Clinical Neuroscience Research, 2025, Volume 3, Issue 3
http://ojs.bbwpublisher.com/index.php/CNR
Online ISSN: 2981-8133
Print ISSN: 3083-4899

Exogenous Compression Caused by a Mass at the Bifurcation of the Carotid Artery

Chih-hsien Lin, Chun-Chung Chen*

Neurosurgery Department, China Medical University Hospital, Taichung, Taiwan, China

**Author to whom correspondence should be addressed.*

Abstract: The bifurcation of the carotid artery is a crucial node for blood supply to the brain. External compression caused by surrounding masses can lead to severe hemodynamic disturbances and neurological abnormalities. This article reviews the pathogenesis, clinical manifestations, diagnostic methods, and treatment approaches of this condition, including the pathological characteristics of common compression lesions such as carotid body tumors and schwannomas. It compares the applicability of imaging examination methods such as ultrasound, CTA, and DSA, analyzes the suitable conditions and treatment effects of surgical removal, vascular reconstruction, and interventional therapy. The research aims to provide standardized diagnostic and treatment concepts for clinical practice, emphasizing the crucial role of individualized treatment plans in improving patient outcomes. It also looks forward to the development trend of precision medicine guided by imaging.

Keywords: Carotid artery bifurcation; External compression; Individualized treatment

Online publication: October 17, 2025

1. Introduction

1.1. Research background

The carotid bifurcation, located in the middle and lower part of the neck, is a crucial site where the common carotid artery divides into the internal carotid artery and the external carotid artery. It primarily supplies blood to the anterior two-thirds of the brain and the eyes. This anatomical region is complex, with nearby important structures such as the vagus nerve, hypoglossal nerve, and cervical sympathetic trunk. Additionally, its hemodynamic conditions are unique, with blood flow impacts at the bifurcation causing local pressure variations, making it a sensitive area for lesions. When a mass appears around the carotid bifurcation, external compression can directly affect the vessel's course and lumen shape, leading to abnormal blood flow velocity, turbulence, and even vascular occlusion. According to clinical data, the occurrence of cerebral ischemia caused by such compression accounts for approximately 3.2%–5.8% of non-traumatic cerebral infarction etiologies. Moreover, due to the subtle early symptoms, treatment opportunities are often missed. With the development of imaging diagnostic techniques, the detection rate of asymptomatic compression has been increasing year by year. However, clinical management strategies for such conditions have been controversial, highlighting the necessity for further exploration into this disease state [1].

1.2. Research objectives and significance

This study aims to systematically organize the pathophysiological mechanisms, clinical diagnostic approaches, and treatment technology developments related to external compression caused by carotid bifurcation masses, by integrating the latest domestic and international research findings. By clarifying the compression characteristics and risk levels of different types of masses, it provides clinicians with normative decision-making references from diagnosis to treatment. The practical significance of this study lies in: first, improving the identification level of early asymptomatic cases, thereby reducing the occurrence of ischemic stroke; second, improving the selection of treatment plans for complex cases, taking into account both tumor resection and vascular protection clinical needs; third, promoting the application of multidisciplinary team (MDT) mode in this field, facilitating collaborative diagnosis and treatment among departments such as radiology, neurosurgery, and vascular surgery, ultimately providing theoretical support and practical guidance for improving patients' quality of life and reducing postoperative complications [2].

2. Anatomical and physiological basis of the carotid bifurcation

2.1. Structure of the carotid bifurcation

The carotid bifurcation is generally located at the level of the fourth cervical vertebra, with a few cases occurring at the level of the third or fifth cervical vertebra. The morphology of the carotid bifurcation varies and is divided into three types: right-angle type, acute-angle type, and obtuse-angle type. The acute-angle type has a large turning angle of blood flow, making it more susceptible to external compression. Here, the common carotid artery divides into the internal carotid artery (ICA) and the external carotid artery (ECA). The ICA has a diameter of approximately 4–5 mm and runs towards the brain [3]. There are no branches inside the carotid sheath, and it directly supplies the anterior-medial part of the cerebral hemisphere and the eyes. The ECA has a slightly smaller diameter and gives off branches such as the superior thyroid artery and the lingual artery to supply the soft tissues of the neck. Behind the bifurcation, there is a branch of the recurrent laryngeal nerve passing through, with the medial side adjacent to the pharyngeal lateral wall and the lateral side being the sternocleidomastoid muscle. These anatomical relationships determine the neurological symptoms and local signs that may accompany mass compression. The vascular wall at the carotid bifurcation is divided into the intima, media, and adventitia. The contraction of smooth muscle cells in the media is important for maintaining vascular tension. Surrounding the adventitia is the carotid sinus, which contains baroreceptors that can sense changes in blood pressure and maintain blood flow stability through neural regulation. When this area is compressed, it may cause a reflexive decrease in blood pressure or a slowing of heart rate [4].

2.2. Hemodynamic characteristics of the carotid artery

The hemodynamic characteristics of the carotid artery are crucial for ensuring normal blood supply to the brain. It functions like a precise "life-sustaining pump", continuously supplying blood to the brain. Under normal circumstances, the blood flow velocity and volume in the carotid artery are relatively stable. The internal diameter of the common carotid artery is relatively large, approximately 5–8 mm, with a blood flow velocity of 40–80 cm/s. The internal diameter of the internal carotid artery is approximately 4–6 mm, and its blood flow velocity falls within the same range. The blood flow velocity in the left carotid artery is relatively faster, averaging 77 cm/s, while the blood flow velocity in the right carotid artery averages 65 cm/s, as shown in **Figure 1** (preoperative MRA and CTA). This stable blood flow velocity and volume ensure that the brain receives sufficient oxygen and nutrients, maintaining its normal physiological functions. The blood test results are shown in **Table 1**. The blood flow in the carotid artery is pulsatile, correlating with the periodic contraction and relaxation of the heart. During heart contraction, blood is rapidly pumped into the aorta and then into the carotid artery, causing an increase in pressure and a speedup in blood flow velocity, forming the peak systolic velocity. During heart relaxation, the pressure in the artery decreases, and the blood flow velocity slows down, forming the end-diastolic velocity [5]. This pulsatile blood flow and its dynamic changes can be clearly displayed through DSA (**Figure 2**), providing a continuous blood supply to the brain and, to a certain extent, massaging the vascular wall, helping to maintain its elasticity and normal function. Under normal physiological conditions, the hemodynamic parameters of the carotid artery are precisely regulated by various factors. From the perspective of neural regulation, when the sympathetic nervous system is excited, blood vessels contract, blood flow resistance increases, and blood flow velocity slows down. Conversely, when the parasympathetic nervous system is excited, blood vessels dilate and blood flow velocity increases. In terms of humoral regulation, hormones such as angiotensin and adrenaline have a significant impact on vascular vasomotor function [6]. When blood pressure decreases in the body, the renin-angiotensin-aldosterone system is activated, and angiotensin II promotes vascular contraction, leading to an increase in blood pressure and thus regulating the hemodynamic parameters of the carotid artery. The autoregulation of the carotid artery is also crucial. When blood pressure fluctuates within a certain range, the carotid artery contracts and relaxes on its own to maintain relative stability in cerebral blood flow, preventing significant changes in cerebral blood flow due to fluctuations in blood pressure [7]. When blood pressure suddenly rises, the smooth muscle of the carotid artery contracts, the vessel diameter becomes smaller, blood flow resistance increases, and cerebral blood flow does not increase excessively; conversely, when blood pressure decreases, the vessel relaxes, and cerebral blood flow remains essentially unchanged. This vasomotor function can be observed under intraoperative microscopy (**Figure 3**), and its structural basis can be verified through pathology specimens (**Figure 4**).

Table 1. Lab data

Laboratory examination	Value	Unit
White blood cell count	15.0*	K/uL
Red blood cell count	5.50	M/uL
C-reactive protein	16.3*	Mg/l
Hemoglobin	14	g/dL
Ca	8.9	Mg/dL
Na	130*	mmol/L
K	3.0*	Mmol/L
TSH	3.8	mIU/L
T3	2.95	mmol/L
T4	177	mmol/L
Plasma free Metanephrines	0.23	nmol/L
Normetanephrines	0.8	nmol/L
24-hour Urine Metanephrines	132	ug/g creatinine
Normetanephrines	221	ug/g creatinine
Chromogranin A	88	ng/mL
Serum Dopamine	323	ug/day
Neuro-Specific Enolase (NSE)	8	ng/mL

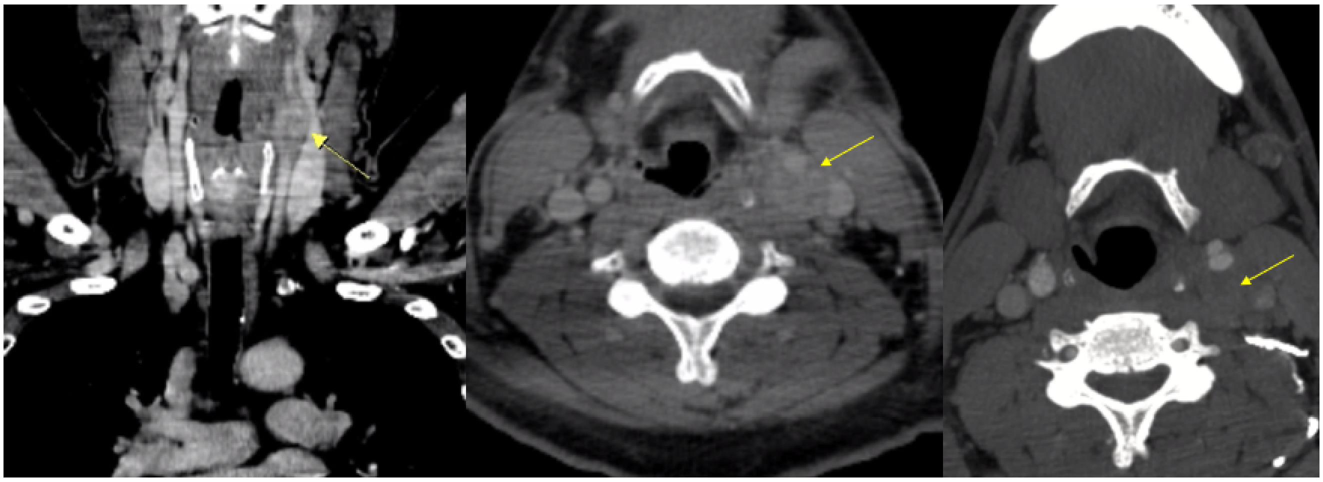

Figure 1. MRA+CTA

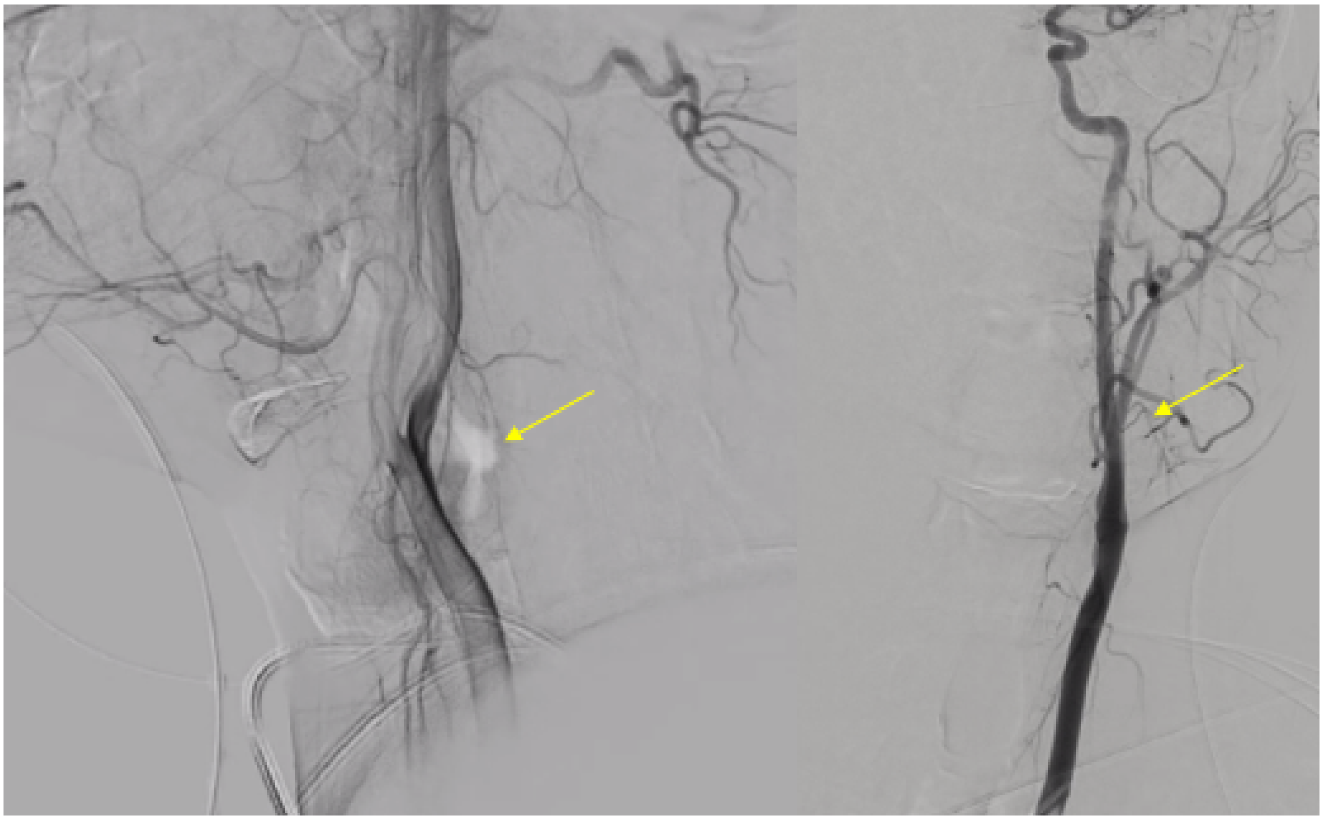

Figure 2. DSA

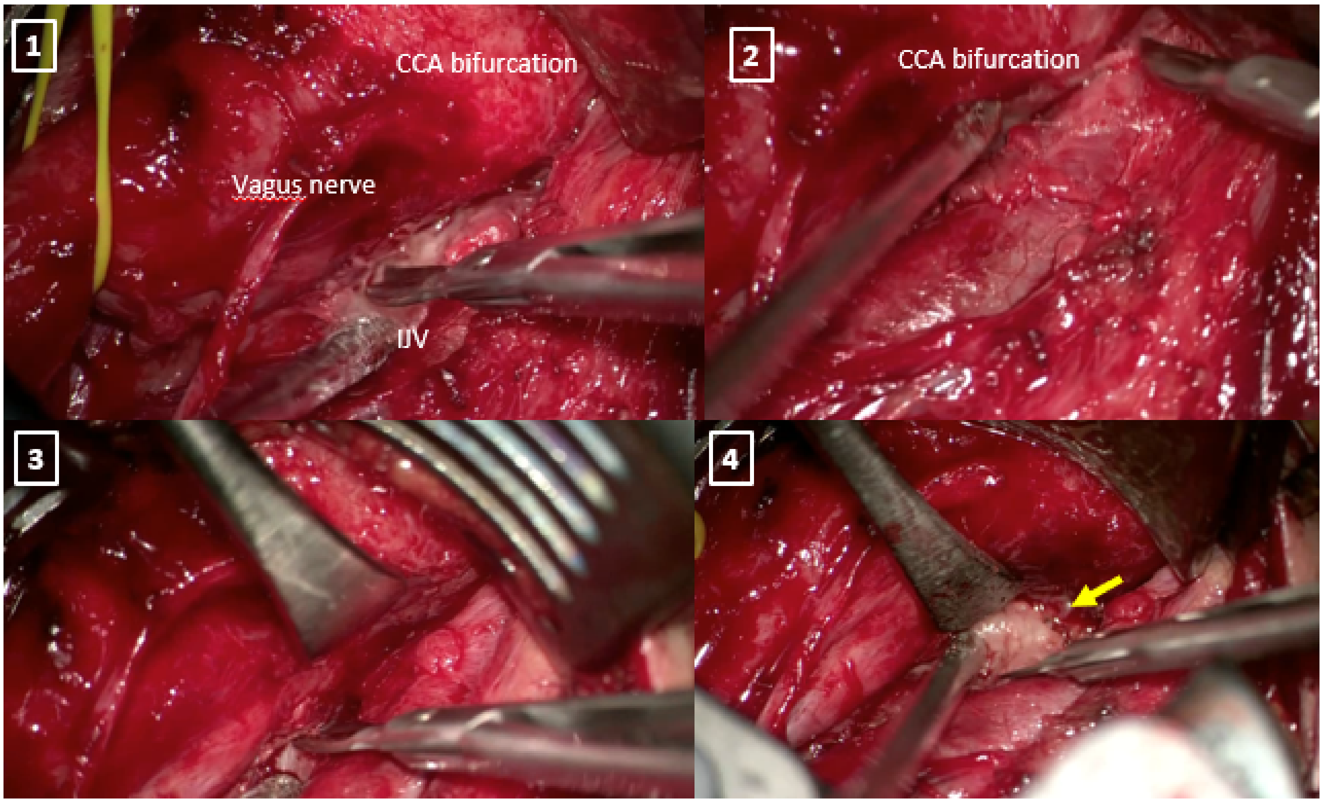

Figure 3. Intraoperative microscope view

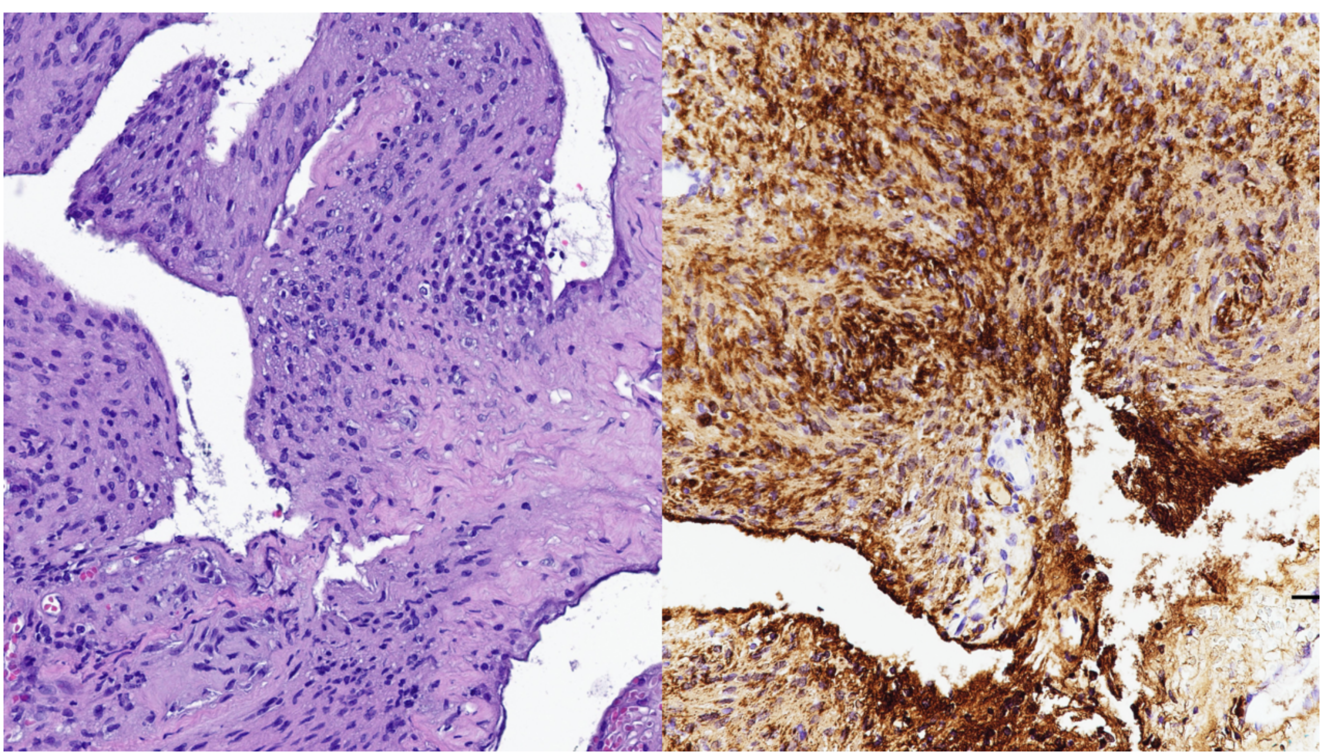

Figure 4. Pathological specimen

3. Causes and mechanisms of external compression of the carotid artery by masses

3.1. Common types of masses causing compression

Carotid body tumor (CBT) is the most common compressive lesion, originating from the chemoreceptor cells of the carotid body and belonging to the category of paragangliomas. Most of them are benign, with a malignant proportion of approximately 5%–10%. The tumor often occurs within the bifurcation angle, with a round or oval shape, medium texture, and is closely connected to the adventitia of the carotid artery [8]. It moves up and down with swallowing and encapsulates the vascular wall, causing compression. Schwannoma (neurilemmoma) ranks second, often originating from the sheath of the vagus nerve or sympathetic nerve, located within the carotid sheath, and presenting as a spindle-shaped tumor with a capsule, clearly demarcated from the blood vessel. However, larger ones can compress or push the carotid artery. Lymphoma manifests as multiple masses in the neck, which are hard and often invade the perivascular adipose tissue, displacing and compressing the blood vessels. Additionally, metastatic lymph node enlargement, such as from thyroid cancer, nasopharyngeal cancer metastasis, branchial cysts, lipomas, etc., can also be a compressive factor. The biological characteristics of different masses determine the mode of compression; CBT is mostly encapsulated compression, Schwannoma is mostly pushing compression, and lymphoma is mostly infiltrative compression [9].

3.2. Discussion of the pathogenesis

External compression of the carotid artery by a mass causes pathological changes through two mechanisms: mechanical mechanics and hemodynamics. Mechanical compression directly deforms the vascular wall. When the compressive force exceeds the elastic limit of the vascular wall, luminal stenosis occurs. At this time, vascular smooth muscle cells undergo compensatory proliferation to maintain the shape of the lumen. Long-term compression can lead to media atrophy, elastic fiber breakage, and decreased vascular wall compliance. In terms of hemodynamics, high shear forces caused by increased blood flow velocity at the stenosis site can damage vascular endothelial cells, activate the coagulation system, and increase the risk of thrombosis. Moreover, vibration of the vascular wall caused by turbulence can trigger local inflammatory reactions, promoting platelet aggregation and the formation of atherosclerotic plaques, thus forming a vicious cycle of “compression-stenosis-thrombosis-ischemia.” When the carotid sinus is compressed, baroreceptor dysfunction can occur, leading to carotid sinus syndrome, which is characterized by paroxysmal dizziness and syncope. This is caused by vagal nerve excitation, slow heart rate, and peripheral vascular dilation. When a mass compresses surrounding nerves, there are corresponding neurological symptoms, such as hoarseness due to compression of the vagus nerve and tongue muscle atrophy due to compression of the hypoglossal nerve [10].

4. Clinical manifestations and diagnostic methods

4.1. Clinical symptoms

Cervical mass is the most common initial symptom, with approximately 82% of patients being able to palpate a painless mass. Its hardness varies depending on the tumor type: CBT has a medium hardness and a pulsatile sensation; schwannoma is soft and elastic; lymphoma is relatively hard and fixed. When the mass enlarges, there is local pain or pressure sensation, which worsens during swallowing. Cerebral ischemia symptoms are the most severe clinical manifestations, including transient ischemic attack (TIA) symptoms such as transient blackouts, limb weakness, and slurred speech, which generally last for several minutes to hours and may recur, indicating a vascular stenosis greater than 70%. In severe cases, acute cerebral infarction may occur, leaving permanent neurological deficits. Symptoms of nerve compression have localization significance: compression of the vagus nerve leads to hoarseness and difficulty in swallowing water; compression of the hypoglossal nerve causes tongue deviation when extended; and compression of the cervical sympathetic nerve leads to Horner's syndrome (miosis, ptosis, and anhidrosis of the face) [11].

4.2. Diagnostic techniques

4.2.1. Imaging examinations

Ultrasound is the preferred screening method, which can clearly visualize the location, size, and relationship with the carotid artery of the mass. Color Doppler can observe changes in blood flow velocity and assess the degree of stenosis, with a sensitivity of up to 95%. However, it may not provide sufficient detail regarding tumor invasion of the vascular wall. Computed Tomography Angiography (CTA) can perform three-dimensional reconstruction of the carotid bifurcation, accurately measure the degree of stenosis, and display the relationship between the mass, bones, and soft tissues. It excels in identifying calcified lesions and is suitable for preoperative anatomical assessment. However, it involves radiation exposure and carries a risk of contrast medium allergy. Magnetic Resonance Angiography (MRA) is radiation-free, provides clear boundaries between the vascular wall and soft tissues, and has specificity in diagnosing liquid-containing lesions such as schwannomas. It can evaluate cerebral perfusion, but the examination time is long, and its evaluation of severe stenosis is slightly inferior to CTA. Digital Subtraction Angiography (DSA) remains the "gold standard" for dynamically observing hemodynamic changes, understanding collateral circulation, and performing interventional therapy. However, it is an invasive examination and is mainly suitable for preoperative assessment of complex cases [12].

4.2.2. Other auxiliary examinations

The focus of physical examination is mass palpation (texture, mobility, pulsatility), auscultation (presence of vascular murmurs), neurological function examination, etc. Laboratory tests, including tumor marker tests, have diagnostic significance for lymphoma and metastatic tumors, and coagulation function tests can determine the risk of thrombosis. Pathological examination determines the nature of the mass through fine needle aspiration or surgical specimen biopsy, which is the most accurate way to distinguish between benign and malignant tumors. However, for tumors with abundant blood supply, such as CBT, there is a risk of inducing bleeding through aspiration, so caution should be taken.

5. Treatment strategies and case analysis

5.1. Surgical treatment

Surgical resection is the curative treatment for most tumors, with the principle of complete removal of the tumor and protection of carotid blood flow as much as possible. For CBT, the surgical procedure is selected based on the Shamblin classification. Type I tumors are small and have less adhesion to blood vessels, so they can be directly removed; Type II tumor partially surrounds blood vessels and require vascular wall repair; Type III tumors completely surround blood vessels, and carotid artery reconstruction is performed simultaneously. Autologous great saphenous vein or artificial blood vessel transplantation is commonly used. Surgery for schwannoma requires protection of nerve bundles, and the use of intracapsular dissection can reduce nerve damage. For malignant tumors such as lymphoma, the resection range needs to be increased, and neck lymph node dissection may be necessary. Surgical risks include intraoperative major bleeding (8%–15%), cerebral ischemia (5%), nerve injury, and vagus nerve injury, with a rate of about 10%. Preoperative evaluation of cerebral collateral circulation is necessary, and temporary carotid artery bypass surgery can be performed to protect cerebral blood flow in patients with an incomplete Willis loop [13].

5.2. Interventional therapy

Interventional embolization is suitable for tumors with abundant blood supply (CBT). Preoperative embolization reduces tumor blood supply and lowers the risk of intraoperative bleeding. Using DSA to insert a microcatheter into the tumor's blood supply artery, injecting gelatin sponge or coil, and performing surgical resection 24–72 hours after embolization can greatly improve the safety of the surgery. For elderly patients or advanced cancer patients who cannot tolerate surgery, stent implantation can be chosen to relieve vascular compression and improve cerebral blood flow, but long-term antiplatelet therapy is needed to prevent stent thrombosis.

5.3. Case study

Case 1: A 45-year-old male patient presented with a right cervical mass for 3 months, accompanied by episodic dizziness. Ultrasound revealed a solid mass at the bifurcation of the carotid artery, encasing the internal carotid artery, with a stenosis rate of 65%. CTA diagnosed it as Shamblin Type II CBT. The patient underwent tumor resection and internal carotid artery repair. During the operation, blood flow was blocked for 15 minutes. Postoperatively, there was no neurological deficit, and no recurrence was observed during a 1-year follow-up.

Case 2: The patient is a 58-year-old female who has had a left cervical mass accompanied by hoarseness for 1 month. MRA revealed a vagal schwannoma compressing the common carotid artery, with a stenosis rate of 40%. An intracapsular tumor resection was performed, with complete preservation of the vagal nerve bundle. Postoperatively, the hoarseness improved, and carotid blood flow was restored [14].

Case 3: The patient is a 72-year-old male with lymphoma-induced carotid artery compression and TIA, who refused surgery due to advanced age. Interventional embolization of the tumor-feeding artery and carotid artery stent implantation were performed. Post-procedure, the symptoms of cerebral ischemia disappeared, and after combined chemotherapy, the tumor volume decreased by 50%.

6. Current research status and future prospects

6.1. Domestic and international research progress

Internationally, scholars in Europe and America primarily focus on molecular genetics research of CBT, discovering that mutations in the SDHD gene are associated with familial CBT, laying the foundation for targeted therapy. Japanese scholars have made breakthroughs in endoscopic minimally invasive surgery for schwannomas, achieving a postoperative nerve function preservation rate of over 90%. Domestic research emphasizes surgical innovation for complex cases, such as hybrid surgery (performing open surgery and interventional therapy simultaneously), which has been applied in type III CBT, reducing the surgical mortality rate to below 2%. Multi-center data show that the 5-year patency rate of carotid artery reconstruction surgery in China is 85%, almost reaching the international advanced level. In terms of diagnosis, ultrasound imaging and elastography enhance the accuracy of qualitative diagnosis of masses; CT perfusion imaging can quantify the degree of cerebral ischemia and guide the timing of treatment.

6.2. Future research directions

Basic research aims to explore the molecular mechanisms of tumor-vessel interactions, such as how mechanical force signaling alters the phenotypic transformation of vascular smooth muscle cells. In terms of diagnostic techniques, multi-modal image fusion technologies, such as real-time fusion of ultrasound and MRA, are being developed to achieve integrated evaluation of anatomical structure and function. In the treatment field, robot-assisted surgical systems may enhance the precision of narrowed vessel reconstruction, and the development of bioresorbable stents may solve the long-term anticoagulation challenges associated with permanent stents. Furthermore, immunotherapy shows potential in the application of malignant tumor-related compression.

7. Conclusion

7.1. Research summary

Exogenous compression caused by a mass at the carotid bifurcation is a complex condition involving multiple disciplines. In diagnosis and treatment, it is necessary to consider both tumor characteristics and vascular protection. This article, after comprehensive analysis, shows that accurate early diagnosis relies on the selection of appropriate imaging techniques. The personalized development of treatment plans (based on the type of mass, degree of vascular stenosis, and the patient's underlying conditions) is key to improving prognosis. Surgical resection combined with vascular reconstruction remains the main curative method, while the application of interventional techniques greatly enhances the safety of treatment [15].

7.2. Clinical practice recommendations

Clinicians should pay attention to vascular evaluation in patients with cervical masses. For asymptomatic individuals, regular ultrasound examinations should be conducted to observe changes in blood flow. For those with symptoms of cerebral ischemia, CTA/MRA should be performed as soon as possible to determine the degree of compression. Treatment decisions should rely on multidisciplinary collaboration, with neurosurgery, vascular surgery, and imaging departments working together to develop a plan. Long-term postoperative follow-up should pay attention to the recurrence of tumors, the patency of blood vessels, and changes in cerebral hemodynamics. Potential ischemic risks should be addressed promptly. With technological advancements, the diagnosis and treatment of this disease will move towards more precise and minimally invasive approaches, thereby improving patients' quality of life.

Disclosure statement

The authors declare no conflict of interest.

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