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# Long-term kinetic papilledema improvement after venous sinus stenting in idiopathic intracranial hypertension

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## **Abstract:**

### **Background and Purpose**

The aim of this study was to assess the safety and effectiveness of lateral sinus stenosis (LSS) stenting in patients with idiopathic intracranial hypertension (IIH) who are refractory to medical treatment, particularly focusing on visual outcomes including papilledema.

### **Materials and Methods**

Retrospective study of consecutive patients with IIH refractory to medical treatment who underwent LSS stenting. Clinical features, visual fields and optical coherence tomography (OCT) retinal nerve fiber layer (RNFL) and ganglion cell layer (GCL) thicknesses were assessed before stenting, at one month post-stenting and at last follow-up. Complications were also recorded.

### **Results**

16 women were included, with a mean age of  $39 \pm 11$  years. All patients had papilledema while 15/16 (94 %) had headaches. Mean visual acuity was (0.036 logMAR [+0.4 to +0.0 logMAR]). RNFL thickness prior to stenting was  $121.7 \mu\text{m}$  ( $\pm 34.7$ ) and mean GCL thickness was  $80.2 \mu\text{m}$  ( $\pm 8.9$ ). Mean follow-up was 19.7 months ( $\pm 11.8$ ). After stenting, acetazolamide was discontinued in 15/16 (94 %) patients. Papilledema improved in 14/16 (88%) of patients. The mean RNFL thickness was significantly decreased one month after stenting ( $96.3 \pm 15.6 \mu\text{m}$ ;  $p < 0.001$ ) and at last visit ( $93.4 \pm 15.3 \mu\text{m}$ ;  $p < 0.001$ ). GCL thickness after LSS stenting was

moderately decreased at the last visit examination:  $80.2 \pm 8.9 \mu\text{m}$  vs  $78.0 \pm 10.7 \mu\text{m}$ ; ( $p < 0.01$ ).

No stent-related complication occurred, while there was one case of restenosis.

## **Conclusion**

Improvement of papilledema after LSS appears to occur relatively rapidly after the stenting.

Our results further support the role of LSS in the treatment of IIH, especially with respect to visual symptoms.

## **Introduction**

One of the main symptoms of idiopathic intracranial hypertension (IIH) is papilledema [1]. IIH is diagnosed by use of the modified Dandy criteria. While lateral sinus stenoses (LSS) are diagnosed in more than 90% of the patients, their cause and pathophysiological role remain controversial [2,3]. Several studies have demonstrated the good safety and efficacy profiles of LSS stenting in these patients.

Patients suffering of IIH experience myriad symptoms which can have a significant effect on quality of life [4,5], including headache, tinnitus and visual symptoms. The traditional first-line treatment for IIH patients includes weight-loss, medication (acetazolamide or topiramate as a second-line agent) or multiple lumbar puncture procedures.

In many centres, LSS stenting has replaced CSF diversion procedures for those patients who are refractory to medical management. However, the trajectory of improvement of papilledema after LSS stenting remains poorly known, and the place of this technique in the management of patients with rapid visual deterioration remains controversial. There remains relatively little published literature regarding the onset and magnitude of improvement in visual symptoms after LSS.

In addition to visual symptoms, vision changes in patients with IIH are generally assessed by using more quantifiable methods. High-definition optical coherence tomography (HD-OCT) allows acute assessment of retinal nerve fiber layer thickness (RNFL) and ganglion cell layer (GCL), which are objective and reproducible markers of severity of papilledema [6].

The purpose of this study is to use OCT parameters to assess the trajectory of improvement of papilledema after LSS stenting in patients with IIH refractory to medical therapy.

## **Material and Methods**

### *Patients*

We retrospectively collected clinical and radiological data of patients treated with LSS stenting for IIH in two tertiary eye-care centers (Pitié Salpêtrière Hospital, Paris, France and Lariboisière Hospital, Paris, France) between July 2015 and August 2017.

The inclusion criteria were:

- Patients treated by LSS stenting for IIH (as defined by the modified Dandy criteria) who were refractory to medical therapy as defined by:
  - Worsening or no improvement of visual field defects and/or papilledema and/or oculomotor nerve palsy despite maximal tolerated dosage of acetazolamide.
  - Recurrence of papilledema after tapering the dose of acetazolamide.
  - Intolerance to acetazolamide with discontinuation of medical treatment.
  - Disabling clinical signs (headaches, tinnitus...) despite medical treatment.

- Patients with a lateral sinus trans-stenotic pressure gradient  $> 4\text{mmHg}$  (measured under local anesthesia) after discontinuation of the treatment with acetazolamide for at least 10 days before the procedure.
- Patients with an ophthalmological assessment performed in our institution at baseline and during follow-up.

The exclusion criteria were:

- Incomplete ophthalmological examination before or after LSS stenting.
- Patients with concomitant ophthalmic conditions, which could potentially confound assessment.
- Patients with fulminant IIH, defined as: [7]
  - acute onset of symptoms and signs of intracranial hypertension;
  - less than 4 weeks between onset of initial symptoms and severe visual loss;
  - rapid worsening of visual loss over a few days

### *Ophthalmic outcome*

Our primary outcome was the thickness of RNFL after the LSS stenting.

All patients had an ophthalmological examination before the stenting, at one month and every 6 months after stenting. All examinations were performed by an experienced senior ophthalmologist, and included visual field assessment, fundus examination, optical coherence tomography imaging and visual acuity (VA) assessment as measured by LogMAR (Minimum

angle of resolution). The LogMAR is a visual acuity scale which has advantages over the more traditionally used Snellen charts, particularly when it comes to statistical analysis of the results. A score of zero indicates 'standard' or average visual acuity, with increasingly positive logarithmic scores indicating progressively poorer vision (1.3 representing blindness), and negative scores indicating better than average vision [8].

Retinal Nerve Fiber Layer (RNFL) and Ganglion cell layer (GCL) thicknesses were measured using a *Cirrus* high-definition Optical Coherence Tomography (HD-OCT) device. According to our institutional practice, OCT scanning was performed as follows: after pupil dilation, the optic disc cube was used to assess peripapillary RNFL thickness and the macular cube was used to assess GCL thickness. The GCL analysis algorithm was included in the Cirrus HD-OCT 6.0 software. It detected and measured the macular Ganglion Cell-Inner Plexiform Layer (GCIPL) thickness within a 6 x 6 x 2-mm elliptical annulus area centered on the fovea. The algorithm has been described previously in detail [9]. The GCL analysis also reported the minimum GCIPL thickness, which is the lowest GCIPL thickness over a single meridian crossing the annulus.

Visual fields (static or kinetic perimetry) were classified as 1) normal, 2) enlargement of the blind spot, 3) nasal or temporal defect, or 4) diffusely constricted [10]. This allowed classifying the clinical evolution of the visual field in worsened, stabilized or improved.

### *Clinical outcomes*

The following clinical data were collected: age, gender, clinical symptoms of IIH (headache, pulsatile tinnitus, visual symptoms), Body Mass Index (BMI), CSF opening pressure, dose of



acetazolamide before and after the procedure. All complications or side effects related to the procedure were also recorded.

### *Radiological data*

All patients underwent a brain MRI to exclude tumors or vascular causes of intracranial hypertension. Radiological signs of intracranial hypertension (i.e empty sella, flattening of the posterior aspect of the globe, distension of perioptic subarachnoid space with or without a tortuous optic nerve) were also assessed. Venous sinus stenoses were evaluated with gadolinium-enhanced T1-weighted 3-dimensional gradient echo sequences and 3-dimensional gadolinium-enhanced venous angiography.

### *Endovascular procedure*

The endovascular procedure was performed according to our institutional protocol which has been detailed previously [11]. Each patient underwent a digital subtraction angiography (DSA) with venous phases acquisitions to assess the venous anatomy. The trans-stenotic gradient was measured with patients under local anesthesia in order to avoid anaesthesia-related fluctuations. Stenting was then performed with patients under general anesthesia [11]. Our protocol was already published in Lenck et al. [11]

The treatment by acetazolamide was discontinued once the endovascular procedure was performed. Patients were rapidly investigated after the procedure to assess the improvement with the discontinuation of acetazolamide.

### *Follow up*

All patients were clinically assessed prior to the stenting, at one week then one month after the stenting and at 6 months intervals until stabilization. Once patients were stabilized from an ophthalmological point of view, the ophthalmological follow-up was then performed by the referring ophthalmologist.

#### *Data analysis*

Data analysis was performed using GraphPad Prism 5 (GraphPad Software, San Diego, CA, USA). Results were presented as means  $\pm$  standard deviations. Continuous data were analyzed using a Wilcoxon test, or the Mann-Whitney test, depending on the data distribution. The Fisher's exact test was used to compare qualitative data. Results were considered statistically significant when  $p\text{-value} < 0.05$ .

#### *Ethical statement*

This study has been approved by the local ethics committee (IRB 00008855). Because this study used data obtained as part of standard of care, the requirement for informed consent was waived.

#### *Data availability*

The authors declare that all data concerning this study are available on request.

## **Results**

#### *Patients' demographics and characteristics*

30 eyes from 16 patients were included; two eyes were excluded because of severe amblyopia. Clinical features at baseline are summarized in **Table1**.

Before stenting, all patients (100%) had bilateral papilledema, 5/16 (31%) had transient visual obscurations and 1/16 (6%) had binocular diplopia caused by a sixth nerve palsy. 15/16(94%) patients had headache, 13/16 (81%) had pulsatile tinnitus. Mean best-corrected visual acuity (BCVA) was 0.036 logMAR [+0.4 to +0.0 logMAR] before stenting. Fourteen (87.5%) patients had a normal BCVA and two (12.5%) patients had an altered BCVA over 0.3 logMAR. Fifteen (94%) patients were taking acetazolamide with a mean dosage of  $660.7 \pm 270.5$  mg/day *range* [250 – 2000 mg/day]. Acetazolamide was not tolerated in one case. The median delay between the onset of IIH symptoms and stenting was 28.2 months ( $\pm$  21.4 months).

#### *Endovascular Treatment and Results*

Stenting of the right LS was performed in 11 patients, while the left LS was stented in 5 cases. The mean trans-stenotic gradient measured under local anesthesia before stenting was  $13 \pm 6$  mmHg. A single stent was deployed in 15/16 (94%) patients without complications. Acetazolamide was subsequently discontinued in 15/16 (94%) patients. One patient had a *de novo* LS stenosis adjacent to the stent which occurred 3 months after stenting without symptom improvement. In this case, a subsequent stenting of this new stenosis was performed 8 months after the first procedure. Despite these stents, the patient failed to show improvement in symptoms and acetazolamide was required to control IIH symptoms and papilledema as well. However, their visual field defects continued to worsen.

### *Clinical Outcomes*

The mean follow-up of patients after stenting was 19.7 ( $\pm 11.8$ ) months. Complete resolution of headaches was noted in 8/15 (53%) patients, while they improve in 5/15 (33%) patients. Headaches were unchanged in 2/15 (13%) patients. Pulsatile tinnitus resolved immediately after stent placement in 12/13 (92%) patients.

At one-month follow-up, papilledema was completely resolved in 14/16 (87.5%) patients and remained unchanged in 2/16 (12.5%). At most recent follow-up, papilledema had resolved in 15/16 (93.8%) patients and remained unchanged in one 1/16 (6.2%).

Concerning our primary outcome, the mean RNFL thickness at initial presentation was  $121.7 \pm 34.7 \mu\text{m}$ . This had significantly decreased at one-month follow-up ( $96.3 \pm 15.6 \mu\text{m}$ ,  $p < 0.001$ ) and further decreased at most recent follow up ( $93.4 \pm 15.3 \mu\text{m}$ ,  $p < 0.001$ ) **Fig 1**.

GCL thickness decreased significantly from  $80.2 \pm 8.9 \mu\text{m}$  before stenting to  $79.8 \pm 9.5 \mu\text{m}$  one month after stenting ( $p=0.23$ ) and  $78.0 \pm 10.7 \mu\text{m}$  at last follow-up ( $p < 0.01$ ) **Fig2**.

All results are summarized in **Table 2**.

Visual fields improved in 16/30 (53.3%) patients at last follow-up, while it remained stable in 13/30 (43.3%) patients and was worsened in one case **Table 2**.

### **Discussion**

Our study demonstrated a significant improvement in visual parameters after LSS stenting for IIH, including papilledema recovery and decrease in RNFL thickness. Of note, this decrease in RNFL thickness was seen as quickly as one month after stenting.

Stenting of LSS was first reported as a potential treatment for IIH in 2002 by Higgins et al [12]. There has been increasing interest in this technique, with many groups reporting their favourable experience, demonstrating the excellent safety profile and efficacy of this technique [11–17]. In a recent meta-analysis, Nicholson et al [18] reported an overall rate of improvement in papilledema in 93.7% of patients, an overall rate of improvement or resolution of headache in 79.6% and resolution in pulsatile tinnitus in 90.3% of patients. The rate of major complications was 1.9% without long-term morbidity or mortality, while the overall rate of recurrence of IIH symptoms was 9.8%. Based on these results, LSS stenting has in many centers progressively replaced CSF diversion procedures as a first-line treatment for those carefully selected patients with IIH refractory to medical therapy. However, there is relatively little evidence regarding the degree of improvement of visual parameters as well as their chronological evolution after LSS.

As for visual outcomes, we found only 4 previous studies looking at RNFL thickness following stenting of LSS in IIH [17,19–21]. The IIHTT study [6] showed that in patients with newly diagnosed IIH, RNFL was strongly correlated with Frisen grade (a grading system which allowed us to grade and track papilledema) [22]. In our study, each patient had OCT measurements including RNFL and GCL, both preceding the stenting and during follow-up visits. The improvement of RNFL thickness after stenting was statistically significant (121.7  $\mu\text{m}$  vs 93.4  $\mu\text{m}$ ) with  $p < 0.001$ , occurred relatively early (with significant changes seen at one-month follow-up), and was sustained throughout the follow-up period (mean follow-up:

19.7 months). There are however several limitations with measuring RNFL thickness in these cases, such as with the combination of atrophy and papilledema, which could be falsely reassuring. The assessment of RNFL thickness must therefore be performed in conjunction with clinical findings as well as other parameters such as GCL. Indeed, in cases of optic nerve atrophy the GCL can be decreased and abnormal. To our knowledge, we are the first group to study the GCL thickness after stenting of LSS in IIH. The GCL thickness is used to monitor the loss of ganglion cell layer in case of glaucoma or optic neuropathy. The GCL thickness is a valuable tool to distinguish an optic nerve injury in case of optic nerve swelling. In case of IIH, the GCL thinning could reflect an early optic nerve injury, while the RNFL can be falsely normal or increased due to optic nerve swelling [6]. We reported a mean GCL thickness of 80.2  $\mu\text{m}$  before stenting and 78.0  $\mu\text{m}$  after stenting (19.7 months). This difference is slight, but within the normal range of GCL thickness. We can therefore infer that our patients had no clinically significant optic nerve atrophy. Therefore, in this study, we can only postulate that changes sustained as a result of intracranial hypertension were not sufficient to induce a significant optic neuropathy. Few studies have looked at GCL thickness in IIH overall – the conclusions of the NORDIC Idiopathic Hypertension Study Group were that GCL thickness was in the normal range in 93% [6]. It appeared to be difficult to find evidence of early axonal and retinal ganglion cell damage in IIH using OCT. The same conclusion has been reached in patients with non-arteritic anterior ischemic optic neuropathy (NAION) or optic neuritis [23]. This is in accordance with our findings. One group monitored GCL thickness in IIH [24], and they did not find any significant alterations in GCL thickness during a mean follow-up of less than ten months. In our study, we found a slight but definite modification of GCL thickness during a longer duration follow-up. To our knowledge, we are the first to assess the long-term changes of GCL thickness in patients with

IIH treated by LSS stenting. In the future, prospective collection of GCL thickness data in larger populations of IIH patients will be needed to further understand how this parameter changes in these patients, and perhaps to integrate that into our clinical practice.

Among patients with IIH, 25% may have permanent visual defects which can ultimately lead to blindness [25], and so it is desirable that any treatment effect on visual field outcomes occurs as quickly as possible. The maximal improvement in visual symptoms in the NORDIC trial was not seen until 6 months after initiation of medication [26]. Indeed, one major point of our study was the early benefit of stenting with regards to papilledema. We observed a significant improvement in papilledema as early as one month after the procedure. This information points to the real and early efficacy of stenting in this respect. It could have relevance in the treatment of patients with fulminant IIH [17]. We also know that compliance with medical therapy in IIH patients can be challenging – in one study looking at tolerability in this patient cohort, only 44.1% of patients were able to tolerate the maximum dose of acetazolamide [27]. Shazly et al have demonstrated that venous sinus stenting significantly shortens the duration of medical therapy, improving visual prognosis [21].

In the NORDIC trial, [27] the mean dosage of acetazolamide was 2500 mg/day. This was a significantly higher dosage than the mean doses used in our patient cohort. However, when compared to the NORDIC trial, our patients appeared to be less severe in terms of BMI, CSF pressure, visual acuity or visual field deficits. In addition, in clinical practice we find that these higher doses are often not tolerated by patients due to side effects.

One other important point from our study was the significant delay between onset of IIH symptoms and stenting (28.2 months). Despite this delay, papilledema still significantly

improved with stenting. This supports the concept of relatively rapid reversibility of papilledema after LSS stenting even in long standing IIH without optic nerve atrophy.

The rate of recurrence of IIH following stent placement is around 10%, supporting the need for long-term ophthalmological follow-up in those patients [16,18,28]. The recurrence of IIH is, in many cases, associated with stent-adjacent stenosis (SAS), i.e. a recurrent area of stenosis in the lateral sinus which occurs adjacent to the initial stent. A second stenting allows the resolution of symptoms of IIH in most cases. Factors which have been associated with recurrence of IIH after stenting include extrinsic stenoses, younger patients and higher CSF opening pressure [29]. Some technical points may allow us to decrease the incidence of this recurrence of IIH after stenting, such as implanting longer (or multiple) stents in young patients with extrinsic stenoses to cover the transverse sinus from the torcula to the sigmoid sinus [11]. Even these technical considerations may not allow us to completely prevent IIH recurrences following stenting. Thus, continued clinical and ophthalmological follow-up is warranted.

### *Limitations*

The main limitation of our study is of course the small patient cohort and its retrospective design. A single prospective study has been published so far, albeit with a short follow-up of the patients [17]. Because of the relatively recent development of this stenting technique, the lack of long-term follow-up clinical data remains one of the main limitations common to all studies of LSS stenting in selected patients with IIH.



Our population is not entirely concordant with other those of other groups who treated IIH patients with LSS. Indeed, we have generally not treated patients with fulminant IIH by venous stenting. Our population also seems to have less severe IIH than in other previously published series since the RFNL thickness is lower than in other papers [29,30]. Eleven (36%) of the eyes included in our study had a normal visual field examination before stenting [10], explaining why the improvement of the visual field seems to be less pronounced in our series than in other published studies [17,21,30]. In our population, 94% of patients experienced disabling headaches despite medical therapy. These patients were selected for a stenting procedure by a multidisciplinary discussion between the neurologist, interventional neuroradiologist and ophthalmologist. In this respect, we did not offer stenting for visual symptoms or findings alone. This could also help explain why our population appeared atypical concerning visual findings pre-stenting. Despite these differences, we feel that our results were still relevant with regards to papilledema improvement.

We had a mean follow-up period of 19.7 months. This was relatively long when compared to some previously published cohorts. As outlined in our methods, once patients were stabilized from an ophthalmological point-of-view we stopped following them centrally at our tertiary centre. Patients were referred back if they disimproved. It is therefore likely that most of our cohort had sustained improvement in symptoms following stenting.

## **Conclusion**

According to our results, patients with medically refractory IIH papilledema experienced significant improvements in RNFL thickness as early as one month after LSS stenting. Improvement in visual parameters was sustained over time without recurrence of clinical

symptoms, and the procedure was associated with no significant complications. Sinus stenting was well tolerated, and was associated with rapid and sustained improvements in both qualitative and quantitative measures of visual outcome. Our study supports the concept of early reversibility of papilledema after LSS stenting in most patients with long-standing IIH. Furthermore, most of our patients (94%) were able to discontinue acetazolamide following the procedure. While larger prospective comparative studies are still needed to assess the place of LSS stenting in IIH, our results point to the usefulness of this technique in the specific treatment of visual symptoms and papilledema.

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Table 1. Patients' characteristics		
Characteristics		Number
Demographics data		
	Patient	16
	Eyes	30
	Female	16 (100%)
	Mean age (y)	39.1±11.3
	Mean weight (Kg)	90.3±16.8
	BMI	31.9±7.9
	Mean CSF OP (cmH2O)	30.9±10.4
Clinical and ophthalmical symptoms		
	Papilledema	30/30 (100%)
	Headache	15/16 (93.8%)
	Diplopia	1/16 (6.3%)
	Pulsatile tinnitus	13/16 (81.3%)
	TVO	5/16 (31.3%)
	BCVA (logMAR)	0.036 [+0.4 to +0.0 logMAR]
Acetazolamide		15/16 (93.3%)
	Mean dosage (mg/day)	660.7±270.5

Table 1. Summarized of patient's characteristics before stenting

BMI, Body Mass Index; BCVA, Best Correct Visual Acuity; CSF OP, CerebroSpinal Fluid Opening Pressure; LogMAR, Logarithm of the Minimum Angle of Resolution; TVO, Transient Visual Obscurations

**Table 2 Results**

Outcome	Before Stenting	1 month after Stenting	p	Final follow-up after Stenting	p
Headache	15/16	6/16	0.16	2/16	<0.001
Tinnitus	13/16	1/16	<0.001	1/16	<0.001
Diplopia	1/16	1/16	1	1/16	1
BCVA	0.036 [+0.4 to +0.0 logMAR]	0.036 [+0.4 to +0.0 logMAR]	1	0.032 [+0.4 to +0.0 logMAR]	0.59
Papilledema	16/16	1/16	<0.001	2/16	<0.001
Mean OCT RNFL	121.7 [73 to 204] $\mu$ m	96.2 [71 to 139] $\mu$ m	<0.001	93.4 [77 to 142] $\mu$ m	<0.001
Mean OCT GCL	80.2 [69 to 90] $\mu$ m	79.8 [74 to 90] $\mu$ m	0.23	78.0 [59 to 90] $\mu$ m	<0.01
<b>Visual Fields results</b>					
<b>Scale</b>					
1	11/30	ND		18/30	0.12
2	10/30	ND		5/30	0.23
3	5/30	ND		4/30	1
4	4/30	ND		3/30	1

Table 2. Summarized of results one month after the stenting and at the last follow-up

BCVA, Best Correct Visual Acuity; GCL, Ganglion Cell Layer; OCT, Optical Coherence Tomography; RNFL, Retinal Nerve Fiber Layer.

Results concerning visual fields assessment.

1) normal; 2) enlargement of the blind spot; 3) nasal or temporal defect; 4) diffusely constricted

## **TABLES LEGENDS**

### **Table 1 Patients' characteristics**

Summarized of patient's characteristics before stenting

BMI, Body Mass Index; BCVA, Best Correct Visual Acuity; CSF OP, CerebroSpinal Fluid Opening Pressure; LogMAR, Logarithm of the Minimum Angle of Resolution; TVO, Transient Visual Obscurations

### **Table 2 Results**

Summarized of results one month after the stenting and at the last follow-up

BCVA, Best Correct Visual Acuity; GCL, Ganglion Cell Layer; OCT, Optical Coherence Tomography; RNFL, Retinal Nerve Fiber Layer.

Results concerning visual fields assessment.



1) normal; 2) enlargement of the blind spot; 3) nasal or temporal defect; 4) diffusely constricted

## **FIGURES LEGENDS**

### **Figure 1 Retinal Nerve Fiber Layer (RNFL) thickness changes over time**

Dot plots of the before stenting values, the first follow-up one month after the stenting and the last visit as well as p values for their comparison. Horizontal lines are median and the interquartile range. Each cross represents one patient.

RNFL were significantly reduced as soon as the first month after the stenting ( $p < 0.0001$ ) and at the last visit ( $p < 0.0001$ )

RNFL, Retinal Nerve Fiber Layer; M1, One month after

### **Figure 2 Ganglion Cell Layer (GCL) thickness changes over time**

Dot plots of the before stenting values, the first follow-up one month after the stenting and the last visit as well as p values for their comparison. Horizontal lines are median and the interquartile range. Each dot represents one patient.

GCL were not significantly reduced at the first month after the stenting ( $p=0.23$ ) and at the last visit ( $p < 0.01$ )

M1, One month after