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# Rescue Venous Sinus Stenting for Acute Intracranial Hypertension Following Ovarian Stimulation

CASE REPORT

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## ABSTRACT

*Background: Idiopathic intracranial hypertension (IIH) is characterized by elevated intracranial pressure (ICP) in the absence of structural or infectious causes. IIH primarily affects young women with overweight or obesity and may be precipitated or aggravated by hormonal fluctuations. Severe presentations require immediate multidisciplinary management to prevent permanent visual loss.*

*Case presentation: We report the case of a young obese woman who developed sight-threatening intracranial hypertension a few days after ovarian stimulation, without ovarian hyperstimulation syndrome. The lumbar puncture opening pressure was 50 cm$H_2O$, and venous manometry demonstrated a bilateral 40 mmHg trans-stenotic gradient across the transverse–sigmoid sinus stenoses. Ophthalmic examination revealed severe bilateral Frisén grade 4 papilledema with macular intraretinal fluid. Given the rapid visual deterioration on the first day of admission, urgent venous sinus stenting (VSS) was performed. The procedure resulted in hemodynamic restoration, clinical improvement, and near-complete resolution of macular intraretinal fluid within 24 hours.*

*Conclusion: This case highlights ovarian stimulation as a potential trigger for acute intracranial hypertension in a patient at risk for IIH. It also underscores the efficacy of emergency VSS as rescue therapy, allowing rapid normalization of ICP and preservation of visual function.*

## KEYWORDS

*endovascular treatment, venous sinus stenting, idiopathic intracranial hypertension, ovarian stimulation, papilledema, fulminant IIH*

## INTRODUCTION

IIH is defined by elevated ICP without an identifiable secondary cause, in the absence of a mass lesion, hydrocephalus, or central nervous system infection.[1,2] It predominantly affects young women with overweight or obesity.[1] Although its precise pathophysiology remains incompletely understood,[2] specific conditions or medications can exacerbate or unmask the disease. Hormonal and metabolic factors—including obesity, polycystic ovarian syndrome (PCOS), and exposure to exogenous hormones—are suspected contributors to the development or aggravation of IIH.[3–6]

Recent evidence suggests that disturbances in estrogen and progesterone signaling may influence cerebrospinal fluid (CSF) dynamics by altering choroid plexus function and intracranial venous outflow resistance.[7] Moreover, assisted reproductive technology (ART) and ovarian stimulation protocols transiently expose patients to supraphysiological estrogen levels, potentially triggering intracranial hypertension in susceptible individuals. A single prior case reported IIH in the setting of ovarian hyperstimulation syndrome following in vitro fertilization.[6] However, the occurrence of acute IIH in the absence of ovarian hyperstimulation syndrome has not been previously documented. In patients with associated transverse venous sinus stenosis, VSS has emerged as an effective therapy,[8,9] particularly for fulminant forms threatening visual function.

We report the case of a young obese woman who developed symptoms of acute intracranial hypertension shortly after ovarian stimulation and was successfully managed with emergency VSS.

## CASE PRESENTATION

A 17-year-old Caucasian woman presented to the ophthalmology emergency department with a 5-day history of sudden-onset binocular horizontal diplopia, headache, and neck stiffness. Her medical history included PCOS diagnosed 1 year earlier. She denied prior headaches, recent weight gain, or symptoms suggestive of obstructive sleep apnea. Her body mass index (BMI) was 34 kg/m$^2$. Recent laboratory investigations were unremarkable, with no evidence of anemia.

Symptoms began 5 days after an oocyte retrieval procedure performed as part of an ART

protocol. Ovarian stimulation included menotropin (Fertistartkit®, Genevrier, France; 112.5–150 IU daily for 10 days), ganirelix (Orgalutran® 0.25 mg, MSD, France; for 5 days), and a single dose of triptorelin (Decapeptyl® 0.2 mg, Ipsen, France). The oocyte retrieval and embryo cryopreservation were uneventful, and no ovarian hyperstimulation syndrome occurred. Ovarian stimulation was performed for elective fertility preservation in the context of PCOS with risk of premature ovarian insufficiency.

She reported progressive headache with nausea, without pulsatile tinnitus or transient visual obscurations. Neurological examination revealed bilateral abducens nerve palsy without focal deficits or relative afferent pupillary defect. Best-corrected visual acuity (BCVA) was 20/20 in the right eye and 20/22 in the left eye. Fundus examination revealed bilateral optic nerve swelling (Frisén grade 4). Spectral-domain optical coherence tomography (SD-OCT) demonstrated severe retinal nerve fiber layer (RNFL) thickening and bilateral peripapillary intraretinal fluid (Figure 1).

Brain magnetic resonance imaging (MRI) showed indirect signs of intracranial hypertension (optic nerve sheath distension, empty sella), and bilateral transverse sinus stenosis, with no evidence of cerebral venous thrombosis or mass lesion. Lumbar puncture and venous manometry via a transfemoral approach were performed. The CSF opening pressure was 50 $cmH_2O$, with normal CSF composition. Venous manometry was performed using a microcatheter advanced sequentially into the cerebral venous sinuses. Pressure measurements were obtained at rest with the patient supine under local anesthesia. Venous manometry revealed intracranial venous hypertension, with pressures of 50 mmHg in the torcular and superior sagittal sinus and a 40 mmHg gradient across both transverse-sigmoid junctions (Figures 2 and 3).

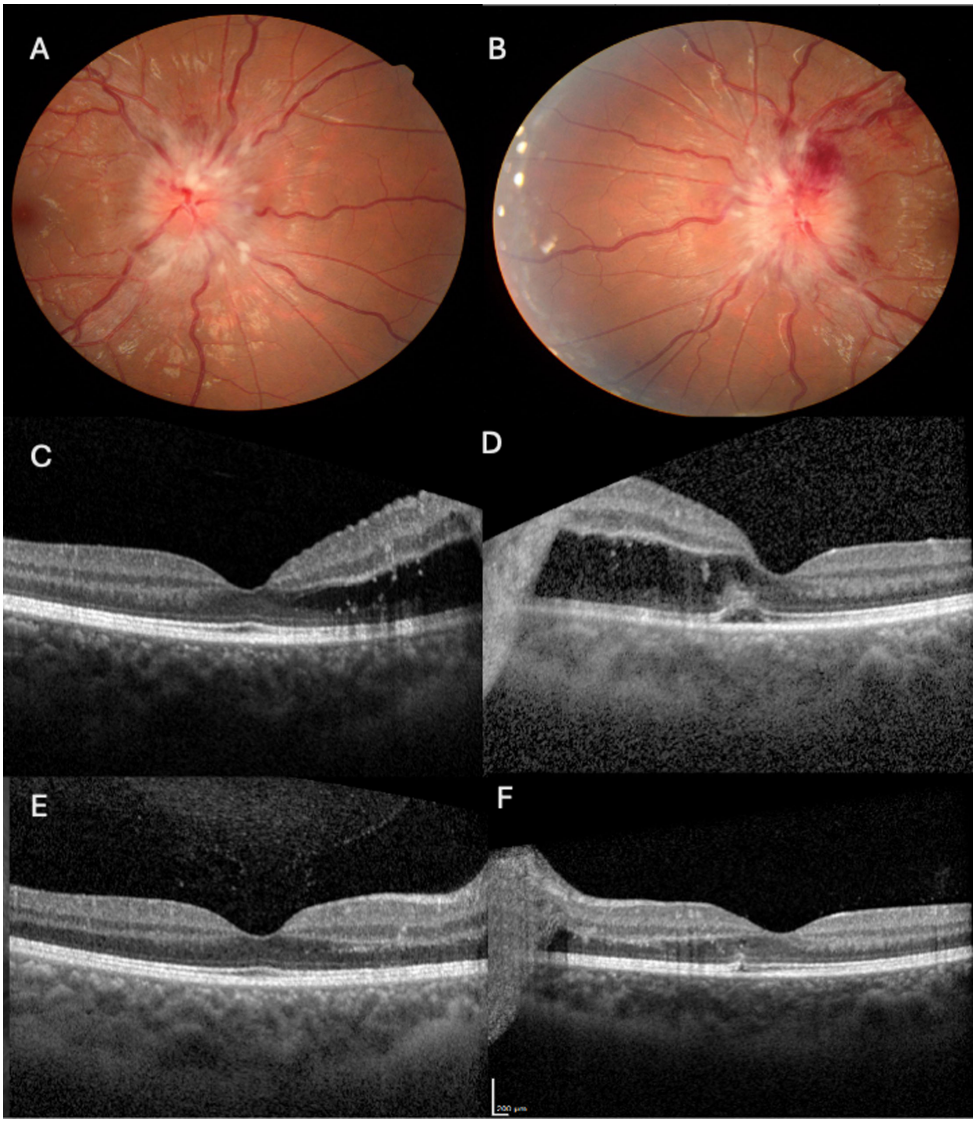


**FIGURE 1.** Severe papilledema and OCT follow-up demonstrating intraretinal fluid. Fundus examination at presentation shows bilateral grade 4 papilledema with peripapillary hemorrhages (A, B). Baseline (SD-OCT, acquired with Spectralis OCT, Heidelberg Engineering, Germany) reveals significant peripapillary intraretinal fluid threatening the macula (C, D). Post-VSS SD-OCT acquired 24 hours after venous sinus stenting demonstrates near-complete resolution of the intraretinal fluid (E, F). SDOCT, spectral-domain optical coherence tomography; OCT, optical coherence tomography; VSS, venous sinus stenting.

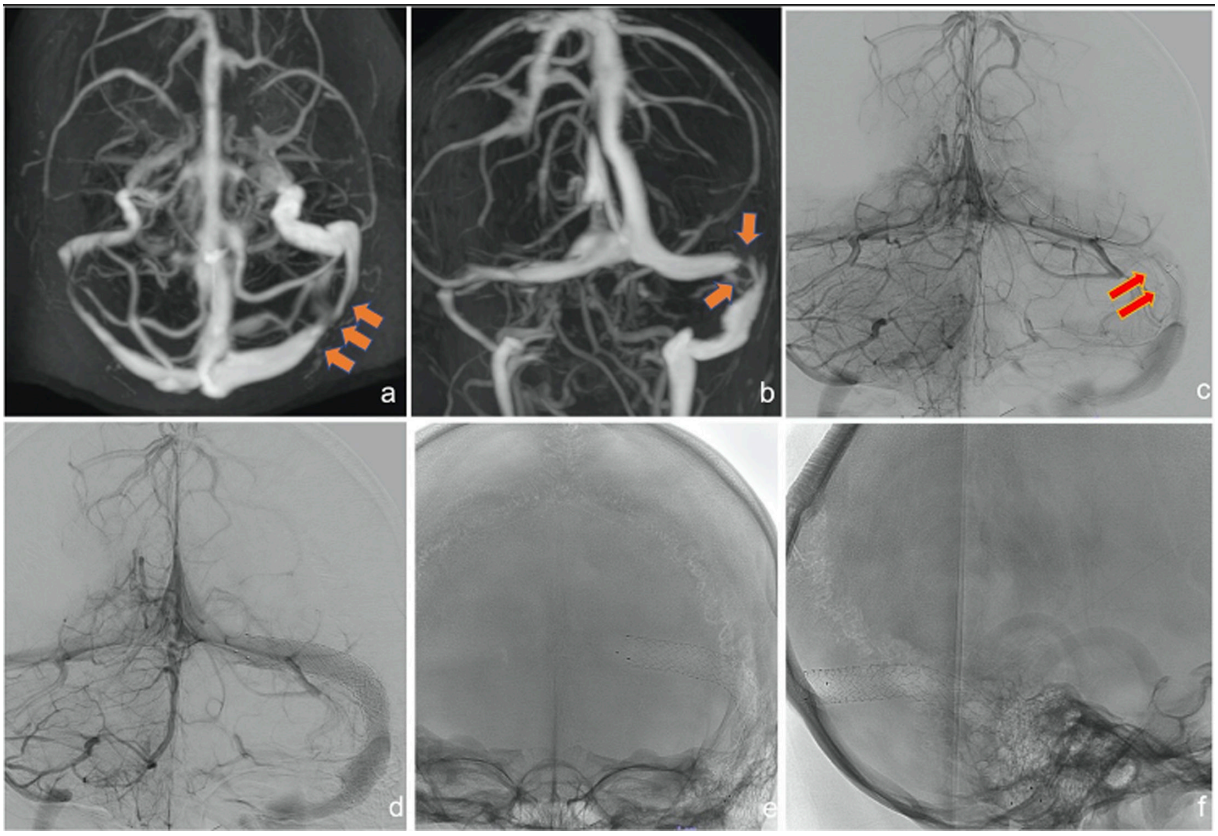


**FIGURE 2.** Cerebral venous sinus stenosis and endovascular reconstruction. Pre-operative venous MR angiography (a, b) and digital subtraction angiography (c) show stenosis of the dominant left transverse sinus (arrows). Angiography after stent placement demonstrates restoration of the venous sinus caliber (d, e, f).

The following day, the patient experienced visual deterioration (BCVA 20/25 in both eyes) with worsening headaches. Urgent left-sided transverse VSS was performed. The dominant left transverse sinus was reconstructed using a 7 mm × 80 mm Tentos® stent (Figure 3). Intraprocedural platelet inhibition was achieved with cangrelor (P2Y12 reaction units = 5; inhibition = 98%), followed by dual oral antiplatelet therapy (ticagrelor 90 mg twice daily and aspirin 100 mg). Initially, the patient's immediate pregnancy plan contraindicated the use of acetazolamide. Within 24 hours after the procedure, headaches and diplopia improved markedly, and macular intraretinal fluid had nearly resolved on OCT (Figure 1). Four days after stenting, follow-up venous manometry demonstrated normalized intracranial venous pressures (15 mmHg in the torcular and superior sagittal sinus) with no residual trans-stenotic gradient. The planned embryo transfer was postponed, and acetazolamide 2,000 mg per day was initiated.

At the 1-month follow-up, while receiving acetazolamide 1,500 mg per day, BCVA was 20/20 in both eyes. Near-complete resolution of bilateral abducens palsy was observed, with progressive RNFL thickness normalization (141 µm in the right eye and 158 µm in the left eye) and no macular ganglion cell complex thinning. Three-month follow-up venous pressures remained normal (14 mmHg in the torcular sinus with no gradient).

At 7 months, papilledema had fully resolved (RNFL thickness was 103 µm in the right eye and 105 µm in the left eye), with preserved macular ganglion cell layer thickness (mean, 52 µm in the right eye and 50 µm in the left eye) with no functional impairment.

## DISCUSSION

This patient rapidly developed symptoms consistent with raised ICP a few days after exposure to high levels of exogenous gonadotropins, without ovarian hyperstimulation syndrome.[6] Whether ovarian stimulation precipitated de novo IIH or unmasked a previously subclinical condition remains uncertain. Indeed, the patient's background (young woman with obesity and PCOS) represents a known risk profile for IIH.[1,2,10] Although a causal relationship cannot be established from this single case report, the temporal sequence and the type of treatment raise this possibility.

This case supports systematic fundus examination in at-risk patients undergoing ART,[11] or at minimum specific screening for symptoms suggestive of IIH during follow-up, including diplopia, headache, and tinnitus.

This case report emphasizes two key points. First, it supports the hypothesis that estrogen excess and hormonal disequilibrium may play a role in the pathogenesis of raised ICP in IIH.[12] Although evidence remains limited, we can hypothesize that rapid and elevated estrogen levels may alter CSF dynamics and venous outflow regulation through effects on the choroid plexus.[7] In this patient, the rapid onset of symptoms following ovarian stimulation suggests that supraphysiological estrogen exposure may have altered CSF production and venous outflow regulation, unmasking or precipitating a severe IIH phenotype. This case therefore strengthens the concept of hormone-related

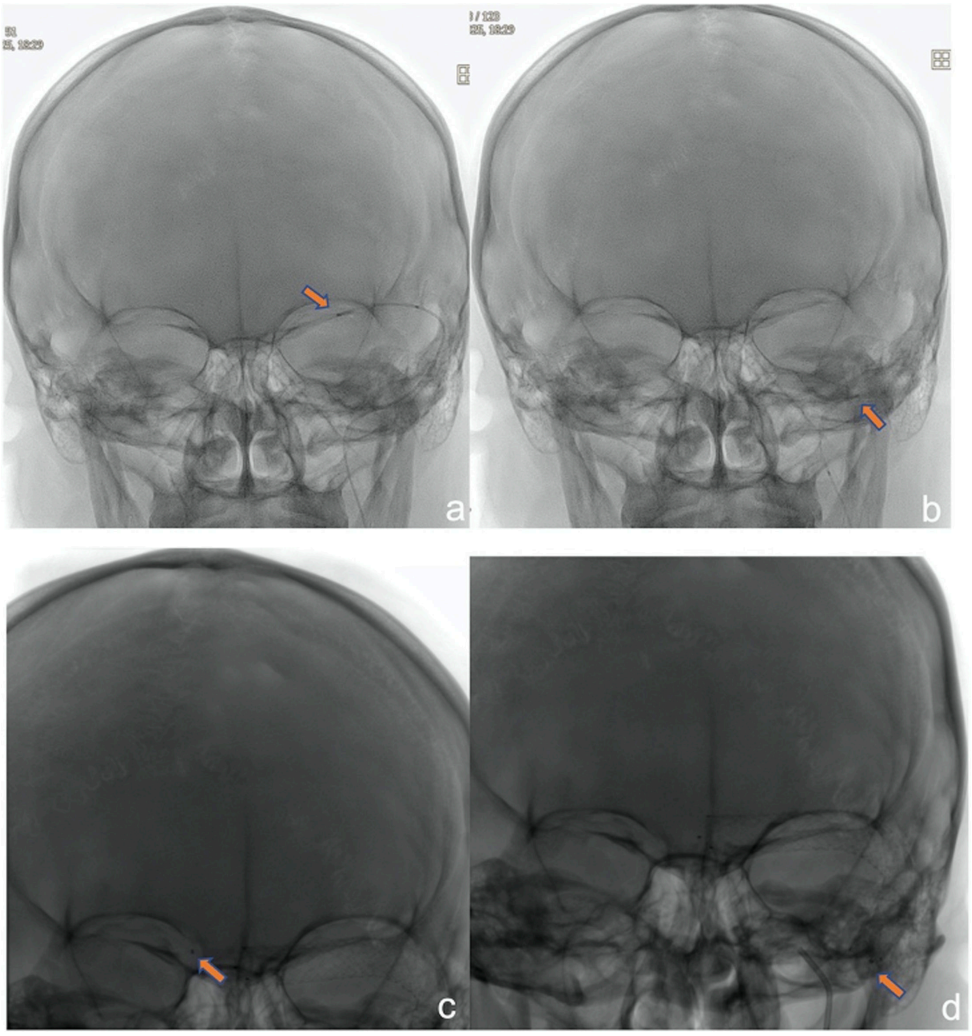


**FIGURE 3.** Intracranial venous manometry and pressure gradient resolution. Measurement of pressure gradients at baseline (a, b) and 4 days after venous stent placement (c, d). The arrow indicates the tip of the microcatheter.

vulnerability in IIH, particularly under conditions of abrupt estrogen fluctuation.

Second, the prompt normalization of venous pressures and resolution of papilledema following VSS underscore the effectiveness of this endovascular treatment in selected cases of severe IIH approaching the definition of fulminant IIH, in which visual function is at risk.[13] While medical management is the traditional first-line approach, our patient presented with severe papilledema, macula-threatening intraretinal fluid, and a severe 40 mmHg trans-stenotic gradient. The immediate normalization of venous pressures following stent deployment correlated directly with the near-complete resolution of macular intraretinal fluid on OCT within 24 hours. A 2024 meta-analysis of 36 studies encompassing more than 1,000 patients confirmed VSS as a safe and effective intervention, with papilledema improvement in 89% of cases.[9] Early recognition and multidisciplinary management remain crucial to prevent permanent visual loss.


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### Patient consent

Informed consent was obtained from the patient and her legal guardian for participation in this case report and for the publication of their case details.

### Contributors

All authors contributed substantially to the conception, design, and execution of this case report. All authors reviewed and approved the final version of the manuscript for submission. Marie Latypov and Antonio Marrazzo were involved in writing, editing, and the literature review for this manuscript. Max Villain, Federico Cagnazzo, Vincent Daien, Vincent Costalat, and Eloi Debourdeau performed critical revisions and editing of the manuscript. Max Villain, Federico Cagnazzo, Cyril Dargazanli, and Marie Duport-Percier were involved in the care of this patient.

### Conflicts of interest

All authors declare no competing interests.


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### Data Availability Statement

All data are available from the corresponding author upon reasonable request.

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