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FROM SYNCOPE TO STROKE: VASCULAR COMPLICATIONS OF SKULL BASE PARAGANGLIOMA

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ABSTRACT

Background: Head and neck paragangliomas are typically slow-growing neuroendocrine tumours, but their proximity to major cervical vessels can result in significant neurovascular complications. Ischemic stroke is rarely described as a direct consequence and is more commonly attributed to catecholamine-mediated hypertension or vasospasm.

Case description: A 48-year-old woman presented with recurrent syncope and was found to have multifocal acute ischemic infarcts on magnetic resonance imaging of the brain. Imaging revealed recurrent jugular foramen paraganglioma with extension along the internal carotid artery and compression of the internal jugular vein. The infarct pattern was embolic in distribution. Although hypertension and recent cocaine use were present, the close anatomic relationship between the tumour and major cervical vessels suggested tumour-related vascular compromise with secondary thromboembolism as a primary mechanism.

Conclusion: This case highlights structural vascular involvement by head and neck paraganglioma as an underrecognized cause of embolic stroke. In patients with skull base tumours and multifocal infarcts, direct tumour-related vascular compromise should be considered to guide appropriate multidisciplinary management and reduce the risk of recurrent events.

KEYWORDS

Paraganglioma, syncope

LEARNING POINTS

- Head and neck paragangliomas can cause ischemic stroke through direct vascular involvement, including carotid artery encasement and jugular vein compression, creating a structural source of thromboembolism.
- Multifocal infarcts across multiple vascular territories should prompt evaluation for embolic and structural causes, even in the presence of traditional risk factors such as hypertension or cocaine use.

INTRODUCTION

Paragangliomas are rare neuroendocrine tumours arising from extra-adrenal paraganglia of the autonomic nervous system. In the head and neck, they most commonly

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originate from the carotid body, jugular bulb, or vagus nerve[1,2]. Although typically slow-growing and histologically benign, these tumours may cause substantial morbidity due to local invasion of adjacent neurovascular structures[3].

Cerebrovascular complications associated with paragangliomas are uncommon. When stroke occurs, it is most often attributed to catecholamine-mediated hypertension, vasospasm, or cardiomyopathy-related embolism in functional tumours[4-6]. Direct vascular invasion or compression resulting in thromboembolism has been described but remains underrecognized as a primary mechanism of ischemic stroke, particularly in skull base paragangliomas involving the internal carotid artery or jugular venous system[7-9].

We describe a patient with recurrent jugular foramen paraganglioma presenting with syncope and subsequently found to have multifocal embolic infarcts in the setting of carotid and jugular involvement. This case emphasizes structural vascular compromise as a clinically significant and potentially underappreciated mechanism of stroke in head and neck paraganglioma.

CASE DESCRIPTION

A 48-year-old African American female with a history of hypertension, newly diagnosed type 2 diabetes mellitus, prior right external auditory canal tumour treated with surgical resection and radiation, and a remote history of resection of a thoracic tumour with partial lung resection, presented with recurrent syncope and presyncope. Historical details were limited due to a recent relocation from another state and incomplete outside medical records.

On the day of presentation, the patient experienced two falls. Earlier in the day, she had an episode of presyncope while standing, during which she was caught by her husband without head trauma or loss of consciousness. Importantly, the presyncopal episode was associated with transient focal neurologic deficits, including right-sided facial paralysis and slurred speech that quickly resolved. Several hours later, she had a witnessed syncopal episode and was found on the ground with suspected head trauma. According to her husband, she was initially unresponsive, followed by approximately one minute of confusion before returning to baseline mental status. There were no witnessed tonic–clonic movements, automatisms, tongue biting, or incontinence. The patient reported a post-fall headache but denied focal neurologic deficits, visual changes, chest pain, dyspnoea, fever, gastrointestinal symptoms, or urinary complaints. Her husband noted increased fatigue and somnolence over several days preceding admission.

She was admitted to the academic internal medicine service for evaluation of syncope and concern for intracranial pathology. Initial non-contrast computed tomography scan of the head demonstrated a right temporal soft tissue density concerning for neoplasm versus infection, with surrounding vasogenic oedema *(Fig. 1 and 2)*. Dexamethasone was initiated for cerebral oedema, and



Figure 1. *Axial contrast-enhanced computed tomography scan of the neck. Demonstration of an enhancing mass (A, yellow arrow) centred upon the right jugular foramen and extending inferiorly, with more than 180 degrees encasement of the right carotid artery (B, yellow arrows), suspicious for glomus jugular paraganglioma. Laterally, the right jugular vein (arrowhead) is slightly displaced. Incidental detection of secretions in the right maxillary sinus and nasopharynx.*

levetiracetam was started for seizure prophylaxis. Neurosurgery was consulted and recommended further evaluation with magnetic resonance imaging (MRI) of the brain. Brain MRI revealed recurrent paraganglioma involving the right jugular foramen with extension along the internal carotid artery, as well as three additional small intracranial lesions consistent with metastatic disease *(Fig. 3 and 4)*. Notably, diffusion-weighted imaging demonstrated multiple areas of restricted diffusion in different vascular territories, consistent with early subacute ischemic infarcts *(Fig. 5)*. These findings raised concern for an embolic stroke mechanism in the setting of vascular invasion and

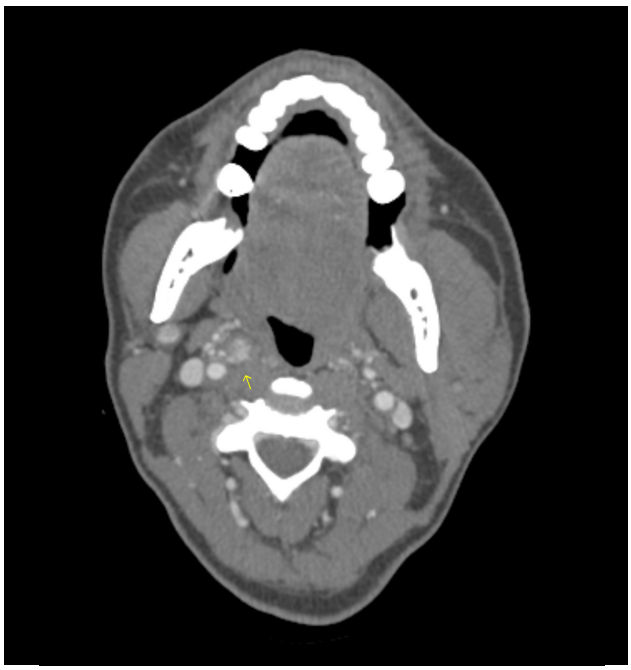


Figure 2. Axial contrast-enhanced computed tomography scan of the neck. Demonstration of a similar-appearing smaller enhancing mass (yellow arrow) anteromedial of the right common carotid artery, without encasement, likely representing an additional paraganglioma or segmentation of the original lesion.

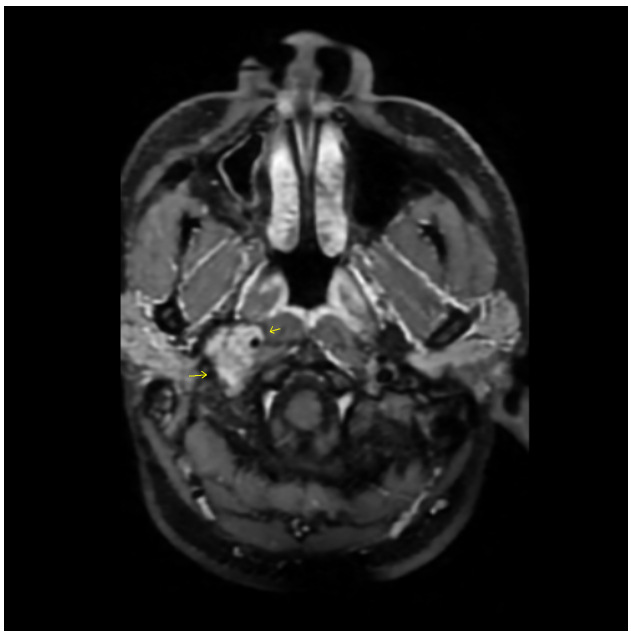


Figure 3. Axial contrast-enhanced magnetic resonance imaging of the brain 3D fast spin echo sequence. Demonstration of an enhancing right jugular lobulated mass with near-complete encasement of the right internal carotid artery.

compression by the paraganglioma, including involvement of the internal carotid artery and internal jugular vein.

Neurology was consulted for management of acute ischemic stroke. Given the multifocal infarct pattern, the leading aetiologies included tumour-related vascular compression and endothelial injury with secondary thromboembolism, catecholamine-mediated hypertension from a functional paraganglioma, and recent cocaine use contributing to vasospasm. The patient was not a candidate for thrombolytic therapy due to unclear symptom onset time. Secondary stroke prevention was initiated with high-intensity statin therapy and optimization of blood pressure control. Psychiatry was consulted after the patient demonstrated emotional lability, circumstantial thought processes, and maladaptive coping behaviours following disclosure of imaging findings and a positive urine toxicology screen. She was started on aripiprazole with plans for outpatient psychiatric follow-up. Physical therapy, occupational therapy, and case management were involved, and the patient was discharged in stable condition with close outpatient follow-up arranged.

DISCUSSION

This case demonstrates an uncommon but clinically significant mechanism of ischemic stroke in the setting of head and neck paraganglioma. Brain MRI revealed multifocal areas of restricted diffusion across distinct vascular territories, consistent with an embolic pattern. Concurrent imaging identified a recurrent jugular foramen paraganglioma with extension along the internal carotid artery and compression of the internal jugular vein, providing a clear anatomic substrate for thrombus formation and subsequent embolization.

Head and neck paragangliomas are hypervascular tumours that frequently encase or displace adjacent vascular structures due to their skull base location[1-3,7-9]. While vascular involvement is well described, stroke is rarely reported as a presenting manifestation. When cerebrovascular events occur in association with paragangliomas, they are more commonly attributed to catecholamine-mediated hypertension, vasospasm, or cardiomyopathy-related embolism[4-6]. In contrast, this case supports a structural mechanism in which direct vascular compression and endothelial injury create a prothrombotic environment, further compounded by altered flow dynamics and venous stasis.

The patient's presenting symptom of syncope is unlikely to be directly attributable to the embolic infarcts. Given the tumour's location at the jugular foramen with extension along the internal carotid artery, involvement of the carotid sinus or glossopharyngeal afferents is a plausible mechanism for reflex-mediated syncope. Loss of consciousness is not a typical feature of ischemic stroke and is generally seen only in cases involving the reticular activating system, basilar artery occlusion, or significantly elevated intracranial pressure. In this case, there were no focal neurologic deficits or imaging findings to suggest such involvement. Instead, syncope is more plausibly explained by tumour-related vasovagal involvement causing reflex-mediated cerebral hypoperfusion. These mechanisms are consistent with prior reports of syncope in patients with carotid body and skull base tumours[10].

The patient's psychiatric manifestations were more consistent with acute stress response in the setting of

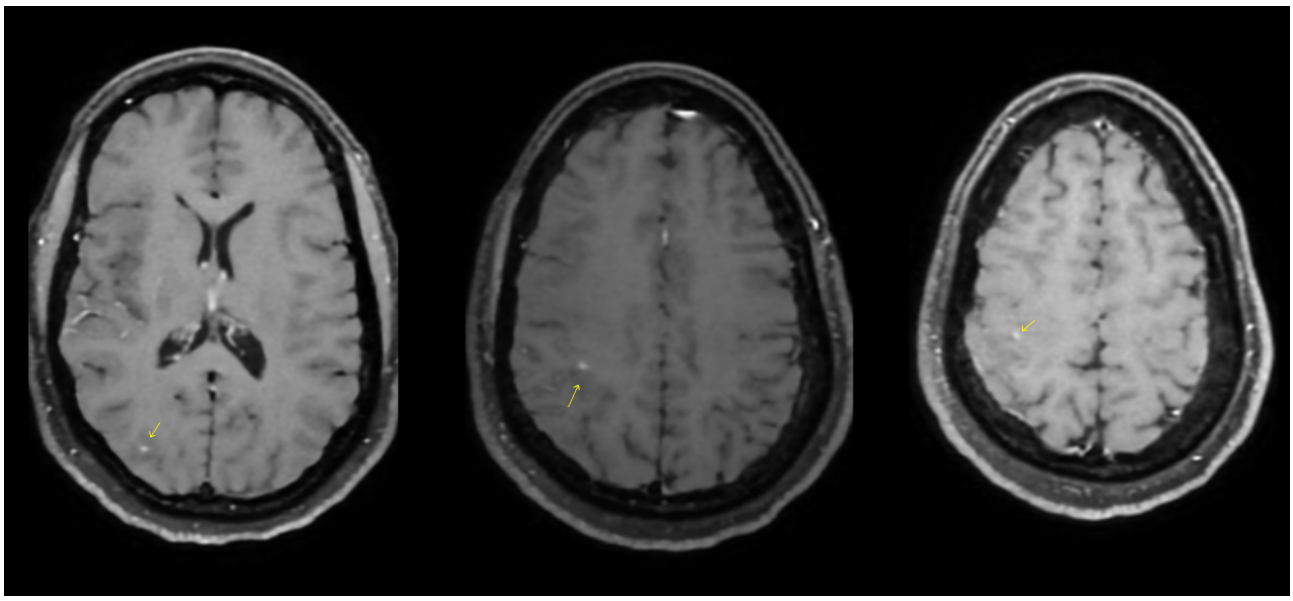


Figure 4. Axial contrast-enhanced magnetic resonance imaging of the brain 3D fast spin echo sequence. Three tiny, scattered foci of enhancement in the posterior frontal, parietal and occipital lobes, concerning for a metastatic process.

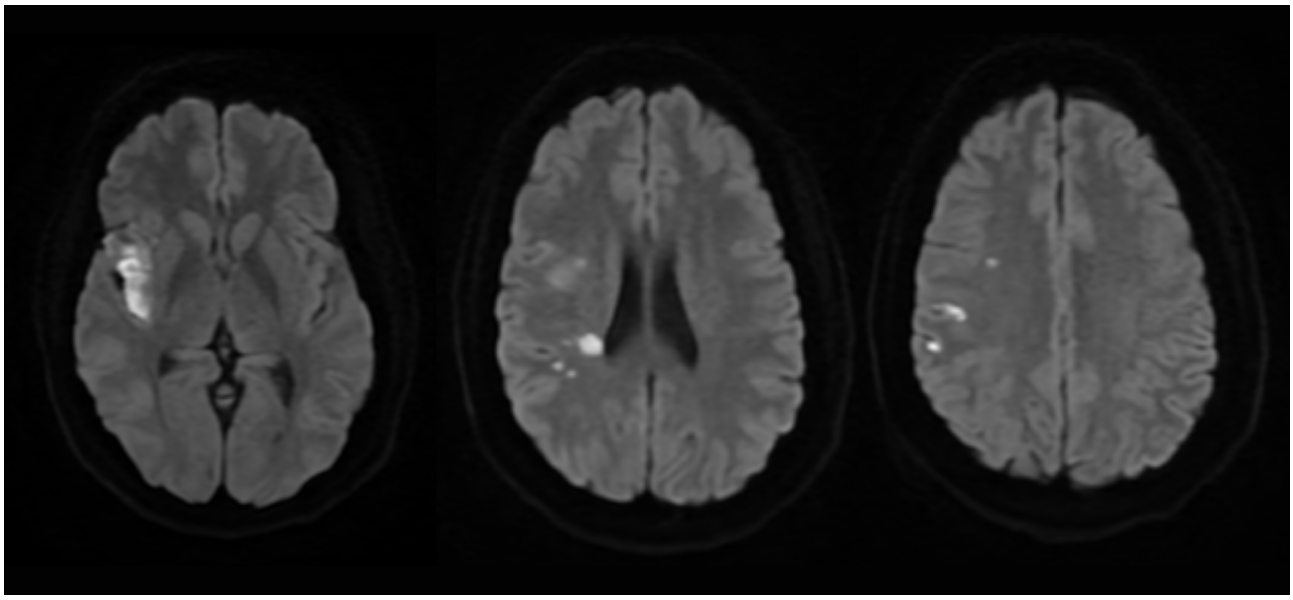


Figure 5. Magnetic resonance imaging of the brain, axial diffusion-weighted imaging sequence. Multiple areas of restricted diffusion in the right insular cortex and temporal lobe, and smaller areas of restricted diffusion in the right caudate nucleus tail and cortical/subcortical right frontal centrum semiovale, with associated T2 fluid-attenuated inversion recovery hyperintense signal suggesting early subacute infarcts. This may be secondary to internal jugular vein compression, embolic source, or flow-related ischemia.

hospitalization and recent substance use rather than multi-infarct dementia. While multi-infarct dementia remains a theoretical consideration in patients with recurrent ischemic events, it typically presents as a chronic, progressive cognitive decline rather than acute behavioural changes[6]. Additionally, the limited burden and subacute nature of the infarcts identified on imaging make vascular dementia less likely in this case. Involvement of the insular cortex and caudate nucleus may also have contributed to acute emotional dysregulation.

Management strategies that could have been considered include systemic anticoagulation for presumed tumour-associated vascular compression. Definitive tumour-directed therapy includes surgical resection or stereotactic radiosurgery, with the latter demonstrating high tumour control rates and lower rates of treatment-related morbidity in selected patients[7-9]. Thrombolytic therapy was not pursued given the unclear timing of symptom onset and concern for bleeding risk in the setting of intracranial lesions. Tumour-directed interventions were deferred due to the patient's recurrent disease, prior radiation exposure, and the complexity of vascular involvement, which increased the procedural risk. Financial constraints also limited surgical intervention given the lack of health insurance and primary care physician. Management decisions were further influenced by significant socioeconomic barriers, including lack of health insurance and limited access to longitudinal care.

The presence of multiple lesions raises the possibility of an underlying hereditary paraganglioma-pheochromocytoma syndrome. Up to half of head and neck paragangliomas are associated with germline mutations, most commonly involving succinate dehydrogenase genes[2]. Genetic testing is recommended, as identification of a hereditary syndrome has implications for surveillance and family screening.

Overall, this case highlights the importance of correlating infarct patterns with structural imaging findings. In patients with skull base tumours, multifocal infarcts should prompt evaluation for direct vascular involvement as a potential embolic source. Recognition of this mechanism may influence both stroke prevention strategies and long-term management planning.

CONCLUSION

Head and neck paragangliomas are often indolent but may cause significant neurologic morbidity through local vascular involvement. This case demonstrates recurrent embolic ischemic strokes associated with carotid and jugular involvement by an invasive jugular foramen paraganglioma, with syncope as the initial presenting feature. Structural tumour-related vascular compromise should be considered in patients with skull base paragangliomas and multifocal infarcts, particularly when conventional stroke risk factors do not fully account for the imaging pattern.

Early recognition may reduce the risk of recurrent ischemic events and guide appropriate multidisciplinary management. This case illustrates an underrecognized mechanism of recurrent embolic ischemic stroke resulting from carotid and jugular involvement by an invasive paraganglioma, with syncope as the presenting manifestation. Clinicians should consider tumour-related embolic stroke in patients with multifocal infarcts and skull base tumours, particularly when conventional risk factors fail to fully explain imaging findings. Early identification of this mechanism is critical to accurate diagnosis and prevention of recurrent ischemic events.

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