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Microsurgical resection of a giant exclusively dopaminesecreting jugular foramen paraganglioma: A case report

Guilherme H. W. Ceccato¹, Júlia S. de Oliveira¹, Pedro H. dos Santos Neto¹, Nick. D. Carvalho¹, Hugo A. Hasegawa¹, Vinícius N. Coelho¹, Vitor P. Barreto¹, Gustavo A. R. Passos^{1,2}, Paulo A. S. Kadri¹, Jean G de Oliveira³, Luis A. B. Borba^{1,4}

¹Department of Neurosurgery, Mackenzie Evangelical University Hospital, Curitiba, Paraná, Brazil, ²CANES Laboratory, University of Miami, Miami, Florida, USA, ³Department of Neurosurgery, Santa Casa de Misericórdia de São Paulo, São Paulo, Brazil, ⁴Department of Neurosurgery, Federal University of Paraná, Curitiba, Paraná, Brazil.

E-mail: Guilherme H. W. Ceccato - guilhermececcatoc4@gmail.com; Júlia S. de Oliveira - jujusantanadeoliveira@gmail.com; Pedro H. dos Santos Neto - pedroh.santosneto@gmail.com; Nick. D. Carvalho - n.dorneli.med@gmail.com; Hugo A. Hasegawa - hugo.akio@hotmail.com; Vinícius N. Coelho - vini_ nicolelli@hotmail.com; Vitor P. Barreto - vitorpbarreto@hotmail.com; Gustavo A. R. Passos - dr.gustavopassos@gmail.com; Paulo A. S. Kadri - kadripas@ hotmail.com; Jean G de Oliveira - jeangol@uol.com.br; *Luis A. B. Borba - luisalencarborba@gmail.com



Case Report

***Corresponding author:** Luis A. B. Borba, M.D., PhD. Rua Paulo Gorski 1240 #7,CEP

Rua Paulo Gorski 1240 #7,CEP 81210220, Department of Neurosurgery, Mackenzie Evangelical University Hospital, Curitiba, Parana, Brazil.

luisalencarborba@gmail.com

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ABSTRACT

Background: Temporal bone paragangliomas are complex pathologies presenting close relationships with many critical neurovascular structures. Exclusively, dopamine-secreting paragangliomas are rare and may present a major hemodynamic challenge during intraoperative and postoperative periods, with great blood pressure lability due to the dose-dependent properties of dopamine. However, preoperative α or β blockage is usually not advised. Microsurgical resection is the treatment of choice; nevertheless, these tumors commonly present with greater size at diagnosis due to their non-specific clinical manifestations.

Case Description: A 43-year-old male patient presented with headache, tinnitus, hearing loss, hypoglossal and facial nerve compromise, as well as vocal cord palsy. Magnetic resonance imaging depicted a giant posterior fossa mass centered in the left jugular foramen extending to the cervical space, associated with important bone erosion. Laboratory investigation depicted elevated serum dopamine concentration of >2.500 pg/mL (reference <30 pg/mL) and increased 24-h urine dosage of 3306 µg (reference <540 µg). Values of epinephrine, norepinephrine, and metanephrines were within normal range. The patient underwent preoperative embolization, and microsurgical resection was performed the next day. Serum and urinary dopamine concentrations were normalized following the procedure. The patient presented a favorable outcome, with no new neurological deficits.

Conclusion: Exclusively dopamine-secreting temporal bone paragangliomas may be successfully resected with a favorable outcome. A multidisciplinary, well-trained team is essential to manage intraoperative challenges up to postoperative rehabilitation adequately. Extensive laboratory training is essential to develop the surgical skills to master this skull-based approach.

Keywords: Case report, Dopamine, Jugular foramen, Paraganglioma, Temporal bone

INTRODUCTION

Head-and-neck paragangliomas (HNPGLs) are neuroendocrine tumors arising from parasympathetic paraganglia, comprising around 0.6% of head and neck neoplasms.^[15] Paraganglia are found close to structures related to the autonomic nervous system and can be well-defined anatomically as carotid bodies or microscopically dispersed.^[7,10] Carotid body paragangliomas

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comprise about 60% of HNPGLs, followed by the temporal bone and vagal tumors; however, up to 20 origin sites have been reported.^[1,14] Tympanomastoid paragangliomas arise from the inferior tympanic or mastoid canaliculi, following the trajectory of the Jacobson or Arnold nerves, respectively, while tympanojugular tumors tend to originate from the adventitia at the jugular bulb.^[14] Histology of these lesions presents chief (usually nonchromaffin ones) and sustentacular cells organized in a characteristic alveolar pattern called *zellballen*.^[17] Chief cells usually contain secretory granules; however, sympathetic HNPGLs comprise <4% of these tumors, while functional tumors secreting catecholamines are more frequently found in the adrenal gland and along the sympathetic chain in the abdomen/pelvis and less frequently thorax.^[5,9]

Exclusively, dopamine-secreting paragangliomas are rare, with <40 cases reported in the literature.^[12] However, up to 30% of HNPGLs may also secrete dopamine.^[16] Symptoms usually are nonspecific and due to tumor compressive effects, often without the classic catecholamine excess presentation of diaphoresis, headache, palpitation, and hypertension.^[9,19]

We present the case of a young male patient with progressive symptoms harboring a giant exclusively dopamine-secreting jugular foramen paraganglioma managed with preoperative embolization followed by microsurgical resection, who presented a favorable outcome.

CASE PRESENTATION

A 43-year-old male presenting progressive headache, hearing loss, and left-sided tinnitus over the past 3 years, with sporadic episodes of left-sided otorrhagia in the last weeks. It was observed on the left side facial nerve palsy House-Brackmann IV, hypoglossal nerve dysfunction, and facial hypoesthesia. Video laryngoscopy depicted left vocal cord palsy [Figure 1]. Examination of the left external auditory canal demonstrated a tumor inside it, while no abnormality on the right side [Figure 1]. There was no history of hypertension or paroxysmal symptoms.

Laboratory investigation demonstrated a high serum concentration of dopamine >2.500 pg/mL (reference <30 pg/mL). Serum values of norepinephrine and epinephrine were in the normal range, 262.8 pg/mL (reference <460 pg/mL) and 21.2 pg/mL (reference <90 pg/mL), respectively [Table 1]. 24-hour urine dosage of dopamine was 3306 μ g (Reference <540 μ g), and other catecholamines and metanephrines were within normality [Table 2].

A computed tomography (CT) scan demonstrated a mass centered in the left jugular foramen with extensions toward intra- and extracranial spaces associated with massive bone erosion, including the destruction of middle ear structures. A comparison between CT scans with 3-year intervals; it is

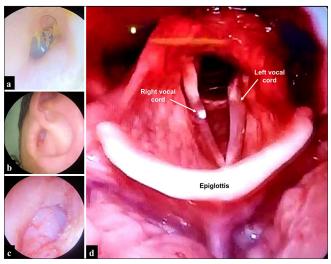


Figure 1: Physical examination. (a) External auditory canal and tympanic membrane with no abnormalities on the right side. (b and c) Ipsilateral external auditory canal filled with tumor. (d) Left vocal cord paralysis.

demonstrated the growth of the lesion and progression of bone destruction [Figure 2].

Magnetic resonance imaging (MRI) demonstrated a giant lesion measuring $13 \times 4.6 \times 8$ cm [craniocaudal (CC) \times anteroposterior (AP)× latero-lateral (LL)] centered in the left jugular foramen, presenting an important mass effect in the posterior fossa and extracranial extension up to the level of C5–C6 [Figure 3]. Comparing 1.5-year interval MRIs, it is also evident growth of the mass [Figure 4].

The patient underwent angiography and embolization of the mass the day before surgical resection [Figure 5]. The tumor was highly vascularized with blood supply from the left external carotid artery, mainly by the occipital, posterior auricular, and ascending pharyngeal arteries, and some nourishment by the posterior meningeal artery. Furthermore, branches from the vertebral artery contributed. It was demonstrated occlusion of the left sigmoid sinus, as well as of the internal jugular vein. Angio-CT scan following embolization depicted degenerative features inside the mass [Figure 5]. As well, it is better to depict the dimensions and relationships of the lesion by the 3D model [Figure 5].

Microsurgical resection was performed [Video 1] employing an infratemporal fossa approach [Figure 6].^[2,3,16,19]

The patient is positioned supine with the head rotated to the contralateral side and extended to increase the sub/ retromandibular space. An arciform incision is performed starting from the level of the pinna to the medial border of the sternocleidomastoid muscle. Following the reflection of the skin flap, the great auricular nerve is identified and dissected along its trajectory over the sternocleidomastoid

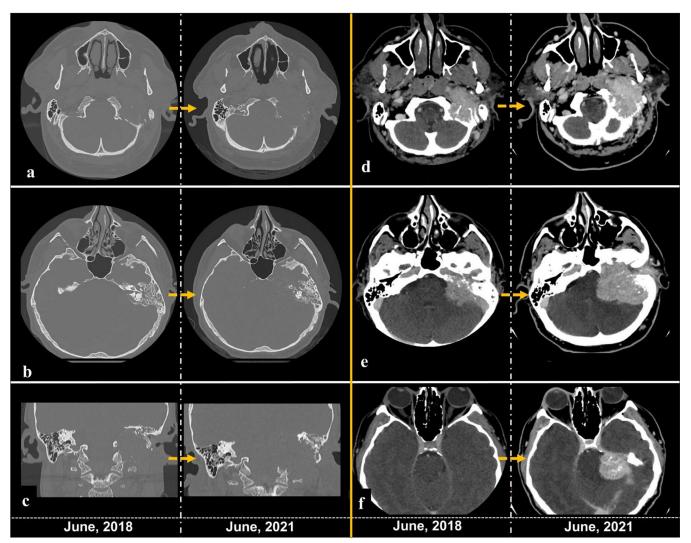


Figure 2: Computed tomography scans comparison between 2018 and 2021. (a-c) Bone window demonstrating the progression of bone erosion over the years, with the greater compromise of the occipital condyle, mastoid, and middle ear structures. (d-f) Postcontrast images depicting the increasing in size of the mass. The yellow arrow means the passage of time between the left and right image.

Table 1: Plama concentrations of catecholamines and metabolites							
Plasma	Preoperative	Reference Values	1 st week Postoperative (under use of vasoactive drugs)	Reference Values	8 Months Post operative	Reference Values	
Dopamine	> 2500 pg/mL	< 30 pg/mL	< 15 pg/mL	< 30 pg/mL	< 30 pg/ml	< 85 pg/ml	
Norepinephrine	262.8 pg/mL	< 460 pg/mL	> 2500 pg/mL	< 460 pg/mL	184 pg/ml	< 420 pg/ml	
Epinephrine	21.2 pg/mL	< 90 pg/mL	696.3 pg/mL	< 90 pg/mL	<15 pg/ml	< 84pg/ml)	
Metanephrine	78.7 µg/ml	< 65 pg/mL	-	-	-	-	
Normetanephrine	396.5 pg/mL	< 196 pg/mL	-	-	-	-	

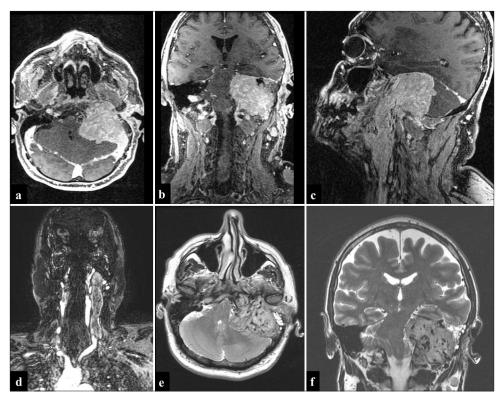


Figure 3: Preoperative magnetic resonance (MR) imaging. (a-c) Post-contrast images expose a large mass with compression of posterior fossa structures and cervical extension through the jugular foramen. (d) MR venography depicts the mass filling the internal jugular vein up to the lower cervical region. (e and f) T2-weighted imaging images depicting many flow voids inside the giant mass, suggestive of a highly vascularized tumor.

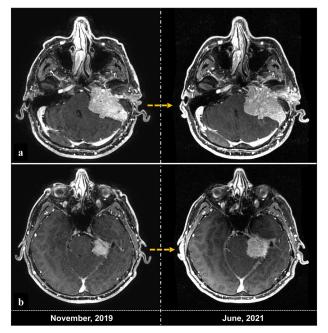


Figure 4: Comparison between MRI scans from November 2019 (left images) and June 2021 (right images). (a) Axial cut at level of middle cerebellar peduncle. (b) Axial cut at level of transition of pons and midbrain. The yellow arrow means the passage of time between the left and right image.

muscle to be used if necessary for facial nerve reconstruction. Temporalis fascia is dissected from the muscle and reflected, kept attached to the sternocleidomastoid muscle, which is mobilized posteriorly. The next step is to obtain neurovascular control in the neck, and the common, internal and external carotid arteries are identified, as well as the internal jugular vein. The external carotid artery branches involved with the tumor blood supply can be ligated. In the cervical area, it is possible to identify the hypoglossal nerve crossing over carotid bifurcation and giving its branch to the ansa cervicalis; accessory nerve runs posteriorly over the internal jugular vein; vagus nerve travels within the carotid sheath between internal carotid artery and internal jugular vein; and glossopharyngeal nerve runs anteriorly crossing over the lateral aspect of distal cervical segment of internal carotid artery.

After cervical dissection, surgery moves to the mastoidectomy. Initially, the antrum is opened for helping to identify the lateral semicircular canal, and then the other semicircular canals; the sigmoid sinus is also skeletonized. Incus is identified in front of semicircular canals and points to the facial canal. In this case, as the patient presented preoperative facial nerve palsy, the canal will be opened to expose and decompress the facial nerve, which will be later grafted with the great auricular nerve if necessary. Dissection

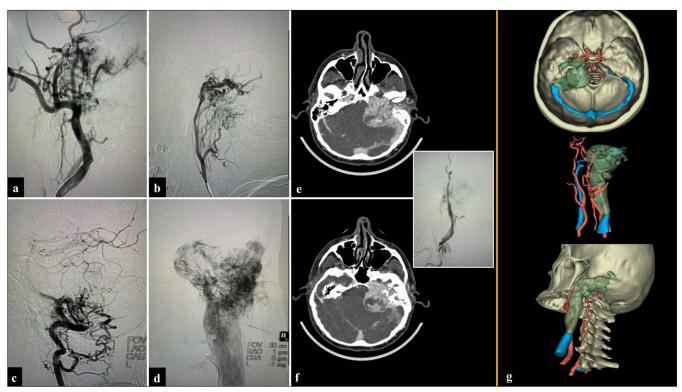


Figure 5: Preoperative embolization. (a-d) Diagnostic angiography demonstrates a highly vascularized mass, with major blood supply from the external carotid (a), ascending pharyngeal (b), and vertebral arteries (c). (d) The angiographic venous phase depicts tumor blush and drainage along the internal jugular vein dilated by the tumor inside it. (e and f) Angio-computed tomography following embolization demonstrates degenerative features inside the mass. The inset image exposes the final embolization view, preserving mainly the superficial temporal artery. (g) 3D model of the tumor, exposing better its vascular relationships and its extensions are also clearly exposed.



Video 1: Intraoperative nuances of microsurgical resection.

includes the removal of the mastoid tip to allow exposure of the whole trajectory of the facial nerve from the mastoid to the extracranial segment. Furthermore, the sigmoid sinus is all the way skeletonized up to the internal jugular vein, including the jugular bulb. All venous outflow is exposed, and the rectus capitis lateralis muscle is mobilized as it relates to the posterior border of the jugular foramen, and if necessary, anterolateral dissection reaches the styloid process. An extradural tumor is first resected, including tumors involving the facial nerve. Extension to the middle ear can be reached through the facial recess, between the facial nerve and chorda tympani. The tumor infiltrates and projects into the lumen of the sigmoid sinus up to the internal jugular vein, and first sigmoid sinus is incised to remove the proximal intraluminal tumor, and then, it is packed and ligated. As the patient already presented several lower cranial nerve deficits, the internal jugular vein is ligated distal to tumor projection, and the whole vein is gradually resected. However, if the patient had preserved lower cranial nerve function, the medial wall of the vein would be preserved. An intrabulbar resection may be performed in the jugular bulb, and bleeding from some emissary veins, especially from the inferior petrosal sinus, may be present and it is controlled with fibrin glue and hemostatic material. The intradural tumor is finally removed, with care to preserve surrounding arachnoid planes.

The closure should be meticulous, employing vascularized flaps as the previous pericranium and temporalis fascia harvested, and the cavity is filled with fat, and fibrin glue. Mastoid air cells should be packed with muscle pieces. Layer by layer must be approximated to avoid most

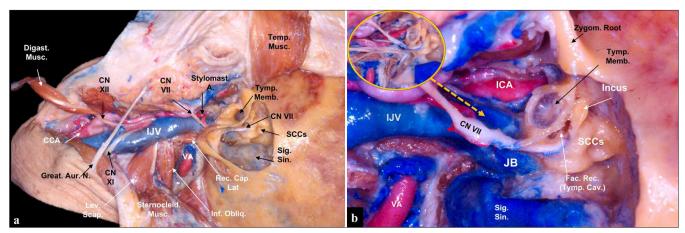


Figure 6: (a) Anatomical view of the surgical approach. It is important to perform a neck dissection to obtain vascular control of major cervical structures. Next, a mastoidectomy is performed, and the extradural tumor may be removed to expose the proximal sigmoid sinus, and the jugular foramen is opened to expose since sigmoid sinus up to the jugular vein. (b) Middle ear structures are accessed through the mastoidectomy, and the incus is an anatomical landmark to point facial canal. A pathway to the middle ear is through the facial recess, anterior to the facial canal, and attention is important as the petrous carotid artery is located deep in this corridor. The great auricular nerve should be harvested from the beginning of dissection to be used to graft the infiltrated facial nerve if necessary. In the inset image (yellow circle)., it is highlighted the lower cranial nerves running underneath the medial wall of the internal jugular vein. CCA: Common carotid artery, CN: Cranial nerve, Digast. Musc.: Digastric muscle, ICA: Internal carotid artery, IJV: Internal jugular vein, Fac. Rec.: Facial recess, Great Aur. N.: Great auricular nerve, Inf. Obliq.: Inferior oblique muscle, JB: Jugular bulb, Lev. Scap.: Levator scapulae muscle, Rec. Cap. Lat.: Rectus capitis lateralis muscle, SCCs: Semicircular canals, Sig. Sin.: Sigmoid sinus, Sternocleid. Musc.: Sternocleidomastoid muscle, Stylomast. A.: Stylomastoid artery, Tymp. Cav.: Tympanic cavity, Tymp. Memb.: Tympanic membrane, VA: Vertebral artery, Zygom. Root: Zygomatic root.

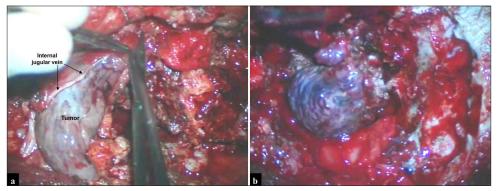


Figure 7: Tumor projections. (a) It is demonstrated tumor fills the lumen of the internal jugular vein as it is incised. (b) The posterior fossa mass is removed.

cerebrospinal fluid leaks. A preemptive lumbar drain may be considered, if necessary, in the immediate postoperative period.

From the middle to the end of the procedure, the patient presented severe blood pressure lability, varying from deep to high values without apparent cause, and demanding usage of an infusion pump of vasoactive drugs alternating norepinephrine, nitroprusside, esmolol, and dopamine recurrently. We removed the intradural and extradural tumor projections, including extension inside the lumen of internal jugular vein and sigmoid sinus, after ligation of these structures [Figure 7]. As the patient already presented preoperative lower cranial nerve dysfunction, the medial wall of the internal jugular vein was also removed, which covers these nerves from a lateral perspective [Figure 6]. As the patient also presented facial nerve palsy, it was opened the facial canal and decompressed the nerve. Pathology confirmed the lesion to be a paraganglioma [Figure 8].

Postoperative imaging demonstrated complete tumor resection [Figure 9]. The patient presented a favorable outcome, with no new neurological deficits on a follow-up of 2 years, presenting with no dysphagia and developing an independent daily life. Just 1 week following the procedure, it was measured serum dopamine concentration of <15 pg/mL (Reference <30 pg/mL). Eight months postoperative, the serum value of dopamine dropped significantly, from

>2500 pg/mL preoperative to <30 pg/mL postoperatively (Reference <85 pg/mL) [Table 1]. Furthermore, the

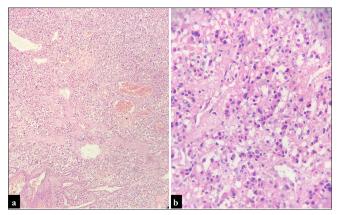


Figure 8: Pathology. (a and b) Hematoxylin and eosin staining is demonstrated, showing a zellballen pattern. Immunohistochemistry was positive for chromogranin and synaptophysin, which are neuroendocrine differentiation tissue markers, with a Ki-67 of 5%. (a) $\times 100$ (b) $\times 400$.

postoperative dosage of homovanillic acid in 24-h urine was 3.7 mg (Reference <6.9 mg/24-h urine) [Table 2]. Serum concentration of norepinephrine was 184 pg/mL (Reference <420 pg/mL), and of epinephrine was <15 pg/mL (Reference <84 pg/mL).

DISCUSSION

Jugular foramen paragangliomas are complex pathologies that can involve both intra- and extracranial spaces, as well as intra- and extradural compartments, and can infiltrate inside of the internal jugular vein and sigmoid sinus, beyond surrounding critical neurovascular structures. Around 40% of HNPGLs are associated with familial disease and the most common mutation is related to a subunit (A-D) of the enzyme succinate dehydrogenase involved in the Krebs cycle, and a germline mutation is commonly identified.^[11,19]

Especially in patients with apparent adrenergic symptoms is important to screen for other possible synchronic tumors, as HNPGLs are mostly associated with the parasympathetic

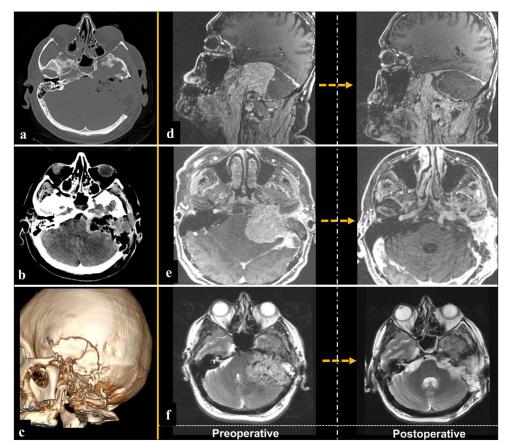


Figure 9: Postoperative imaging. (a-c) Computed tomography scans demonstrating the surgical corridor. (d-f) Comparison between pre- and postoperative magnetic resonance imaging, highlighting complete tumor. The yellow arrow means the passage of time between the left and right image.

Table 2: 24-Hour urine concentration of catecholamines and metabolites						
24-Hour Urine	Preoperative	Reference values	8 Months postoperative	Reference values		
Dopamine	3306 µg	< 540 µg	-	-		
Homovanillic acid	-	-	3.7 mg	< 6.9 mg		
Norepinephrine	74 µg	< 97 µg	-	-		
Epinephrine	9 µg	< 90 µg	-	-		
Metanephrine	119 µg	< 124 µg	-	-		
Normetanephrine	284 µg	< 538 µg	-	-		

system, while thoracic, abdominal, and pelvic tumors are more likely linked with the sympathetic system.

Exclusively, dopamine-secreting tumors are rare, and these tumors have a median age of 48 years at diagnosis and a male-to-female ratio of 1:1.5.^[13] Diagnostic tests include plasmatic and 24-h urine dopamine dosage, as well as its metabolites 3-methoxytyramine and homovanillic acid, preferably in serum or 24-h urine samples, respectively.^[15] Symptomatology is nonspecific, and usually, diagnosis is delayed until the tumor is large enough to cause a mass effect.^[6,19]

Dopamine has a dose-dependent hemodynamic effect, varying from a vasodilatory effect at low concentrations up to inotropic effects at moderate dosage through β -receptor stimulation and, at higher concentrations, a vasopressor effect due to α receptor affinity.^[12] Usually, dopamine-secreting paragangliomas present their plasmatic concentrations in a low range, in this way manifesting mainly vasodilatory effects due to stimulation of dopamine receptors.^[12] Preoperative α or β receptor blockage is controversial in dopamine-secreting paragangliomas; however, it seems that these tumors are more linked to hypotension scenarios, and preoperative α or β receptor blockage should be avoided.^[18]

Management of HNPGLs includes wait-and-scan, radiosurgery, and microsurgical resection; however, the therapeutic decision should be individualized in each case.^[4] Usually, more aggressive treatment is indicated for symptomatic and secretory cases, as well as for young patients under 50 years, as there is a higher incidence of growth in this age group.^[8] However, it is important to mention that patients can get worse clinically without a radiological increase in about 64% of cases, despite the fact that in the present case, radiological growth was documented.^[8]

CONCLUSION

Exclusively dopamine-secreting paragangliomas may be successfully resected; however, it is essential to be wellprepared to deal with possible major hemodynamic instability. It can occur since preoperative embolization, especially during the intraoperative period, and be apparent up to the 1st days following microsurgical resection. Laboratory training is essential to the surgeon's better understanding of surgical approaches to jugular foramen, and a multidisciplinary team is critical to manage these patients adequately.

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