Multimodality Management of Cavernous Sinus Hemangiomas—An Institutional Experience

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J Neurol Surg B 2017;78:399–407.

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Abstract

Objective Cavernous sinus hemangiomas (CSHs) are benign lesions accounting for less than 2% of the cavernous sinus tumors. They provide a formidable surgical challenge because of their vascularity and their being surrounded by critical neurovascular structures. In this study, one of the largest in available literature, we analyze our experience in the management of these unusual tumors and review the available literature.

Materials This is a retrospective analysis of patients who were managed surgically (both microsurgical and Gamma knife radiosurgery [GKRS]) for CSH at our Institution from 2007 to 2015. Complete demographic, clinical-radiologic surgical records were analyzed. Follow-up data were collected from the hospital records.

Results Total 23 patients were managed. Among these, 15 patients underwent microsurgery (group 1) whereas 8 underwent GKRS (group 2). Predominant clinical presentation in both the groups included headache and involvement of multiple cranial nerves. Five patients in group 1 had deteriorating vision. The volume of tumors ranged from 29 to 115 cm³ (mean = 64.57 cm³) in group 1 and from 2.1 to 11.6 cm³ in group 2. GKRS was performed with a mean dose of 13 Gy, an average isodose line of 50% with an average coverage of 96%. In group 1, the follow-up period ranged from 6 to 62 months (mean = 29.4 months). The extraocular movement (EOM) preservation rate in our series was not favorable, as most patients presented late with large tumors and established deficits. Recurrence/residual tumor was seen in two cases. In group 2, the follow-up was 5 to 48 months. All of them showed significant reduction in size.

Conclusion Both surgery and radiosurgery are highly effective in the management of CSHs. They are complementary to each other, with individual characteristics—the size and volume of the lesion—being the main factors in deciding the choice of treatment.

Keywords

- hemangiomas
- cavernous sinus
- Gamma knife radiosurgery
- interdural approach

Introduction

Cavernous sinus hemangiomas (CSH), arising within the confines of cavernous sinus (CS), are very unusual tumors.^{1,2} They do not breach the dural confines of the CS and can attain large sizes without symptoms. In published literature, there

have been several short series describing the management of CSH,^{3–6} but despite the advances in surgical techniques, they continue to pose a formidable challenge to the operating surgeon, mainly due to the surrounding critical neurovascular structures and high vascularity. Current treatment modalities include microsurgical resection, fractionated

received
April 20, 2016
accepted after revision
March 24, 2017
published online
June 5, 2017

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Stuttgart · New York

DOI <https://doi.org/10.1055/s-0037-1602790>.
ISSN 2193-6331.

radiation therapy, and stereotactic radiosurgery (SRS)⁷. Total resection provides a cure but may be at the cost of profuse intraoperative bleeding and new cranial nerve (CN) deficits.^{2,8} There have been successful reports of Gamma knife radiosurgery (GKRS) in these tumors.^{9,10} In this article, we analyze our experience in the management of these unusual tumors and review the available literature. We have analyzed our experience with both microsurgery and GKRS.

Materials and Methods

This retrospective study included all the cases of CSH who underwent treatment (both microsurgery and GKRS) at our tertiary level referral center during 2007–2015. The clinical data were analyzed retrospectively. The demographic profile, clinical features, radiologic findings, management strategies, postoperative complications, length of hospitalization, and outcome (both immediate and long term) were noted. The pathology of the tumors was reconfirmed via reexamination of the histologic slides. The patient data were divided into two groups: group 1 consisted of patients who underwent microsurgery whereas group 2 consisted of patients who underwent GKRS.

Results

Group A

There were a total of 15 patients (7 males) with a median age of 34 years (range: 17–58 years). The duration of the symptoms varied from 6 to 62 months. Severe headache and involvement of the CNs passing through the CS were the most common presenting symptoms. Five cases presented with diminution of vision, one with hemiparesis, and one with pituitary dysfunction. Detailed patient characteristics have been outlined in ►Table 1.

All the patients underwent preoperative computed tomography (CT) and magnetic resonance imaging (MRI) scans. (►Fig. 1A–D) The tumor volume was 29 to 115 cm³ (median = 64.57 cm³). CT images showed fairly well-defined parasellar lesions, mild to moderately hyperdense in plain scans, with intense homogenous enhancement. On MRI, CSH were well-delineated parasellar lesions, hypo- to isointense in T1-weighted images and brightly hyperintense in T2-weighted images. Gradient-echo sequences were used to identify any hemorrhagic component, which was seen in two of the cases. Most lesions showed intense homogenous enhancement. In one case in which the enhancement was poor on immediate imaging, delayed imaging showed intense enhancement. This finding is also characteristic of CSH. The characteristic imaging finding following contrast administration was a nipple-like projection of tumor toward sella, which was seen in all cases. In all but five cases, internal carotid artery (ICA) was seen encased within the tumor mass without affecting the vessel diameter. Digital subtraction angiography (DSA) was performed in three cases. The characteristic DSA finding was lack of tumor blush in capillary phase and pooling of contrast in late venous phase, seen in all cases. A prominent meningo-hypophyseal

(inferolateral trunk) branch of cavernous ICA was seen in nine cases.

All patients underwent a frontotemporal craniotomy along with an orbitozygotomy (FTOZ) and tumor excision. The sphenoid ridge was drilled, the superior orbital fissure opened, and the anterior clinoid process (ACP) removed extradurally. An extradural temporopolar approach was used in 13 patients whereas a combined extradural-intradural (ED + ID) was performed in 2 patients. The middle meningeal artery was identified, coagulated and cut at the foramen spinosum. The V2 and V3 branches were exposed, and then an interdural approach to the CS was used. This involved peeling away the outer membranous dural layer from the inner membranous layer using a combination of sharp and blunt dissection. This leads to the lateral wall of CS where the nerves (III, IV, and V1) are exposed. The tumor bulge was clearly made out and the tumor could be accessed between these nerves. As these tumors were highly vascular, they were decompressed rapidly using a Cavitron ultrasonic suction aspirator (CUSA). Feeders from the meningo-hypophyseal trunk of the intracavernous ICA were exposed and coagulated early in the course of the surgery (►Fig. 2A, B). Care was taken to recognize and prevent injury to the ICA and the VI CN. Once the tumor was removed, including the part medial to the ICA, hemostasis was achieved relatively easily using degraded cellulose (Surgicel, Johnson & Johnson Medical Ltd., New Yorkshire, United Kingdom). The keys to good hemostasis were gross total excision and devascularization at an early stage. In one of our cases, blood loss was profuse the moment tumor decompression was started. In this case, the procedure was abandoned, and DSA was done to look for tumor blush and rule out any associated aneurysm. Following this, gross total resection (GTR) was performed at reexploration.

GTR or near-total resection (NTR) (i.e., > 95% of tumor removal) was attained in 13 cases. Histopathology in all cases was confirmatory of cavernous hemangiomas (►Fig. 3A, B). Though there was no mortality, morbidity was encountered in 12 cases. These included worsening of extraocular movement (EOM) in 11 cases, hemiparesis in 1 case, and wound infection in 1 case. In the patient with hemiparesis, there was a postoperative hematoma with brainstem compression. However, as the patient was well preserved, it was managed conservatively and the patient gradually and completely improved at follow-up. Among the 15 patients, 5 developed complete ophthalmoplegia. Among these, two showed partial improvement whereas three remained without any improvement. Five patients had transient and partial ophthalmoplegia and all improved at follow-up. Fourteen patients were available for a detailed follow-up. The follow-up duration varied from 6 to 62 months (mean = 29.4 months). Postoperative MRI was obtained at 3 to 6 months after surgery and then at 6-month intervals. Two cases showed recurrence/residual lesion. In one of them, GKRS was administered, and in the other, in view of the large remnant, resurgery has been advised. In the others, there has been no recurrence or significant increase in size of residual lesions.

Table 1 Demographics and clinical characteristics of patients in our series

Patient	Age/sex	side	Symptoms	Clinical signs/structures involved	Complications	Follow-up status	Follow-up duration	Volume	Extent of resection	Blood loss (mL)
1	20/M	Right	Diplopia and redness 4 mo	B/L 6/6, VI, V-1	None	EOM full	6 mo	45	GTR	150
2	58/F	Left	Headache 4 mo, ptosis 4 mo	R-6/24, L-6/9, III, VI	None	Same	6 mo	34	NTR	350
3	22/F	Left	Left ptosis and facial hypoesthesia 2 y	L-partial III, V1V2 50% loss	Complete ophthalmoplegia	Same	1 y	102	NTR	450
4	45/M	Right	Headache 3 mo right UL LL weakness 3 mo	Right UMN VII	Complete ophthalmoplegia	Same	14 mo	96	STR	450
5	22/M	Right	Right ptosis 3 mo, headache 6 mo, blurring 3 mo	Right inferior medial field cut, right III, V1V2 25% loss	Transient ophthalmoplegia	EOM full	20 mo	32	NTR	400
6	29/M	Left	Ptosis, proptosis left facial hypoesthesia and difficulty in chewing 6 mo	L-III, IV, V1V2 with motor, VI, B/L 6/24	Complete ophthalmoplegia	Partially improved	30 mo	29	GTR	650
7	21/F	Right	Right blind, diplopia- 1 y, facial pain 1 mo	R PL-, III, V2 10–15% loss	Transient ophthalmoplegia	EOM full	36 mo	88	NTR	350
8	28/F	Left	Ptosis, facial numbness 2 y	B/L 6/6, L: III, IV, VI	Status quo	Same	46 mo	78	GTR	550
9	32/F	Left	Ptosis, amenorrhea, blurring 6 mo	L 6/36, R 6/9, temporal hemianopia, III	Transient ophthalmoplegia	EOM full	54 mo	72	STR	450
10	17/F	Right	Headache 4 mo, amenorrhea and galactorrhea 5 mo	R PL-, L 3/60, L temporal cut	Transient ophthalmoplegia	EOM full, blind on right	62 mo	92	NTR	1,500
11	33/M	Right	Headache 4 mo, partial ptosis 2 mo	B/L 6/6, L III	Transient ophthalmoplegia	EOM full	54 mo	54	GTR	550
12	50/F	Left	Headache 5 mo, partial ptosis 4 mo	B/L 6/6, L III, IV, VI	Status quo	Partially better	36 mo	115	GTR	650
13	48/M	Right	Headache 4 mo, ptosis with diplopia 3 mo	R III, IV	Complete ophthalmoplegia	Partially better	30 mo	28	GTR	550
14	55/M	Left	Headache, facial numbness, partial ptosis 7 m	BR 6/36, III, IV, V1V2 25%	Complete ophthalmoplegia	Same	6 mo	39	GTR	600
15	38/M	Left	Hyponatremia, headache, diplopia 2 wk	Vision 6/6, Left III, IV, VI paresis	Partial ophthalmoplegia	Same	1 mo	78	NTR	1,500

Abbreviations: EOM, extraocular movement; F, female; GTR, gross total resection; M, male; NTR, near-total resection.

Fig. 1 (A) T1 weighted MRI axial images showing an intracavernous lesion. (B) T2-weighted MRI axial lesions, which is hyperintense. (C) T1-weighted (postcontrast) MRI axial images showing brilliant enhancement on contrast. (D) Postoperative MRI (6 months) showing complete excision of lesion.

Group B

GKRS was administered as in eight patients (six primary and two residual tumors). It was performed using the Model 4C machine (Elekta, Sweden). The Leksell Stereotactic frame (Elekta, Sweden) was fixed on all patients. An MRI of the brain was performed (1-mm sequences, axial T1-weighted images pre- and postgadolinium contrast). Treatment was planned with an aim of maximal coverage, maximal conformity, and protection of optic pathways (< 8 Gy). The mean tumor volume was 5.09 cm^3 . The radiation dose administered varied from 12 to 15 Gy with a mean of 13.25 Gy and an average isodose of 50%. The tumor coverage ranged from 92 to 98% (mean: 95%). There was no complication related to GKRS. The mean follow-up duration was 22.62 months. The tumor size decreased in seven patients and remained constant in one patient (—Fig. 4A, B). Though preoperative ocular dysmotility was present in all eight patients, it improved only in three patients. CN deficits improved in three cases. The mean percentage reduction in volume was 75% (—Table 2).

Discussion

Incidence

CSHs are, in fact, hamartomas or malformations of the micro-circulation, rather than true vascular neoplasms.^{11,12} When they grow and start to compress neighboring structures, they behave like real tumors.¹³ CSH are usually diagnosed in middle-aged patients and women.^{2,4,9} Though estrogen/progesterone has been postulated to the pathogenesis of these tumors, the exact etiology remains unclear.⁷ In our series too, 62.5% were women. The mean age in our series was 34.28 years, with a range of 17 to 58 years.

Clinical Presentation

Clinical presentations are usually in the form of acute and subacute dysfunction of the CNs traversing the CS and the optic nerve. The onset is usually insidious and gradual. In contrast to intracerebral hemangiomas, CSHs do not have the propensity to bleed.^{2,14} In our series, two cases had evidence of asymptomatic bleed on radiology. In large tumors, visual deficits and field defects are encountered. In our series, most

Fig. 2 (A) Intraoperative photomicrograph showing the tumor after exposure. Note the cranial nerves splayed on the surface of the tumor (black arrow). (B) Intraoperative photomicrograph showing the tumor after excision. Note the internal carotid artery.

Fig. 3 Histopathology (A) H&E staining showing vascular channels lined by single endothelial layer. (B) Masson trichrome stain showing dense collagen in between thin vascular channels.

Fig. 4 (A) T1-weighted (postcontrast) MRI axial images with Leksell frame fixed (pre-GKRS). (B) MRI (postcontrast T1-weighted images) showing significant reduction in size (6 months post-GKRS).

Table 2 Details of patients who underwent GKRS

Patient	Age/sex	Clinical presentation	Volume (cm ³)	Dose (Gy)	Isodose line (%)	% coverage	Outcome	Follow-up (mo)	Volume at last follow-up (cm ³)
1	27/F	VI nerve only	2.85	14	50	98	Good (same)	8	No change
2	44/F	Headache, III, IV, VI	8.7	12 y	52	92	Good (decreased)	30	1.1
3	38/F	VI nerve only	2.1	12	50	97	Good (same)	32	No change
4	22/M	Headache, III, IV, VI	7.3	12	50	95	Good (decreased)	28	1.3
5	43/F	Partial ptosis	3.2	15	50	92	Good (decreased)	36	0.6
6	40/F	III, VI nerve palsy	4.8	13	52	95	Good (same)	13	1.8
7	26/M	Partial ptosis, VI nerve	5.6	15	51	97	Good (same)	18	0.6
8	28/M	Partial ptosis, VI nerve	6.2	13	50	96	Good (same)	16	NA

Abbreviations: F, female; GKRS, Gamma knife radiosurgery; M, male; NA, not available.

patients in group 1 had a delayed presentation and significant CN deficits, as already described. Though seizures, facial numbness, and neuralgia have been reported,^{2,6,15–17}, we did not encounter them. Pituitary dysfunction is a rare finding reported in the literature^{2,8} and was seen in one of our cases. Another unusual finding in our series was pyramidal weakness in one patient.

Radiology

CSH is the only lesion that is purely intracavernous; thus, even giant lesions remain within the dural confines of the CS. Large CSHs usually extend toward the sella, superior orbital fissure, and Meckel’s cave—this was observed in all our cases. The most probable mechanism of growth is considered to be progressive ectasia of the blood vessels or their autonomous development at the edges of the lesion. These lesions appear as an iso/hyperdense mass on nonenhanced CT scans, enhancing intensely with contrast in most cases. Erosion of the sphenoid bone can also be seen occasionally. DSA can be normal and show a relatively avascular mass or a discrete-to-moderate tumoral blush, with feeding vessels originating from branches of the external carotid or cavernous internal carotid.^{14,18,19} The characteristic DSA finding is lack of tumor blush in arterial and capillary phase and pooling of contrast in the late venous phase, which was seen in all our cases. The ICA is often encircled by the lesion in its cavernous portion, without affecting the diameter. There are reports of successful embolization prior to surgery.^{2,8} In our series, though a prominent inferolateral trunk was seen, as there was no tumor blush, embolization was not attempted. Unlike cerebral cavernous angiomas and their characteristic popcorn appearance, their CS counterparts do not have a pathognomonic appearance on MRI. However, they are well-delimited parasellar lesions, hypo- or isointense in T1-weighted images, and brightly hyperintense in T2-weighted images. A dumbbell-shaped mass can sometimes be seen, with a small suprasellar component and a large CS component.^{14,18,19} Instead of the dumbbell mass, a nipple-like projection toward the sella was seen in all our cases with large tumors. Enhancement postcontrast is intense and

homogeneous. Though the progressive contrast “filling in” in CS lesions on conventional contrast-enhanced sequences is not specific, it is suggestive of the diagnosis of CSH and was seen in one of our cases.

The most important differential diagnosis (and the most common preoperative misdiagnosis) is a parasellar meningioma. Although differentiation between these two conditions could be difficult, a homogeneously enhancing mass enwrapping the ICA without significant reduction in its caliber should lead diagnosis toward a CSH. Other possible differential diagnosis in this context is parasellar schwannoma.^{14,18,19}

Surgical Management

Because the onset is insidious, these lesions usually attain a large size before the symptoms start. Therefore, surgical excision is the most favored modality for management, considering the benign nature of the pathology and potential curability. Linskey and Sekhar reported the use of a combined intra- and extradural approach to treat three patients with CSH, and they performed total excision with minimal blood loss and preservation of CN function in two patients.² They further hypothesized that the tumor arises within the CS and, when small, it takes its blood supply from the intracavernous carotid artery. Goel reported 13 patients with CSH, 7 of whom were operated on using Dolenc technique of an entirely extradural approach.⁶ Of these, 12 patients had a complete tumor resection. The CN outcome in their series was poor, perhaps because of the large size of the tumors at diagnosis. Shi et al retrospectively analyzed 10 patients with CSH.²⁰ Among the 10 patients, total tumor removal was performed in 4 patients, partial removal in 2 patients, and 4 patients had tumor biopsies. Both patients who underwent partial removal developed complete ophthalmoplegia and diminished sensation in the distribution of CN VI after surgery; one patient developed contralateral paralysis. Zhou et al published 20 surgically treated cases of CSH, 13 of which were treated using the extradural approach.¹⁷ Complete tumor removal was achieved in 12 patients, and at a mean follow-up of 3 years, all patients in this group improved without tumor recurrence.

Bansal et al¹⁵ shared their experience of treating 22 patients with CSH over a period of 14 years (1999–2012), 13 of whom were treated using a purely extradural trans-cavernous surgical approach, whereas 9 were given primary GKRS. Gross total excision was achieved in all but one patient. Transient CN dysfunction was present for 2 to 3 months in all but three patients. One patient developed permanent VI nerve palsy. Vision improved in all patients with preoperative visual diminution but not in those with preoperative complete vision loss. They further opined that because CN VI is the only CN truly within the CS, it will most often run through the substance of the tumor; CNs III, IV, and V (V1, V2, and V3) will always be found stretched over the surface of the tumor in the overlying dura. Because of the presence of the pseudo capsule, there will always be a potential plane between the tumor and the overlying dura and CNs. This can be utilized to preserve CNs III, IV, and V (V1, V2) when removing a hemangioma rather than when removing a CS meningioma that arises from the dura that invests these nerves.

We opine that the key to preservation of CNs is opening the superior orbital fissure first and tracing the nerves posteriorly toward the CS. We recommend avoiding disturbing CN III unnecessarily, as it is very sensitive. The space between CN IV and V1 is sufficient to detach the antero-medial aspect of the tumor. Furthermore, as CN VI lies within the substance of the tumor, it is best preserved during the initial part of the surgery. With careful preservation of the nerves, deficits are usually temporary and will improve during follow-up, unless CN palsy was present prior to treatment. Sometimes tumor in the anteromedial part may be densely adhered to the ICA and CN III and may not be removed easily. In this circumstance, subtotal or partial resection is the wise choice, followed by GKRS. In our series, CN deficits were higher, as most of the cases presented late, thereby attaining large sizes with established deficits. Janjua et al¹² reported a separate layer, which they called the intermediate layer, between the outer meningeal layer and inner membranous layer in the lateral wall of the CS in fresh cadaver heads. The meningeal dura and intermediate layer adhered closely to each other at the SOF, making it easier to separate these two layers from the membranous layer. During surgery, we did not encounter any difficulty in stripping the two layers of the dura. In our experience, the key to CSH removal are good exposure through a wide craniotomy with de-roofing of the orbital roof to prevent unnecessary traction, stripping of the two layers of the dura to preserve CNs, early identification of the VI nerve to maintain full range of EOM, avoidance of unnecessary dissection of the III nerve, piecemeal removal of the tumor instead of en block excision, and early devascularization of the tumor.

The results of CN dysfunction were higher in our series, as the cases were referred late. In fact, as per available literature, the mean volume of cases in our series was the largest (64.57 cm³). However, all the patients are stable and are relieved of disabling symptoms of headache and V nerve involvement.

Radisurgery

Though surgical resection is the treatment of choice, especially in large tumors, GKRS is a viable alternative for partially resected and small tumors located away from the optic pathways. Historically, there have been reports of using fractionated radiation previously, for unresectable or residual cranial base hemangiomas.²¹ Shibata and Mori published three cases of CSH in which radiation was used successfully to alleviate symptoms or facilitate surgical removal of middle fossa cavernous hemangiomas. In 1999, Iwai et al reported the use of GKRS for the first time in CSH.⁷ Following this, there have been several reports of the successful use of GKRS in CSH with significant reduction in volume.^{9,16,22} The proposed mechanism is radiation-induced endothelial proliferation, vessel wall hyalinization, and subsequent vessel obliteration in addition to direct cytotoxicity to the tumor cells. Nakamura et al proposed that GKRS should be used as primary therapy in small tumors without risking damage to the optic pathways.²³ In large tumors and those close to the visual pathway, they suggested surgical debulking followed by GKRS for the residual portion.¹⁰ Chou et al used GKRS as both primary and secondary treatment modalities for CSH. The largest size of tumor in which GKRS was used in his series was 23.1 cm³ and average reduction in volume was 82%. The largest series of CSH treated by GKRS was published by Yamamoto et al.²¹ It was a multi-institutional study that included 30 cases, and GKRS was used both as primary and secondary therapy. The mean volume of tumor was 11.5 cm³, mean dose used 13.8 Gy, and mean follow-up duration 53 months. They analyzed the dose treatment responses and found that tumor shrinkage is directly proportional to the radiation dose received. They further concluded that a peripheral dose of 14 to 15 Gy is sufficient to control the growth of CSH and that a dose of 10 to 12 Gy is the threshold level for tumor growth control. Yamamoto used staged GKRS in two large cases of CSH and put forward the concept of radiosurgical thrombolisation, further vindicating the hypothesis of Lindsay and Sekhar described previously.²¹ Though some of these studies advocate using GKRS as a primary modality even in large tumors and achieved good tumor control, we do not follow this policy. We use GKRS as primary therapy only when volume is amenable and the dose to the optic apparatus does not exceed the safety limits of radiation. In our series, there has been no complication related to GKRS. CN involvement improved in two of our cases and the rest showed no new deficits. The mean decrease in volume in our study was 75%, which was comparable to available literature. This reduction in volume is perhaps unique, as no other tumor treated with GKRS demonstrates a consistent and significant reduction in size.

To the best of our knowledge, along with Bansal et al, ours is the only other study available in literature that studied the results of both surgery and Gamma knife for CSH (– Table 3). We feel that CSH need a tailored approach for proper management, and decisions regarding treatment modality must be individualized. Surgery and radisurgery are complementary to each other and the choice should be

Table 3 Summary of reviewed reports of CSH

Study	No. of cases	Tumor volume reduction	Ocular motility disorder (Pre-operative)	Posttreatment ocular motility disorder (long-term follow-up)
<i>Radiosurgery</i>		> 50%		
Thompson et al	3	2	3	3 (3 improved)
Kida et al	3	3	3	3 (1 improved, 2 stable)
Nakamura et al	3	1	1	0 (1 resolved)
Peker et al	5	5	4	4 (2 improved, 2 stable)
Yamamoto et al	30	18	20	18 (2 resolved, 11 improved, 7 stable)
Chou et al	7	7	6	1 (5 resolved, 1 stable)
<i>Our series</i>	8	5	8	3 improved, 5 no new deficits
<i>Surgery</i>		> 90%		
Linskey and Sekhar	3	3	3	2 (1 resolved, 1 improved, 1 stable)
Shi et al	10	4	9	8 (1 resolved, 4 stable, 4 worse)
Zhou et al	20	12	10	NA
Goel et al	13	12	11	10 (2 resolved, 9 stable, 1 new)
Suri et al	7	6	5	1 (4 resolved, 1 worse)
Yin et al	22	18	12	7 (6 resolved, 2 improved, 4 stable, 1 new)
<i>Our series</i>	15	15	12	6 resolved, 3 improved, 4 new

Abbreviations: CSH, cavernous sinus hemangioma; NA, not available.

individualized based on the size and radiation risk to optic apparatus. The residual tumor, if small, can be followed up at regular intervals or GKRS can be offered to treat the residue.

Conclusion

Both microsurgical excision and GKRS are effective modalities to treat CSH. Though surgery poses formidable challenges, with proper techniques, most CSH can be excised. Established CN involvement prior to surgery usually does not reverse. Though surgery is the preferred modality in large tumors, GKRS is a safe option for cases with small, residual, or recurrent lesions, and can supplement surgery wherever possible.

Funding

No funding was received for this research. Consent for this type of study formal consent is not required.

Source of Support

Nil.

Conflict of Interest

None declared.

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