ORIGINAL ARTICLE

Papilloedema from Dural Venous Sinus Compression by Meningiomas

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ABSTRACT

Intracranial mass lesions may cause intracranial hypertension secondary to venous hypertension when they compress the dural venous sinuses (DVS) and may present with isolated papilloedema, mimicking idiopathic intracranial hypertension. We report a series of 16 patients with isolated papilloedema related to meningiomas compressing the DVS seen from 2012 to 2016 at three institutions. Correct diagnosis was delayed in 10/16 patients and treatment required a multi-disciplinary approach, often with multiple sequential interventions, including combinations of acetazolamide, cerebrospinal fluid-shunt, optic nerve sheath fenestration, surgical resection of the meningioma, radiation therapy, and endovascular venous stenting. Two patients also received anticoagulation for venous thrombosis secondary to venous sinus compression.

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Introduction

Meningiomas are intracranial neoplasms arising from the arachnoid cap, and may occur at any location along the meninges encasing the central nervous system.¹ They are the most common intracranial neoplasms in adults, with an increasing prevalence with age, and a 2 to 1 female predominance.² Symptoms of elevated intracranial pressure (ICP) usually develop in association with focal neurologic symptoms or seizures from very large meningiomas.³ Meningiomas arising from meninges in close proximity to the dural venous sinuses (DVS) provide unique surgical challenges to neurosurgeons tasked with their management.^{3–10} Indeed, meningiomas that involve the major DVS can produce impaired venous drainage and venous hypertension, resulting in intracranial hypertension secondary to decreased passive resorption of the cerebrospinal fluid (CSF).³ Isolated intracranial hypertension secondary to venous hypertension from meningiomas or other neoplasms involving the DVS

represents a unique diagnostic challenge, especially when the mass is small (Table 1).^{11–20} We present a case series describing the occurrence of isolated intracranial hypertension mimicking idiopathic intracranial hypertension (IIH) secondary to meningiomas engaging the major DVS.

Materials and methods

The study was approved by the Institutional Review Boards of each of the participating institutions.

We reviewed the medical records of the neuro-ophthalmology services at three tertiary care hospitals to identify patients with isolated intracranial hypertension and papilloedema who were found to have a solid intracranial mass suggestive of meningioma involving one or more DVS. We included cases with histologically proven meningiomas, as well as patients with a diagnosis of meningioma made radiologically. We excluded cases with diffuse leptomeningeal carcinomatosis. We also excluded patients in whom the diagnosis of

Table 1. Summary of prior cases of discreet neoplastic lesions involving the venous dural sinuses resulting in papilloedema. Superior sagittal sinus (SSS). Transverse venous sinus (TVS).

Author	Year	Patients	Pathology	Location	Treatments
Marr ¹¹	1966	2	Meningioma	SSS	Surgery Shunt
Repka ¹²	1982	2	Meningioma	SSS	Surgery Furosemide Corticosteroids Acetazolamide
Kim ¹³	2000	1	Metastasis	SSS	Radiation
Bodkin ¹⁴	2008	3	Meningioma, eosinophilic granuloma, depressed skull fracture	SSS	Surgery
Ganesan ¹⁵	2008	1		SSS	Stent Radiation
Higgins ¹⁶	2008	1	Meningioma	Straight sinus	Shunt Stent Surgery Radiation
Zilani ¹⁷	2009	1	Meningioma	Right Jugular vein	Stent
Chausson ¹⁸	2010	1	Meningioma	Left TVS	Stent
Shah ¹⁹	2016	4	Meningioma	SSS	Surgery Shunt
Marvin ²⁰	2017	1	Metastasis	SSS	Surgery Acetazolamide Corticosteroids

intracranial mass was made because of focal neurologic symptoms or seizures.

We recorded each patient's gender, age, body mass index (BMI), initial diagnosis, time to correct diagnosis from presentation, CSF opening pressure, tumour location, radiologic aspects of the intracranial venous sinuses, presenting symptoms, visual acuity, visual field assessment, optic disc characteristics, and treatment modalities. The medical records and imaging studies of each patient were reviewed to determine the reasons for delay in correct diagnosis and appropriate management when applicable.

Results

A total of 16 patients with intracranial hypertension secondary to compression of the DVS by a mass consistent with meningioma were identified (Table 2). They included 11 women and 5 men, with a mean age of 46 years, and a range of 28–80 years. The median BMI was 28 kg/m², with a range of 21–34. Median CSF opening pressure was 29 cm of water with a range of 20–45.

All patients had an intracranial mass involving one or more DVS responsible for venous hypertension. All lesions were radiographically consistent with dural meningiomas as determined by one expert neuroradiologist (AS) and all patients

underwent venous imaging by CT venogram, MRV or catheter angiography at some point during their evaluation; illustrative cases are shown in Figure 1. Tissue diagnosis was obtained in two patients who underwent surgical resection (#1,5). A total of 18 meningiomas were identified in the 16 patients, with two patients having two meningiomas. In one of these patients (#5), both meningiomas involved the dominant transverse venous sinus (TVS) and the superior sagittal sinus (SSS), and in the other patient (#14), one tumour involved the TVS and the other did not involve any DVS. There were 10 patients with a tumour involving the posterior SSS, and seven patients with meningiomas involving the right TVS, which was identified to be the dominant sinus in six of those cases, with the left TVS hypoplastic or stenotic. Of the 10 patients with SSS meningiomas, four also had a stenosis of one or both TVS, and one patient had a hypoplastic TVS. A total of seven patients (#4,6,8,9,11,13,15) had additional areas of TVS stenosis remote from the DVS involved by tumours, presumably secondary to external TVS compression from elevated ICP.

All patients presented with symptoms of isolated intracranial hypertension such as chronic or new onset headaches, pulsatile tinnitus, blurry vision, transient visual obscurations, and diplopia

Table 2. Case descriptions.

Pt#	Age Sex	BMI	Diagnostic delay	Misdiagnosis	CSF- OP	Tumour location	Transverse sinus characteristics	Presenting symptoms	Optic disc OD	Optic disc OS	Treatment
1	34W	28	1 month ^a	IIH	30	Right TVS	Dominant right Hypoplastic left	Headache Blurred vision Diplopia	Oedema	Oedema	Acetazolamide Radiation Resection, stent
2	37W	30	1 month ^a	IIH	20	Right TVS	Dominant right Hypoplastic left	Headache Blurred vision	Oedema	Oedema	Acetazolamide LP shunt
3	64M	33	6 weeks ^a	IIH	High	SSS	Dominant right Hypoplastic left	Incidental papilloedema	Oedema	Oedema	Acetazolamide
4	44M	33	1 month ^a	IIH	29	Right TVS	Multifocal narrowing left and right	Vision loss	Oedema Shunt vessels	Oedema	VP shunt
5	28W	24	None	None	27	Right TVS SSS	Right resected Left poorly visualized	Blurred vision	Oedema	Oedema	Radiation Resection VP shunt
6	33W	24	7 months	Venous thrombosis ^c Bilateral ONSM	32	Right TVS	Stenotic left	Vision loss	Oedema	Oedema Shunt vessels	Anticoagulation ^b
7	47M	34	4 years	Venous thrombosis ^c (Meningioma missed on MRI)	23	SSS	Normal	Vision loss	No view due to previous trauma	Oedema Shunt vessels	Anticoagulation ^b ONSF
8	80W	25	None	None	24	Right TVS	Dominant right Stenotic left	Vision loss	Atrophy	Oedema	None, did not follow-up
9	51W	29	3 years	IIH (meningioma missed on MRI)	45	SSS	Bilateral stenosis	Vision loss	Atrophy	Oedema	Acetazolamide LP Shunt
10	40W	28	None	None	24	SSS	Normal	TVO	Oedema	Oedema	Radiation Acetazolamide
11	62M	26	None	None	N/A	SSS	Stenotic right	TVO	Oedema	Oedema	Acetazolamide
12	35W	32	None	None	N/A	SSS	Normal	Blurred vision	Oedema	Oedema	Acetazolamide
13	33W	26	3 weeks	Venous thrombosis	29	SSS	Dominant, stenotic right	Vision loss	Pallid oedema	Pallid oedema	Acetazolamide, VP shunt, ONSF
14	46W	27	None	None	30	Right TVS and right falx	Stenotic right	Headache, tinnitus, TVOs	Oedema	Oedema	Acetazolamide VP shunt
15	49W	26	3 months	IIH (meningioma missed on MRI)	23 ^d	SSS	Dominant left, stenotic right	TVOs, tinnitus	Oedema	Oedema	Acetazolamide
16	46M	21	6 months	IIH (meningioma missed on MRI)	40	SSS	Dominant right	Headache, tinnitus, TVOs, neck pain	Oedema	Oedema	Acetazolamide

Cases 1–9 were collected at Emory University. Cases 10–13 were collected at the University of Minnesota. Cases 14–16 were collected at Wills Eye Hospital.

^aAlthough the meningioma was correctly identified on the initial brain MRI, no MRV had been obtained and the meningioma was diagnosed as an incidental finding (described as “too small to cause the patient’s symptoms”); its mass effect on the venous sinus was not recognized.

^bPatients with cerebral venous sinus thrombosis associated with the tumour were treated with anticoagulation.

^cThese patients were diagnosed correctly with cerebral venous sinus thrombosis, but the dural meningiomas (responsible for venous thrombosis) were either considered irrelevant or were missed.

^dThis patient’s opening pressure was measured while on acetazolamide.

Body mass index (BMI), cerebrospinal fluid opening pressure (CSF-OP) provided in centimetres of water, idiopathic intracranial hypertension (IIH), lumboperitoneal (LP), optic nerve sheath fenestration (ONSF), optic nerve sheath meningioma (ONSM), superior sagittal sinus (SSS), transient visual obscuration (TVO), transverse venous sinus (TVS), ventriculoperitoneal (VP).

Diagnostic delay is the time from symptom onset to correct diagnosis.

(Table 2). Papilloedema was present in all patients, although three patients (#8,9,13) had already developed secondary optic atrophy and pallor from chronic papilloedema at the time of

diagnosis, and three had retino-choroidal collaterals (#4,6,7) from chronic papilloedema. Except for three patients with profound visual acuity loss (in both eyes in two (#4,7), and in one eye in one

Figure 1. (A and B) MRIs of meningiomas invading the right transverse venous sinuses in two patients. (C and D) MRIs of meningiomas invading the posterior superior sagittal sinus in two patients. (E and F) MR venograms showing occlusion of right transverse venous sinuses (dark arrows) with a hypoplastic left transverse venous sinus (E) and a stenotic left transverse venous sinus (F) (light arrows).

(#6)) from untreated chronic papilloedema, all patients had relatively good central visual acuity at presentation, consistent with papilloedema (Table 3). However, visual field testing demonstrated visual field defects in 15 out of 16 patients, including four with enlarged blind spots alone, six with nasal depression or arcuate defects in addition to enlarged blind spots, and five with generalized constriction. The three patients with visual acuity loss at presentation (#4,6,7) already had chronic appearing oedema with retino-choroidal collaterals and did not improve despite treatment. One additional patient (#13) with good visual acuity, but with severe visual field defects at presentation, had worse visual function at last follow-up despite treatment.

The correct diagnosis of venous hypertension secondary to compression of the DVS by a meningioma and appropriate management was delayed

in 10/16 patients (63%) and was only made after a neuro-ophthalmology consultation. Seven patients (44%) were misdiagnosed with IIH. There were three categories of misdiagnosis. In the first category, three patients (#9,15,16) had brain imaging with MRI obtained appropriately when bilateral optic disc oedema suggestive of papilloedema from elevated ICP was identified, but the meningiomas were not identified by the radiologists reviewing the MRIs. All three of these tumours were located in the SSS. The correct diagnosis in this group was made by expert review of the available brain MRIs without additional tests being necessary in two patients (#15,16), and with a supplemental MRV in the third patient (#9). In the second category, four patients (#1,2,3,4) had meningiomas that were correctly identified on brain imaging but were not considered related to the patient's clinical syndrome of elevated ICP

Table 3. Visual function at baseline and at final visit.

Pt#	Initial visual evaluation				Final visual evaluation				Time between first and final evaluations
	First visual acuity OD	First visual acuity OS	First visual field OD	First visual field OS	Last visual acuity OD	Last visual acuity OS	Last visual field OD	Last visual field OS	
1	20/20	20/20	Enlarged blind spot	Enlarged blind spot	20/20	20/20	Full	Full	4 years
2	20/20	20/20	Enlarged blind spot	Enlarged blind spot Nasal depression	20/20	20/20	Enlarged blind spot Nasal depression	Enlarged blind spot Nasal depression	6 months
3	20/20–1	20/20	Enlarged blind spot Nasal depression	Full	20/20–2	20/20–1	Full	Full	4 years
4	20/200	Count fingers	Generalized constriction	Generalized constriction	20/400	Count fingers	Cecocentral scotoma	Cecocentral scotoma	4 months
5	20/20–2	20/20–2	Full	Enlarged blind spot Inferior arcuate defect	20/20 + 2	20/20–1	Full	Enlarged blind spot Inferior arcuate defect	5 months
6	20/40	Hand motion	Generalized constriction	Generalized constriction	20/30 + 2	Hand motion	Generalized constriction	Generalized constriction	6 months
7	NLP	Count fingers	Unable	Generalized constriction	NLP	Count fingers	Unable	Generalized constriction	3 years
8	20/20	20/80	Superior arcuate defect	Generalized constriction	Unavailable	Unavailable	Unavailable	Unavailable	No follow-up
9	20/20	20/20	Enlarged blind spot Nasal depression	Enlarged blind spot Nasal depression	20/20	20/20	Enlarged blind spot	Enlarged blind spot	
10	20/20	20/20	Enlarged blind spot	Normal	20/20	20/20	Normal	Normal	7 years
11	20/15	20/15	Normal	Normal	20/15	20/15	Normal	Normal	4 years
12	20/20	20/20	Enlarged blind spot	Normal	20/20	20/20	Enlarged blind spot	Normal	8 months
13	20/40	20/20	Generalized constriction	Arcuate	20/60	HM	Generalized constriction	Central scotoma	5 years
14	20/20	20/20–1	Inferior arcuate defect Enlarged blind spot	Inferior arcuate defect	20/20	20/25	Inferior arcuate defect	Inferior arcuate defect	2 years
15	20/25	20/20	Enlarged blind spot	Mild inferonasal constriction	20/20	20/20	Mild nonspecific changes	Mild inferonasal constriction	4 years
16	20/20	20/20	Enlarged blind spot	Enlarged blind spot	20/20–	20/20–	Enlarged blind spot	Enlarged blind spot	3 years

Cases 1–9 were collected at Emory University. Cases 10–13 were collected at University of Minnesota. Cases 14–16 were collected at Wills Eye Hospital.

All patients underwent formal visual field testing with Humphrey or Goldmann visual fields.

No light perception (NLP).

because they were considered too small to induce intracranial hypertension. The fact that these meningiomas involved an intracranial venous sinus was missed. One of these four patients had a poor visual outcome, with reduced visual function both on initial assessment and at the conclusion of his treatment. Only one of these patients had MRV at initial diagnosis of the tumour. In the third category, an alternative diagnosis of venous sinus thrombosis was made in three patients (#6,7,13). In this group, two patients (#6,7) were correctly identified to have venous sinus

thrombosis, although the underlying associated meningioma (which caused the venous thrombosis) was not appreciated on initial imaging. There was additional confusion in patient #6, who initially underwent MRI with dedicated orbital views, which was incorrectly interpreted as showing bilateral optic nerve enhancement suggestive of bilateral optic nerve sheath meningiomas. Patient #13 had a meningioma compressing the SSS, which was misinterpreted as a venous sinus thrombosis. These patients all had poor visual outcomes (Table 3). Patients #6 and #13 underwent initial

imaging with MRI only, and required subsequent acquisition of a dedicated MRV to clarify their diagnosis. One patient (#7) had an MRI that was read as normal initially, but had a venous sinus thrombosis identified on a conventional angiogram, with a later MRV identifying the associated meningioma.

Among all 10 misdiagnosed patients, the correct diagnosis was delayed by 3 weeks to 4 years (median of 9 weeks). In four patients, the delay in appropriate treatment resulted in profound visual loss from chronic papilloedema.

Treatments were mostly aimed at reducing ICP, and most patients were initially treated similarly to IIH patients, with an initial lumbar puncture followed by acetazolamide in 11 out of 16 cases. The two patients with venous thrombosis (#6,7) received anticoagulants in addition to an optic nerve sheath fenestration (ONSF) in one (#7). Treatments varied once the correct diagnosis of meningioma compressing the DVS was made. There were two patients who underwent surgical resection of the meningioma with adjuvant radiation therapy (#1, 5), associated with placement of a CSF shunt in one (#5). A single patient underwent radiosurgery in addition to treatment with acetazolamide (#10). There were five patients with normal visual acuity and only mild visual field changes (#3,11,12,15,16) who received acetazolamide alone. A CSF-shunt (three ventriculoperitoneal (VP) shunts and two lumboperitoneal (LP) shunts) were placed in five patients (#2,4,9,13,14), among whom one (#13) also had an ONSF.

Discussion

We describe here the largest case series of patients with isolated intracranial hypertension and papilloedema due to involvement of the DVS by mass lesions, presumably meningiomas. We confirm prior reports that meningiomas compressing or invading the DVS can cause symptomatically elevated ICP, presumably by limiting cerebral venous outflow, with resultant venous hypertension, and subsequent reduction of passive CSF resorption causing elevated ICP. Intracranial hypertension can occur even with a small meningioma as long as the meningioma involves an intracranial venous

sinus, causing venous stenosis or venous stenosis with secondary intracranial venous hypertension.

The patients in our series are representative of epidemiologic trends in patients with intracranial meningiomas, with a 2:1 female to male ratio. The meningiomas were located most commonly in the posterior aspect of the SSS. Prior literature has demonstrated that the posterior aspect of the SSS is the most common location for a neoplastic lesion to result in symptomatic intracranial hypertension (Table 1). However, the previous body of literature only reported in detail one TVS-involving meningioma resulting in high ICP¹⁸, whereas our current series was notable for seven meningiomas involving a TVS (Table 2). All meningiomas involving a TVS in our series occurred in the dominant TVS, with no case of symptomatically elevated ICP from a meningioma involving a non-dominant TVS.

Interestingly, seven of our patients had areas of TVS stenosis distant from the meningioma, suggesting that venous hypertension from the initial venous compression by a meningioma may have led to enough elevation in ICP to cause a secondary stenosis of another DVS, perhaps contributing to worsening of intracranial hypertension. Indeed, increased ICP has been proposed as a contributory mechanism of TVS stenosis in IIH.^{21–23} Prior literature has demonstrated that meningiomas are the most common neoplastic lesion to result in high ICP from obstruction of the DVS, with rare reports of solitary metastases, with one case from prostate adenocarcinoma¹³ and one from renal cell carcinoma.²⁰ Both of these cases were also notable for diagnostic error: of the third type (a misdiagnosis of venous sinus thrombosis) in the case of renal cell carcinoma metastasis, and of the second type (the mass was not considered related to elevated ICP) in the case of the prostate cancer metastasis. In the case with the prostate cancer metastasis, the tumour was initially felt to represent a meningioma, and was also initially felt to be unrelated to the patient's intracranial hypertension symptoms. A high prostate specific antigen in this patient led to the correct diagnosis and proper treatment. Therefore, while it appears that meningiomas are by far the most likely cause of symptomatic involvement of a neoplastic process associated with the DVS, metastases are also

possible and appropriate workup is indicated, especially given the important implications for both management and prognosis.

Of our 16 total patients, 10 (63%) were misdiagnosed, resulting in a delay of appropriate treatment by a median of 9 weeks, but as long as 4 years. All 4 patients in our series with poor visual outcomes were misdiagnosed initially. The most common error in misdiagnosis was to assume that a correctly identified meningioma involving the DVS was not responsible for a clinical syndrome of elevated ICP because it was too small. Although this was the most common error, it was associated with comparatively short diagnostic delays compared to the other patients. This diagnostic error occurred despite previously reported cases in the literature of patients with symptomatically elevated ICP in the setting of meningiomas involving the DVS (Table 1).^{11–20} Our large series of 16 cases should persuade clinicians to not assume that mass lesions involving a venous sinus found during a workup for elevated ICP are incidental. Additionally, four of our patients who had diagnostic delay underwent MRV as a subsequent study after an initial brain MRI was read as normal or was misinterpreted. We usually obtain a coincident MRV with MRI (with contrast) urgently in patients with bilateral optic disc oedema suggestive of papilloedema.

Although all our patients with poor visual outcomes had a delayed diagnosis, the patients in the third category, whose diagnosis was either confused with, or complicated by a venous sinus thrombosis, had by far the worst visual outcomes. As two of these patients did indeed have venous sinus thrombosis associated with a meningioma, this may suggest a worse visual prognosis in that specific scenario.

In our case series, treatments offered were similar to what has been described in previous case reports, including medical therapy^{12,20}, CSF shunting^{11,16,19}, endovascular venous stenting^{15–18} as well as surgical resection and radiotherapy of the meningioma.^{3–16,19,20} Two of our patients had successful ONSF. Surgical treatments directed specifically to treat papilloedema from elevated ICP, namely CSF shunting, endovascular venous sinus stenting, and ONSF, were utilized in patients with visual field defects worse than blind spot

enlargement, and in patients with reduced visual acuity. The patient who underwent treatment with venous stenting maintained good visual function, but was chronically noncompliant with acetazolamide, necessitating further intervention.

Although surgical reconstruction of DVS compromised by a meningioma is possible, this is an invasive procedure, which necessitates opening the involved dural sinus, which can lead to major complications including venous thrombosis and air embolus.⁵ If reconstruction of the dural sinus is not technically feasible, ligation of the sinus may be attempted, but this can result in venous infarction, as noted in large neurosurgical series^{4,6–9}, or worsening of the intracranial hypertension with potentially increased papilloedema and further visual loss. In our own series, only two patients underwent surgical resection of any sort (#1,5), with a subtotal resection in patient #1 without surgical entry into the dural sinuses. In patient #5, the right TVS was sacrificed to complete a total resection of the associated meningioma. In both cases, measures in addition to surgical resection were necessary to treat persistent intracranial hypertension and chronic papilloedema.

In our series, the necessity of CSF shunts, ONSF, and stenting was determined primarily on a case by case basis, and all were effective. It seems that the primary factor in the patients with poor outcomes was delayed diagnosis, rather than the specifics of their treatment modality. Six patients with less severe vision loss were medically compliant and did well with acetazolamide treatment alone. Radiotherapy alone was not effective in reducing ICP, and was administered primarily to limit growth of the presumed meningiomas in three cases.

The need for serial monitoring of papilloedema and additional measures to control ICP even following surgical intervention is important.¹² In our series, the two patients who underwent surgical resection required additional measures thereafter to effectively control ICP. Several patients underwent radiation therapy in order to halt growth of the presumed meningiomas, but this treatment modality would not be expected to reduce ICP. We suggest that patients with meningiomas involving the DVS be examined systematically by ophthalmologists or neuro-ophthalmologists affiliated with neurosurgical centres where patients with these meningiomas are

commonly managed in order to prevent vision loss from papilloedema.

Conclusion

Meningiomas invading the DVS can cause elevated ICP and vision loss due to papilloedema. Thorough imaging review and a high degree of suspicion are crucial in making a timely diagnosis and preventing vision loss. Effective treatment requires diligent monitoring of visual function and may involve the expertise of neurosurgeons, neuro-ophthalmologists, neuroradiologists, radiation oncologists, and orbital surgeons, depending on a patient's course.

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Declaration of Interest

The authors declare that there are no conflicts of interest. The authors alone are responsible for the writing and content of the article.

Notes on contributors

Nancy J. Newman is a consultant for Gensight Biologics (France) and serves on the Data Safety Monitoring Board for Quark Pharmaceuticals (Israel) clinical trial, has provided expert testimony on the topic of optic disc oedema, and has received research support from NIH grants (R01-NS089694, P30-EY006360) and Research to Prevent Blindness Lew R. Wasserman Merit Award.

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References

- Langford LA. Pathology of meningiomas. *J Neuro-Oncol*. 1996;29(3):217–221. doi:10.1007/BF00165651.
- Mcneill KA. Epidemiology of brain tumors. *Neurol Clin*. 2016;34(4):981–998. doi:10.1016/j.ncl.2016.06.014.
- Sindou M, Auque J, Jouanneau E. Neurosurgery and the intracranial venous system. *Acta Neurochir Suppl*. 2005;94:167–175.
- Sindou MP, Alvernia JE. Results of attempted radical tumor removal and venous repair in 100 consecutive meningiomas involving the major dural sinuses. *J Neurosurg*. 2006;105(4):514–525. doi:10.3171/jns.2006.105.4.514.
- Mazur MD, Cutler A, Couldwell WT, Taussky P. Management of meningiomas involving the transverse or sigmoid sinus. *Neurosurg Focus*. 2013;35(6):E9. doi:10.3171/2013.8.FOCUS13340.
- Mathiesen T, Pettersson-Segerlind J, Kihlström L, Ulfarsson E. Meningiomas engaging major venous sinuses. *World Neurosurg*. 2014;81(1):116–124. doi:10.1016/j.wneu.2013.01.095.
- Mantovani A, Di Maio S, Ferreira MJ, Sekhar LN. Management of meningiomas invading the major dural venous sinuses: operative technique, results, and potential benefit for higher grade tumors. *World Neurosurg*. 2014;82:455–467. doi:10.1016/j.wneu.2013.06.024.
- Han MS, Kim YJ, Moon KS, Lee KH, Yang JI, Kang WD, Lim SH, Jang WY, Jung TY, Kim IY, Jung S. Lessons from surgical outcome for intracranial meningioma involving major venous sinus. *Medicine (Baltimore)*. 2016;95(35):e4705. doi:10.1097/MD.0000000000004705.
- Anthofer J, Seidel-Schulz R, Proescholdt M, Brawanski A, Schebesch KM. Meningiomas adjacent to major venous sinuses-clinical outcome and recurrence. *World Neurosurg*. 2017;104:560–566. doi:10.1016/j.wneu.2017.05.025.
- Debernardi A, Quilici L, La Camera A, Boccardi E, Cenzato M. Torcular meningioma with multi-venous sinus invasion: compensatory drainage veins and surgical strategy. *World Neurosurg*. 2018;109:451–454. doi:10.1016/j.wneu.2017.10.120.
- Marr WG, Chambers JW. Occlusion of the cerebral dural sinuses. *Am J Ophthalmol*. 1966;61:45–49.
- Repka MX, Miller NR. Papilledema and dural sinus obstruction. *J Clin Neuroophthalmol*. 1984;4:247–250.
- Kim AW, Trobe JD. Syndrome simulating pseudotumor cerebri caused by partial transverse venous sinus obstruction in metastatic prostate cancer. *Am J Ophthalmol*. 2000;129:254–256.
- Bodkin PA, Hassan MF, Kane PJ, Brady N, Whittle IR. ‘Surgical’ causes of benign intracranial hypertension. *J R Soc Med*. 2008;101(5):259–261. doi:10.1258/jrsm.2008.070386.
- Ganesan D, Higgins JN, Harrower T, Burnet NG, Sarkies NJ, Manford M, Pickard JD. Stent placement for management of a small parasagittal meningioma. *J Neurosurg*. 2008;108(2):377–381. doi:10.3171/JNS/2008/108/2/0377.
- Higgins JNP, Burnet NG, Schwindack CF, Waters A. Severe brain edema caused by a meningioma obstructing cerebral venous outflow and treated with venous sinus stenting. *J Neurosurg*. 2008;108(2):372–376. doi:10.3171/JNS/2008/108/2/0372.
- Zilani G, Pereira EAC, Baig F, Uberoi R, Kuker W, Kerr RS. Venoplasty and stenting of a jugular foramen meningioma. *Br J Neurosurg*. 2009;23:557–560.
- Chausson N, Bocquet J, Aveillan M, Olindo S, Signaté A, Merle H, Smadja D. Intracranial hypertension

- caused by a meningioma compressing the transverse sinus. *J Clin Neurosci.* 2010;17(12):1589–1592. doi:[10.1016/j.jocn.2010.03.039](https://doi.org/10.1016/j.jocn.2010.03.039).
19. Shah AH, Ivan ME, Komotar RJ. Pseudotumor-like syndrome and cerebrospinal fluid leak in meningiomas involving the posterior third of the superior sagittal sinus: report of 4 cases. *J Neurosurg.* 2016;125(1):62–66. doi:[10.3171/2015.7.JNS15770](https://doi.org/10.3171/2015.7.JNS15770).
 20. Marvin E, Synkowski J, Benko M. Tumor cerebri: metastatic renal cell carcinoma with dural venous sinus compression leading to intracranial hypertension; a case report. *Surg Neurol Int.* 2017;8:175. doi:[10.4103/sni.sni_69_17](https://doi.org/10.4103/sni.sni_69_17).
 21. Farb RI, Vanek I, Scott JN, Mikulis DJ, Willinsky RA, Tomlinson G, terBrugge KG. Idiopathic intracranial hypertension: the prevalence and morphology of sinovenous stenosis. *Neurology.* 2003;60:1418–1424.
 22. Bidot S, Saindane AM, Peragallo JH, Bruce BB, Newman NJ, Bioussé V. Brain imaging in idiopathic intracranial hypertension. *J Neuro-Ophthalmol.* 2015;35(4):400–411. doi:[10.1097/WNO.0000000000000303](https://doi.org/10.1097/WNO.0000000000000303).
 23. Ridha MA, Saindane AM, Bruce BB, Riggeal BD, Kelly LP, Newman NJ, Bioussé V. Magnetic resonance imaging findings of elevated intracranial pressure in cerebral venous thrombosis versus idiopathic intracranial hypertension with transverse sinus stenosis. *Neuro-Ophthalmology.* 2013;37(1):1–6. doi:[10.3109/01658107.2012.738759](https://doi.org/10.3109/01658107.2012.738759).