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Parasellar meningioma presenting by stroke and bilateral occlusion of the internal carotid arteries: A case report

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Abstract

Cerebrovascular stroke caused by skull base meningioma has been rarely reported. A 30-year-old male presented (April 2015) with acute right-sided hemiplegia. His brain neuroimaging (computerized tomography and magnetic resonance imaging) showed left ischemic infarction in the territory of middle cerebral artery. Magnetic resonance imaging also showed a right parasellar solid lesion which extended to the right basisphenoid and cavernous sinus and attenuated the right internal carotid artery. It also had left smaller parasellar extension. The lesion enhanced uniformly and strongly following gadolinium injection. Digital subtraction angiography using selective catheterization of both common carotid and left vertebral arteries (07/13/2015) showed occlusion of both internal carotid arteries and faint visualization of left terminal internal carotid artery and its bifurcation. The right internal carotid artery and its branches were not visualized. Left vertebral injection showed prominent left vertebral and basilar arteries and filling of both internal carotid arteries through posterior communicating arteries. A faint blush of contrast was noticed at the parasellar region coinciding with meningioma. The patient received three treatment sessions of gamma knife radiosurgery as follow: 20 cc of the tumor was treated with 12 Gy (15 August 2015), 1.7 cc was treated with 10 Gy (31 January 2016), and 2.5 cc was treated with 11 Gy (13 August 2016) which resulted in complete clinical recovery and tumor size reduction. Compensation from the posterior communicating and external carotid arteries might explain the complete clinical recovery after tumor size reduction with gamma knife radiosurgery.

Keywords

Infarction, internal carotid artery, cavernous sinus, parasellar meningioma, gamma knife radiosurgery

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Introduction

The parasellar region is defined as the area immediately surrounding the pituitary (sella turcica). It is anatomically complex as it has no specific landmark boundaries. Its arbitrary boundaries include the cavernous sinus (laterally), nasopharynx, and the medial parts of the temporal lobes. The boundaries may also involve embryonic remnants.[1] The cavernous sinus is bounded inferiorly by the basisphenoid and sphenoid sinus and superiorly by diaphragma sella, suprasellar subarachnoid spaces containing the optic nerves and chiasm, hypothalamus, tuber cinereum, and anterior third ventricle.[2] Parasellar lesions include pathologies which arise from the structures surrounding the sella turcica (neoplastic, inflammatory/infectious, developmental, and vascular lesions).[2] These structures include the meninges, visual pathways, other cranial nerves, major blood vessels, hypothalamus, pituitary gland, brain parenchyma, and skull base. The manifestations of parasellar pathologies are diverse depending on the location, size, and growth potentials of each lesion and its compression on or damage to the adjacent specific structures.[3] It has been observed that 9% of parasellar lesions are due to extension of primary pituitary adenoma (a sellar mass) to the parasellar region.[3] These manifestations may include headache, increase intracranial tension, visual loss, cranial nerve palsies, diencephalic syndrome, diabetes insipidus, hypopituitarism, and hyperprolactinaemia (if there is compression on the pituitary stalk).[3]

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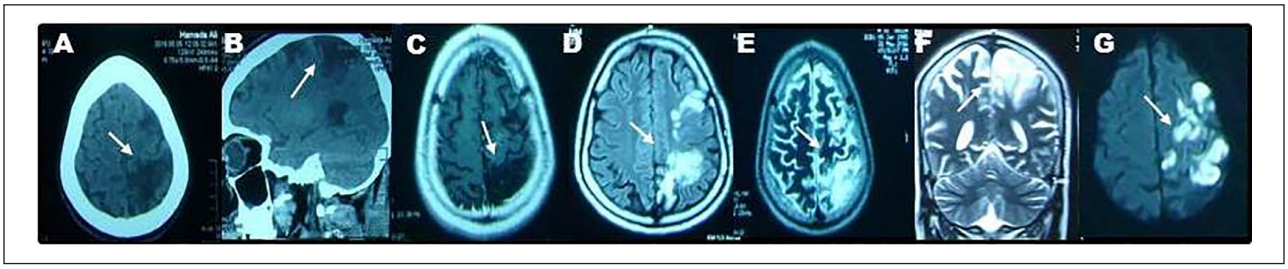

Figure 1. Axial and sagittal CT brain (A and B) scans showed left fronto-parieto-temporal hypodense lesion (ischemic infarction) (Date: 04/24/2015). MRI-brain showed left white matter lesion in the left fronto-parieto-temporal region (wedge shape) which was hypointense in axial T1W1 (C) with no detected enhancement in post contrast scans, hyperintense in axial FLAIR (D), axial and coronal T2WI (E and F) and DWI (G) scans (i.e. subacute ischemic infarction) (Date: 04/25/2015).

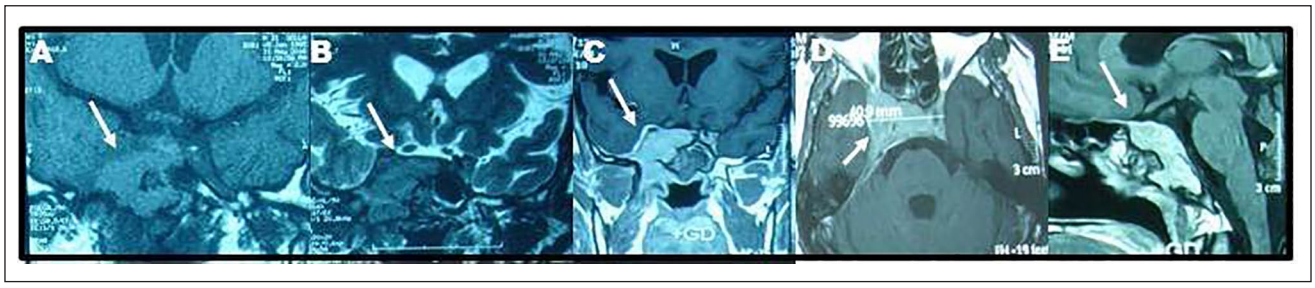

Figure 2. MRI-brain scans [coronal T1W1 (A) and T2WI (B) and coronal (C), axial (D) and sagittal (E) T1W1 with gadolinium enhancement (Gd)] showed a solid lesion which extended caudally into the right sphenoid sinus and greater wing of sphenoid bone and basisphenoid. The lesion also expanded the right cavernous sinus and attenuated the right ICA. It had a smaller left parasellar extension. It had no suprasellar extension and the pituitary stalk was kept central. The pituitary gland and optic chiasm were normal. Following Gd injection, it enhanced uniformally and strongly. The lesion measured about 4.1X4X3.5 cm in its maximum diameter. (Date: 04/25/2015).

Meningioma is the second commonest (~10%–15%) parasellar lesion after pituitary adenoma. It is the commonest tumor of the anterior and central skull base.[4,5] It is the commonest primary, extra-axial, non-glial intracranial space-occupying tumor, comprises 15% of primary brain tumors, and has an incidence rate of 3–13/100,000. It appears at any age with a peak incidence in adult women in their 40s.[6] Meningioma commonly locates in the parasagittal region and frequently originates in the suprasellar (diaphragma sella and tuberculum sella), frontobasal, temporobasal, sphenoid wing (medial lesser wing of sphenoid), anterior clinoid or petroclival regions, cavernous sinus wall, and optic nerve sheath.[3,7] Meningioma usually manifests by slow and progressive signs. The usual clinical presentations are visual disturbance and occasionally endocrine dysfunction with mild increase in blood prolactin concentrations. It has been observed that the malignant behavior of meningioma may occur with any grade.[3,7] Meningioma rarely occupies the sella mimicking a pituitary adenoma.[8] Cerebrovascular stroke is a rare presentation of parasellar space occupying lesion particularly meningioma of skull base due to compression on or encroaching the cavernous portion of internal carotid artery (ICA) or its branches.[4,9–11]

Description of the case

A 30-year-old right-handed man presented with acute weakness of the right upper limb (24 April 2015) which increased in severity and progressed within 2 days to involve the right lower limb. The patient denied presence of headache or seizures. Neurological examination showed right upper motor neuron facial muscles' weakness and weakness of the right hand of 2/5 Medical Research Counsil (MRC) scale, shoulder, toes, and ankle (3/5) and knee and hip muscles (4/5); diminished sensation on the right side of the body (hemihypoalgesia); diminished right tendon jerks (biceps, brachioradialis, triceps, quadriceps, and ankle); and right Babinski sign. He had normal other cranial nerves and fundus. His National Institute of Health Stroke scores (NIHSS)[12] was 12 and his modified Rankin Scale (mRS) grade was 4 (i.e. moderately severe disability: unable to do bodily needs or walk unassisted).[13] Computed tomography (CT) of the brain (24 April 2015) showed left fronto-temporo-parietal hypodense area consistent with ischemic infarction in the territory of the middle cerebral artery (MCA) (Figure 1). Magnetic resonance imaging (MRI) (1.5 T) (25 April 2015) also showed a right parasellar solid lesion extended caudally into the right sphenoid sinus and greater wing of sphenoid bone and basisphenoid and also expanded the cavernous sinus and attenuated the right ICA. It also had a smaller left parasellar extension. There was no suprasellar extension. The pituitary stalk was kept central. The optic chiasm was normal. This lesion enhanced uniformly and strongly following gadolinium injection. The lesion measured about 4.1X4X3.5 cm in its maximum diameter (Figure 2). Treatment with dexamethasone as

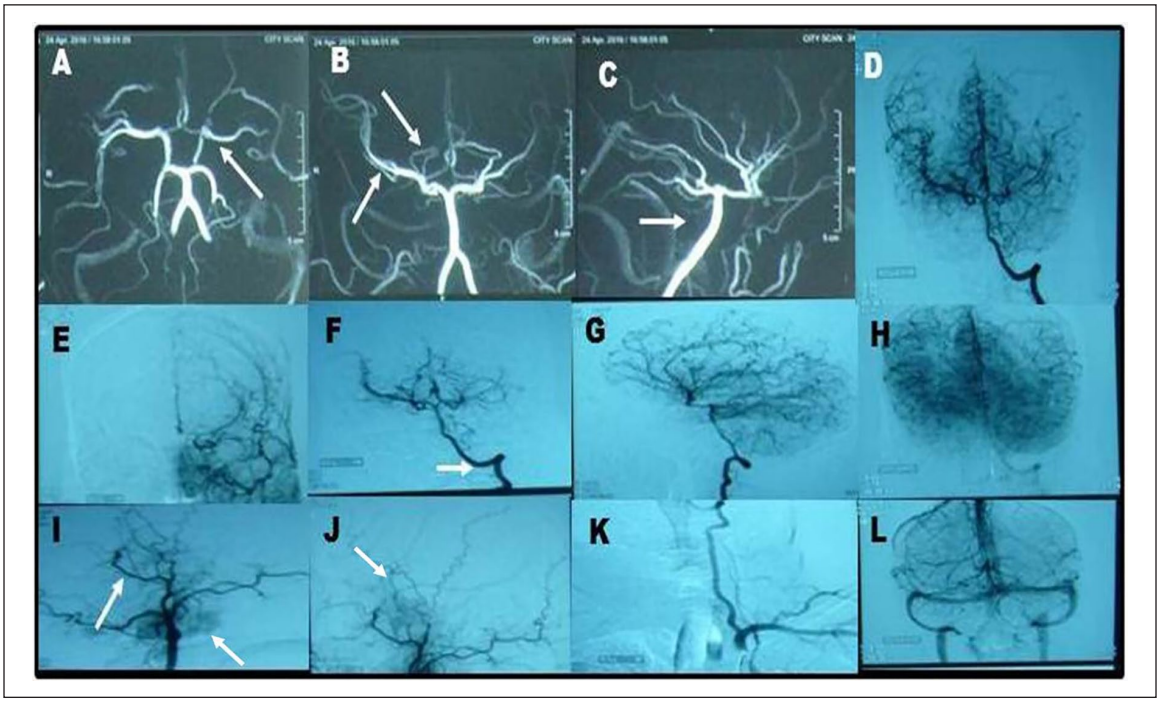

Figure 3. MRA showed absence of both ICAs (A and B). ACA and MCAs had normal courses and calibers and mostly fed via PCAs (C). The vertebral and basilar arteries had normal flow void signals, courses, calibers and enhancements (A, B and C) (Date: 04/25/2015). Digital subtraction angiography using selective catheterization of both CCAs and left vertebral arteries showed occlusion of both ICAs. There was faint visualization of the left terminal ICA and its bifurcation via faint collaterals from ophthalmic artery. The right ICA or its branches were not visualized (D-F). Left vertebral injection showed prominent left vertebral and basilar arteries (F-H) and filling of both ICAs through patent PCAs. Both ACA and MCA were opacified but there was slight and slower filling on the left side from ECA. ECAs, CCAs and subclavian arteries had normal caliber (I and J). A faint blush of contrast was noticed at the parasellar region coinciding with meningioma (H-K). Venous circulation was normal (L) (Date: 07/13/2015).

dehydrating agent for 2 weeks (8 mg intravenously twice daily) resulted in partial recovery of motor symptoms (mRS grade = 3, i.e. moderate disability or able to do bodily needs with some help and walk unassisted). Magnetic resonance angiography (MRA) showed absence of both ICAs. The anterior (ACA) and middle (MCA) cerebral arteries had normal courses and calibers and mostly fed via the posterior communicating arteries (PCAs). The vertebral and basilar arteries had normal flow void signal, courses, calibers, and enhancements (25 April 2015). Upon catheterization of both common carotid arteries (CCAs), occlusion of both ICAs was noted almost at their origins (13 July 2015). ACAs and MCAs had normal courses and calibers and mostly fed via the PCAs. There were faint visualization of the left terminal ICA and its bifurcation via faint collaterals (ophthalmic artery). The right ICA and its branches were not visualized. Left vertebral injection showed prominent left vertebral and basilar arteries as well as filling of both ICAs through patent both PCAs. Both ACAs and MCAs were opacified but there was slight and slower filling on the left side which might be due to partial filling from external carotid artery (ECA) or a relatively smaller left PCA. A faint blush of contrast was noticed in the parasellar region coinciding with meningioma in concern which apparently got its blood supply from the right internal maxillary artery. External carotid (ECAs), common carotid (CCAs), and subclavian arteries had normal calibers. The venous circulation was unremarkable (Figure 3). Hormonal profiles for thyroid (thyroid-stimulating hormone, T3 and T4), reproductive (follicle-stimulating hormone and luteinizing hormone), growth, cortisol, and prolactin hormones were unremarkable. Automated perimetry (central 24, 2, Sita-standard strategy) (30 May 2015) showed mild generalized depression of retinal sensitivity in both eyes and bilateral significant field defects. Transoesophogeal echocardiography and patient's coagulation profile were unremarkable. The patient underwent focused gamma knife radiosurgery (GKRS) using Leksell Model C or Perfexion (Elekta Inc.) (first session) (15 August 2015). The lower part of the tumor (20 cc) (which showed tumor's extension around the visual pathway) was treated with 12 Gy to the 50% isodose with 98% cover. Complete clinical recovery was observed within 3 weeks after the first GKRS session. In the second session, 1.7 cc was treated with 10 Gy to the 50% isodose with 93% cover (31 January 2016). In the third session, 2.5 cc was treated with 11 Gy to the 50% isodose with 94% cover (13 August 2016). On each session, the maximum dose to the visual pathway was 8 Gy which was considered safe. Radiological follow-ups of the patient were done every 6 months for 2 years after the third session (29 August 2018) and showed reduction of the tumor size (3.9×3×3 cm)

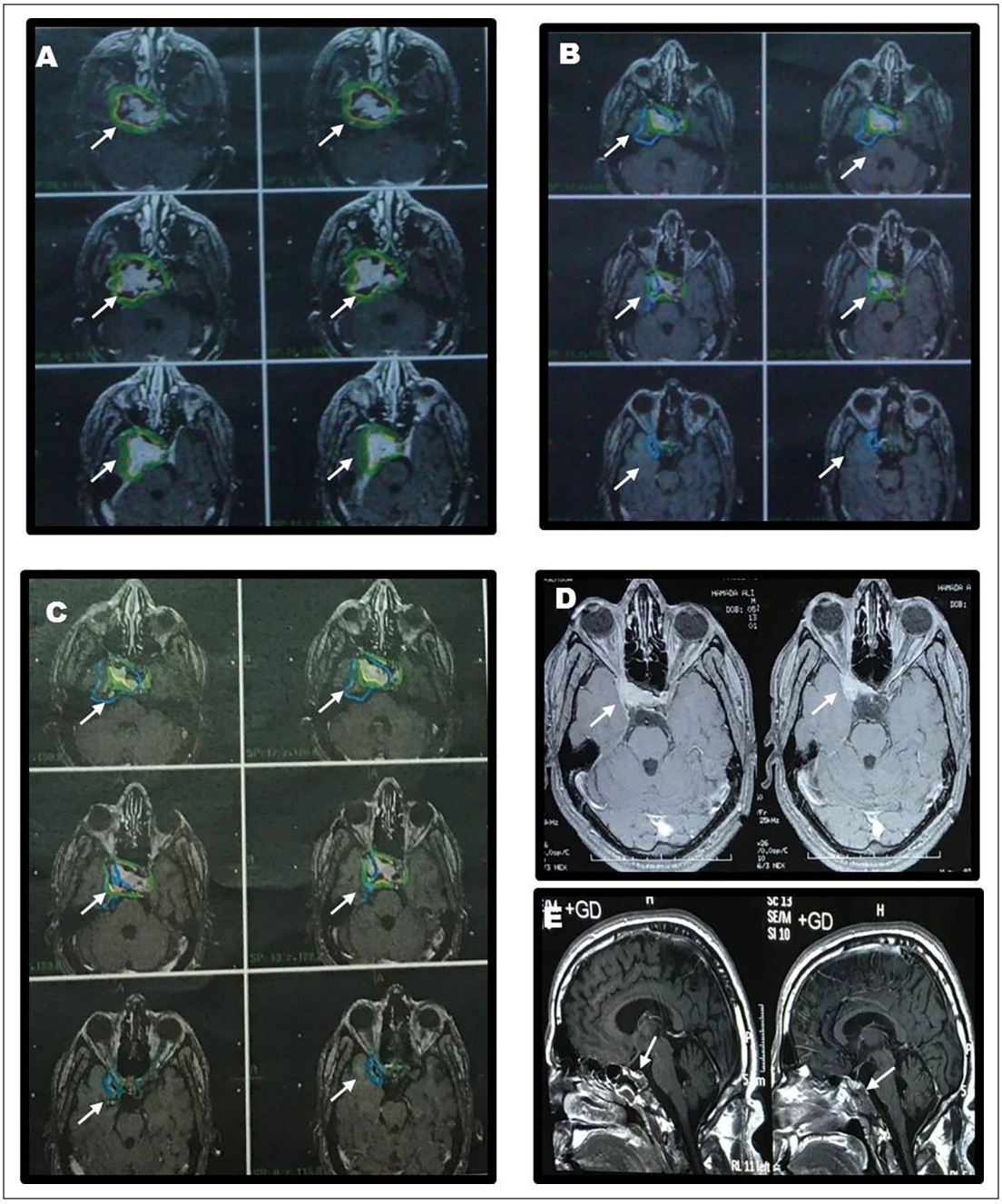

Figure 4. Follow-up MRI-brain scans showed significant reduction of the tumor size after GKRS (3.9X3X3 cm) [Dates: 08/13/2016 (B), 01/31/2017 (c), 05/15/2017 (D) and 01/29/2018 (E)] compared to his initial scan (4.1X4X3.5 cm) [Date: 08/15/2015 (A)].

(Figure 4) which coincided with his unremarkable neurological examination (grade 0 on mRS or no symptoms) (15 June 2019). This study was conducted according to the principles established in Helsinki. Informed written consent was obtained from the patient to publish the details of his clinical history, laboratory, and neuroimaging data.

Discussion

The differential diagnosis of parasellar tumors remains challenging even with the current advances in neuroimaging. A multidisciplinary approach (neurological, endocrinological, ophthalmological, neuroimaging, and histopathological) is necessary for proper diagnosis. Because of the location of the parasellar lesion in our patient, the diagnosis of meningioma was based on the neuroimaging findings, being isointense to the gray matter in T1WI and T2WI MRI-brain scans, and its homogeneous vivid enhancement.[2] Meningioma differs from pituitary adenoma by its differential uniform enhancing pattern and normal hormonal profile and from other parasellar tumors by its potential to encase the ICA, narrow its lumen, and compromise the cerebral blood blow. The occurrence of

Table 1. Case reports of patients presented with cerebrovascular strokes due to skull base meningioma.

Reference	Type of study	Clinical presentation	Neuroimaging findings	Cerebral angiography findings	Treatment modalities
Komotar et al.[9]	Case reports (n = 2) 1-A 49-year-old right-handed man	Left-sided hemiplegia, hemihypesthesia, and upper motor neuron facial muscle paralysis.	MRI-brain showed meningioma in the right cavernous sinus, encased the ICA, and resulted in ischemic infarction in the territory of the right MCA.	Occlusion of the right ICA with the meningioma, reconstitution of the flow in the supraclinoidal ICA from ECA via the ophthalmic artery, and cross filling to the right hemisphere via the anterior communicating artery.	Focused radiation therapy resulted in gradual improvement of patient's symptoms. Six months later, he developed worsening of the left leg weakness. MRI-brain showed acute right MCA and right watershed distribution infarction caused by a fresh thrombus in the right cavernous part of ICA. The patient was treated with anticoagulants. He had no further neurological events over the next 30 months.
	2-A 31- year-old right-handed man	Acute onset of complete painless visual loss in the right eye. The patient felt that he could compensate sufficiently and did not seek medical attention. Two years later, he developed sudden left-sided numbness, became collapsed, and was unable to sit up.	MRI-brain showed right parietal infarction due to olfactory groove meningioma which compressed the right optic nerve and encased the right ICA at the apex of the right orbit. The mass also compressed the right optic nerve.	Cerebral angiography showed occlusion of the right distal ICA. The majority of perfusion to the right hemisphere was due to cross filling from the left ICA.	The patient underwent complete tumor resection. Over the next several weeks, his neurological manifestations improved to great extent but he was left with right blindness and left arm numbness.
	Retrospective study. The authors reviewed the medical records of 1617 patients with meningioma in their institution (from year 1985 to year 2001). They identified 3 patients with involvement of the ICA by meningioma.	1-A patient with progressive left hemiparesis. 2-A patient with right upper extremity paresthesia. 3-A patient with left hemiparesis	1. Parasellar/medial sphenoid wing meningioma that narrowed the cavernous portion of the right ICA. 2. Petroclival meningioma which encased the left ICA. 3. Fonto-basal meningioma which involved the right ICA.	N/A	N/A

(Continued)

Table 1. (Continued)

Reference	Type of study	Clinical presentation	Neuroimaging findings	Cerebral angiography findings	Treatment modalities
Masuoka et al.[10]	Case report (n = 1) A 31-year-old man	Recurrent transient ischemic attacks followed by left lower limb weakness due to stroke in the territory of right ACA.	Meningioma in the planum sphenoidale and the right petrous apex which measured 20 mm and 25 mm in diameter and caused direct invasion of the right ACA.	Cerebral angiography showed occlusion of the right A2 portion after the branching of the frontopolar artery but no stenotic lesion of the right ICA. The right A1 portion was not opacified. Three-dimensional CT angiography showed that the left A1 coursed forward to the planum sphenoidale, and the left A2 ascended along the posterior margin of the tumor. The right A2 from the left A1 was encased and occluded by the tumor. The right A1 portion was invisible on three-dimensional constructive interference in steady state MRI-brain, suggesting right A1 aplasia.	The patient was initially treated for cerebral infarction and his neurological deficit gradually improved with rehabilitation. Three months after the stroke, he was referred for surgical treatment of the tumor. The tumor involved the right A2 portion, and the orbitofrontal and frontopolar arteries. The postoperative course was uneventful except for transient cerebrospinal fluid leakage, and the patient was discharged without new neurological deficits.
Lévêque et al.[5]	Case series of midline skull base meningioma	The authors reported that 32.7% of meningioma of the midline caused infiltration of ACA.	N/A	N/A	Surgical removal
Ossou-Nguiet et al.[11]	Case report (n = 1) A 52- year-old woman from Congo	A right hemiplegia (lower limb > upper limb) and grasp reflex	CT showed frontobasal meningioma associated with infarction in calloso-marginal territory of ACA.		Treatment with corticosteroid (prednisolone 1 mg/kg) and motor rehabilitation for 1 month were associated with partial clinical recovery. The patient was transferred to the neurosurgical department and then lost to follow-up.

MRI-brain: magnetic resonance imaging of the brain; CT: computed tomography; ICA: internal carotid artery; ECA: external carotid artery; ACA: anterior cerebral artery; MCA: middle cerebral artery; N/A: not applicable.

stroke due to meningioma has been rarely reported. Previous reports observed that skull base meningioma may compress the carotid artery and produce transient neurological symptoms including loss of consciousness, hemiparesis, paresthesias, and global amnesia.[14–19] Few case reports documented cerebral infarction due to compression of ICA by skull base meningioma (summarized in Table 1).

The rate of development of cerebral ischemia as a complication of meningioma is unknown. In an attempt to estimate the incidence of cerebral ischemia due to compression of the ICA by meningioma, Komotar et al.[9] retrospectively reviewed the medical records of 1617 patients with meningioma from their institution and identified three with meningiomal involvement of ICAs. The authors estimated that the incidence of meningioma-related cerebral ischemia by carotid artery compression was 0.19% (3/1617). Lévêque et al.[5] observed that 32.7% of midline meningioma caused infiltration to ACAs.

The mechanisms of cerebrovascular stroke associated with meningioma are either direct compression or vascular infiltration by the tumor, or indirectly by coagulation disorders or a combination of hypoperfusion and thromboembolization.[9,10,14,20,21] We suggest that the complete recovery of our patient was due to reconstitution of blood flow through the PCAs and efficient collaterals from ECAs. Some authors suggested that although meningioma commonly involves the ICA or even completely encases the ICA and its bifurcation into MCA and ACA, its presentation by cerebrovascular stroke is rare because (1) meningioma is a slowly growing and noninvasive tumor. It does not exert sufficient external force to significantly compress the high pressure arterial vasculature compared with venous vessels which can be easily compressed or injured; (2) The slow growth rate of meningioma allows the development of substantial collateral drainage;[22] (3) the thick muscular media segment of the ICA usually offers substantial resistance to vascular compromise.[23]

The optimal treatment of meningioma and other benign parasellar tumors is complete surgical removal by a craniotomic route if they are symptomatic or growing lesions. Endovascular intervention is sometimes used before surgery to reduce the size of bulky meningioma.[21] Transsphenoidally (TSS) debulking approaches are currently used to remove lesions involving the tuberculum sella, suprasellar region, cavernous sinus or clivus with minimal lesion to adjacent vital structures.[24] However, parasellar tumors usually have irregular margins and adhere to vital neurovascular structures and do not allow complete resection without the danger of affecting critical brain areas;[25] therefore, surgical debulking and radiotherapy are preferred for such cases and for those with known or suspected residual disease or recurrence after previous surgery.[24] Stereotactic fractionated radiosurgery, which delivers a single fraction of high-dose ionizing radiation on mapped targets, keeping the exposure of adjuvant tissues to a minimum, is preferred to treat unresectable and recurrent sellar and parasellar meningiomas after resection.[3] The mechanism of fractionated radiosurgery is to alter DNA of the tumor (i.e. arrest cell division) resulting in stable tumor size or its shrinkage due to reduction of angiogenesis. The selected dose of radiotherapy is based on an empirical algorithm that evaluated tumor volume, proximity to critical structures (such as the optic apparatus and pituitary gland), preexisting neurological deficits, and previous history of treatment with fractionated radiation therapy. Among published series of 100 or more patients, the rates of significant long-term complications of fractionated radiotherapy ranged from 0% to 16% in centers utilizing median doses of 12–15 Gy and 8 Gy or less has been found to be safe for the optic apparatus.[7] Some reported susceptibility to visual decline despite the 8 Gy dogma.[26,27]

For the presented patient, treatment with GKRS looks optimal, because resection of the tumor was considered excessively risky and the patient was unwilling to accept the risks of resection. It has been estimated that the risks of injury of adjacent vital structures and persistent neurological deficits after surgery of skull base meningioma is about 60%.[28] Although, the patient had complete clinical recovery for 3 years after the third GKRS session (15 June 2019) and reduction of the tumor size (Figure 4) and absence of visual side effect using the same doses as recommended in the literature, were noted,[26,27] we cannot guess the future prognosis as it needs follow-up for at least 5–10 years.

Many authors observed good outcomes with GKRS treatment for parasellar or sellar meningioma.[27–31]Lee et al.[27] followed-up 159 patients with cavernous sinus meningioma and treated with radiosurgery for a mean of 35 months (range 2–138 months). The authors observed improvement of neurological status in 29%, stabilization of manifestations in 62%, and worsening in 9%. Kondziolka et al.[29] followed-up 972 patients with meningioma in diverse locations and treated with GKRS (a median tumor margin dose of 13 Gy) at the University of Pittsburg. The authors reported tumor control rates in 93% at 5 years and 87% at both 10 and 15 years. These results confirmed an earlier study of 159 patients from the same institution in which tumor volumes decreased in 3%, remained stable in 60%, and increased in 6% of patients. Minniti et al.[30] in their review of 18 studies with 2919 skull base meningioma and treated with GKRS, observed that the 5-year tumor control rate was 91%. Seven of the studies (1626 skull base meningioma) reported an average of 10-year control rate of 87.6%. Sheehan et al.[31] in their multicenter study on 763 patients with sellar and/or parasellar meningioma and treated with GKRS (some of their patients did at least one resection before GKRS or more than one GKRS session), the authors did follow-up for patients for median of 66.7 months (range 6–216 months) and reported stabilization or reduction of tumor volumes in 90.2% and the progression-free survival rates were 3, 5, 8, and 10 years were 98%, 95%, 88%, and 82%, respectively. The authors observed that tumor margin dose of less than 13 Gy significantly increased the likelihood of tumor progression after GKRS. They also noted that in the last clinical follow-up, no change or improvement in neurological conditions in 86.2% of patients,

symptom progression in 13.8%, and new or worsening cranial nerve deficits in 9.6%. The long-term worsening has been attributed to the re-growth of the tumor and/or edema and necrosis as adverse effects from radiation. However, some authors reported a poor correlation between clinical and radiographic changes. For example, Bindal et al.[32] reported that 54.5% of patients with neurological progression did not have changes in neuroimaging, and 27.2% had simultaneous neurological and radiographic progression.

Conclusion

We describe an adult male with cerebral infarction as a result of ICA compression by a skull base meningioma encased the cavernous sinus. Angiography showed occlusion of both ICAs at their origins. Compensation from PCAs and partial filling from ECA may explain the complete recovery of neurological manifestations after GKRS. To our knowledge, massive parasellar meningioma with occlusion of both ICAs has not been reported before in the literature.

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Declaration of conflicting interests

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Ethical approval

Our institution does not require ethical approval for reporting individual cases or case series.

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Informed consent

Written informed consent was obtained from the patient(s) for their anonymized information to be published in this article.

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References

1. Smith JK. Parasellar tumors: suprasellar and cavernous sinuses. *Top Magn Reson Imaging* 2005; 16(4): 307–315.
2. Rennert J and Doerfler A. Imaging of sellar and parasellar lesions. *Clin Neurol Neurosurg* 2007; 109(2): 111–124.
3. Freda PU, Wardlaw SL and Post KD. Unusual causes of sellar/parasellar masses in a large transsphenoidal surgical series. *J Clin Endocrinol Metab* 1996; 81(10): 3455–3459.
4. Ishikawa M, Nishi S, Aoki T, et al. Predictability of internal carotid artery (ICA) dissectability in cases showing ICA involvement in parasellar meningioma. *J Clin Neurosci* 2001; 8(Suppl. 1): 22–25
5. Lévêque S, Derrey S, Martinaud O, et al. Superior interhemispheric approach for midline meningioma from the anterior cranial base. *Neurochirurgie* 2011; 57(3): 105–113.
6. van Alkemade H, de Leau M, Dieleman EM, et al. Impaired survival and long-term neurological problems in benign meningioma. *Neuro Oncol* 2012; 14(5): 658–666.
7. Ko KW, Nam DH, Kong DS, et al. Relationship between malignant subtypes of meningioma and clinical outcome. *J Clin Neurosci* 2007; 14(8): 747–753.
8. FitzPatrick M, Tartaglino LM, Hollander MD, et al. Imaging of sellar and parasellar pathology. *Radiol Clin North Am* 1999; 37(1): 101–121, x.
9. Komotar RJ, Keswani SC and Wityk RJ. Meningioma presenting as stroke: report of two cases and estimation of incidence. *J Neurol Neurosurg Psychiatry* 2003; 74(1): 136–137.
10. Masuoka J, Yoshioka F, Ohgushi H, et al. Meningioma manifesting as cerebral infarction. *Neurol Med Chir* 2010; 50(7): 585–587.
11. Ossou-Nguiet PM, Gnonlonfoun DD, Obondzo-Aloba K, et al. Anterior cerebral infarction by fronto-basal meningioma. *Neurosci Med* 2013; 4: 277–279.
12. Lyden P, Raman R, Liu I, et al. National institutes of health stroke scale certification is reliable across multiple venues. *Stroke* 2009; 40(7): 2507–2511.
13. Wilson JT, Hareendran A, Grant M, et al. Improving the assessment of outcomes in stroke: use of a structured interview to assign grades on the modified Rankin Scale. *Stroke* 2000; 33: 2243–2246.
14. Launay M, Fredy D, Merland JJ, et al. Narrowing and occlusion of arteries by intracranial tumors. *Neuroradiology* 1977; 14(3): 117–126.
15. Kaur U, Chopra JS, Kak VK, et al. Meningioma presenting as recurrent transient cerebral ischemia and intracranial hemorrhage. *Surg Neurol* 1982; 17(2): 120–122.
16. Davidovitch S and Gadoth N. Neurological deficit-simulating transient ischemic attacks due to intracranial meningioma. Report of 3 cases. *Eur Neurol* 1988; 28(1): 24–26.
17. Araga S, Fukada M, Kagimoto H, et al. Transient global amnesia and falcotentorial meningioma—a case report. *Jpn J Psychiatry Neurol* 1989; 43(2): 201–203.
18. Cameron EW. Transient ischaemic attacks due to meningioma—report of 4 cases. *Clin Radiol* 1994; 49(6): 416–418.
19. Fazi S and Barthelemy M. Petroclival meningioma mimicking the presentation of a transient ischemic attack. *Acta Neurol Scand* 1994; 89(1): 75–76.
20. Kreisl TN, Toothaker T, Karimi S, et al. Ischemic stroke in patients with primary brain tumors. *Neurology* 2008; 70(24): 2314–2320.
21. Fernandez-Valverde F, Jimenez-Gomez E, Roldan-Romero E, et al. Presurgical endovascular treatment of symptomatic carotid stenosis caused by a meningioma of the planum sphenoidale. *Rev Neurol* 2013; 56(3): 157–160.
22. Kiya K, Satoh H, Mizoue T, et al. Postoperative cortical venous infarction in tumours firmly adherent to the cortex. *J Clin Neurosci* 2001; 8(Suppl. 1): 109–113.
23. Halbach VV, Higashida RT, Hieshima GB, et al. Venography and venous pressure monitoring in dural sinus meningiomas. *AJNR Am J Neuroradiol* 1989; 10(6): 1209–1213.
24. Marosi C, Hassler M, Roessler K, et al. Meningioma. *Crit Rev Oncol Hematol* 2008; 67(2): 153–171.
25. Torres RC, Frighetto L, De Salles AA, et al. Radiosurgery and stereotactic radiotherapy for intracranial meningiomas. *Neurosurg Focus* 2003; 14(5): e5.

26. Eustacchio S, Trummer M, Fuchs I, et al. Preservation of cranial nerve function following gamma knife radiosurgery for benign skull base meningiomas: experience in 121 patients with follow-up of 5 to 9.8 years. *Acta Neurochir Suppl* 2002; 84: 71–76.
27. Lee JY, Niranjan A, McInerney J, et al. Stereotactic radiosurgery providing long term tumor control of cavernous sinus meningiomas. *J Neurosurg* 2002; 97(1): 65–72.
28. Sughrue ME, Rutkowski MJ, Aranda D, et al. Treatment decision making based on the published natural history and growth rate of small meningiomas. *J Neurosurg* 2010; 113(5): 1036–1042.
29. Kondziolka D, Mathieu D, Lunsford LD, et al. Radiosurgery as definitive management of intracranial meningiomas. *Neurosurgery* 2008; 62(1): 53–58; discussion 58–60.
30. Minniti G, Amichetti M and Enrici RM. Radiotherapy and radiosurgery for benign skull base meningiomas. *Radiat Oncol* 2009; 4: 42.
31. Sheehan JP, Starke RM, Kano H, et al. Gamma knife radiosurgery for sellar and parasellar meningiomas: a multicenter study. *J Neurosurg* 2014; 120(6): 1268–1277.
32. Bindal R, Goodman JM, Kawasaki A, et al. The natural history of untreated skull base meningiomas. *Surg Neurol* 2003; 59(2): 87–92; discussion 92.