

Recurrent syncope after hysteroscopy finally diagnosed as cerebral venous sinus thrombosis: a case report



Yongqing Zhang, MD; Hongxing Ye, MD; Danqing Chen, MD, PhD; Guohui Yan, MD; Zhanfu Li, MD; Qianhui Xie, MD; Guodong Shan, MD; Zhaoxia Liang, MD, PhD

Cerebral venous sinus thrombosis (CVST) is a rare cerebrovascular condition that causes obstruction of venous blood flow or cerebrospinal fluid circulation, leading to intracranial hypertension symptoms such as syncope and vomiting. Transurethral Resection of the Prostate (TURP) syndrome, a complication of hysteroscopic surgery, can also present with cerebral edema and symptoms similar to CVST, making differentiation challenging. Cases of unexplained recurrent syncope posthysteroscopy diagnosed as CVST have not been previously reported. We present a case of a 33-year-old woman who underwent hysteroscopy for abnormal uterine bleeding. Five hours postoperation, she experienced syncope, which resolved spontaneously. Ten hours later, she had recurrent syncope, nausea, and vomiting. Head computed tomography suggested cerebral edema, potentially indicating TURP syndrome. However, the brief surgery and minimal fluid imbalance (200 mL) made TURP syndrome unlikely. Further investigation with cranial magnetic resonance venography revealed multiple venous sinus thromboses, explaining the recurrent syncope linked to minimal fluid volume expansion postsurgery. Subsequent screening identified hyperhomocysteinemia as a contributing factor. Treatment with anticoagulants, folic acid, and vitamin B6 led to a favorable outcome. This case involves recurrent syncope after hysteroscopy secondary to CVST. If symptoms such as recurrent syncope, nausea, and vomiting, indicating increased intracranial pressure, arise posthysteroscopy and cannot be attributed to typical TURP syndrome, primary intracranial vascular conditions like CVST should be considered, as even a minor increase in blood volume can exacerbate these intracranial pressure symptoms.

Key words: cerebral venous sinus thrombosis, hyperhomocysteinemia, hysteroscopy, syncope

Introduction

Cerebral venous sinus thrombosis (CVST) refers to a type of cerebrovascular disease characterized by the formation of thrombi in the cerebral venous sinuses, leading to obstruction of blood return or cerebrospinal fluid circulation, resulting in intracranial hypertension and focal damage.¹ Previously reported incidence rates range from 1.3 to 2.6 per 100,000.^{2,3} Typically affecting younger patients, CVST is believed to result from a variety

of underlying factors, including genetic or secondary thrombophilic conditions (such as Factor V Leiden mutation, prothrombin G20210A mutation, hyperhomocysteinemia, deficiencies in protein C, protein S, or antithrombin III), pregnancy, postpartum state, oral contraceptives, obesity, and infections.^{1–3}

Hysteroscopic surgery is a common minimally invasive gynecological procedure used to diagnose and treat abnormalities of the uterine cavity. Among known

risks, Transurethral Resection of the Prostate (TURP) syndrome—usually associated with excessive absorption of irrigating fluid—can sometimes present with symptoms of brain edema, including cerebral edema and altered consciousness.^{4,5}

Here, we report a case of a 33-year-old woman who experienced recurrent syncope following hysteroscopic surgery, where typical TURP syndrome could not explain the observed symptoms. MRI venography (MRV) revealed CVST, which

From the Department of Obstetrics and Gynecology, Women's Hospital, School of Medicine, Zhejiang University, Hangzhou, China (Zhang, Chen, Yan, and Liang); Department of Obstetrics and Gynecology, People's Hospital of Haixi Autonomous Prefecture, Delingha, Qinghai, China (Zhang, Li, Xie, and Liang); Department of Neurosurgery, The First Affiliated Hospital of Zhejiang University, Hangzhou, China (Ye); Department of Neurosurgery, People's Hospital of Haixi Autonomous Prefecture, Delingha, Qinghai, China (Ye); Department of Gastroenterology, The First Affiliated Hospital of Zhejiang University, Hangzhou, China (Shan); Department of Gastroenterology, People's Hospital of Haixi Prefecture of Qinghai Province, Delingha, China (Shan)

Y.Z. and H.Y. have contributed equally to this work.

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Corresponding author: Zhaoxia Liang, MD, PhD. xiaozaizai@zju.edu.cn

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accounted for the intracranial hypertension symptoms triggered by a slight increase in systemic fluid after the hysteroscopic surgery. Further etiology screening revealed a hereditary heterozygous missense mutation (CT heterozygote) in the methylenetetrahydrofolate reductase (MTHFR) gene and hyperhomocysteinemia, which may have contributed to the formation of the CVST. The patient achieved a favorable recovery with anticoagulation therapy, folic acid, and vitamin B6 supplementation. This case highlights the importance of monitoring unusual symptoms after hysteroscopic surgery, considering a broad differential diagnosis including CVST, and the need for comprehensive postoperative evaluation and multidisciplinary management to optimize patient outcomes.

Case presentation

A 33-year-old woman presented to a tertiary medical institution with “irregular vaginal bleeding for 15 days.” She is Gravida 1, Parity 1, having delivered a 3300 g baby boy at full term in her 20s. The medical history shows that she has a history of bilateral tubal ligation and appendectomy. She was born in a county located at an altitude of 3100 meters. She does not smoke or consume alcohol. Her menstrual cycles have been regular, with no history of dysmenorrhea. This is her first occurrence of abnormal uterine bleeding (AUB). However, the bleeding was significantly different from her usual menstrual cycles, lasting for 15 days without resolution. The patient has not used emergency contraceptives or hemostatic drugs before her hospitalization. Family history reveals that her father passed away about 10 years ago from hypertensive nephropathy, while her mother and two sisters are in good health.

Her physical examination revealed no abnormalities. She is 170 cm tall and weighs 52.5 kg (body mass index of 18.2 kg/m²). Gynecological examination showed only a small amount of dark red blood in the vagina, with no abnormalities detected in the uterus, cervix, or adnexa. Ultrasound indicated that the uterus and bilateral adnexa were normal, with an endometrial thickness of 0.6 cm

(double-layer). Laboratory tests revealed the following: complete blood count showed a white blood cell count (WBC) of $4.0 \times 10^9/L$ and hemoglobin (Hb) of 138 g/L, with normal coagulation function and D-dimer at 0.23 mg/L (reference: 0–0.55 mg/L). The beta-human chorionic gonadotropin (β -HCG) test ruled out pregnancy (β -HCG <5 U/L); blood glucose was 4.51 mmol/L; potassium was 3.76 mmol/L, sodium was 137.4 mmol/L, and chloride was 104.4 mmol/L. The electrocardiogram (ECG) indicated sinus bradycardia (49 beats per minute).

The patient underwent hysteroscopy and diagnostic curettage on the third day after her hospitalization, due to “abnormal uterine bleeding.” Intravenous anesthesia with a laryngeal mask was administered during the procedure, and 5% glucose was used as the uterine distention medium at a pressure of 100 mmHg. The uterine cavity depth measured 9 cm, and intraoperative findings revealed endometrial thickening on the posterior wall, while other areas appeared normal. A comprehensive curettage was performed, and after completing the procedure, the hysteroscope was reinserted to confirm that the endometrium had been thoroughly curetted. The estimated blood loss was approximately 5 mL. The surgery lasted 30 minutes, with a fluid input of 1000 mL and an output of 800 mL, leaving a difference of 200 mL. A small sample of endometrial tissue was sent for pathological examination. The patient had fasted for 12 hours prior to the procedure.

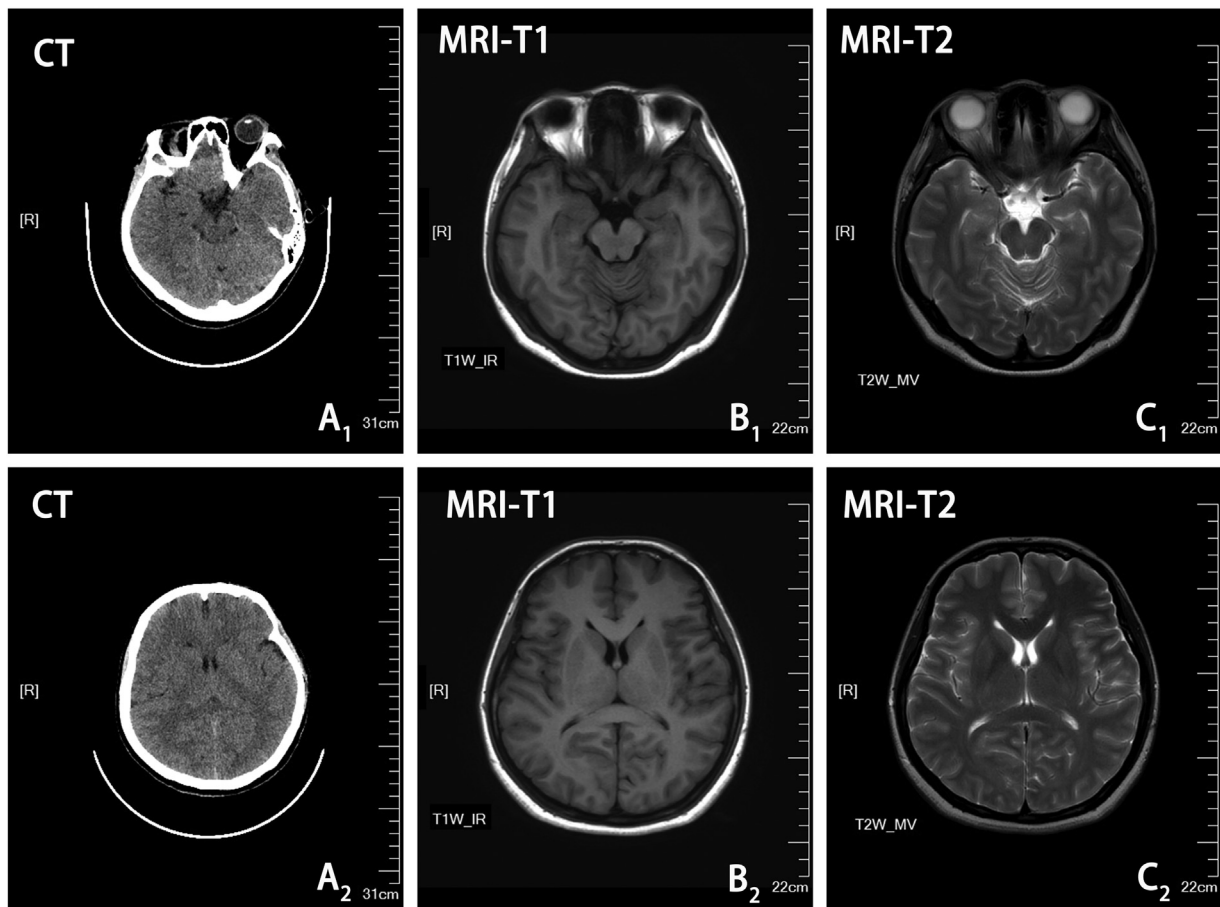
After surgery, the patient was transferred back to the ward, with vital signs showing a temperature of 36.2°C, pulse 74 bpm, respiration 18 bpm, blood pressure 100/60 mmHg, and clear consciousness. She received 0.6 g of clindamycin (q8h) for infection prevention and 0.5 g of tranexamic acid with 500 mL of 5% glucose for hemostasis. Five hours later (at 16:00), the patient reported chest tightness, followed by drowsiness and unresponsiveness. Continuous ECG monitoring showed a transient heart rate of 120 to 127 bpm, oxygen saturation at 98%, and blood pressure at 127/80 mmHg. Oxygen was

administered at 3 L/min, and she regained consciousness after about a minute but still felt chest tightness. All medications were discontinued, with no rashes observed. Consultation with cardiology indicated sinus rhythm with a heart rate of 59 bpm. Lab tests showed WBC $8.24 \times 10^9/L$, Hb 127 g/L, D-dimer 0.29 mg/L, blood glucose 7.4 mmol/L, potassium 3.48 mmol/L, sodium 137.1 mmol/L, and chloride 107.8 mmol/L. Dexamethasone 10 mg was administered to prevent allergic reactions. The patient’s symptoms improved, but she remained fatigued.

Ten hours postsurgery (at 21:00), the patient experienced nausea and vomiting, followed by a syncope episode lasting about 1 minute, which resolved spontaneously. She appeared fatigued, with a blood pressure of 120/80 mmHg and blurred vision. Bedside ECG monitoring was performed, and the intensive care unit (ICU) was consulted. Pupillary response was normal, tongue extension showed no deviation, and there was no neck stiffness. The Glasgow Coma Scale score was 13 (Eye Opening 4, Verbal Response 5, Motor Response 4). Ten minutes later, she experienced syncope again for 1 minute but recovered spontaneously. The ICU physician suspected increased intracranial pressure and administered 125 mL of mannitol intravenously and 5 mg of dexamethasone to reduce cerebral edema. Head CT scan revealed mild cerebral edema (Figure 1, A1 and A2). After treatment, the patient improved, with no further syncope and increased urine output. Repeat tests showed WBC $8.07 \times 10^9/L$, Hb 137 g/L, D-dimer 0.29 mg/L, potassium 3.47 mmol/L, sodium 133.6 mmol/L, chloride 102.7 mmol/L, and normal cardiac enzyme levels. NT-proBNP was 213 ng/L (reference value: 0–133.0 ng/L). Coagulation function was normal with a D-dimer of 0.220 mg/L. Mannitol and dexamethasone continued, and her condition stabilized. Considering the hysteroscopy history and electrolyte status, she likely experienced water intoxication, with fluid overload and dilutional hyponatremia (TURP syndrome).

However, several questions remain regarding this case: (1) The patient’s

FIGURE 1
CT and MRI findings of the patient



Parts (A1 and A2) represent the intracranial CT scan of the patient on the day of hysteroscopy, revealing swollen brain tissue in the ambient cistern and frontal horn levels, with unclear sulci and gyri, indicating signs of cerebral edema. Parts (B1 and C1) correspond to the T1-weighted and T2-weighted images at the ambient cistern level on the second postoperative day; parts (B2 and C2) correspond to the T1-weighted and T2-weighted images at the frontal horn level. After dehydration and intracranial pressure reduction treatment, MRI shows reduced brain tissue swelling, with clearer sulci and gyri, indicating relief of cerebral edema.

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surgery lasted only 30 minutes, with a fluid input-output difference of just 200 mL. The fluid entering the body was minimal, and postoperative electrolyte tests indicated mild hypokalemia, while sodium levels were normal (137.1 mmol/L). Despite this, the patient exhibited significant symptoms of increased intracranial pressure (cerebral edema), which cannot be fully explained by TURP syndrome alone. (2) The patient also experienced repeated episodes of syncope postoperatively—could there be an underlying neurological issue?

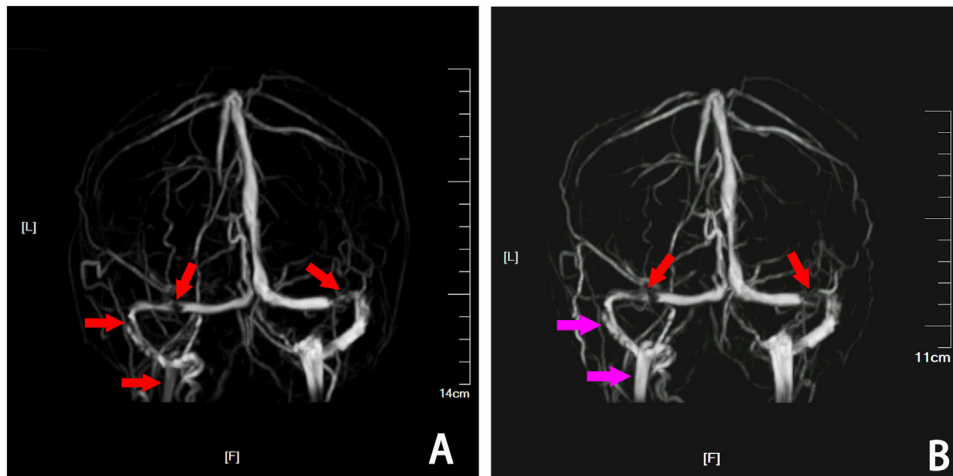
Following a neurosurgical consultation, cranial MRI and MRV were

performed. The cranial MRI revealed no significant abnormalities (Figure 1, B1, B2, C1, C2); however, the MRV indicated possible chronic thrombosis in the bilateral internal jugular veins, sigmoid sinus, and transverse sinus (Figure 2, A, indicated by the red arrows). Further history revealed intermittent headaches over the past 2 years, treated with ibuprofen, with no family history of thrombotic disorders or autoimmune diseases. The endometrial pathology results indicated a proliferative endometrium. According to the findings from the hysteroscopy, preoperative ultrasound, and endometrial pathology report, as well as the FIGO

(International Federation of Gynecology and Obstetrics) classification system for AUB, the most likely cause of this patient's AUB is AUB-O (ovulatory dysfunction).⁶

Based on the patient's MRV results, multiple chronic venous sinus thromboses were identified intracranially. This has increased the resistance of her intracranial venous return to the right atrium. During hysteroscopic surgery and postoperative fluid infusion, even a slight and short-term increase in blood volume can significantly elevate this resistance, leading to symptoms of intracranial hypertension, such as syncope and vomiting. The diagnosis is

FIGURE 2
MRV findings of the patient



(A) On the second postoperative day, the MRV indicated linear and nodular low-signal shadows in the transverse sinus, sigmoid sinus, and internal jugular vein, suggesting chronic thrombosis (as indicated by the red arrows). (B) Follow-up MRV at 1.5 months still showed chronic thrombosis in the transverse sinus, sigmoid sinus, and internal jugular vein; however, there was significant improvement in the patency of the left sigmoid sinus and left internal jugular vein (as indicated by the pink arrows), while other intracranial venous sinuses showed no significant changes in filling defects (as indicated by the red arrows).

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now clear and includes: (1) chronic CVST; (2) AUB-O; and (3) secondary intracranial hypertension following hysteroscopy. After 3 days of treatment with mannitol to reduce intracranial pressure and dexamethasone to prevent cerebral edema, the patient's symptoms have resolved, and she is now fully mobile. However, the patient declined further lumbar puncture and cerebral vascular Digital Subtraction Angiography examination.

The differential diagnosis of CVST includes acute cerebral infarction, intracerebral hemorrhage, and brain tumors. The main distinguishing features between CVST and these other conditions are the findings of corresponding venous sinus occlusion and poor venous enhancement on MRV examination.

Is the hypercoagulable state in this case a compensatory change due to AUB? We conducted further discussion and concluded that in this case, CVST is not considered to be caused by this episode of AUB for the following reasons: (1) The patient's AUB occurred within the past 15 days, and she had no history of oral contraceptive use. If CVST had

developed within such a short period, the patient would typically present with acute symptoms such as headache, vomiting, or signs of increased intracranial pressure. However, the patient did not exhibit any such symptoms prior to the surgery. (2) Acute CVST is usually associated with a significant increase in coagulation markers, especially D-dimer. However, the patient's preadmission coagulation test showed a D-dimer level of 0.23 mg/L, which is within the normal reference range of 0 to 0.55 mg/L. Considering these two factors, it is unlikely that the CVST was caused by a hypercoagulable state induced by this episode of AUB.

We conducted a comprehensive screening to investigate the causes of CVST in this patient. The antinuclear antibody test showed no abnormalities, and the lupus anticoagulant and antiphospholipid antibody tests did not support a diagnosis of antiphospholipid syndrome. Genetic screening revealed no Factor V Leiden mutation or Prothrombin G20210A mutation. However, thrombophilia screening identified a heterozygous mutation (CT type) in the

MTHFR gene, which can impair folate metabolism, leading to hyperhomocysteinemia and increasing the risk of thrombosis. Therefore, we further monitored the patient's homocysteine levels, which were elevated at 15.4 $\mu\text{mol/L}$ (reference value $<15 \mu\text{mol/L}$), confirming our hypothesis.

The treatment plan we provided for this woman was as follows: rivaroxaban 15 mg once daily for anticoagulation, folic acid 800 mg once daily, and vitamin B6 10 mg once daily. After 1 month of follow-up via phone, she reported no recurrence of symptoms such as headache or syncope and was living normally. After 1.5 months, a follow-up MRV of the cranial region still showed chronic thrombosis in the transverse sinus, sigmoid sinus, and internal jugular vein; however, there was significant improvement in the patency of the left sigmoid sinus and left internal jugular vein, while other intracranial venous sinuses showed no significant changes in filling defects (Figure 2, B, indicated by the pink arrow). The repeat homocysteine level had decreased to 10.3 $\mu\text{mol/L}$. All of the above indicates that

our treatment has shown initial effectiveness. However, considering that thrombus absorption may take a longer time, we decided to continue the current treatment plan and monitor her intracranial thrombus status and homocysteine levels.

Discussion

This is a case of recurrent syncope following hysteroscopy, diagnosed by a multidisciplinary team as secondary to CVST. The most common cause of syncope after hysteroscopy is water intoxication (TURP syndrome), typically caused by the absorption of large amounts of distension fluid (>1000 mL), which can lead to serious complications such as pulmonary and cerebral edema.⁵ In this case, the patient also had CVST, where even a small increase in circulating fluid triggered recurrent syncope. Although rare, clinicians should consider the possibility of intracranial pathology, such as CVST, when evaluating unexplained syncope after hysteroscopy to prevent severe intracranial hypertension and life-threatening complications due to fluid overload.

We conducted a search in *PubMed* and *Web of Science* using the keywords “hysteroscopy and cerebral venous sinus thrombosis” or “hysteroscopy and cerebral thrombosis,” revealing our case as a rare cause of syncope that occurred after hysteroscopy and has not been previously documented in the literature. Typically, when syncope occurs after hysteroscopy, clinicians will first consider TURP syndrome and initiate treatment without further investigation. However, in this case, the presentation differed from typical TURP syndrome. When conventional causes could not explain the symptoms, a multidisciplinary consultation was conducted, leading to a definitive diagnosis through cranial MRI and MRV. This represents a significant aspect of innovation in this case. The presence of CVST obstructed venous return in the brain, and even a

small increase in circulating volume was sufficient to raise intracranial pressure, resulting in recurrent episodes of syncope.

After the patient was diagnosed with CVST, we investigated related factors, including genetic or secondary thrombophilic conditions. We identified abnormalities in MTHFR mutation and hyperhomocysteinemia, both reported as risk factors in CVST.^{1–3,7} Hyperhomocysteinemia may contribute to thrombosis through mechanisms such as oxidative damage to the vascular endothelium, smooth muscle hyperplasia leading to lumen narrowing, increased platelet activation and aggregation, and disruption of the coagulation balance.⁸ This case highlights the importance of multidisciplinary collaboration in diagnosing recurrent syncope following hysteroscopic surgery and demonstrates the value of MRV as a diagnostic tool. Additionally, it emphasizes the necessity of etiological screening for CVST, which is crucial for subsequent targeted treatment.

Conclusion

Our study emphasizes the importance of ongoing assessment and attention after hysteroscopy, particularly when patients continue to report discomfort. If symptoms such as recurrent syncope, nausea, and vomiting—suggestive of increased intracranial pressure—occur after the procedure and cannot be explained by typical TURP syndrome, the possibility of primary intracranial vascular conditions, such as CVST, should be considered, as even a minor increase in circulating blood volume may trigger these symptoms. Etiological screening for CVST is essential, as it plays a crucial role in guiding subsequent targeted treatment. ■

CRediT authorship contribution statement

Yongqing Zhang: Writing – original draft, Conceptualization. **Hongxing Ye:**

Writing – original draft, Data curation. **Danqing Chen:** Formal analysis. **Guohui Yan:** Methodology. **Zhanfu Li:** Formal analysis. **Qianhui Xie:** Resources, Investigation. **Guodong Shan:** Writing – review & editing, Supervision. **Zhaoxia Liang:** Supervision. ■

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