Hypertensive crisis during embolization unveils occult catecholamine-secreting glomus jugulare tumor: A case report and management protocol

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Received September 9, 2025; Accepted April 8, 2026

DOI: 10.3892/etm.2026.13170

Abstract. Glomus jugulare tumor (GJT) is a rare paraganglioma arising from neural crest cells. Although the majority of cases are non-functional, a minority of GJTs can secrete catecholamines. Such functional variants are frequently overlooked due to their rarity and may trigger perioperative crises. The present report documents a 53-year-old female patient with a giant occult secretory GJT (65 mm) presenting with prolonged cranial nerve deficits. Preoperative blood pressure was normal, but the patient developed hypertensive crisis (220/130 mmHg) during embolization. Biochemical tests confirmed catecholamine excess. Hemodynamic stability was maintained using calcium channel blockers without α-blockade after embolization. Tumor resection was performed within 72 h, achieving total resection without intraoperative crisis. Catecholamine levels normalized postoperatively with marked neurological improvement. The present case highlights the importance of recognizing occult secretory GJTs and discusses key management considerations regarding preoperative preparation, timing of surgery and anesthetic management. Increased awareness may improve diagnosis and optimize outcomes in these challenging cases.

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Key words: head and neck paragangliomas, glomus jugulare tumor, occult catecholamine secretion, hypertensive crisis, multidisciplinary management, embolization, ectopic pheochromocytoma

Introduction

Glomus jugulare tumors (GJTs) are rare neuroendocrine neoplasms originating from paraganglion cells within the adventitia of the jugular bulb. These paraganglion cells are derived from the embryonic neural crest and belong to a category of head and neck paragangliomas (HNPGLs). Previous epidemiological studies have indicated that HNPGLs account for ~0.6% all head and neck tumors, with an annual incidence of 1-8 per million population (1-7). The most common sites of involvement, in descending order, are the carotid body (carotid body paragangliomas), jugular bulb (GJ), vagus nerve (vagus nerve paragangliomas) and tympanic cavity (tympanic paragangliomas) (7). The vast majority of HNPGLs are non-functional (parasympathetic type), whilst only 1-4% secrete catecholamines (sympathetic/functional type) (8,9). It is therefore estimated that the annual incidence of functional GJTs is <3.2 per 10 million population.

The rarity of functional GJTs has resulted in limited understanding and the lack of standardized treatment guidelines (8). Furthermore, GJTs are highly vascular tumors, frequently necessitating preoperative embolization to minimize intraoperative hemorrhage. The complex neurovascular anatomy of the jugular foramen region also demands meticulous surgical planning and technical expertise (10). Catecholamine secretory variants further complicate management due to the potential for hemodynamic instability and arrhythmias, significantly increasing perioperative risk (11-14). To the best of our knowledge, 15 cases of secretory jugular paragangliomas (JPGLs) have been identified (Table I) (9,14-24). Amongst these, four cases experienced cardiac arrest during treatment and one case of mortality occurred during postoperative care. In addition, it has been previously emphasized that preoperative biochemical screening and multidisciplinary coordination are crucial for reducing risks associated with managing this rare and complex condition (10,14,23,25).

The present report presents a challenging case of a large JPGL with occult catecholamine secretion, which was unmasked during embolization, highlighting a gap in preoperative recognition. Through subsequent multidisciplinary management, the tumor was successfully resected without

intraoperative crisis, where the patient recovered favorably. Based on this experience, a protocol that integrates biochemical monitoring, crisis-prepared anesthesia and a time-sensitive surgical approach to optimize outcomes in secretory GJTs was proposed.

Case report

Case presentation. A 53-year-old female patient initially presented with hoarseness and no other symptoms 3 years prior, leading to the diagnosis of a left jugular foramen mass at a local hospital. Subsequent evaluation confirmed a GJT and the patient underwent transarterial embolization (TAE). Following TAE, the patient's hoarseness showed no significant clinical improvement. However, because the patient was informed by the treating physician that surgical resection carried a high risk of damaging the posterior cranial nerves and a substantial likelihood of permanent dysphagia, surgical resection was deferred.

At 1 month before admission to Sanbo Brain Hospital (Beijing, China), the patient developed dysphagia, progressive left lower limb weakness and somnolence. Neurological examination revealed left-sided sensorineural hearing loss (cranial nerve VIII deficit, with a duration of ~1 year), left facial hypoesthesia (impaired pain and light touch perception; cranial nerve V deficit, with a duration of ~1 year), incomplete left eyelid closure, ipsilateral nasolabial fold flattening, leftward uvular deviation (progressive cranial nerve VII deficit, ultimately presenting as House-Brackmann grade IV (26), with a duration of ~6 months), diminished left gag reflex and left tongue deviation on protrusion (cranial nerves IX, X and XII deficits, with a duration of ~1 month). Left lower extremity motor strength was graded 4/5 (with a duration of ~1 month). The patient's medical history included 6 months hypertension managed with oral nifedipine. Notably, during the patient's prior hospitalization, the blood pressure remained stable without signs suggestive of pheochromocytoma (27).

Imaging findings. Contrast-enhanced MRI demonstrated a 65-mm dumbbell-shaped tumor centered in the left jugular foramen, extending into the cerebellopontine angle (CPA). The lesion compressed the brainstem, cerebellum and fourth ventricle, resulting in obstructive hydrocephalus (Figs. 1A, S1A and SB). Radiological classification was Fisch type D2 (28) and Glasscock-Jackson type IV (29). Bone-window CT revealed jugular foramen and carotid canal erosion (Fig. 1B). Adrenal CT and echocardiography showed no abnormalities (Fig. S1C).

Given the stable blood pressure on admission, the absence of hypertensive episodes during prior interventions, and most importantly, insufficient awareness of the occult secretory potential in such rare tumors, the catecholamine secretory nature of the tumor was not initially recognized. Consequently, biochemical testing was omitted.

Preoperative preparation. A multidisciplinary team (neurosurgery, neurointerventional radiology, anesthesiology, neurocritical care and nutrition) collaboratively optimized preoperative management. Given the hypervascularity of the tumor, preoperative embolization was performed (25,30). External ventricular drainage was additionally placed to mitigate intracranial pressure.

Biochemical evaluation. Post-embolization 24-h urinary catecholamines were found to be elevated: Epinephrine, 53.53 μg/24 h (normal range, 0-21), norepinephrine, 138.93 μg/24 h (normal range, 0-80) and dopamine 281.67 μg/24 h (normal range, 0-400). Plasma levels similarly showed marked norepinephrine elevation (2,247.60 pg/ml; normal range, 70-750, supine), confirming ectopic catecholamine secretion (31,32). Post-resection normalization (norepinephrine, 70.80 pg/ml) validated the diagnosis.

Endovascular intervention and anesthetic management. Under local anesthesia, diagnostic angiography was performed to confirm the vascular supply of the tumor, originating from the left external carotid, internal carotid and vertebral arteries (Fig. 1C). The use of local anesthesia at this stage allowed continuous neurological assessment and informed the subsequent decision to proceed with general anesthesia for definitive embolization. Balloon occlusion testing of the internal carotid artery confirmed adequate collateral circulation through the anterior communicating artery (Fig. S1G). Following general anesthesia induction, super-selective embolization of feeders from the posterior auricular, occipital meningeal and ascending pharyngeal arteries achieved >90% devascularization (Fig. 1C).

Notably, contrast administration during angiography triggered hypertensive crises (180/100 mmHg), persisting for 15 min. A second surge (220/130 mmHg) occurred during embolization, necessitating intravenous nicardipine (administered as an intravenous bolus of 0.5 mg, followed immediately by continuous intravenous infusion at a maintenance dose of 1-5 mg/h, titrated according to blood pressure), a first-line antihypertensive agent for intraoperative hypotensive anesthesia and catecholamine-induced hypertension (which was recommended in current guidelines and literature) (33,34), to rapidly stabilize hemodynamics (Fig. 2A).

Second preoperative multidisciplinary meeting. Based on the specific characteristics of the patient's condition, literature review (Table I) and accumulated clinical experience, a multidisciplinary team discussion was conducted to formulate a meticulous surgical plan: Given the tumor's compression of cranial nerves and the brainstem, early surgical excision was recommended, with tumor resection attempted on day 3 following interventional embolization. α/β-adrenergic blockers and magnesium sulfate injections were prepared for intraoperative use, where the anesthesiology team managed the patient's condition in accordance with the real-time anesthetic course.

Surgical intervention

Tumor exposure. Following induction of general anesthesia with stable hemodynamic monitoring (Fig. S1C), the surgical team initiated a two-phase approach. A periumbilical incision first provided autologous adipose tissue for subsequent reconstruction. A standardized retroauricular C-shaped incision was

Table I. Summary of patients with GJT in the previous literature.

Author, year	Patient (age/sex)	Associated symptoms	Preoperative CA level	Administration of medication	Administration effect	Embolization effects/crisis	Surgery	Intraoperative crisis in surgery	Surgery effect	Radiation treatment and effect	(Refs.)
Chweya *et al*, 2019	1 case (45/F)	FS: HA/HTN CND: VIII	Urine: Increased NE	α-Blockers (phenoxybenzamine; p.o., 3 months)	HTN ↓	N/A	Yes, STR	N/A	CA: N/A HTN ↓	Yes GKRS/ decreased CA/FS↑	(15)
	1 case (38/F)	FS: HA/HTN/ SNT/SNHL/ ARRH CND: N/A	Urine: Increased NE	α-Blockers (phenoxybenzamine; p.o., 7 days)	N/A	N/A	Yes, STR	N/A	N/A	GKRS/ decreased CA, FS↑	
Ibrahim *et al*, 2017	1 case (60/M)	FS: HTN/TNP/ ARRH CND: V and VIII	Urine: Increased MN and VMA	N/A	N/A	N/A	N/A	N/A	N/A	Yes, GKRS/ decreased CA, FS↑	(16)
Teranishi *et al*, 2014	1 case (31/F)	FS: HTN/SD/ VCP CND: IX-XII	Plasma: Increased NE Urine: Increased NE and VMA	α-Blockers (doxazosin mesylate; 3 months, p.o.); Magnesium sulfate (during iodine contrast test, i.v)	Increased weight; HTN ↓	Yes Crisis: N/A Effect: Tumor stain was reduced by 90%.	Yes, GTR	Cardiac arrest (resuscitation)/ hypotension	Reduce CA; FS: HTN/ SD/VCP↑	N/A	(14)
Fussey *et al*, 2013	1 case (37/F)	FS: SNT/ SNHL/VCP CND: VIII/ IX~XII	Plasma: Increased NMN Urine: Increased NE	α-Blockers (phenoxybenzamine; p.o., 7 days)	N/A	N/A	No	N/A	N/A	Yes, GKRS/ decreased CA and tumor volume; FS↑	(17)
Castrucci *et al*, 2010	1 case (47/M)	FS: HTN/ SNT/SNHL CND: VIII	Plasma: Increased NMN Urine: Increased NE, VMA and NMN	α-Blockers (phenoxybenzamine; p.o., 10 days) + β-blockers (labetalol; p.o., 10 days)	N/A	N/A	N/A	N/A	N/A	Yes, GKRS/ CA↓, decreased tumor volume; FS↑	(18)
Colen *et al*, 2009	1 case (65/F)	FS: HTN/HA	Urine: Increased NMN and VMA	α-Blockers (phenoxybenzamine; p.o., >14 days) + β-blockers (propranolol; p.o., N/A)	HTN↓	Yes Crisis: HTN↑ (post-embolization night) Effect: N/A	Yes, GTR	HTN↑	CA: N/A FS: HTN↑/ HA↑	N/A	(19)

Table I. Continued.

Author, year	Patient (age/sex)	Associated symptoms	Preoperative CA level	Administration of medication	Administration effect	Embolization effects/crisis	Surgery	Intraoperative crisis in surgery	Surgery effect	Radiation treatment and effect	(Refs.)
Goutcher *et al*, 2006	1 case (69/M)	FS: HTN/FNP/ SNHL CND: VII/VIII	Plasma: Increased NE Urine: Increased NMN	α-Blockers (phenoxybenzamine; p.o., 8 weeks) + β-blockers (propranolol; p.o., 8 weeks); Magnesium sulfate (during iodine contrast test, i.v.)	N/A	Yes Crisis: N/A Effect: N/A	Yes, GTR	HTN↑ Cardiac arrest (resuscitation)	CA: N/A FS: HTN↓/ FNP↓	N/A	(9)
	1 case (59/M)	FS: SNT/ SNHL CND: VIII	Plasma: Increased NE Urine: Increased NE and DA	α-Blockers (phenoxybenzamine; p.o., 4 weeks) + β-blocker (atenolol; p.o., 4 weeks); Magnesium sulfate (during iodine contrast test, i.v.)	N/A	Yes Effect: N/A	Yes, GTR	N/A	CA: N/A FS: FNP↓/ SNHL-	N/A	
Ueda *et al*, 2002	1 case (51/F)	FS: HTN/SNT/ SNHL CND: VIII	Plasma: Increased NE Urine: Increased NE and VMA	N/A	N/A	Yes Effect: No change	N/A	N/A	CA: -	Yes, GKRS/ N/A	(20)
Kremer *et al*, 1989	1 case (43/F)	FS: HTN/ palpitation CND: N/A	Urine: Increased NE and VMA	α-Blockers (prazosin; p.o., 1 week)	FS: HTN↓/ palpitation↓	Yes Crisis: HTN↑/cardiac rhythm↑/ cardiac failure Effect: N/A	Yes, GTR	HTN↑/arrest↑	Reduced CA	N/A	(21)
Schwaber *et al*, 1988	1 case (58/F)	FS: HA/HTN CND: N/A	Plasma: NE ↑ Urine: NE↑, MN↑	α-Blocker (phenoxybenzamine; p.o., N/A)	HTN↓	N/A	N/A	N/A	N/A	Yes, 4,750-rad Cobalt-60 radiation/-	(22)
Matishak *et al*, 1987	1 case (39/M)	FS: HTN CND: N/A	Plasma: Increased NE Urine: Increased NE and MN	α-Blockers (phenoxybenzamine; p.o., N/A) + β-blockers (propranolol; p.o., N/A)	Reduced HTN	Yes Crisis: Hypotension/ HTN↑/cardiac arrest Effect: N/A	Yes, GTR	N/A	Reduced CA FS: FNP↓/SD↓/ VCP↓	N/A	(23)

Table I. Continued.

Author, year	Patient (age/sex)	Associated symptoms	Preoperative CA level	Administration of medication	Administration effect	Embolization effects/crisis	Surgery	Intraoperative crisis in surgery	Surgery effect	Radiation treatment and effect	(Refs.)
Schwaber *et al*, 1984	1 case (31/F)	FS: HTN/VCP CND: IX-XII	Plasma: Increased DA and NE Urine: Increased VMA and MN	α-Blockers (phenoxybenzamine; p.o., 2 weeks) + β-blockers (propranolol; p.o., 2 weeks)	N/A	N/A	Yes, GTR	N/A	Succumbed (pulmonary embolism)	N/A	(24)
	1 case (22/M)	FS: SNT/ SNHL/VCP CND: IX-XII	First surgery: N/A Second surgery: i) Plasma: Increased NE; ii) Urine: Increased VMA; increased MN	First surgery: N/A Second surgery: α-Blockers (phenoxybenzamine, p.o.; N/A)	N/A	N/A	Yes, GTR	First surgery: Cardiac arrest/ HTN↑ (terminated) Second surgery: N/A	CA: N/A FS: Improved SNT/ improved SNHL/i mproved VCP	N/A	

Symbols: '↑' indicates elevated hormone levels or improvement in dysfunction; '↓' indicates decreased hormone levels or worsening dysfunction/decline in HTN, '-' indicates no change or no effect. M, male; F, female; FS, functional symptoms; CND, cranial nerve dysfunction; GKRS, gamma-knife radiosurgery; STR, subtotal resection; GTR, gross-total resection; TNP, trigeminal nerve paralysis; HA, headache; HTN, hypertension; FNP, facial nerve paralysis; ARRH, arrhythmia; SNT, sensorineural tinnitus; SNHL, sensorineural hearing loss; SD, swallowing disorder; VCP, vocal cord paralysis; CA, catecholamines; NE, norepinephrine; DA, dopamine; NMN, normetanephrine; MN, metanephrine; VMA, vanillylmandelic acid; N/A, not applicable; p.o., per os; i.v., intravenously.

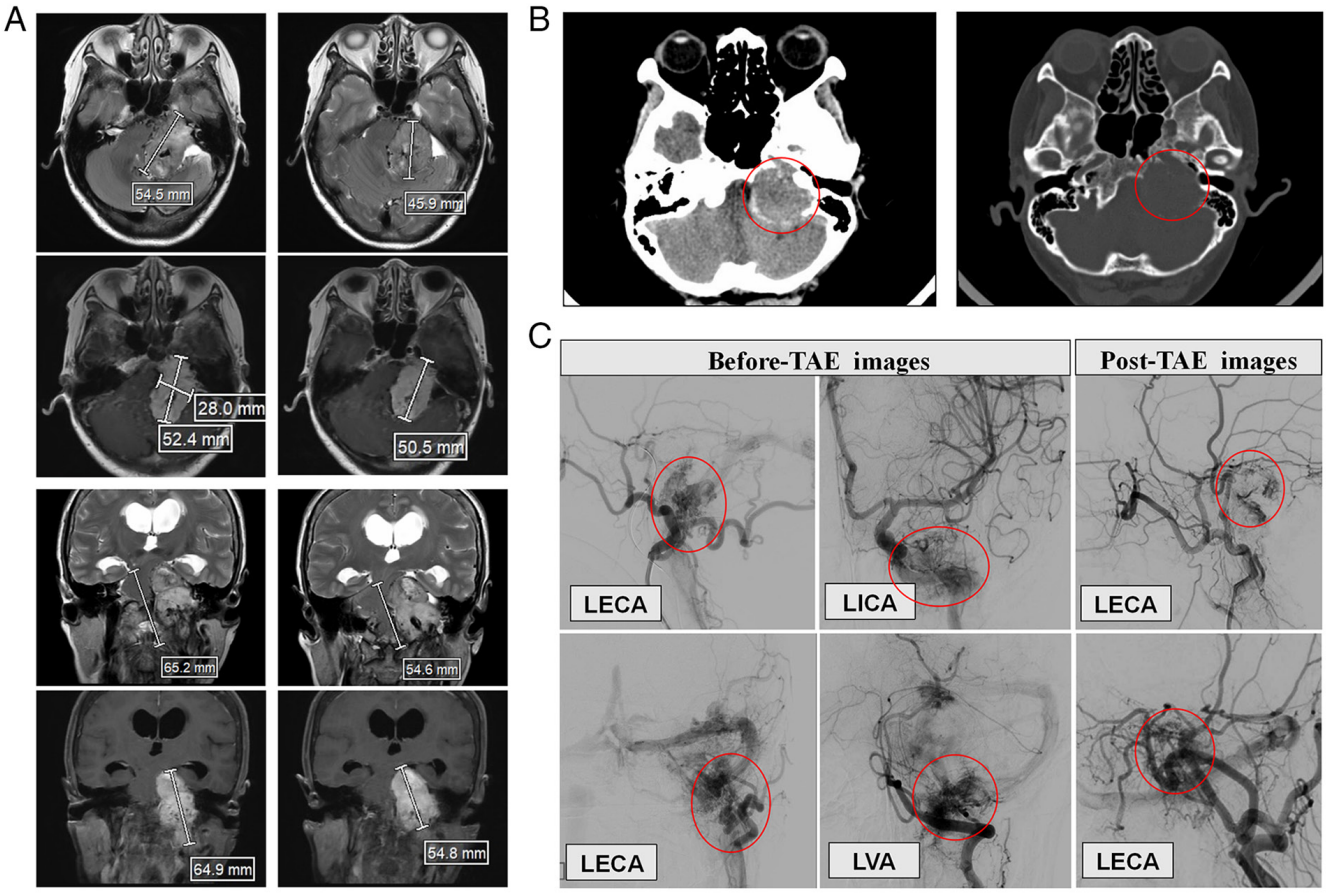

Figure 1. Preoperative neuroimaging and endovascular evaluation. (A) T2-weighted and contrast-enhanced T1-weighted MRI demonstrating a dumbbell-shaped tumor centered in the left jugular foramen, with intracranial extension compressing the brainstem and cerebellar hemisphere. (B) CT images in bone and brain windows showing erosive destruction of the left jugular foramen and partial involvement of the carotid canal. (C) DSA illustrating the arterial supply and venous drainage of the tumor, followed by post-embolization DSA after transcatheter arterial embolization, which shows a marked reduction in tumor vascularity. The red ellipses indicate the anatomical location of the tumor on (B) MRI and its angiographic appearance on (C) angiography. DSA, digital subtraction angiography; LECA, left external carotid artery; LICA, left internal carotid artery; LVA, left vertebral artery; TAE, transarterial embolization.

then extended along the left neck, permitting systematic exposure of the sternocleidomastoid muscle, internal jugular vein (IJV) and internal carotid artery (ICA). The tumor presented a bimodal configuration: i) An intraluminal IJV component extending caudally to C4; and ii) a deep extravenous portion nestled between the ICA anteriorly and vertebral artery posteriorly. Mastoidectomy with facial nerve canal decompression (preserving petrous, tympanic and labyrinthine segments) and sigmoid sinus skeletonization preceded intracranial access. The intracranial extension of the tumor demonstrated CPA involvement with middle ear extension, displaying characteristic hypervascular, grayish-red morphology adherent to the facial nerve's petrous segment.

Extracranial tumor resection. After complete exposure, sequential vascular control was achieved through IJV and facial vein ligation. Meticulous microsurgical technique facilitated circumferential dissection of tumor from the cervical ICA (including vertical and partial horizontal segments). Jugular foramen decompression enabled *en bloc* resection of the petrous portion, with intraoperative hemostasis maintained by using an absorbable gelatin sponge compression during tumor debulking (Fig. S2).

Intracranial tumor resection. The intracranial portion of the tumor was primarily located in the left side of the CPA area, with its surface covered by proliferative pathological blood vessels. The trigeminal nerve was displaced superiorly by the tumor, whilst the facial and vestibulocochlear nerves were compressed ventrally and inferiorly. The tumor was tightly adhered to the brainstem but was ultimately resected in segments after sufficient decompression. Surrounding tissues were preserved intact. Adipose tissue was used to fill the jugular foramen area for structural support (Fig. S2).

Anesthetic management process. Prior to surgery, the anesthesiologist had prepared a comprehensive treatment plan for the potential massive catecholamine release, including the use of α-adrenergic blocker (phenoxybenzamine), which were ultimately not administered due to concerns about the risk of hypotension. During the surgical procedure, β-adrenergic antagonists (landiolol) and calcium channel blockers (nicardipine) were used for maintaining stable heart rate and blood pressure. Throughout the procedure, blood pressure remained stable and there were no episodes of sudden or uncontrollable hypertension (Fig. 2B).

Intraoperative management. Postoperatively, the patient received treatment in the intensive care unit and was able to breathe spontaneously ~8 h after surgery, leading to the removal of the endotracheal tube. On the following day, the patient was transferred back to a general ward without

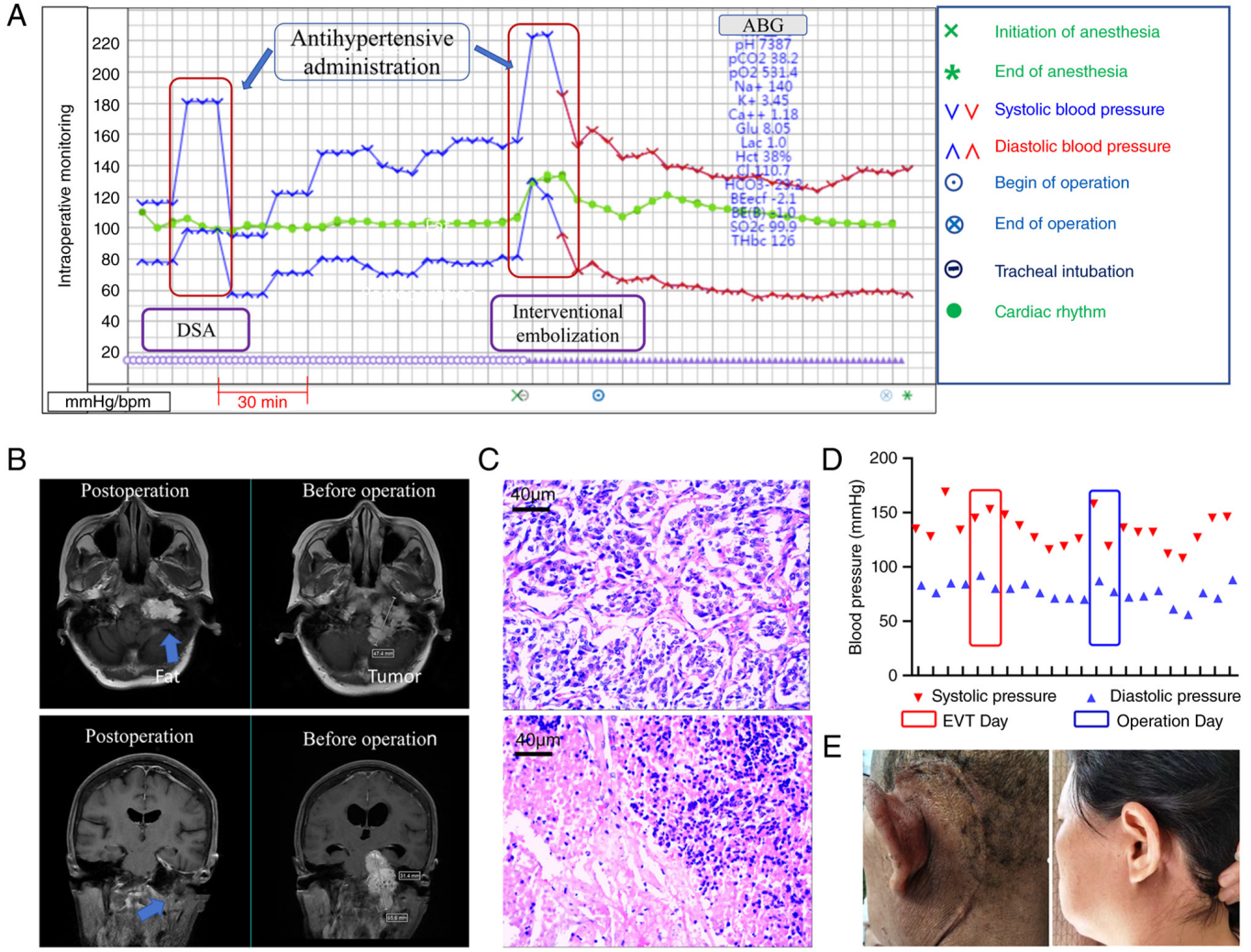

Figure 2. Perioperative management and outcomes. (A) Anesthesia record during endovascular embolization demonstrating a hypertensive crisis (peak systolic pressure, 220 mmHg) following contrast administration and particle embolization, stabilized with nicardipine infusion. The red box indicates the blood pressure line on the anesthesia record during the hypertensive crisis, and blue arrow indicates that blood pressure management was performed during this period. (B) Postoperative contrast-enhanced T1-weighted MRI showing complete resection of the jugular foramen mass with brainstem decompression and fat graft placement. The blue arrows indicate the MRI appearance after tumor resection. (C) H&E staining revealing a nested and trabecular architecture of tumor cells separated by abundant thin-walled and reticulated vascular channels, with focal areas of necrosis. (D) Blood pressure trends over the first 11 days of hospitalization (recorded at 8 AM and 6 PM daily). Red boxes indicate the EVT day; blue boxes indicate the day of surgery. (E) Images of the patient at 1 month and 1 year postoperatively. DSA, digital subtraction angiography; ABG, arterial blood gas; EVT, endovascular treatment.

experiencing hypotension or hypoglycemia due to reduced plasma catecholamine levels. The posterior cranial nerve symptoms did not worsen postoperatively and they continued to receive nutrition through the gastric tube that had been inserted prior to surgery. In total, 20 days after the operation, there was an improvement in the posterior cranial nerve symptoms and limb muscle strength, allowing for a smooth discharge from the hospital. After returning home, the patient continued nasogastric tube feeding, rested at home and gradually recovered. Subsequently, 1 month postoperatively, the patient was able to eat orally and manage daily activities independently.

H&E and immunohistochemistry (IHC) staining results. Formalin-fixed, paraffin-embedded tissue sections (4 μm) were processed using a VENTANA automated immunostainer following the manufacturer's protocols. Antigen retrieval was performed with CC1 buffer (pH 8.0) at 95˚C for 24 min. Primary antibodies were incubated at 37˚C for 32 min, followed by HRP-labeled secondary antibody and DAB detection. Sections were counterstained with hematoxylin. Microscopy was performed on a Leica MD3000 microscope. Antibody dilutions of 1:100-1:500 were applied.

H&E and IHC staining revealed the classic structure of paragangliomas and their immunomarkers. H&E staining observations: Tumor cells exhibited a nest-like and trabecular distribution of tumor cells, with the cell nests separated by abundant thin-walled and reticular blood vessels, accompanied by focal necrosis (Fig. 2C).

IHC staining observations were as follows: Synaptophysin(+), tumor protein 53)(-), microtubule-associated protein(-), myelin basic protein(-), chromogranin A(+), neuron-specific enolase(+), CD31(-), coagulation factor VII(-), S-100 protein(focally positive), vimentin(+), neuronal nuclei(-), CD34(-), SRY-box transcription factor

10(focally positive), histone H3 trimethylated at lysine 27(+), epithelial membrane antigen(-), CD56(+), glial fibrillary acidic protein(-), oligodendrocyte transcription factor 2(-), epidermal growth factor receptor(-), succinate dehydrogenase B(+), neurofilament(focally positive) and Ki-67 (proliferation marker; positive in <1% of tumor cells).

Discussion

According to the widely accepted Glasscock-Jackson and Fisch classification systems (28,29), the term 'GJTs' in the strict sense refers to paragangliomas originating from the paraganglion cells located at the dome of the jugular bulb. However, in clinical practice, due to close anatomical proximity, tumors arising from the tympanic cavity frequently extend inferiorly to involve the jugular bulb, whilst those originating from the jugular bulb frequently invade the tympanic cavity. Therefore, these are collectively termed JPGLs in a broader context (35,36). In the present case, the initial symptoms included hoarseness without hearing loss, with auditory decline manifesting only at a later stage. Preoperative imaging revealed a large mass in the jugular foramen, which was confirmed by intraoperative findings to be a true GJT in the narrow sense. The present case is notable for its exceptionally low incidence and the absence of typical symptoms of catecholamine excess, such as hypertension, diaphoresis, palpitations or headache, which led to a lack of awareness regarding its secretory potential prior to interventional procedures.

Historical data indicate that the perioperative mortality rate for undiagnosed pheochromocytoma can reach 25% (12). Although advances in medical technology have substantially reduced this rate, contemporary clinical reports continue to document life-threatening crises, including cases of intraoperative cardiac arrest. A review of previously treated GJT cases (Table I) similarly reflects the high risks associated with managing these tumors. Given that GJTs share physiological similarities with pheochromocytomas, they can be regarded as a form of extra-adrenal pheochromocytoma (34). The present case underscores the diagnostic and therapeutic challenges posed by secretory GJTs. Even in the absence of classic symptoms, the potential for catecholamine secretion must be considered. Therefore, preoperative screening of blood and urine for catecholamines and their metabolites is likely essential. In addition, adrenal pheochromocytoma and ectopic pheochromocytomas in other locations (such as thoracic or abdominal) should be ruled out.

A critical oversight in the present case was the omission of preoperative biochemical screening for catecholamines. Despite the absence of typical symptoms such as paroxysmal hypertension, diaphoresis or palpitations, and despite stable blood pressure at admission and during prior interventions, the tumor's occult secretory potential was not initially suspected. This diagnostic gap reflects the low index of suspicion for functional paragangliomas in the absence of classic clinical features, and underscores a key learning point: Routine biochemical screening for catecholamine excess should be considered in all patients with GJT, regardless of blood pressure status or symptomatology. Delayed recognition may lead to life-threatening hypertensive crises during angiography, embolization or surgery, as occurred in the present case. Therefore, a low threshold may be suggested to perform plasma or urinary metanephrine testing in all cases of head and neck paragangliomas, particularly when embolization or surgical intervention is planned.

Another significant insight from the present case is that catecholamine secretion can be provoked during interventional procedures, such as the injection of iodinated contrast (such as ioversol) or embolization, leading to hypertensive crisis (220/130 mmHg). This observation is consistent with previous case reports (Table I) (19,21,23). The underlying pathophysiological mechanism remains incompletely understood, but may involve direct mechanical stimulation of tumor cells or transient ischemia induced by the high viscosity of the contrast agent. This phenomenon highlights the risk of catastrophic cardiovascular events resulting from an unexpected hypertensive crisis during embolization or imaging studies. Therefore, continuous hemodynamic monitoring and the presence of an anesthesiologist are mandatory during interventional procedures, regardless of initial biochemical screening results.

Another point of discussion is the use of preoperative α- and β-adrenergic blockade. According to established guidelines for pheochromocytoma management (34), α- and β-blockers are typically administered preoperatively to control hypertension, tachycardia and other catecholamine-related symptoms. In the present case, however, calcium channel blockers (such as nifedipine and nicardipine) were sufficient to maintain blood pressure within an acceptable range throughout the embolization, resection and postoperative phases. This suggests that catecholamine secretion by the tumor was significant but not excessively active, where the embolization procedure substantially suppressed its functional activity. It is worth emphasizing that although an α-blocker (phentolamine) and magnesium sulfate [which blocks the action of catecholamines (14)] was prepared for intraoperative use, it was not administered due to concerns about arrhythmia and hypotension.

Regarding the timing of surgery, given the presence of significant neurological deficits and the large tumor volume, it was decided to perform resection within 72 h after embolization. This approach aimed to minimize secondary injury to nerves and the brainstem caused by post-embolization inflammatory edema. Although certain protocols recommend an interval of 1-2 weeks between embolization and surgery, the timing in the present case balanced the risk of acute catecholamine release and hypertensive crisis against the urgency of relieving brainstem compression. The successful outcome suggests that with thorough evaluation and multidisciplinary preparation, accelerated surgical intervention may be feasible and beneficial in cases with severe neural compression.

Postoperative management is equally critical. The present case involved a large tumor, complex anatomy, prolonged operative time, considerable blood loss and secretory function, all of which complicated postoperative care. These observations suggest that monitoring in an intensive care unit for 24-48 h is essential. This period should be extended

if lower cranial nerve deficits are present. During this time, vigilance is required for hypotension due to the abrupt decline in catecholamine levels after tumor removal and for the risk of venous thrombosis, particularly deep vein thrombosis of the lower limbs. A previous case of fatality due to pulmonary embolism has been reported postoperatively (24). In addition, early initiation of enteral nutrition and rehabilitation is essential. Nasogastric tube feeding within 24 h helps address malnutrition resulting from dysphagia caused by cranial nerve palsy, whilst early involvement of rehabilitation specialists promotes functional recovery of swallowing and mobility (36-40).

In conclusion, the present case underscores the importance of recognizing occult secretory GJTs and presents a successful example of surgical resection performed within a short period after endovascular embolization, without the prolonged use of α-adrenergic blockers. Throughout the diagnostic and treatment process, a proactive and protocol-based multidisciplinary approach was adopted, integrating expertise from neurovascular surgery, interventional radiology and specialized anesthesiology, which ultimately yielded favorable outcomes. This case further enriches the treatment strategies for secretory GJTs and enhances the understanding of this disease entity, which may offer an optimized pathway for the diagnosis and management of this rare and challenging tumor.

Acknowledgements

The authors would like to thank Dr Mingwang Zhu (Department of Neuroradiology, Sanbo Brain Hospital Clinical Center, Beijing, China) for diagnostic imaging analysis and Ms Jianhua Huang (Neurosurgical Nursing Team, Sanbo Brain Hospital Clinical Center, Beijing, China) for postoperative care.

Funding

This work was supported by the PhD Research Fund of Weifang People's Hospital, Shandong Second Medical University (Weifang, China).

Availability of data and materials

The data generated in the present study may be requested from the corresponding author.

Authors' contributions

HWZ, WHN and SCJ designed the case report and coordinated the clinical evaluation. HWZ and WHN are co-corresponding authors. SCJ and TP were responsible for data collection and analysis, clinical interpretation and assessments, with equal contributions. YMQ, KG, GJS and XNL participated in the diagnosis and treatment of the patient, in addition to the analysis and interpretation of clinical data and supervised the study process. All authors contributed to the writing of the original draft and preparation of figures. The authenticity of all raw data was confirmed by all authors. The final version of the manuscript has been read and approved by all authors.

Ethics approval and consent to participate

The present case report involving human participants was reviewed and approved by the Institutional Review Board of Capital Medical University (approval no. SBNK-YJ-2025-017-01).

Patient consent for publication

Written informed consent was obtained from the patient for publication of clinical details and accompanying images. The patient was fully informed about the publication's purpose and provided explicit consent for the use of images and other potentially identifiable information.

Competing interests

The authors declare that they have no competing interests.

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