted less than 24 hours (P>0.05).

However, there was statistically significant relation between occurrence of death among the studied cases and (No postoperative complications, postoperative Ischemic stroke and Hemorrhagic stroke) (P=0.008, 0.015 and 0.015, respectively). Death happened in all cases with post-operative stroke, meanwhile other cases did not die within 30 days postoperatively.

Table (4): Relation between death and socio-demographic characteristics, comorbidities, diagnosis, side and postoperative complications of studied cases.

	Death		**Test of significance**
	No N=17	Yes N=3	
Age / years Mean ±SD	 64.41±5.026	 73.67±3.215	t=3.042 p=0.007*
Sex Male Female	 12(70.6) 5(29.4)	 2(66.7) 1(33.3)	χ^2=0.019 p=1.0
Comorbidities			

DM Hypertension Smoker IHD	14(82.4) 13(76.5) 3(17.6) 1(5.9)	2(66.7) 2(66.7) 1(33.3) 1(33.3)	P=0.531 P=1.0 P=0.509 P=.284
Diagnosis Asymptomatic Stroke TIA	 3(17.6) 8(47.1) 6(35.3)	 0 0 3(100)	 χ^2=4.31 p=0.116
Plaque stability Unstable Stable	 6 11	 3 0	 χ^2=4.314 p=0.038*
Postoperative complications Negative Ischemic stroke hemorrhagic stroke femoral hematoma brain edema for 24 hours	15(88.2) 0 0 1(5.9) 1(5.9)	0 1(33.3) 2(66.7) 0 0	P=0.008* P=0.015* P=0.015* P=0.667 P=0.667

FET: Fisher exact test, t: Student t test, χ2=Chi-Square test

DISCUSSION

The study group had a mean age of 65.80±5.818, with 70% being male and 30% female. Among the participants, 80% had diabetes mellitus (DM), 75% had hypertension (HTN), and smaller proportions were affected by smoking (20%), hyperthyroidism (5%), and ischemic heart disease (IHD) (10%). Clinically, 45% of the cases presented with transient ischemic attacks (TIA), 40% with stroke, and 15% were asymptomatic. [7]

Consistent with our findings, Kim and Ha (2023) evaluated outcomes of CAS and CEA. In their study, CAS patients had a mean age of 68.9 ± 10.6 years, with males comprising 84%. HTN was present in 76% of cases, while smoking and coronary vascular disease were noted in 36% and 12% of cases, respectively. [8]

Similarly, fuse et al. (2020) reported a mean age of 72.2 ± 6.94 years in CAS patients, with 84.9% being male. The majority (78%) had HTN, while smoking and coronary vascular disease were observed in 26% and 48% of cases, respectively. At the time of initial treatment, 92% had severe stenosis (≥70%).[9]

A study by Jalbert et al. (2015) analyzing the data of patients undergoing CAS between 2005 and 2009 found that the mean age was 74 years, and males constituted 59.8% of the study group. Hypertension was present in 82% of cases, and diabetes in 39%. Perioperative stroke and death rates were observed at 4.1% and 1.7%, respectively, highlighting the importance of careful patient selection as certain individuals may have a higher risk of complications. Factors such as age, anatomical considerations, and existing health conditions (especially hypertension) should be evaluated to ensure the benefits of the procedure outweigh the potential risks. [10]

Furthermore, Bonati et al. (2018) in the International Carotid Stenting Study (ICSS) demonstrated that the 30-day stroke or death risk was 7.4% in symptomatic patients undergoing CAS compared to 4.0% in those undergoing CEA. However, the study stated that the outcomes of CAS improve as the operator experience increases. [11]

In our study, the mean degree of stenosis was 73.3±14.305%, with a mean peak systolic velocity (PSV) of 248.75 ± 82.26 cm/s. Postoperatively, 75% of cases experienced no complications. However, some complications were reported like hemorrhagic stroke (10%), ischemic stroke (5%), femoral hematoma (5%), and transient brain edema (5%). The mortality rate was 15%.[12]

Comparatively, Ozaki et al. (2023) reported lower complication rates, including cerebral infarction (2–3%), cerebral hemorrhage (1%), and hyperperfusion syndrome (0.5–1.0%). Mortality rates were between 1.0% and 1.5%, primarily due to hemorrhage associated with hyperperfusion syndrome. In our study one hyperperfusion syndrome was reported. Fortunately, it was self- limited and lasted for 24 hours. [13]

The EVA-3S trial (Endarterectomy Versus Angioplasty in patients with Symptomatic Severe carotid stenosis) (Eckstein et al., 2008) reported a 3.9% risk of stroke or death at 30 days for symptomatic patients undergoing CEA versus 9.6% for those undergoing CAS. The SPACE trial also corroborated these findings, highlighting higher perioperative risks with CAS in less experienced centers. [14]

Fuse et al. (2020) found a restenosis incidence of 7.2%, with mortality occurring in 13% of patients over the observational period. The observation period varied among patients, with a median follow-up time of 44.8 months. In our study, no cases of restenosis were detected during the 24-month follow-up period. [15]

CAS-CARE Trial (Carotid Artery Stenting with Cilostazol Addition for Restenosis) was conducted to evaluate the inhibitory effect of adding cilostazol on in-stent restenosis in patients treated with CAS. The incidence of in-stent restenosis (ISR) after carotid artery stenting (CAS) over a 2-year follow-up period was:

- 10.8% in the cilostazol group (patients who received cilostazol in addition to standard antiplatelet therapy)
- 19.6% in the control group (patients who received standard antiplatelet therapy only)

This study confirmed that cilostazol reduced the incidence of restenosis by nearly half compared to standard therapy alone. [16]

The current study identified no statistically significant relationships between the degree of stenosis (50–69% vs. >70%) and sex ($P > 0.05$). However, ischemic and hemorrhagic strokes were significantly associated with increased mortality ($P = 0.008$ and $P = 0.015$, respectively). Also advanced age was statistically related to increased death.

Contrasting findings include de Weerd et al. (2009), who reported a significant influence sex on the prevalence of moderate stenosis, with higher rates observed in older males. Meanwhile Gröschel et al. (2005) results copes with our study, identified advanced age as a predictor of hemodynamic instability and mortality following CAS, possibly due to impaired cerebral autoregulation. Furthermore, age and diabetes mellitus were highlighted as predictors of 30-day stroke and mortality rates in Schlüter et al. (2007) and Arif et al. (2016). [17] [18]

Howard et al. (2021) demonstrated that the degree of stenosis was significantly associated with stroke risk in asymptomatic patients. Their systematic review found a 5-year ipsilateral stroke risk of 18.3% in those with 80–99% stenosis compared to 1.0% in those with 50–79% stenosis. This highlights the necessity of individualized risk assessment beyond stenosis severity. [19]

In our study nine cases (45%) had the criteria of the unstable plaque, and the other eleven plaques (55%) were stable. There was a significant relation between the Unstable plaque and the occurrence of death in the peri-procedural period ($p=0.038$).

Regarding the type of stents and its embolic protection devises, in our study eighteen protégé (shouldered-open cell) stents were deployed under the spider FXTM filter protection with no dissections recorded. Meanwhile, two carotid (closed) wall stent with the FilterWire EZ™ caused one case of carotid dissection and subsequent ischemic stroke and death. The explanation for the reported dissection is that the CarotidWall stent is a closed-cell stent with less flexibility than the Protégé stent (open-cell) which provide more flexibility. [20]

A Japanese study by Iko et al. (2014) compared the treatment outcomes of CAS using same types of stents and filter-based embolic protection devices. This study assessed 121 consecutive cases of CAS performed with FilterWire EZ™ with the Carotid Wallstent and 37 consecutive cases of CAS performed with the Spider FX™ with the protégé. No significant differences were observed in the incidence of complications between the groups. [21]

American College of Surgeons National Surgical Quality Improvement Program (ACS-NSQIP) evaluated the outcomes of carotid artery stenting (CAS) with and without the use of embolic protection devices (EPDs). This study stated that CAS performed without EPDs was associated with a 4-fold increase in the likelihood of perioperative stroke. In the study titled “Carotid Stenting without Embolic Protection Increases Major Adverse Events: Analysis of the National Surgical Quality Improvement Program” (Nazari P ,et al 2021). The authors examined 3,098 patients who underwent carotid artery stenting (CAS) using data from ACS-NSQIP between 2011 and 2018. This study found that the stroke incidence is 1.5% with EPD vs 6.5% without the EDP. [22]

Recommendations

This study highlights the importance of assessing plaque morphology using carotid-enhanced Doppler ultrasound and MRI, rather than relying solely on the degree of stenosis.

CTA plays a crucial role in evaluating aortic arch anatomy and identifying challenges such as a bovine arch, which can complicate the CAS procedure.

Maintaining strict blood pressure control during the peri-procedural period is essential. It is advisable to postpone the procedure if the patient develops stress-induced hypertension preoperatively, as uncontrolled hypertension is a major factor contributing to reperfusion injury and hemorrhagic stroke.

CAS should be performed by an experienced surgeon who has independently completed at least 12 procedures and is proficient in the monorail technique.

Transfemoral CAS is not advisable for unfavorable aortic arch anatomy, such as a heavily calcified aortic arch or type 3 aortic arch. The arch anatomy should be carefully assessed using CTA prior to performing a CAS procedure, as the presence of a bovine arch or a Type II or III aortic arch can make the procedure significantly more challenging.

Selecting the appropriate stent type and embolic protection device is critical for treating carotid artery stenosis. Shouldered Protégé stents are preferred for common lesions at the carotid bifurcation due to their flexibility, which reduces the risk of carotid dissection. However, as an open-cell stent, it carries a higher risk of plaque material embolization through its wider cells. In contrast, the Carotid Wallstent is a closed-cell stent, which is less likely to cause embolization but has a higher risk of causing carotid dissection.

The first 24 hours postoperatively are the most critical for strict blood pressure control. Once this period is managed successfully, the overall outcomes of CAS are likely to be optimal. Mannitol plays a significant role in preventing brain hyper-perfusion after CAS.

Cilostazol 100 mg twice daily for 12 months plays a significant role in preventing In-Stent Restenosis within 24 months follow-up.

Further research involving larger patient cohorts and extended follow-up is necessary to validate these findings and address the limitations associated with small sample sizes and regional variations in management practices.

Conclusions

The findings of this study demonstrate favorable outcomes with carotid artery stenting regarding stroke prevention and low complication rates when patient selection is optimized. Continued efforts to refine treatment protocols and improve patient outcomes are essential for minimizing stroke-related morbidity and mortality.

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