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**Single Case – Headache**

# Trigeminal Neuralgia as a Rare Complication of Idiopathic Intracranial Hypertension

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**Keywords**
Idiopathic intracranial hypertension · Trigeminal neuralgia · Meningocele · Stenting

**Abstract**
***Introduction:*** Idiopathic intracranial hypertension (IIH) is a rare cause of chronic headaches. Usually, patients with IIH present headaches and papilledema with no focal neurological signs. Classical MRI findings feature characteristic signs, i.e., dilated perioptic nerves sheath and empty sella turcica. Rarely, cranial nerve palsies existed, especially abducens nerve palsy. ***Case Presentation:*** We reported herein another rare clinical feature of IIH: trigeminal neuralgia in association with meningocele. This 35-year-old obese woman initially presented with chronic headaches and papilledema. Cerebral MRI showed classical IIH findings. The CSF opening pressure was increased. A treatment coupling acetazolamide and iterative lumbar punctures led to the regression of papilledema, but headaches were difficult to control. Ten years later, while she was lost to follow up and treated with acetazolamide by her *general practitioner*, she developed extremely painful neuropathic pain in the left trigeminal nerve territory, in association with a recurrence of the chronic headaches. A new MRI showed new bilateral cavum trigeminal meningoceles, predominantly on the left side, associated with an atrophy of the cisternal segment of the left trigeminal nerve. Angio-CT showed transverse sinus stenosis, treated by stenting. After this treatment, IIH symptoms disappeared, while trigeminal neuralgia amplified: a surgical procedure led to its complete disappearance. ***Conclusion:*** To our knowledge, this is the first case reported of trigeminal neuralgia associated with meningocele formation in IIH. Our

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case illustrates the great efficacy of venous stenting in IIH, and one may wonder whether earlier stenting could have avoided the subsequent development of meningoceles and subsequent neuralgia.



## Introduction

Idiopathic intracranial hypertension (IIH) is a rare cause of chronic headaches. Its incidence is 1/100,000 in the general population, but it can be as high as 15/100,000 in overweight young women [1]. Its classical presentation combines headaches and papilledema without focal neurological signs and with characteristic MRI findings (dilated perioptic nerves sheath and empty sella turcica) [2, 3]. Untreated, IIH can lead to progressive visual loss.

Cranial nerve palsies, especially abducens nerve palsy, and, more rarely, sensorineural hearing loss, tinnitus, or olfactory dysfunction have also been reported in IIH [1]. We reported herein another rare clinical feature of IIH: trigeminal neuralgia in association with meningocele.

## Case Presentation

A 35-year-old woman with no past medical history presented with chronic holocranial headaches increased in supine position without any other features, particularly no visual deterioration. Body mass index was 35.8. Systematic fundoscopic examination showed bilateral papilledema. Cerebral MRI showed distension of the optic nerve sheaths and empty sella suggestive of IIH, with no meningocele. The CSF opening pressure during lumbar puncture was increased (35 mm Hg). A treatment coupling acetazolamide and iterative lumbar punctures was started, with regression of papilledema on follow-up, but difficulties to control the headaches.

Ten years later, while she was lost to follow up and irregularly treated with acetazolamide by her *general practitioner*, she developed extremely painful neuropathic pain in the left trigeminal nerve territory, mainly in segments V2 and V3, in association with a recurrence of the chronic holocranial headaches. This trigeminal pain occurred in brief episodes, often due to stimulating a trigger zone while chewing or pressing the cheek. Pain was initially alleviated by carbamazepine, but eventually required an increase of dosing over the course of a year. A new MRI showed bilateral cavum trigeminal meningoceles, predominantly on the left side, associated with an evident atrophy of the cisternal segment of the left trigeminal nerve (absent on previous MRI; Fig. 1a–d). Due to increasing difficulties in pain management despite lumbar punctures, the case was collectively discussed. Venous angio-CT showed bilateral transverse sinus extrinsic stenosis (Fig. 1e), which prompted treatment by transverse sinus stenting (carotid wall stent 8 × 40). Before stenting, right transverse sinus stenosis gradient was 10 mm and superior sagittal sinus pressure was 20 mm, versus 13 mm after the stent placement procedure. After this treatment, IIH symptoms disappeared, with sole persistence of the left trigeminal neuralgia. A new lumbar puncture 6 months later showed normal opening pressure, while MRI signs of IIH regressed. Conversely, the trigeminal neuralgia amplified, and a surgical procedure was eventually decided. No neurovascular conflict was found during surgical exploration of the cerebellopontine angle cistern; therefore, section of the inferolateral part of the right V at the level of the pars major was performed, with excellent outcome and complete disappearance of the neuralgia.

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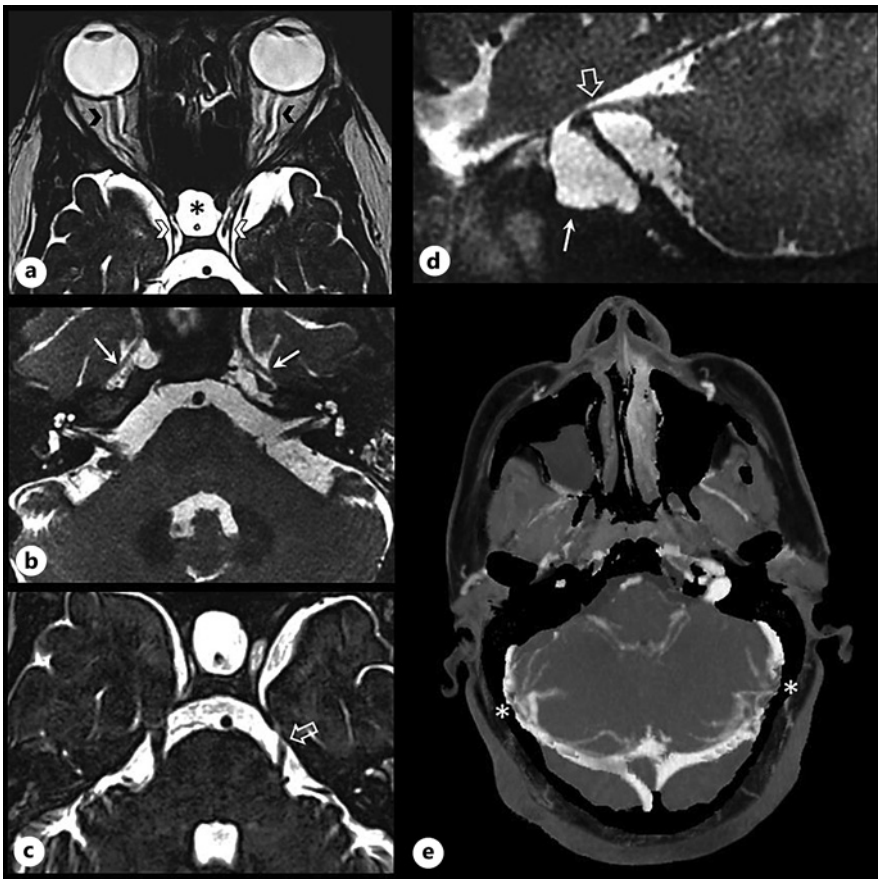


**Fig. 1.** Brain MRI (**a–d**) and venous angio-CT (**e**). **a** Axial HR T2 showing bilateral distension of optic nerve sheaths (black arrowheads), distension of oculomotor nerve sheaths within cavernous sinuses (white arrowheads), and empty sella (black asterisk). **b** Axial HR T2 showing bilateral cavum trigeminal meningoceles (white arrows). **c** Axial HR T2 showing atrophy of left trigeminal nerve (empty arrow). **d** Sagittal HR T2 showing atrophy of left trigeminal nerve (empty arrow) and left cavum trigeminal meningocele (white arrow). **e** Bilateral venous transverse sinus stenosis before stenting (white asterisk). HR, high resolution.

## Discussion

In this paper, we reported a patient with an unusual complication of IIH, namely trigeminal neuralgia secondary to compression by an ipsilateral cavum trigeminal meningocele. A meningocele is a herniation of meninges through a weakness in the skull, which is typically categorized as congenital, iatrogenic, or spontaneous. Meningoceles are asymptomatic most of the time, but can sometimes compress adjacent structures or induce CSF leaks. Some previous reports indicated that they were more frequent in IIH: in a large retrospective radiological survey, 9% of 79 IIH patients were found to have at least one meningocele versus none in the control population [4]. Authors of this study suggested that meningoceles may be counted as an additional classical imaging sign of IIH. Another study reported a case series of 5 patients with idiopathic meningoceles; all of them had empty sella turcica, and the mean pressure in their lumbar puncture was increased (mean: 29 cm $H_2O$) [5]. Elsewhere, a radiological study showed the same association between empty sella and meningocele, suggesting a common etiology between the two radiological features [6].

To this day, the association between IIH and meningocele is not totally understood. Some authors have suggested that the pressure and progressive pulsatile force may gradually erode the cranial vault, leading to meningocele formation [7]. Increasing the available space for CSF, due to inherent weaknesses in the meningocele wall, they may induce small chronic leaks of CSF, helping to alleviate the hyperpressure [4]. This may explain why our patient never developed any ophthalmologic complication apart from a discreet papilledema. Indeed, if Bialer et al. [4] did not report that meningoceles were associated with decreased CSF opening pressure, better visual outcome, or fewer complications, however, patients with meningoceles tended to require less surgical or invasive treatment, although this was not statistically significant.

## Conclusion

Our case report shows the great efficacy of venous stenting in IIH with a complete regression of symptomatology. One may wonder whether earlier stenting could have avoided the development of meningoceles and subsequent trigeminal neuralgia.

The CARE Checklist has been completed by the authors for this case report, attached as online supplementary material (for all online suppl. material, see https://doi.org/10.1159/000544077).

## Statement of Ethics

Written informed consent was obtained from the patient for publication of the details of their medical care and any accompanying images. Ethical approval is not required for this study in accordance with local or national guidelines.

## Conflict of Interest Statement

The authors have no conflicts of interest to declare.

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## Author Contributions

Study concept and design: Mariana Sarov-Riviere and Christian Denier. Acquisition of data: Mariana Sarov-Riviere, Claire Ancelet, Jildaz Caroff, Ghaidaa Nasser, Nozar Aghakhani, and Christian Denier. Analysis and interpretation of data: Mariana Sarov-Riviere, Claire Ancelet, Ghaidaa Nasser, and Christian Denier. Drafting of the manuscript: Mariana Sarov-Riviere, Claire Ancelet, and Christian Denier. Revising it for intellectual content: Jildaz Caroff, Ghaidaa Nasser, and Nozar Aghakhani. Final approval of the completed manuscript: Mariana Sarov-Riviere, Claire Ancelet, Jildaz Caroff, Ghaidaa Nasser, Nozar Aghakhani, and Christian Denier.

## Data Availability Statement

All data generated or analyzed during this study are included in this article and its online supplementary material files. Further inquiries can be directed to the corresponding author.

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