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Review Article

Carotid body tumors: a review

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ABSTRACT

Carotid body tumor or paraganglioma, is located periadventitially in relation to the carotid bifurcation. Generally, the blood supply of carotid tumors is abundant, it comes mainly from the branches of the external carotid arteries, although cases have been reported where the irrigation comes from the internal carotid artery, vertebral artery, ascending pharyngeal artery and superior thyroid. Although its etiology is unknown, different factors have been associated that contribute to its incidence, such as genetic factors and states of chronic hypoxia. Histologically, they are well vascularized tumors. Between the capillaries there are groups of tumor cells known as Zellballen's pseudoalveolar pattern. These are cells with eosinophilic granular cytoplasm and small round or oval nuclei. The tumor is mobile, but with limitation to mobilization in the cephalocaudal direction. The mass can transmit the carotid pulse due to its proximity to the carotid, and it may also be accompanied by a murmur or thrill. Usually the carotid glomus is identified by clinical examination, so consideration of multiple differential diagnoses. The treatment of choice is surgical excision. The need for preoperative embolization is controversial. Embolization before surgery is recommended to improve surgical success with reduced blood loss as well as reduce the risk of cranial nerve injury. The decision to perform presurgical embolization or not depends on the surgeon.

Keywords: Carotid body tumor, Paraganglioma, Chemodectoma, Zellballen's cell, Carotid glomus

INTRODUCTION

Carotid body tumor or paraganglioma, is a rare type of benign tumor with low malignant potential, it has an embryological origin of paraganglionic tissue derived from the neural crest. It is the only disease that affects the carotid body.[1] It is located periadventitially in relation to the carotid bifurcation. This type of tumor has different names; chemodectomas, paragangliomas, carotid glomus among others and there are different classifications, among them the anatomical classification stands out according to their location.[2] Different variants of the carotid glomus have been described, among which are the sporadic and the familial. Different mutations have been demonstrated, including that of the enzyme succinate dehydrogenase, which will end up causing an accumulation of succinate, which will overactivate angiogenesis through the associated vascular endothelial growth factor pathway.[3] The most frequent alterations are in the subunits of the SDH gene: SDH-B, SDH-C and SDH-D, in addition, other mutations are found in the genes: MAX, SDHA, SDHAF2, TMEM127, all of which represent an autosomal dominant pattern. Each child of an individual with carotid glomus caused by genetic alteration and especially if the glomus comes from the father, has a 50% chance of inheriting it, and is at greater risk if the father is the one who develops the disease. Male patients under 40 years of age and a family history of carotid glomus are at high risk of suffering from carotid glomus due to possible genetic mutation, which is why these patients should be offered genetic analysis.[4] Von Haller is credited as the first person to describe the carotid glomus in 1743. It was his students Taube, in 1743, and Berckelmann, in 1744, who were responsible for making its description in their doctoral theses to finally be taken up by Van Haller in his work. Disputationum Anatomicarum in 1743. Years later, in 1880, Reigner made the first description of a complete resection of a carotid glomus; his patient died. In 1886 Mayld performed the resection successfully and his patient survived, although leaving him with the consequences of hemiplegia and aphasia. In 1971, Shamblin proposed a classification that, after some modifications over time, remains in force today for preoperative classification.[5,6] The 4 main anatomical areas of the head and neck where it is distributed are: carotid bifurcation, inferior region of the ganglion and cervical portion of the vagus nerve, jugular bulb region, and inside the middle auditory canal.[7] The carotid body is an ovoid mass with a variable size of around 3×5 mm and an average weight of 12 mg, located at the carotid bifurcation. Its irrigation comes from the third pharyngeal arch. Under normal conditions, the common carotid artery gives rise to the glomus arteries, and these penetrate the connective tissue of the carotid body. On some occasions, branches of the ascending pharyngeal artery have been found, originating from the posterior surface of the external carotid, providing an accessory supply to the carotid body that must be considered when surgical approach in the resection of the carotid glomus. Generally, the blood supply of carotid tumors is abundant, it comes mainly from the branches of the external carotid arteries, although cases have been reported where the irrigation comes from the internal carotid artery, vertebral artery, ascending pharyngeal artery and superior thyroid.[8]

The carotid body receives afferent innervation from the fibers of the petrosal ganglion, through the carotid sinus nerve. These fibers will later join the glossopharyngeal nerve and the signals will be integrated into the respiratory and vasomotor centers.[9] The carotid space extends from the lower edge of the jugular carotid foramen to the aortic arch and is delimited by three layers of deep cervical fascia. This carotid space includes suprahyoid and infrahyoid regions. Lesions that exist in the carotid body can arise from asymmetry of the normal vasculature, inflammatory or infectious processes, and benign or malignant tumors, including those of metastatic disease.[8] Although the exact etiology of carotid glomus is still unknown, a higher incidence has been found in patients with obstructive pulmonary diseases and patients living in higher altitude regions (1000-2000 meters above sea level) where they are exposed to chronic hypoxia. because the cells in the carotid body are constantly sensing partial pressure changes of oxygen, carbon dioxide and PH.[10,11] The diagnosis of these tumors is often late given that patients are frequently asymptomatic and their growth pattern is slow, so many years may pass before the patient consults.[12] The carotid glomus represents a great surgical challenge due to the difficulties brought about by the great vascularity of the tumor, its proximity and possibility of infiltration of the carotid bifurcation, in addition, compression of cranial nerves at the cervical level and at the base of the skull, which represents a higher risk of complications. Due to the rarity of this type of tumor and the risk that surgical intervention represents, there is no statistical confidence that predicts the benefit of the treatment and its practicality in patients.[13]

EPIDEMIOLOGY

Carotid glomus, like other types of paragangliomas, are rare types of lesions that represent only 0.5% of all head and neck tumors, among which carotid glomus represents 65% of all paragangliomas.[14] The Mayo Clinic has the largest report of series, in which they describe 153 cases in a period of 50 years.[15] Multicenter studies have shown a higher prevalence in women, between 20-60 years old.[16] In the study carried out by Gabiño et al in 2011 in Mexican population, it was found that there is no age predisposition, the average age was 43 years with presentations from 6 to 82 years, with a higher prevalence in women than in men.[17] In the literature it is described that 57% present on the right side, 25% on the left side, 17% bilateral and 10% are malignant.[8] The specific cause why it predominates in women has not been found. It is believed that perhaps it is because the greater lung capacity and affinity for sports in men prevent chronic hypoxia, women also have periods of blood loss during menstruation. Even among populations that live at high altitudes, the female sex predominates, perhaps for the same reasons.

ETIOLOGY

Although its etiology is unknown, different factors have been associated that contribute to its incidence, such as genetic factors and states of chronic hypoxia. Histologically, they are well vascularized tumors. Between the capillaries there are groups of tumor cells known as Zellballen's pseudoalveolar pattern. These are cells with eosinophilic granular cytoplasm and small round or oval nuclei.[10,11] Furthermore, tumor cells are reactive with chromogranin and synaptophysin staining in the immunohistochemistry technique, as well as neuroendocrine markers such as CD56. The cell population can generally be identified at the periphery of the nests and are believed to be modified Schwann cells; They are spindle-shaped and can be highlighted with S-100 protein stain.[15]

The association of paragangliomas and pheochromocytomas with hereditary syndromes such as multiple endrocrine neoplasia type 2 (MEN2), neurofibromatosis type 1 (NF1), Von Hippel Lindau (VHL) has been studied and has been related to the existence of cases of familial paragangliomas.[17] Among the present cases of familial paragangliomas, in 30% mutations can be identified in a subunit of the succinyl dehydrogenase enzyme (SDH, SDHD, SDHA, SDHC, SDHB), of these most of the mutations affect the B and D subunits. Patients who have hereditary paraganglioma syndrome and undergo routine surveillance may have their tumors detected at an earlier stage.

CLINICAL PRESENTATION

The glomus pharyngeus most frequently manifests as a mass in the neck, anterior to the sternocleidomastoid and at the level of the hyoid bone, asymptomatic and slow growing (Figure 1). If symptomatic, it is associated with the presentation of symptoms caused by space-occupying lesions such as headache (72%), sweating (69%) and palpitations (51%), other studies have reported pain, hoarseness, Horner's syndrome, tinnitus, hearing loss, dysphagia, odynophagia, stridor.[8,17] The tumor is mobile, but with limitation to mobilization in the cephalocaudal direction. (Fontaine's sign) The mass can transmit the carotid pulse due to its proximity to the carotid, and it may also be accompanied by a murmur or thrill.[18] These tumors are generally non-functional, although they can present as catecholamine-producing tumors and produce paroxysmal hypertension. Any history of hypertension or uncontrolled tachycardia could suggest a catecholamine-producing tumor. Functional tumors can secrete: Histamine, serotonin, adrenaline, or norepinephrine.[15] In addition, local symptoms such as pulsatile tinnitus or hearing loss and carotid sinus syndrome that manifests as repetitive syncopal episodes have been reported.[14]

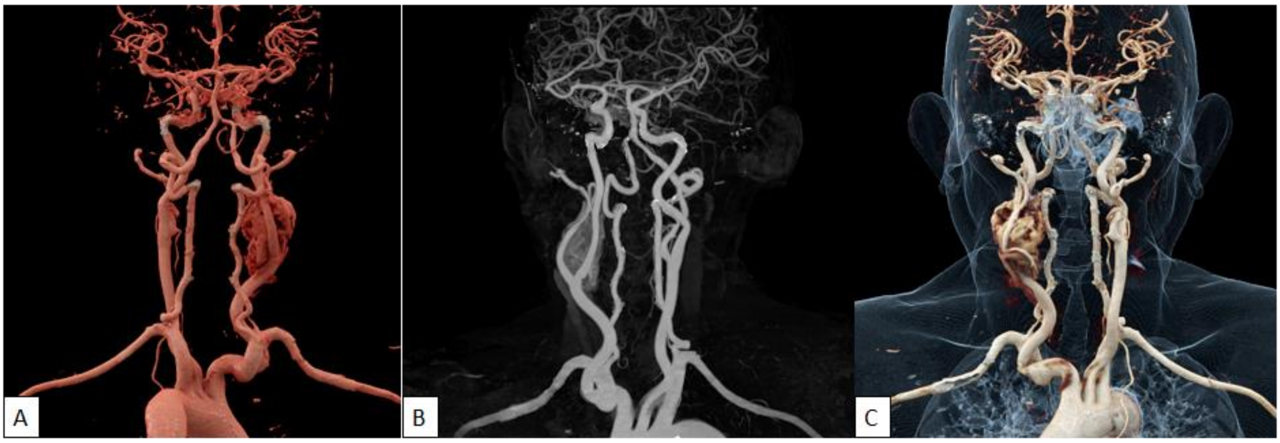

Figure 1 (A-C): Angiotomography with 3D reconstruction showing right carotid glomus.

DIAGNOSIS

Usually the carotid glomus is identified by clinical examination, so consideration of multiple differential diagnoses should always be present in these patients (Table 1).[19] In order to make a timely diagnosis, the finding during routine clinical examination in the office may yield findings that increase the suspicion of some hidden pathology in the case of an asymptomatic carotid glomus or some symptomatic pathology as the main reason for consultation. Inspection and palpation of the neck will show a mass in the neck, it is common to find the Fontaine sign and the Chevassu sign (reexpansion of the tumor followed by pulsations after applying firm pressure) or as an incidental finding in imaging studies. The final diagnosis will depend on imaging, among which, Doppler ultrasound, tomography, resonance, and angiography play a crucial role.[8,14] Doppler ultrasound is considered the ideal initial study, because it is non-invasive, a solid, well-defined, hypoechoic and hypervascularized mass is observed. Although angiotomography is considered the gold standard, it serves to evaluate its relationship with adjacent structures and see cranial extension, which will allow an adequate surgical plan, just like magnetic resonance imaging.[20] CT typically reveals well-circumscribed soft tissue masses with homogeneous enhancement, and angiography is also sensitive for the detection of small lesions, maintaining an arterial anatomical record and the relationship of the tumor with the carotid bifurcation.[14,21] Different classifications have been described to describe and approximate the clinical approach to the carotid glomus. The Shamblin classification defines 3 types of carotid glomus in its relationship with the blood vessels and indirectly a measure to determine its size and the intraoperative risk it represents; type I: localized tumors, type II: tumor adhered and partially surrounded by blood vessels, type III: tumors intimately surrounded by the carotid.[22] Also, the modified Shamblin classification was proposed: localized tumors with widening of the carotid bifurcation, but little attachment to the carotid vessels; resection is usually possible with minimal risk of vascular or cranial nerve complications; tumors partially surround the carotid vessels, complete, resection is more challenging; and tumors intimately surround the carotid. Complete resection is very challenging and often requires temporary interruption of cerebral circulation for vascular reconstruction. The risk of permanent vascular and neuronal defects is significantly higher. than for class I.[19] The classification of Hallet's Zones is also frequently used, this divides the neurovascular surgical field into three zones in order to avoid injuring structures during excision: zone I includes the carotid bifurcation and the adjacent vagus nerve, zone II includes the territory of the external carotid, the overlying hypoglossal nerve and the underlying superior laryngeal nerve, and zone III includes the territory of the internal carotid artery, the mandibular branch of the facial nerve, the proximal portion of the hypoglossal and vagus nerves, the pharyngeal branch of the vagus, the spinal accessory nerve and the glossopharyngeal nerve.[23,24]

Table 1: Differential diagnoses of carotid body tumor.

Variables	Diagnoses
Reactive diseases	Lymphadenitis, Sjogren's syndrome, Kimura's disease, sialadenitis, sialolithiasis, thyroiditis, goiter
Infectious	Tuberculosis, sarcoidosis, cat scratch disease, actinomyces, tularemia, toxoplasmosis, coccidioidomycosis, infectious mononucleosis, parotitis
Cysts	Dermoid, brachial epidermoid, salivary glands, thyroglossal duct
Benign neoplasm	Lipoma, hemangioma, lymphangioma, neurofibroma, Schwannoma, desmoplastic fibroma, paraganglioma, thyroid adenoma, teratoma, hyaline trabecular adenoma
Malignant neoplasm	Thyroid carcinoma (medullary and papillary), neuroendocrine tumor, Hodgkin and non-Hodgkin lymphoma, neck carcinoma
Others	Neck metastasis, extracapsular extension of neoplasms

Table 2: Comparative table classification of: Shamblin, modified Shamblin, Hallet and PUMCH.

S. no.	Shamblin	Modified Shamblin	Hallet	PUMCH
1	Localized tumors	Localized tumors with widening of the carotid bifurcation, but little attachment to the carotid vessels. Resection is usually possible with minimal risk of vascular or cranial nerve complications	Includes the carotid bifurcation and the adjacent vagus nerve	Upper margins of the tumors were below the mandibular angle
2	Tumor adhered and partially surrounded by blood vessels	The tumors partially surround the carotid vessels. Complete, resection is more challenging	Includes the territory of the external carotid, the overlying hypoglossal nerve, and the underlying superior laryngeal nerve	Upper margins of tumors above the mandibular angle, but below the mastoid tip, without enclosure, partial enclosure, and complete coverage of the internal carotid artery (ICA) and external carotid artery (ECA), respectively
3	Tumors intimately surrounded by the carotid artery	The tumors intimately surround the carotid. Complete resection is very challenging and often requires temporary interruption of cerebral circulation for vascular reconstruction	It includes the territory of the internal carotid artery, the mandibular branch of the facial nerve, the proximal portion of the hypoglossal and vagus nerves, the pharyngeal branch of the vagus, the spinal accessory nerve, and the glossopharyngeal nerve	Upper margins of tumors above the mandibular angle, but below the mastoid tip, without enclosure, partial enclosure, and complete coverage of the internal carotid artery (ICA) and external carotid artery (ECA), respectively
4	-	-	-	Upper margins of tumors above the mandibular angle, but below the mastoid tip, without enclosure, partial enclosure, and complete coverage of the internal carotid artery (ICA) and external carotid artery (ECA), respectively
5	-	-	-	Upper margins of tumors above the mastoid tip

Finally, the classification was proposed according to the anatomical zone proposed by the Peking Union Medical University Hospital (PUMCH), which classifies the carotid glomus from I-V and was defined based on the envelope of the carotid artery and vertical extension of tumors revealed by preoperative imaging - type I: upper margins of tumors were below the mandibular angle; type II, III and type IV: the upper margins of the tumors are above the mandibular angle, but below the mastoid tip, without enclosure, partial enclosure and complete coverage of the internal carotid artery (ICA) and the external carotid artery (ECA) respectively; and type V, the upper margins of the tumors were above the mastoid tip (Table 2).[24]

TREATMENT

The treatment of choice is surgical excision. The need for preoperative embolization is controversial among different authors (Figure 2). Some support its use because it could reduce blood loss and the sacrifice of vascular structures. Others argue that it is not recommended based on the principle that the vessels feeding the tumor arise primarily from the adventitia surrounding the main vessels and these cannot be adequately embolized. Some literature supports the idea of recommending it mainly for large carotid glomus or those with Shamblin II/III classification.[25] Guichard et al reported that the occasion in which preoperative embolectomy is performed in the carotid glomus is rare since it is reserved more specifically for specific cases without reporting a specific prevalence between embolized versus non-embolized cases.[4] If embolization has occurred, once performed, excision is suggested within the next 48 hours to avoid revascularization (Figure 3).[11] In a retrospective, observational, longitudinal and comparative study with a sample of 29 patients with a radiological diagnosis of carotid paraganglioma, a comparative analysis was carried out in 2 groups in which the surgical time and the average bleeding were taken into account: group A, a single session of selective embolization of the carotid glomus was performed, the average bleeding and surgical time was 60 ml and 1.4 hours, respectively. In group B, the average bleeding and surgical time was 318 mL and 3 hours, respectively. The Mann-Whitney U test was performed to compare the differences, with a result of a statistically significant value of $p<0.001$. Concluding that transarterial embolization of hypervascular tumors represents an important advantage, reducing the average surgical time and intraoperative bleeding significantly.[26] However, Gözen et al demonstrated in a retrospective study the need or not for preoperative embolization for the resection of carotid paraganglioma. Concluding that it does not significantly reduce neurovascular injury in the group with preoperative embolization, they indicated that preoperative embolectomy should not be considered as a routine measure to reduce intraoperative blood loss, as it does not influence the operating time due to blood loss, they concluded by talking about the need to conduct a randomized controlled study with a larger sample size to determine the effectiveness of preoperative embolectomy.[25] Wernick et al demonstrated in their study of contemporary management of carotid body tumors in an academic center that preoperative embolization does not reduce blood loss or the incidence of death, stroke, or nerve damage.[27] In a study carried out in a tertiary center with retrospective data collection between 2012-2019, the usefulness or not of previous embolization to reduce bleeding and associated complications such as damage to nervous structures in carotid glomus surgery, 14% were classified with a Shamblin I, 53.8% with a Shamblin II and 30% with a Shamblin III, without finding statistically significant differences between the non-embolized group and the embolized group.[25] As part of the therapeutic spectrum when making the diagnosis, the surgical approach is the management option and must be carried out in a considerable amount of time to avoid tumor progression that generates new surgical challenges. The reasons for performing surgical management are: probability of being a malignant tumor, although the percentage of malignancy is low, puncture biopsy is not acceptable due to the high risk of hemorrhage in an intensely vascularized tumor; there is no reliable screening mechanism for cytological monitoring of tumor progression; there is no evidence that correction of hypoxemia, if possible, will result in tumor regression; the risk of vascular injury is acceptable in expert medical centers; and all tumors can eventually become symptomatic.[1] Therefore, the optimal surgical approach is recommended for patients with secretory tumors, malignant tumors, rapidly growing tumors or increased symptoms and when radiotherapy is not possible. If the above criteria are not met, conservative management could be established with continuous surveillance or radiotherapy since good local control at 5, 10 and 15 years of 99%, 95% and 86% has been demonstrated in tumors larger than 5cm, multiple tumors and patients with high surgical risk.[16] Carotid body tumor surgery requires meticulous dissection, careful planning, and patience during surgery.[4] In the development of surgical excision, the most important step is the control of the blood vessels at the upper and lower level, for this reason it must be clear in the identification of the internal jugular vein and common and internal carotid artery and place vascular loops in each a. Until intraoperative tumor dissection is performed, it will be difficult to determine the need for the use of a carotid reverse saphenous vein graft. If an adequate separation between the tumor and the artery is not found, both can be completely excised, and a graft will subsequently be performed. Likewise, carotid injuries will require clamping of the common or internal carotid with subsequent heparinization and vascular reconstruction. Postoperative complications play a fundamental role that must be considered, due to the intimate relationship that the carotid maintains with other vascular and nervous structures. Complications have been observed more frequently in tumors larger than 5 cm and/or a classification of Shamblin III.[28] The risk of stroke as an intraoperative consequence has been shown to be lower in Shamblin I tumors, and more frequently in Shamblin II and

III tumors.[29] Vagus and hypoglossal nerve injuries have been reported as the most frequent, other cranial nerves also affected are: facial (VII), glossopharyngeal (IV), spinal (XI), cervical sympathetic nerves (Horner's syndrome), recurrent laryngeal and pharyngeal superior.[30] A complete neurological evaluation should be performed to evaluate the integrity of the nerve. In patients with injury to the hypoglossal nerve, dysarthria and swallowing problems will be found; in injuries to the vagus nerve, dysphonia is common when its portion of the branch of the ascending laryngeal nerve is affected; in addition, disorders in regulation may be found of heart rate and gastric acid secretion. The lesion of the facial nerve will result in facial paralysis, asking the patient to change facial gestures and evaluate their sensitivity in a comparative way will be enough to reach the diagnosis, the lesion of the glossopharyngeal nerve is found in a patient with deviation of the uvula when asking them to generate some sound, this maneuver will also help in the evaluation of the vagus nerve. For the evaluation of the spinal nerve, the patient is asked to perform flexion, rotation and abduction of the shoulder, which will be impossible due to paralysis. Also, injuries to the cervical sympathetic nerves have been reported, leading to the appearance of Horner's syndrome, which is characterized by partial eyelid ptosis, miosis and facial anhidrosis. As a postoperative complication, it was recorded that the risk of suffering a cerebral infarction increased in cases where there was intraoperative vascular injury, a personal history of type 2 diabetes mellitus and carotid artery disease.[31]

Figure 2: (A) Initial image in carotid glomus, and (B) embolization of the occipital artery with 2 interlook 35 coils measuring 40 mm by 20 mm, in the ascending pharyngeal artery and the nutrient branches of the tumor, 15 ml of bellblock microparticles of 300 to 500 microns are injected.

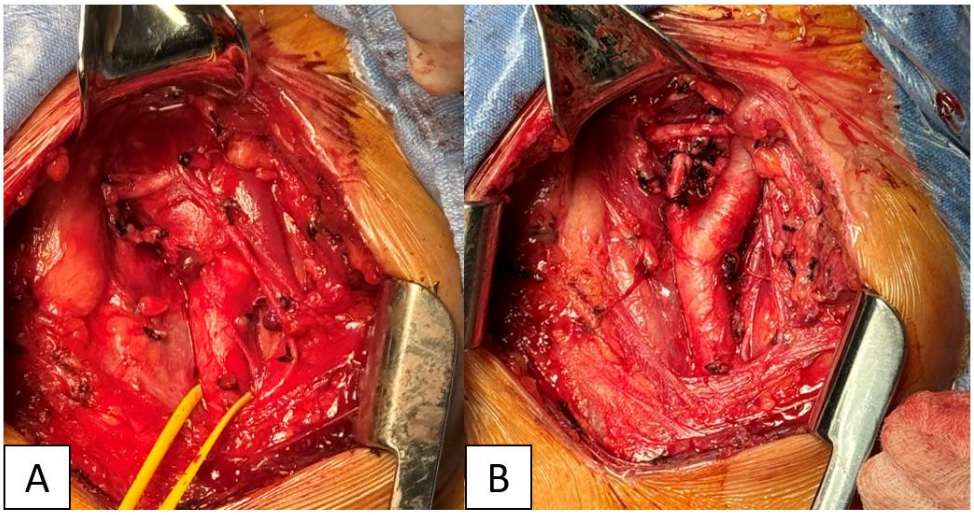

Figure 3: (A) Dissection and ligation of nutrient vessels of the carotid body tumor, and (B) the carotid bifurcation can be seen after resection.

DISCUSSION

Carotid body tumors are considered rare neuroendocrine neoplasms that arise near the carotid bifurcation within glomus cells derived from the embryonic neural crest.[23] The carotid body is a small, round, reddish-brown, well-ciscunscribed, highly specialized chemoreceptor organ located in the adventitia, posteromedial aspect of the carotid bifurcation. It is very well vascularized, supplied mainly by the vessels derived from the external carotid artery, most commonly the ascending pharyngeal branch, and it is innervated through the glossopharyngeal and vagus nerves.[8] Physiologically it is related to the homeostasis of pH, pO_2 and pCO_2, which they control by modulating cardiovascular and respiratory function with the release of neurotransmitters.[32] Normally the carotid body measures 2 to 6 mm in diameter, in some people who live in regions with higher altitudes they can reach larger dimensions, it functions as a chemoreceptor organ that is stimulated by acidosis, hypoxia and hypercapnia and manages to play a role in the autonomic control of blood pressure, heart rate, respiration, and blood temperature in response to changes in these parameters by increasing sympathetic outflow.[8]

Hyperplasia of the carotid body has been associated with states of chronic hypoxemia, chronic obstructive pulmonary disease and cyanotic heart diseases, this may explain why in geographical areas located above 2000 meters above sea level.[17] Usually the carotid glomus are asymptomatic, the clinical presentation is mostly volume dependent, commonly as incidental findings in radiological imaging studies.[33] Functional carotid tumors can rarely produce neuroendocrine secretions that cause catecholamine-related symptoms, such as palpitations, headaches, hypertension, tachycardia, flushing.[8] Although the classic clinical presentation of paragangliomas is the presence of a pulsating tumor that can be painful and is frequently associated with headache. In some cases, the first form of presentation is an increase in volume, so making an adequate diagnosis is based on suspicion and clinical examination.[34] The clinical history can guide a timely diagnosis, especially in patients who present hereditary paraganglioma syndrome in whom a guided interview and physical examination can be performed for early detection. Radiological study using Doppler ultrasound, computed tomography and magnetic resonance imaging are useful radiological tools to establish the diagnosis. Color Doppler ultrasound is widely used for the detection of carotid tumors and follow-up, due to its availability and being a non-invasive study. They have a relatively high diagnostic specificity and sensitivity for carotid tumor. The use of a linear transducer with high frequency and resolution (7–10 MHz) allows an adequate morphological assessment where a rounded or ovoid, hypoechoic-solid mass can be evident, whose limits are correctly appreciated, located at the level of the bifurcation of the carotid artery. Using color Doppler ultrasound, it is possible to identify a hypervascularized mass that occupies the carotid body and displaces the vessels, which allows it to be differentiated from other regional masses such as salivary gland tumors. In addition to the Doppler study, a low resistance flow pattern can be detected.[35] Angiotomography is capable of defining the size of the tumor, its relationship with bone landmarks; which allows tumors to be classified as well as to adapt the surgical approach. This tumor causes the carotid bifurcation to widen, which is called "Lyra sign". On non-contrast tomography, it has soft tissue density and with contrast, the highly vascularized nature provides severe enhancement.[36] Magnetic resonance imaging allows for greater anatomical assessment of the involved structures, as well as more detailed tumor characterization in relation to findings. It does not use ionizing radiation, and the precision is greater than tomography. In this study, the carotid tumor can be visualized as a focal, elliptical image, with precise limits, located in the carotid bifurcation, which it displaces, separating the external and internal carotid in an anteroposterior direction; It presents intermediate-low signal in T1 and DP sequences, as well as high signal in T2 and T2 fat-sat sequences.[35]

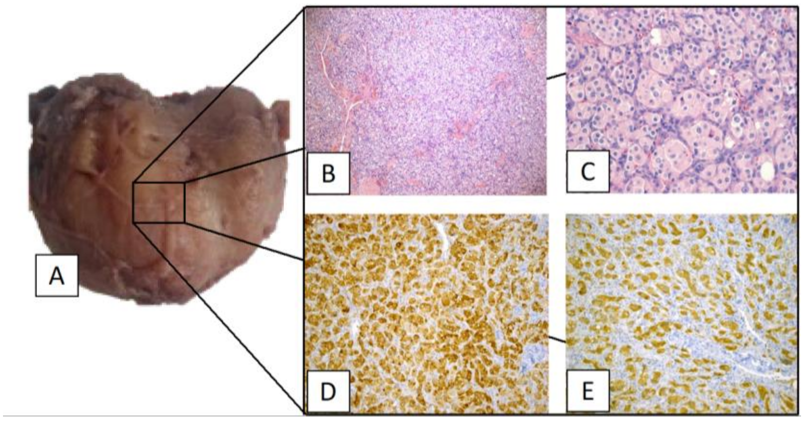

Figure 4: (A) A macroscopic image of encapsulated carotid body tumorectomy; (B) neoplasm composed of epithelioid cells, separated by fibrovascular septa; (C) cells arranged in a nested pattern ("Zellballen"), with abundant eosinophilic cytoplasm and round nuclei, vesicular or "salt and pepper" chromatin; (D) immunohistochemistry reaction, chromogranin, with diffuse expression and granular cytoplasmic pattern; and (E) synaptophysin immunohistochemistry reaction, with diffuse expression and cytoplasmic pattern.

Carotid angiography is considered the gold standard for diagnosis as well as a therapeutic study that allows visualization of the vessels that supply irrigation to the tumor. It is considered a confirmatory study, with a specificity of 97.1%. Additionally, preoperative embolization can be planned. Biopsy in these tumors is contraindicated due to hypervascularization and proximity to various vascular and nervous structures. Conditioning a risk of massive hemorrhage, dissemination as well as pseudoaneurysm formation and carotid thrombosis. Surgical resection is considered the main treatment with or without presurgical embolization, which currently has a greater tendency in its use with better results and fewer complications due to advances in the diagnostic approach, surgical technique, anesthetics as well as adequate postoperative care (Figure 4).[12] Tumor embolization prior to surgical resection is preferred especially in cases when bilateral tumors occur or when invasion is excessive. That is, in large tumors, with the purpose of occluding the nutrient vessels of the tumor, reducing intraoperative bleeding as well as morbidity, since it is considered to facilitate the resection technique, achieving a shorter surgical time, in addition to the closure of arterial supplies not accessible to the surgeon.[33,37]

CONCLUSION

Surgical resection of the carotid glomus can be challenging, especially in cases where it is large. Embolization before surgery is recommended to improve surgical success with reduced blood loss as well as reduce the risk of cranial nerve injury. The decision to perform presurgical embolization or not depends on the surgeon.

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