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Cranial nerve injuries post carotid endarterectomy: a 15-year prospective study with routine otolaryngologist and neurological evaluation

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ABSTRACT

Objective: The aim of this prospective monocentric cohort study was to analyze the risk of otolaryngologist-assessed cranial nerve injuries (CNIs) following carotid endarterectomy (CEA) in our academic center during a 15-year period, and to identify possible risk factors for CNI development.

Methods: From January 2007 to December 2022, 3749 consecutive CEAs were performed and their data prospectively recorded in a dedicated database. CNIs were assessed and defined according to a standardized protocol. Instrumental ear, nose, and throat (ENT) evaluations were conducted within 30 days before intervention and before discharge. Preoperative neurological assessments were carried out in all patients with symptomatic carotid stenosis, whereas postoperative neurological evaluations were performed in all patients. Patients with newly onset CNIs underwent follow-up assessments at 30 days and, if necessary, at 6, 12, and 24 months. Perioperative results, including mortality, major central neurological events, and postoperative CNIs, were analyzed. Regression or persistence of lesions during follow-up visits was assessed, and multivariate analysis (binary logistic regression) was conducted to evaluate clinical, anatomical, and surgical technique factors influencing the occurrence of CNIs.

Results: CEAs were performed more frequently in male patients (2453 interventions; 65.5%) than in females (1296 interventions; 34.5%). The interventions were performed in asymptomatic patients in 3078 cases (82%). In 66 cases, the interventions followed a previous ipsilateral CEA. At preoperative ENT evaluation, no cases of ipsilateral pre-existent CNI were recorded. The 30-day stroke and death rate was 1%. In 113 patients (3%), a postoperative neck bleeding requiring surgical revision and drainage was noted. Pre-discharge ENT evaluations identified 259 motor CNIs, accounting for 6.9% of the entire study group. Eighteen patients had lesions in more than one cranial nerve. ENT and neurological evaluations at 30 days showed the complete resolution of 161 lesions, whereas in 98 cases (2.6%), the CNI persisted. At 1 year, the rate of persistent CNI was 0.4% (10 patients), whereas at 2 years, it was 0.25% (6 cases), in all but one asymptomatic. At multivariate analysis, urgent intervention in unstable patients, secondary intervention, a clamping time >40 minutes, a hematoma requiring revision, and a postoperative stroke were independent predictors of CNIs.

Conclusions: Data from this prospective monocentric cohort study showed that the occurrence of CNI following CEA was low, even when an independent multi-specialist evaluation was performed. The percentage of persistent lesions at 2 years was negligible and, in most cases, asymptomatic. (J Vasc Surg 2024;■:1-7.)

Keywords: Carotid endarterectomy; Cranial nerve injury; ENT assessment; Persistence; Risk factors

Cranial nerve injury (CNI) is a quite frequent complication of carotid endarterectomy (CEA), referring to partial or total loss of function of one or more of the cranial nerves in patients operated on of CEA. The average rate of CNIs in the literature is approximately 5%[1]; however, with an extremely wide range of reported values, from 2% to 50%, in the main published series.[2] This fact derives from the extreme heterogeneity in the assessment of CNI following CEA, with regards to the specialist who assesses the lesion (vascular surgeon, neurologist, otolaryngologist), to the timing (only postoperative, pre- and postoperative), and frequency (at discharge, at 1 month postoperatively, or later on) of the assessment, to the

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kind of examined lesion (sensor deficits or purely motor injuries) and to the method (clinical or instrumental) of the assessment. Even if the natural history of CNIs is relatively benign, with a low rate of persistent neurological deficits[3] and a limited impact on the patients' quality of life,[4,5] in a few cases they can be potentially serious and, moreover, there are few data concerning the predictors and the possible strategies of prevention of such injuries.[3,6]

The aim of this prospective monocentric observational cohort study was to analyze the risk of neurologist- and otolaryngologist-assessed motor CNIs following CEA in our academic center during a 15-year period, after the application of an appropriate multidisciplinary protocol, and to identify possible risk factors for CNI development.

ARTICLE HIGHLIGHTS

- **Type of Research:** Single-center prospective cohort study
- **Key Findings**: Multi-specialist (otolaryngological and neurological) assessment of cranial nerve injury (CNI) following 3749 carotid endarterectomies demonstrated a 30-day rate of CNI of 6.9%, with almost complete resolution of the deficits at 2 years.
- **Take Home Message**: the occurrence of CNI following carotid endarterectomy is low, even when an independent multi-specialist evaluation is performed, allowing also the activation of an effective rehabilitation pathway in determining its regression.

MATERIAL AND METHODS

Study group, indications for treatment, and preoperative assessment. In late 2006, a working table involving vascular surgeons; ear, nose, and throat (ENT) specialists; neurologists; and hospital management was established at our institution, and a multidisciplinary protocol of assessment and treatment of CNIs following CEA was created. The details of the protocol are explained further in the text. Until 2006, we did not perform any preoperative ENT assessment: we defined postoperative CNIs by ourselves without ENT assessment, which was selectively reserved only for patients with severe postoperative injuries. The multidisciplinary protocol has been part of our clinical routine since January 2007, and its results were collected using a dedicated database that was prospectively maintained and consisted of 146 numerical fields, encompassing key variables related to perioperative classification and surgical and anesthetic techniques, as well as medium- and long-term follow-up data, following suggested reporting standards.[7]

From the beginning of the protocol application (January 2007) to December 2022, 3749 consecutive carotid interventions were performed at our institution. In the same period of time, 145 carotid artery stenting (CAS) interventions were performed; the indications for CAS were the presence of the so called "hostile neck," of early (<24 months) restenosis following CEA, and of the paralysis of the contralateral vocal cord demonstrated at the preoperative ENT evaluation.

All patients underwent a preliminary Doppler ultrasound examination of the supra-aortic trunks. The examinations were conducted using state-of-the-art imaging equipment. For symptomatic patients with a stenosis ≥50% and for asymptomatic patients with carotid stenosis exceeding 60%, a computed tomography angiography of the neck and intracranial vessels, along with the study of cerebral parenchyma, was routinely performed. In cases where patients had contraindications to the use of iodine contrast material, magnetic resonance imaging was preferred. The indication for CEA in symptomatic patients was the presence of a ≥50% carotid stenosis; in asymptomatic patients, prior to 2018, the indication for surgery was based solely on the presence of stenosis exceeding 60% in patients with low-to-intermediate surgical risk. However, following the publication of the new European Society for Vascular Surgery guidelines,[8] surgery in asymptomatic patients was recommended for patients with a life expectancy greater than 3 years and the presence of a >60% plaque with characteristics considered at high risk of embolization.

In the preoperative setting, the CNI protocol included ENT evaluation within 30 days before intervention; the evaluation, which was performed by the same experienced otolaryngologist consultant by using a stroboscopic light fibreoptic laryngoscopy, included the assessment of phonation and swallowing, the evaluation of vocal cord motility, and the eventual pre-existence of CNIs. Moreover, preoperative neurological assessments were carried out by neurologists in all patients with symptomatic carotid stenosis in the days before the intervention. All the patients gave their informed consent for using their clinical data for future investigations. Moreover, considering that the protocol was approved by our hospital as a routine clinical activity, the approval of the local Ethical Committee was not mandatory. The manuscript adheres to the Strengthening the Reporting of Observational Studies in Epidemiology (STROBE) guidelines.[9]

Anesthetic and surgical details. For patients undergoing general anesthesia, cerebral perfusion monitoring was conducted through somatosensory evoked potentials (SSEPs), with the insertion of a selective shunt in case of critical modifications in the potentials. This strategy was generally used in patients who did not accept local anesthesia with clinical monitoring of the neurological status or were poorly compliant with it. In patients receiving loco-regional anesthesia or cooperative patient general anesthesia (CoPaGeA),[10] which was routinely adopted in the years following 2010, any

hemispheric clinical impairment observed during clamping was considered a criterion for selective shunt insertion. In cases where SSEP was not assessable or patients were unresponsive or intolerant to local anesthesia or CoPaGeA, a shunt was immediately placed. Prior to clamping, all patients received intravenous heparin prophylaxis at a dose of 30 IU/kg, with continuous monitoring of the activated clotting time.

The surgical procedure followed an institutional standardized technique. Initially, the internal carotid artery was clamped, and a long arteriotomy was performed to extensively overpass the lesion. Then, open endarterectomy of the common and internal carotid arteries and eversion endarterectomy of the external carotid artery were conducted. Arteriotomy closure was achieved by interposing a polyurethane patch in the majority of the cases. In selected cases, where there was kinking and coiling of the internal carotid artery or when the lesions were limited to the proximal part of the vessel, the eversion technique was preferred. Immediately after removing the clamps, an intraoperative quality check was routinely performed using angiography or Doppler ultrasound. At the end of the intervention, drainage in the surgical site was routinely placed. Once the surgery was completed, patients were transferred to the intensive/sub-intensive care unit or to the ordinary ward, based on the preoperative anesthetic evaluation.

At discharge, which usually occurred in the second postoperative day, except in the presence of complications requiring prolonged hospitalization, postoperative medical management consisted of single antiplatelet treatment, along with high-dose statins. In patients requiring oral anticoagulation for comorbidities, antiplatelet treatment with 100 mg acetylsalicylic acid was associated for the first postoperative month.

CNI evaluation, endpoints, and statistical analysis. All patients underwent instrumental ENT examination before discharge home. Patients with motor CNIs of new onset underwent further follow-up assessments at 30 days, and, if the lesion persisted, at 6, 12 and 24 months. In patients with persistent lesions beyond 30 days, a rehabilitative pathway was started. Moreover, at 30 days after the procedure, all patients underwent a comprehensive neurological assessment to evaluate neurological outcomes, including transient ischemic attack (TIA), and minor or major strokes.

The results were analyzed in terms of perioperative mortality, major neurological events, and CNIs. Among major neurological events, we considered TIAs, defined as a single episode of focal neurological dysfunction and/or amaurosis fugax, not associated with cerebral infarction and spontaneously resolved within few minutes to hours, minor strokes (defined as any neurological event lasting more than 24 hours with recovery in several days without residual functional impairment), and major strokes (any neurological event lasting more than 24 hours with residual stabilized neurologic deficit). CNIs were considered resolved if the otolaryngologist follow-up visits showed complete resolution of the clinical signs and symptoms, whereas they were considered persistent if the lesion did not resolve, even in the presence of clinical and patient-reported improvements.

Statistical analysis was performed with the SPSS 28.0 for Windows program (SPSS Inc). Clinical, demographic, and anatomical characteristics were analyzed. Continuous variables were tested for normality through the Shapiro-Wilk test; quantitative variables were compared through the Student *t*-test. The χ^2 test was employed to compare qualitative variables between subgroups and to identify factors associated with postoperative CNI occurrence. To gain insight into factors independently associated with CNI, all variables with values of $P < .2$ in the previously described bivariate analyses were used to develop a multivariable regression model. Multivariate analysis was conducted using a binary logistic regression with Wald's backward stepwise model.

RESULTS

Study cohort: demographic characteristics, risk factors, and comorbidities. CEAs were performed more frequently in male patients (2453 interventions; 65.5%) than in females (1296 interventions; 34.5%). The mean age of patients at the time of the intervention was 73.8 ± 7.7 years. The details and the definitions of risk factors and comorbidities can be found in Table I.

Anatomical and clinical features of extracranial carotid disease. The interventions were performed in asymptomatic patients in 3078 cases (82%); among the

Table I. Demographics and comorbidities

	Factor
Age, years	73.8 ± 7.7
Octogenarians	754 (20)
Smoking history	2212 (59)
Hypertension	3082 (82)
Diabetes	818 (22)
Coronary artery disease	1358 (36)
Peripheral arterial obstructive disease	610 (16)
Hypercolesterolemia	2483 (66)
Chronic renal failure	196 (5.5)

Data are presented as number (%) or mean ± standard deviation.
Hypertension is defined as arterial hypertension in medical treatment.
Diabetes is defined as the need for specific antidiabetics drugs.
Coronary artery disease is defined as prior myocardial infarction or surgical or percutaneous revascularization.
Peripheral arterial obstructive disease is defined as ankle brachial index value lower than 0.9 and higher than 1.4.
Hypercolesterolemia is defined as hypercolesterolemia in medical treatment.
Chronic renal failure is defined as estimated glomerular filtration rate <60 ml/min/1.73 m^2.

671 interventions performed for symptomatic stenosis, 436 were performed for previous TIA, 117 and 62 for previous stable minor or major stroke, respectively, whereas 56 were performed in patients with unstable neurological symptoms (recent TIA -<48 hours-, crescendo TIAs, stroke in evolution, acute/recent minor stroke -<7 days-). In 151 cases, patients reported in their clinical history a previous symptom due to the contralateral internal carotid artery. The majority of patients had 80% to 99% carotid stenosis at the operated side (2062 cases; 55%), whereas a 60% to 80% stenosis was present in 1397 cases (37.3%). Two hundred and forty-six interventions were performed for carotid near occlusion (6.5%), whereas the remaining 44 patients (1.2%) had a 50% to 60% stenosis.

In 66 cases, the interventions followed a previous ipsilateral CEA. In 139 cases, occlusion of the contralateral internal carotid artery was present, and in 343 cases, a contralateral CEA had been performed. At preoperative ENT evaluation, no cases of ipsilateral pre-existent CNI were recorded.

Intraoperative details. CoPaGeA with clinical monitoring of neurological status was used in the majority of the cases (2432 interventions; 63%); in 961 cases (25.5%), general anesthesia with SSEP monitoring was used, whereas the remaining 437 interventions (11.5%) were performed under pure local anesthesia. The rate of intraluminal shunt placement was 16% (598 cases). Patch closure was used in 95% of the cases (3558 interventions); primary closure and eversion were performed in 85 (2.3%) and 82 (2.2%) cases, respectively, whereas 24 patients (0.5%) had a bailout common-to-internal carotid bypass. In 10 cases, completion study showed significant defects at the site of endarterectomy, requiring immediate re-exploration and correction. The mean time of carotid clamping (including both time of eventual shunting and time of ischemia) was 39 ± 11.5 minutes. We were not able to identify any intra-operative CNIs.

Perioperative results: mortality, neurological events, cardiovascular events, and local complications. There were 13 perioperative deaths, with a mortality rate of 0.3%. In two cases, the cause of death was a major perioperative stroke. Four patients suffered from a fatal acute myocardial infarction, and two patients had postoperative intracranial bleeding. In the remaining five cases, the causes of death were acute respiratory distress in two cases, sepsis with multi-organ failure in two cases, and rupture of the bladder in the remaining one. Non-fatal neurological events occurred in 36 patients: eight had a postoperative TIA (0.2%), fifteen had a minor stroke (0.4%), and 11 had a major stroke (0.3%). The 30-day stroke and death rate was 1% (37 cases). Non-fatal postoperative acute myocardial infarction was recorded in 25 patients (0.6%). In 113 patients (3%), a postoperative neck bleeding requiring surgical revision and drainage was noted.

Cranial nerve injuries: perioperative and follow-up results. At discharge, ENT evaluation identified 259 motor CNIs in 241 patients, accounting for 6.9% of the entire study group. Of these, 116 affected the hypoglossal nerve, 92 the facial/marginalis nerves, 48 the laryngeal nerve, and three other nerves such as the glossopharyngeal and oculomotor nerves. Eighteen patients had lesions involving two cranial nerves (hypoglossal and facial in 12 cases, facial and laryngeal in three cases, and hypoglossal and laryngeal in the remaining three). In 151 of 259 CNIs, the lesion was asymptomatic.

ENT and neurological evaluations at 30 days showed the complete resolution of 161 lesions, whereas in 98 cases (2.6%; 66 asymptomatic patients) the CNI persisted. At 1 year, the rate of persistence CNI was 0.4% (10 patients, eight asymptomatic), whereas at 2 years, it was 0.25% (six cases). The details regarding numbers and involved nerves in the cases of persistent lesions at 1, 12, and 24 months are reported in Table II. In all the patients who had a persistent laryngeal injury at 1 year, there was no improvement of the lesion at 2 years. Indeed, all the persistent lesions involving the laryngeal nerve were asymptomatic, whereas the patient with a persistent lesion of the hypoglossal nerve showed mild dyslalia.

At bivariate analysis, urgent interventions in patients with unstable neurological status and patients operated on for carotid restenosis had a higher risk of developing CNI; the same was in patients operated on general anesthesia, in those who had a total clamping time of more than 40 minutes, in patients who had re-exploration of the internal carotid artery following unsatisfactory completion study, in the case of neck hematoma requiring surgical revision, and in the patients who suffered from a postoperative stroke (Table III). At multivariate analysis, urgent intervention in unstable patients, secondary intervention, a clamping time >40 minutes, a hematoma requiring revision, and a postoperative stroke were independent predictors of CNI (Table IV). In the Fig, the role of each predictor in determining the injury of the involved CNs is reported. Postoperative stroke was more frequently associated with facial and hypoglossal lesions, whereas urgent CEA in unstable patients was more frequently associated with laryngeal lesions.

DISCUSSION

The reported incidence of CNIs following CEA in the literature varies from 5% to 20% depending on the case series, with resolution in the months following surgery and permanent damage reported in less than 1% of cases.[3] Our case series is broadly in line with the literature, with an incidence of CNIs of 6.9%, which is, however, slightly higher than the average frequency of about 5% reported in the literature.[1] This finding can be easily justified by the complex multi-disciplinary and multi-

Table II. Cranial nerve injuries (*CNIs*) at discharge and during follow-up

	At discharge No. (%)	At 1 month No. (%)	At 12 months No. (%)	At 24 months No. (%)
Overall CNI rate	259 (6.9)	98 (2.6)	10 (0.4)	6 (0.25)
Affected nerves				
Facial/marginalis	92 (2.5)	34 (0.9)	2 (0.1)	–
Hypoglossal	116 (3.1)	39 (1)	3 (0.1)	1 (0.05)
Laryngeal nerve	48 (1.3)	24 (0.65)	5 (0.2)	5 (0.02)
Glossopharyngeal	2 (0.1)	1 (0.05)	–	–
Other	1 (0.05)	–	–	–

Data are presented as number (%).

Table III. Bivariate association of preoperative and intraoperative variables with cranial nerve injury (*CNI*)

	No.	CNI, %	*P*
Age >80 years	754	7.6	.1
Male gender	2453	6.8	.6
CAD	852	7.7	.1
Arterial hypertension	3082	6.1	.4
Diabetes	818	5.8	.3
Symptomatic patient	671	7.9	.07
Urgent intervention	56	12.5	.04
Secondary intervention	66	15	.004
Contralateral ICA occlusion	139	9.3	.1
General anesthesia	961	8.4	.003
Shunt insertion	598	5.6	.4
Clamping time >40 minutes	1146	9.5	<.001
Patch closure	3558	5.5	.4
Immediate revision of ICA defects	10	33	.02
Hematoma requiring reintervention	113	19.5	<.001
Postoperative stroke	26	23	<.001

CAD, Coronary artery disease; *ICA*, internal carotid artery.

Table IV. Independent predictors for cranial nerve injury (CNI) occurrence

	95% CI	HR	*P*
Urgent intervention	1.02-2.1	1.5	.03
Secondary intervention	1.08-4.6	2.2	.003
Clamping time >40 minutes	1.9-3.8	2.8	<.001
Hematoma requiring reintervention	1.9-5.9	3.3	<.001
Postoperative stroke	1.4-11.6	4.1	<.001

CI, Confidence interval; *HR*, hazard ratio.

specialist strategy we adopted in our institution, making it possible to identify lesions that would not be probably detected by non-specialists, such as the vascular surgeons themselves. In the literature, we found very few old studies adopting the same strategy of complex pre- and postoperative ENT and neurological evaluation, whereas in the majority of the papers, the evaluation of CNIs is extremely heterogeneous and often surgeon- or patient-driven.[11] Only Assadian et al[12] reported a 6% CNI rate in 170 interventions detected with a similar pre- and postoperative protocol of CNI assessment and using a transverse neck incision.

Consistent with the results of previous studies, in our experience, a high percentage of CNIs resolved in the first postoperative month, and about 1% persisted at 1-year follow-up.[13] Moreover, we found a complete resolution of the nerve deficit in almost all cases, with only six patients (0.25% of the study group) having a persisting injury at 24 months post-surgery. In our study, the time limit for evaluating CNIs and their potential irreversibility was set at the 24th month post-surgery, whereas other the majority of studies have this cutoff at 6 or 12 months.[14-16] Even if a deficit lasting more than 6 to 12 months is generally considered permanent, there are anecdotal cases of complete recovery even after 4 years from the intervention,[14] showing that the recovery process from CNIs can extend well beyond 12 months; indeed, our study recorded a further reduction in persistent CNIs, with the incidence decreasing from 0.4% to 0.25% between the first and the second postoperative year. Of interest, the majority of persistent injuries involved the laryngeal nerve, with complete vocal cord paralysis, which was, however, completely asymptomatic. Such a result can be favored by the routine program of rehabilitation and speech therapy started in patients with persistent lesions beyond 30 days, supporting the recovery of the motility of the vocal cord and the compensation through the contralateral vocal cord.

Several studies tried to identify different predictors of CNI after CEA: Bennett et al[6] found that older age, bleeding disorders, a long operative time, and secondary interventions were independent predictors of CNI, whereas in the study from Fokkema et al,[3] urgent cases, immediate revision of the internal carotid artery, and return to operating room, both for neurological events and for postoperative bleeding, were independently associated with CNI development. In the present study, urgent interventions in patients with unstable neurological

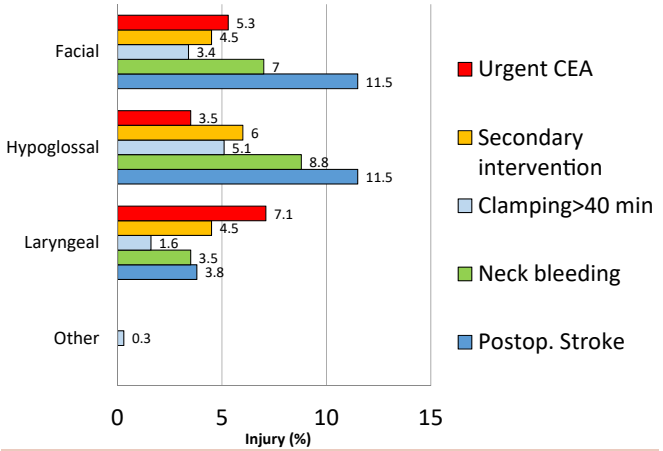

Fig. Role of predictors in each nerve injury. *CEA*, Carotid endarterectomy.

status, secondary interventions, a clamping time longer than 40 minutes, the presence of neck hematoma requiring revision, and postoperative stroke were found to be independent risk factors for the occurrence of post-surgery CNI. All these factors imply the occurrence of complex anatomical conditions, of extensive lesions, or particularly high carotid bifurcations, requiring more vigorous retraction, extensive use of electrocautery, or urgent settings where time is crucial and a quick access to the carotid bifurcation is mandatory.[2] Contrary to what has been described in other studies,[17] we have not identified a correlation between the occurrence of CNI and female sex or older age, whereas general anesthesia was found to be associated with an increased risk of CNI only on univariate analysis but not on multivariate analysis, contrary to the findings of Grieff et al, who identified general anesthesia as an independent risk factor for CNI.[18]

In these instances, the use of perioperative adjustments to mitigate potential cranial nerve damage or expedite possible recovery has been suggested. The identification of preoperative and intraoperative factors associated with increased risk of CNI could allow the implementation of an intraoperative and postoperative prevention strategy in such high-risk patients. Possible precautions include performing preoperative ultrasound mapping of the carotid bifurcation to reduce the extent of the surgical incision, a limited use of retractors, the use of bipolar forceps to limit electrocautery damage to nerve tissues, a careful isolation and retrieval of nerve branches on vessel loops, a careful activated clotting time monitoring to prevent thrombotic or hemorrhagic complications that could lead to early reinterventions, and the ligation of peri-nerve satellite veins. Also, the administration of dexamethasone pre- and postoperatively and postoperative supplements with lipoic acid, L-acetyl carnitine, and citicoline to accelerate the recovery in case of CNI has been suggested.[19] However, in our experience, we never used such strategies, because we firmly believe that only proper surgical technique and a rehabilitation pathway can respectively prevent and regress CNIs.

The main limitations of this study are its single-center nature and the fact that the majority of cases were performed using the same surgical technique and that we were not able to analyze the impact of the single operating surgeon on the occurrence of CNI. Moreover, we did not analyze the occurrence of sensory nerve damage, considering that a certain degree of skin sensitivity alterations is inevitable during carotid surgery. Finally, this kind of study requires a multidisciplinary and multiprofessional protocol, and this may make its complete reproducibility more difficult.

On the other hand, the strengths include the prospective nature of this large case series, the fact that all examined patients were treated by a small number of experienced surgeons with comparable levels of expertise, the extended follow-up to 24 months, which was performed by the same ENT specialist who performed preoperative and early postoperative assessment, and the performance of a multi-specialist assessment including clinical and instrumental evaluation. Moreover, a path such as the one adopted in our experience was, in our opinion, also extremely useful from the patient's perspective. Not only was the patient informed of the possibility of being affected by postoperative CNI, but he or she also knew that, should this occur, an effective diagnostic and rehabilitation pathway was predefined. In short, the patient feel him/herself constantly followed up and helped. This claim is well-supported by the fact that, during the period under study, there were no medical-legal disputes concerning the occurrence of CNI following CEA.

CONCLUSION

Data from this prospective monocentric cohort study showed that the occurrence of CNI following CEA was low, even when an independent multi-specialist evaluation was performed. The majority of injuries completely resolved at 1 year of follow-up, and the percentage of persistent lesions at 2 years was negligible and, in most cases, asymptomatic. In this context, the collaboration of a multidisciplinary team is crucial both for the early identification of CNI and for activating an effective rehabilitation pathway in determining its regression. The identification of preoperative and intraoperative factors associated with an increased risk of CNI could allow the implementation of an intraoperative prevention strategy in high-risk patients.

AUTHOR CONTRIBUTIONS

Conception and design: WD, LD, CS, CP, RP
Analysis and interpretation: WD, SS, EG, AF, RP
Data collection: WD, LD, MC, RD, CS, MN

Writing the article: WD, MC, MN
Critical revision of the article: WD, SS, EG, LD, AF, RD, CS, CP, RP
Final approval of the article: WD, SS, EG, MC, LD, AF, RD, CS, MN, CP, RP
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DISCLOSURES

None

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