Case Report Brain Conflux

Interception of Lipid-rich Emboli Using an Embolic Protection Device during Carotid Artery Stenting: A Case Report

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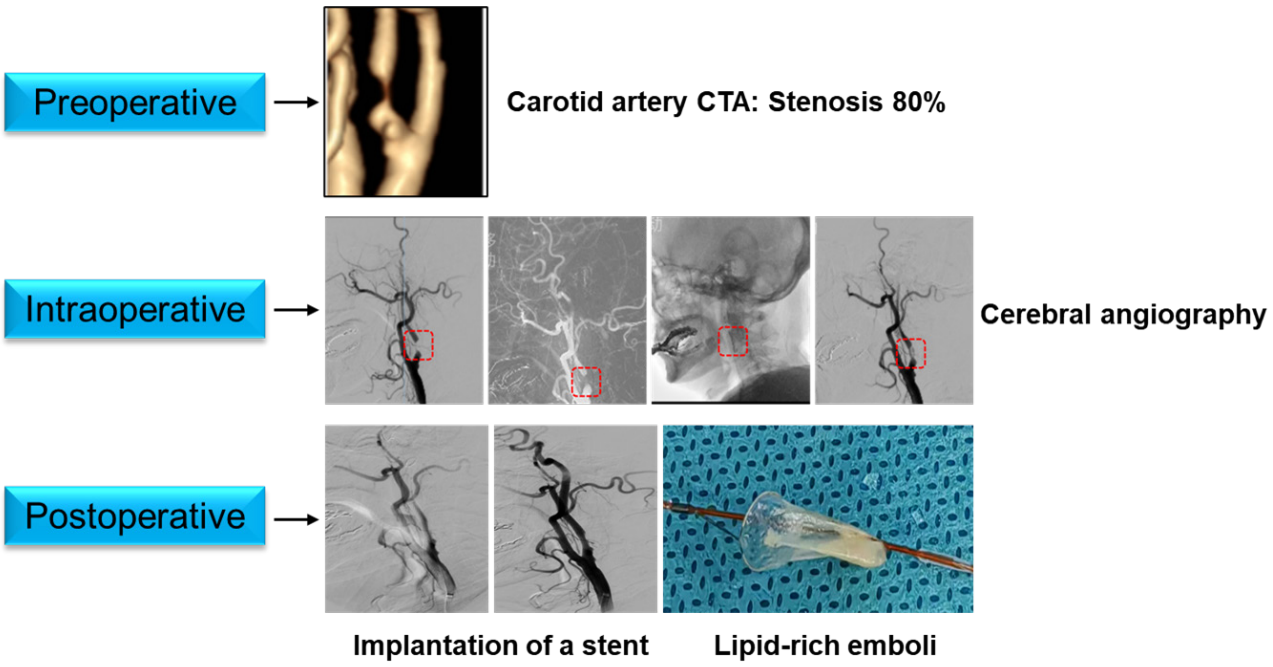
Graphical Abstract



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Case Report

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Interception of Lipid-rich Emboli Using an Embolic Protection Device during Carotid Artery Stenting: A Case Report

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Abstract

Carotid artery stenosis is a major risk factor for ischemic stroke, and carotid artery stenting (CAS) has become a crucial minimally invasive treatment, especially for patients with poor surgical tolerance. This case report focuses on a 73-year-old male patient with severe right internal carotid artery stenosis (80%), complicated by hypertension, a history of cerebral infarction, and recurrent transient ischemic attack (TIA). Preoperative dual anti-platelet therapy was administered, and CAS was performed using a distal filter-type embolic protection device (EPD) to intercept lipid-rich embolism. Importantly, transient bradycardia and hypotension occurred due to carotid sinus reflex but were successfully managed with atropine and dopamine. Postoperative digital subtraction angiography (DSA) showed that the residual stenosis rate was reduced from 80% to <10%, with unobstructed blood flow. During 1-month follow-up, the patient's symptoms resolved, and carotid ultrasound confirmed no in-stent re-stenosis. This case demonstrates that CAS with EPD is safe and effective for severe carotid artery stenosis, as the EPD can significantly reduce the risk of cerebral embolism by intercepting dislodged emboli. The findings provide valuable clinical experience for improving treatment outcomes and patient prognosis in similar cases.

Keywords: Carotid Artery Stenting; Embolic Protection Device; Severe Carotid Artery Stenosis; Transient Ischemic Attack

Introduction

Carotid artery stenosis holds an extremely important position in cerebrovascular diseases and is one of the key risk factors for ischemic stroke. With the intensification of population aging and changes in lifestyle, the incidence of carotid artery stenosis has been increasing year by year, seriously threatening human health and quality of life. According to relevant statistical data, ischemic stroke accounts for more than 4/5 of all cerebrovascular diseases worldwide [1]. In China, due to unhealthy factors such as high-salt and high-fat diets of the public, the incidence of basic diseases such as hypertension, diabetes, and hyperlipidemia is relatively high, which leads to a much higher incidence of carotid artery stenosis than the global average, about 5% to 7% [2].

As the main blood vessel that supplies oxygen-rich blood to the brain, once carotid artery stenosis occurs, it will lead to insufficient blood supply to the brain, causing a series of serious clinical symptoms. Patients with mild to moderate carotid artery stenosis may experience symptoms such as dizziness and limb weakness due to insufficient blood supply to the brain; while patients with severe carotid artery stenosis, due to severe vascular blockage, have a more serious situation of cerebral ischemia, with a risk of cerebral ischemia as high as 26% within two years, and are extremely prone to cerebral infarction, leading to hemiplegia, aphasia, hemisensory disturbance and other severe neurological deficits, and even life-threatening [3]. In addition, unstable plaques at the carotid artery may also detach and enter the brain with the blood flow, causing multiple cerebral infarctions and repeated attacks, causing severe pain in patients and seriously affecting their self-care ability and social function.

Carotid artery stenosis is mainly caused by atherosclerosis. When the vessel wall is gradually eroded by fat, cholesterol and other substances, forming plaques, the vessel diameter narrows, hindering blood flow. This pathological process is often insidious, and many patients are unaware of the disease progression until severe clinical symptoms occur, at which point the condition may be quite serious, making treatment more difficult and the prognosis relatively poor.

Currently, the main treatment methods for carotid artery ste-

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nosis include drug therapy, carotid endarterectomy (CEA), and carotid artery stenting (CAS) [4-5]. Drug therapy is mainly used for patients with mild carotid artery stenosis or as an adjunct to surgical treatment, by controlling blood pressure, blood sugar, and blood lipids, and using antiplatelet drugs and statins, etc., to delay the progression of the disease. However, for patients with moderate to severe carotid artery stenosis, the effect of drug therapy alone is limited. CEA is a traditional surgical method that removes atherosclerotic plaques in the carotid artery to restore vascular patency, effectively reducing the risk of stroke in patients with carotid artery stenosis, and is widely used in Europe and America. However, CEA has a relatively large surgical trauma, high requirements for the patient's physical condition, and relatively high surgical risks, and there are certain postoperative complications, such as cranial nerve palsy, wound infection, and cervical hematoma, etc.

With the advancement of medical technology and the development of medical materials, CAS, as a minimally invasive, safe and effective method, has become an important means of treating carotid artery stenosis, especially suitable for patients with poor physical conditions who cannot tolerate CEA or have contraindications to surgery [6]. It restores cerebral blood supply by implanting a stent to expand the narrowed vessel, and an embolic protection device (EPD) is placed at the distal end of the stenosis during the operation to intercept detached emboli, reducing the risk of cerebral embolism and enhancing the safety of the operation.

Materials and Methods

Patient Information

A 73-year-old male patient with:

Chief complaint: Numbness and weakness in the left hand, progressive worsening over 3 days.

Comorbidities: 15-year history of hypertension (max blood pressure: 200/130 mmHg, managed with sacubitril/valsartan and amlodipine besylate), 2-year history of cerebral infarction (residual slurred speech), dyslipidemia (total cholesterol: 6.8 mmol/L, triglycerides: 2.5 mmol/L, low-density lipoprotein cholesterol: 4.5 mmol/L), and poor glycemic control (glycated hemoglobin: 7.5%).

No history of smoking or alcohol consumption.

Preoperative Examinations

Imaging tests:

Carotid ultrasound (2 years prior to admission): Multiple plaques in the origin of the right internal carotid artery, 50–69% stenosis, and a 13.8 mm × 5.0 mm unstable hypoechoic plaque.

Cranial magnetic resonance imaging (MRI) (admission): Multiple acute cerebral infarctions in the right cerebral hemisphere, with multiple old ischemic infarcts (Figure 1).

Through cranial MRI, it was found that the patient's brain tissue contained multiple acute cerebral infarction lesions, mainly distributed in the right frontal lobe, basal ganglia, occipital lobe and other brain regions.

Computed tomography angiography (CTA) (admission): Severe stenosis (80%) at the origin of the right internal carotid artery, irregular vessel walls, and visible plaque shadows (Figure 2).

Through the patient's cranial CTA scan, it was found that there was severe stenosis (more than 80%) at the beginning segment of the right internal carotid artery.

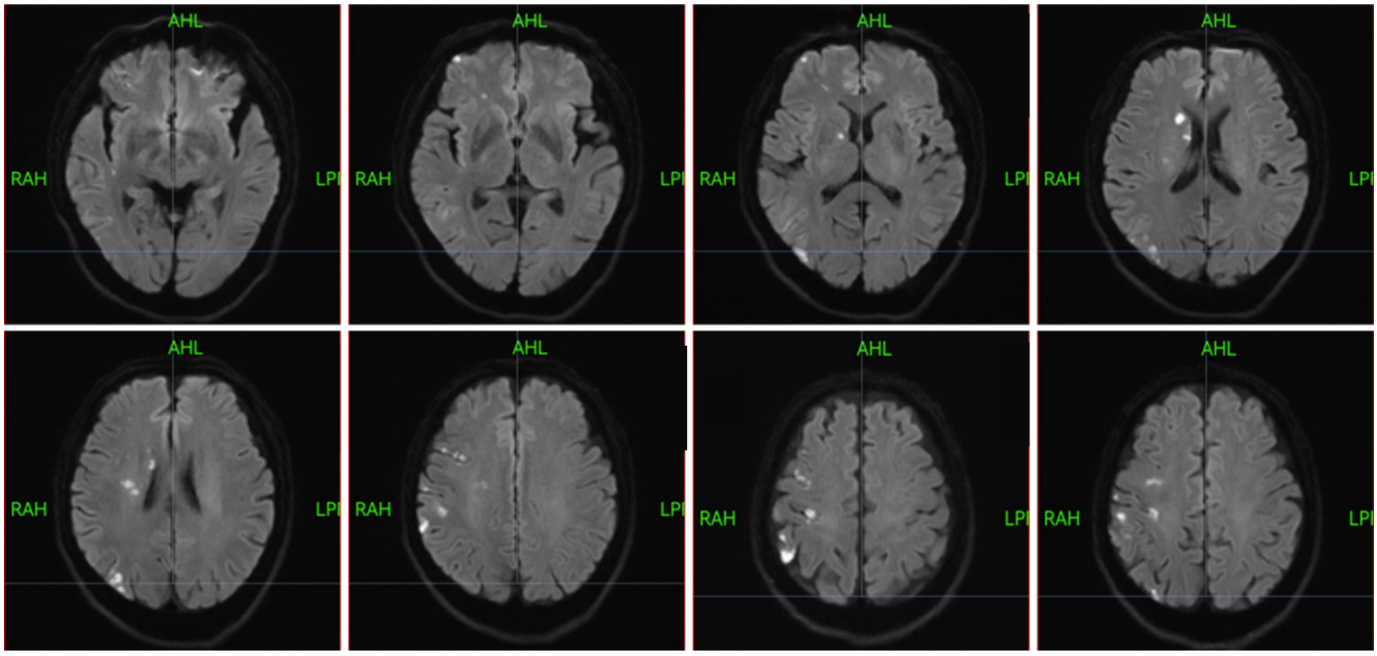
Other tests: Electrocardiogram (ECG) showing sinus arrhythmia and ST-T segment changes; blood tests confirming dyslipidemia and poor glycemic control.

Surgical Indications and Contraindications

Indications: Symptomatic severe carotid artery stenosis (80%) with recurrent TIAs, consistent with the 2017 Chinese Guidelines for the Diagnosis and Treatment of Carotid Artery Stenosis (symptomatic stenosis ≥50% for interventional treatment).

Contraindications: No absolute contraindications (e.g., active bleeding, severe coagulation disorders, or contrast agent aller-

Figure 1. Preoperative cranial MRI of the patient.



gy) were identified.

Surgical Instruments and Medications

Instruments: hydrophilic-coated loach guidewire, microguidewire, 8F guiding catheter, self-expanding nitinol stent (size-matched to the vessel), and distal filter-type EPD (matching the diameter of the distal internal carotid artery).

Medications:

Preoperative: Dual antiplatelet therapy (aspirin 100 mg/day + clopidogrel 75 mg/day) for 5 days.

Intraoperative: Atropine (for bradycardia), dopamine (for hypotension), and non-ionic contrast agent.

Postoperative: Continued dual antiplatelet therapy, nifedipine controlled-release tablets (for blood pressure), metformin + insulin (for blood glucose), and atorvastatin calcium tablets (for lipid regulation and plaque stabilization).

Surgical Procedure

Anesthesia: Local anesthesia (to monitor neurological function intraoperatively); Vascular access: Right femoral artery puncture using the Seldinger technique, followed by insertion of an 8F arterial sheath.

Guiding catheter placement: The guiding catheter was advanced to the common carotid artery (2 cm proximal to the lesion) under fluoroscopic guidance; EPD deployment: A shaped EPD was advanced to the distal C1 segment of the internal carotid artery under roadmap guidance and opened to fully cover the vessel lumen; Balloon angioplasty: A balloon (1–2 mm smaller than the internal carotid artery diameter) was used for pre-dilation of the stenotic lesion, followed by rapid deflation and withdrawal; Stent implantation: A self-expanding stent was advanced along the guidewire to the stenotic lesion, deployed, and post-dilated if necessary to ensure adequate apposition; EPD retrieval: The EPD (with intercepted emboli) was retrieved after confirming unobstructed blood flow and stable stent position via angiography; Intraoperative monitoring: Continuous monitoring of vital signs (heart rate, blood pressure) and neurological function.

Postoperative Follow-Up

Short-term: Postoperative DSA immediately after surgery; cranial CT on postoperative day 1; neurological examination and vital sign monitoring daily during hospitalization.

Long-term: 1-month follow-up including carotid ultrasound and assessment of symptoms (e.g., dizziness, TIAs).

Results

Intraoperative Findings

The surgery proceeded smoothly overall, with all steps completed as planned:

Vascular access: Successful one-time puncture of the right femoral artery; the 8F arterial sheath and guiding catheter were positioned correctly (parallel to the common carotid artery axis) without vessel injury.

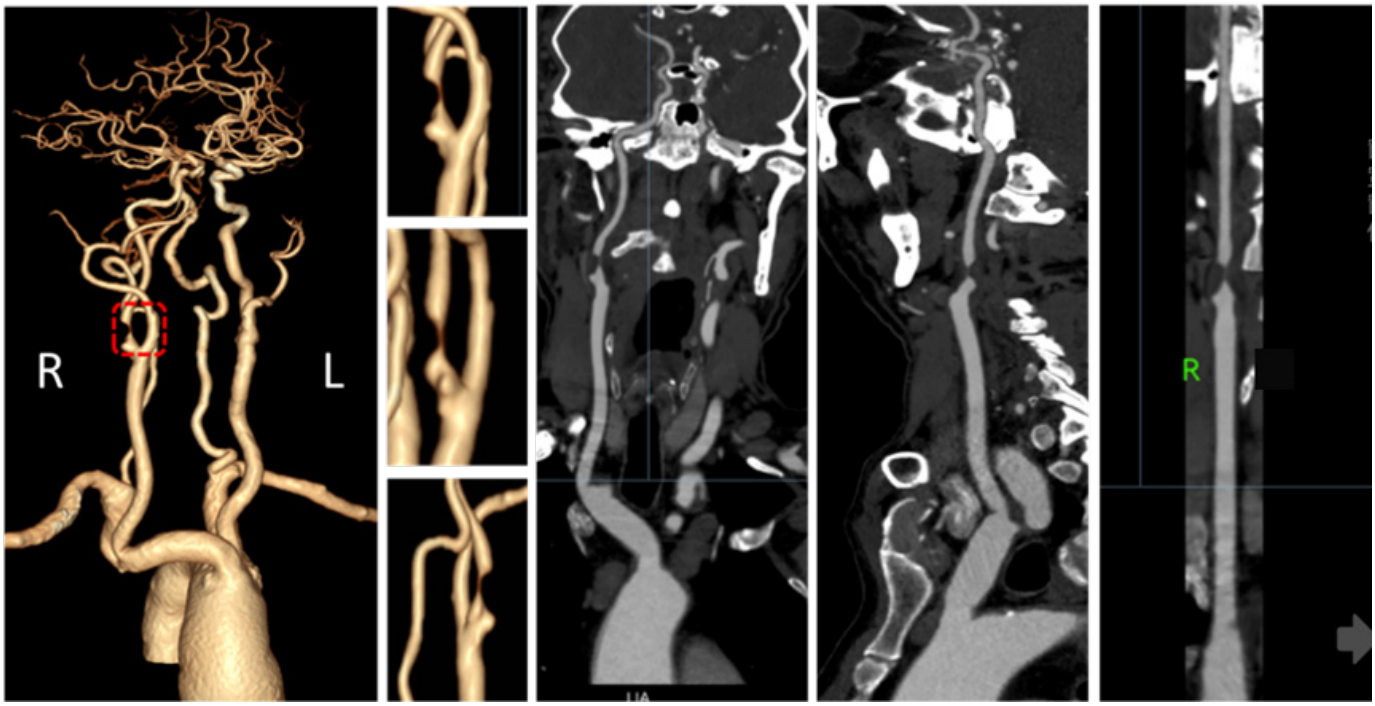
EPD deployment: The EPD was advanced through the stenotic segment without resistance, opened completely at the target "landing zone" in the distal C1 segment, and apposed closely to the vessel wall without displacement.

Balloon angioplasty: The balloon expanded fully in a cylindrical shape; post-angioplasty angiography showed significantly reduced residual stenosis, no dissection or thrombosis, and stable EPD position.

Stent implantation: The stent was deployed accurately, fully covering the stenotic segment with good wall apposition; post-deployment angiography showed residual stenosis <50%, no in-stent thrombosis, and normal vessel status.

Complication management: Transient bradycardia (minimum heart rate: 50 bpm) and hypotension (systolic blood pressure:

Figure 2. Preoperative cranial CTA of the patient.



90 mmHg) occurred after balloon angioplasty (attributed to carotid sinus reflex). Intravenous atropine (0.5 mg) and dopamine (2–5 μg/kg/min via intravenous pump) were administered, and vital signs stabilized within 5 minutes (heart rate: ~70 bpm, blood pressure: 120/70 mmHg).

Immediate Postoperative Outcomes

Postoperative DSA showed:

Accurate stent positioning at the origin of the right internal carotid artery, fully covering the lesion. Good stent expansion and close apposition to the vessel wall, with no gaps or displacement. Significant improvement in stenosis: Preoperative stenosis rate (80%) reduced to postoperative residual stenosis rate (<10%). Unobstructed blood flow: Increased contrast agent flow velocity through the stent segment, clear visualization of distal vessels, and good filling of branch vessels.

Postoperative Recovery

In-hospital recovery:

The patient returned to the ward in stable condition, with heart rate maintained at 70–80 bpm and blood pressure at 130–140/80–90 mmHg.

Postoperative day 1: Dizziness resolved; no recurrent TIAs; neurological examination showed improved biceps, triceps, and patellar reflexes; right Babinski sign turned negative; the patient could ambulate with assistance; cranial CT showed no intracranial hemorrhage or new cerebral infarction.

Postoperative day 3: The patient could ambulate independently and perform activities of daily living; continued dual antiplatelet therapy and management of comorbidities.

Postoperative day 7: No adverse symptoms; the patient was discharged.

1-month follow-up:

The patient reported complete resolution of dizziness and no recurrent TIAs; quality of life improved significantly. Carotid ultrasound showed unobstructed in-stent blood flow and no in-stent restenosis.

Discussion

Necessity and Advantages of CAS

CAS is a critical minimally invasive treatment for carotid artery stenosis, offering unique advantages for high-risk patients:

Minimally invasive nature: Compared with carotid endarterectomy (CEA)—a traditional open surgery requiring a large cervical incision and direct carotid exposure—CAS is performed via femoral artery puncture, with only a small puncture wound in the groin. This minimizes damage to normal cervical tissues and reduces the risk of CEA-related complications (e.g., cranial nerve palsy, wound infection, cervical hematoma) [7]. In this case, the patient (elderly with multiple comorbidities) tolerated CAS well, with no surgical site complications.

Rapid recovery: CAS has a shorter operative time and faster postoperative recovery than CEA. The patient in this case experienced reduced dizziness on the day of surgery, ambulated with assistance on postoperative day 1, and was discharged on day 7—far quicker than the typical 2–3-week recovery period for CEA [8]. This reduces hospital costs and improves patient quality of life.

Efficacy in stroke prevention: CAS restores blood flow by expanding the stenotic vessel with a stent, alleviating cerebral ischemia and reducing TIA and stroke risk [9]. Clinical studies (e.g., ACST-2, SPACE-2) have confirmed that CAS is non-inferior to CEA in preventing stroke in carotid stenosis, and may be superior in elderly or comorbid patients [10, 12]. This case further validates CAS efficacy: the patient's TIA recurrence ceased, and no new cerebral infarction occurred postoperatively.

Critical Role of EPDs in CAS

Mechanism of Embolus Interception

The distal filter-type EPD used in this case acts as a "mechanical barrier" to prevent embolic complications:

Structural design: The EPD consists of a fine metal mesh with precisely sized pores (small enough to trap lipid-rich emboli, large enough to allow normal blood flow). After deployment in the distal internal carotid artery, it forms a complete filter covering the vessel lumen.

Embolus capture: During balloon angioplasty or stent deployment, unstable atherosclerotic plaques may rupture, releasing lipid-rich emboli. These emboli are intercepted by the EPD's mesh and prevented from entering the cerebral circulation. Upon EPD retrieval, the captured emboli are removed from the body, eliminating the risk of cerebral embolism [13]. In this case, plaques were observed in the retrieved EPD, confirming successful embolus interception.

Reduction in Cerebral Embolism Risk

Cerebral embolism is a severe perioperative complication of CAS, with a reported incidence of 5–10% without EPD use [15]. EPDs significantly reduce this risk to 1–3% [16], as validated by clinical evidence:

A network meta-analysis by Giannopoulos et al. [16] showed that EPD use during CAS reduces the odds of perioperative stroke by 60–70% compared with no EPD.

In the CASWEP trial [15], patients undergoing CAS without EPD had a 3.2-fold higher cerebral embolism rate than those with EPD.

In this case, no cerebral embolism occurred, likely due to effective EPD use. Without EPD protection, the lipid-rich emboli intercepted during surgery could have caused new cerebral infarctions, leading to severe neurological deficits (e.g., hemiplegia, aphasia) and poor prognosis.

CAS Risks and Management Strategies

Common Risks

Despite its safety, CAS is associated with several risks:

Vascular perforation: Caused by excessive force during guidewire/catheter manipulation or mismatched instrument size, leading to hemorrhage and hematoma.

In-stent thrombosis: Related to hypercoagulability, platelet adhesion to the stent surface, or inadequate antiplatelet therapy [17-18].

In-stent restenosis: Occurs in 5–10% of patients within 1 year, due to intimal hyperplasia and smooth muscle cell proliferation [19-20].

Carotid sinus reflex: Triggered by balloon/stent stimulation of the carotid sinus, leading to bradycardia and hypotension (as observed in this case).

Contrast agent allergy: Manifesting as rash, pruritus, or severe anaphylaxis.

Puncture site hematoma: Caused by improper puncture tech-

nique or inadequate postoperative compression.

Prevention and Management

Vascular perforation: Preoperative vascular assessment (via CTA/MRI) to select appropriately sized instruments; gentle, precise manipulation during surgery. If perforation occurs, surgical intervention is required.

In-stent thrombosis: Preoperative dual antiplatelet therapy (≥5 days) to inhibit platelet activation; postoperative continuation of antiplatelet therapy for 6–12 months. For established thrombosis, thrombolysis or thrombectomy is recommended [18].

In-stent restenosis: Postoperative control of risk factors (hypertension, dyslipidemia, hyperglycemia); long-term statin use to stabilize plaques. Restenosis may be treated with repeat angioplasty or re-stenting [20].

Carotid sinus reflex: Preoperative atropine (0.5–1 mg) for high-risk patients; intraoperative monitoring of vital signs.

Contrast agent allergy: Preoperative allergy history inquiry; premedication with antihistamines/corticosteroids for high-risk patients. Severe anaphylaxis requires emergency treatment (e.g., epinephrine, corticosteroids).

Puncture site hematoma: Standardized puncture technique; 15–20 minutes of postoperative manual compression. Small hematomas are managed with cold compresses; large hematomas require surgical evacuation.

Conclusion

This case reports a 73-year-old male patient who developed severe stenosis of the initial segment of the right internal carotid artery due to high-risk factors such as long-term hypertension and diabetes, accompanied by frequent dizziness and transient ischemic attacks. After the lesion was confirmed through preoperative examination and met the surgical indications, carotid artery stent implantation was performed for treatment. During the operation, hydrophilic guidewires, 8F guiding catheters, self-expanding nitinol stents and distal filter-based EPD and other instruments were selected. The steps of establishing the puncture access, placing the EPD, balloon dilation, stent insertion and retrieval were completed in a standardized manner. Although transient bradycardia and hypotension occurred intraoperatively, vital signs were stabilized with intervention, and the procedure was completed successfully. Postoperative DSA showed that the stent position was accurate, the stenosis rate decreased from 80% to less than 10%, and the blood flow was unobstructed. The patient has recovered well, with alleviated dizziness, no recurrence of transient cerebral ischemia, improved neurological function and enhanced quality of life. During the one-month follow-up, carotid ultrasound showed that the blood flow within the stent was unobstructed and there was no restenosis. This case demonstrates the effectiveness and safety of the surgery, highlighting the crucial role of the EPD and providing a reference for clinical practice.

Abbreviations

CAS: Carotid Artery Stenting; TIA: Transient Ischemic Attack; EPD: embolic protection device; DSA: Digital Subtraction Angiography; CEA: Carotid Endarterectomy; MRI: Magnetic Resonance Imaging; CTA: Computed Tomography Angiography; ECG: Electrocardiogram; bpm: Beats Per Minute; ACST-2: Second Asymptomatic Carotid Surgery Trial; SPACE-2: Stenting and Angioplasty with Protection in Patients at High Risk for Endarterectomy-2; CASWEP: Carotid Artery Stenting Without Embolic Protection.

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Author Contribution

Ruixiao Yu, involved in drafting and finalizing the manuscript, and Chenlu Zhu, as the corresponding author, contributed to the study concept and supervision. All authors have approved the final version of the manuscript.

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Ethics Approval and Consent to Participate

Not applicable.

Competing Interests

The authors declare that they have no competing interests.

Data Availability

The datasets used and analyzed during the current study are available from the corresponding author on reasonable request.

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