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CASE REPORT

The Paradox of Clot and Bleed: A Rare Presentation of Extensive Cerebral Venous Thrombosis with Subarachnoid Hemorrhage in a Young Woman

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ABSTRACT

Background: Cerebral venous thrombosis (CVT) is an uncommon but important cause of stroke, more frequently seen in young females. Several risk factors have been identified, including pregnancy, the puerperium, anemia, polycystic ovarian syndrome (PCOS), and the use of oral contraceptive pills (OCPs).

Case Presentation: We report the case of a 23-year-old woman who presented to the emergency department with a sudden-onset, severe thunderclap headache. She had been on combined oral contraceptive pills (ethinyl estradiol 0.03 mg + desogestrel 0.15 mg, Miliana) for the past three months for menstrual cycle regulation. Neuroimaging revealed cerebral venous thrombosis complicated by subarachnoid hemorrhage (SAH) a rare and diagnostically challenging presentation.

Management and Outcome: The patient was managed with anticoagulation therapy under close neurological monitoring, with careful discontinuation of OCPs. Despite the initial severity, she made a complete neurological recovery.

Conclusion: This case highlights the need for heightened clinical suspicion of CVT in young women presenting with severe headache, especially those on hormonal contraceptives. Early diagnosis and appropriate anticoagulation, even in the presence of SAH, can result in favourable outcomes. A multidisciplinary approach is essential in managing the complexities of such cases

KEYWORDS

• Cerebral venous thrombosis • Subarachnoid hemorrhage • Oral contraceptives • Anticoagulation • Stroke • Young female • Venous sinus thrombosis • Hemorrhagic infarction

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INTRODUCTION

Cerebral venous thrombosis (CVT) accounts for less than 1% of all strokes, yet it carries significant morbidity, particularly in young females.[1] Cerebral venous thrombosis (CVT) is characterized by thrombosis of intracranial venous sinuses or cortical veins and is less frequent than the thrombotic and embolic arterial infarctions.[1] Impaired venous drainage leads to increased intracranial pressure and, in severe cases, venous infarction or hemorrhage. CVT predominantly affects young adults, particularly women, due to hormonal factors. CVT can present with a wide spectrum of clinical manifestations, including isolated headache, seizures, focal neurological deficits, altered consciousness,[2,3] focal seizures, and coma. Risk factors include pregnancy and the postpartum period, immobilization during surgery or long-haul flights, dehydration, thrombophilias, malignancies, hematologic and immunologic disorders, infections, and exogenous hormone use, such as oral contraceptives, which are associated with a fourfold to 13-fold increased risk. In particular, estrogen-containing oral contraceptives contribute to a hypercoagulable state through multiple mechanisms, including increased levels of coagulation factors (fibrinogen, factor VII, factor VIII, and factor X), reduced levels of anticoagulant proteins (protein S and antithrombin III), and impaired fibrinolysis. SAH is a rare but recognized manifestation of CVT, seen in ~ 3-5% of cases, often presenting as cortical sulcal bleeding rather than classic aneurysmal patterns.[4] The combination of CVT with SAH poses diagnostic and therapeutic challenges. We report a rare case of extensive right-sided CVT with sulcal SAH in a previously healthy young woman who recovered neurologically despite extensive sinus venous thrombosis after receiving timely anticoagulation.

Case Presentation:

A 24-year-old female with recently diagnosed polycystic ovarian disease on **tablet Miliana** (ethinyl estradiol (0.03 mg) + desogestrel (0.15 mg)) presented to the Emergency Department on 22/02/2025 with complaints of sudden-onset severe headache of two days' duration. The headache was described as thunderclap in nature, localized to the right side, and associated with neck pain. She also reported multiple episodes of vomiting. On the morning of 22/02/2025, at approximately 07:00 am, the patient developed sudden-onset weakness of the left upper limb, which progressed to involve the left lower limb by around 05:30 pm the same day. She also complained of blurring of vision in the left eye since then, and that is when attenders rushed her to our hospital emergency medicine department.

Primary survey:

Airway - patent, no gurgling or pooling of secretions.

Breathing - Respiratory rate of 24/min, oxygen saturation - 99% on room air, bilateral normal breath sounds.

Circulation - Blood pressure: 140/70 mmHg, heart rate: 61/min, extremities were warm.

Disability - Glasgow Coma Scale (GCS): E4V4M6, pupils 3 mm in size bilaterally reactive to light. GRBS: 144 mg/dl.

Exposure - No external rashes found.

Examination Findings:

On arrival at the Emergency Department, the patient was alert but irritable. She was obeying commands. Paucity of movement was noted in both the left upper and lower limbs. Vital parameters were within normal limits: Temperature: Afebrile. Neurological examination revealed a Glasgow Coma Scale (GCS) score of 14/15 (E4V4M6). Pupils were bilaterally equal and reactive to light (3 mm). Significant neurological findings included left-sided hemiparesis (Medical Research Council [MRC] grade 1/5), left-sided upper motor neuron (UMN) facial palsy, left homonymous hemianopia, and left-sided neglect. Decreased sensation to light touch over the left face and left upper limb. Fundoscopic examination was within normal limits. No signs of papilledema or retinal hemorrhages were noted. No signs of meningeal irritation were noted.

Systemic examination of the cardiovascular, respiratory, gastrointestinal, and musculoskeletal systems was unremarkable.

History:

The patient had been recently diagnosed with polycystic ovary syndrome (PCOS) and had been on combined oral contraceptive pills (OCPs), the tablet Miliana (ethinyl estradiol (0.03 mg) + desogestrel (0.15 mg), for the past three months for regularization of her menstrual cycles. She had a history of on-and-

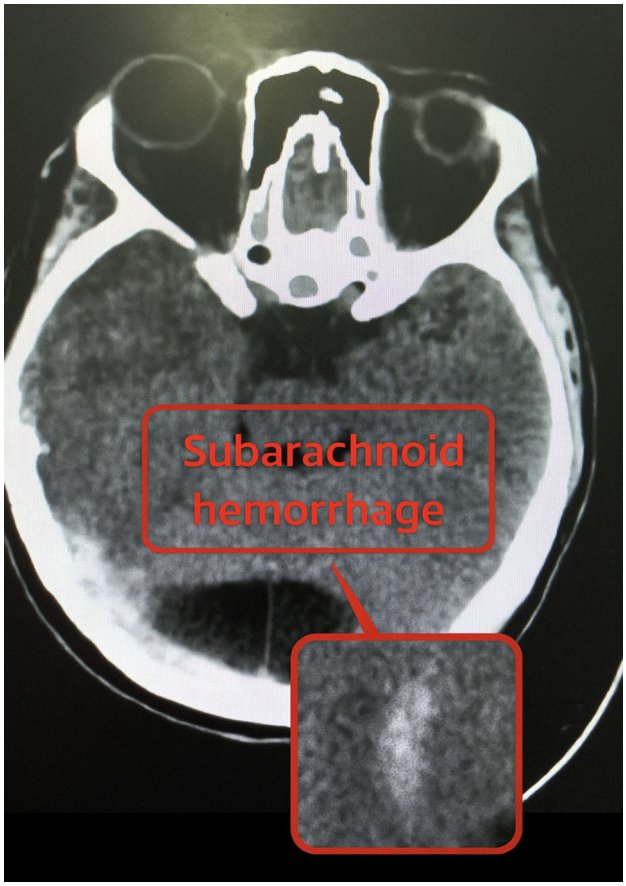
off headaches from the past 2 months. She had no prior history of migraine, thrombotic events, or neurological disorders. There was no family history of stroke, epilepsy, or thrombotic events. Social history was unremarkable. She did not smoke, consumed alcohol occasionally, and denied any illicit drug use.

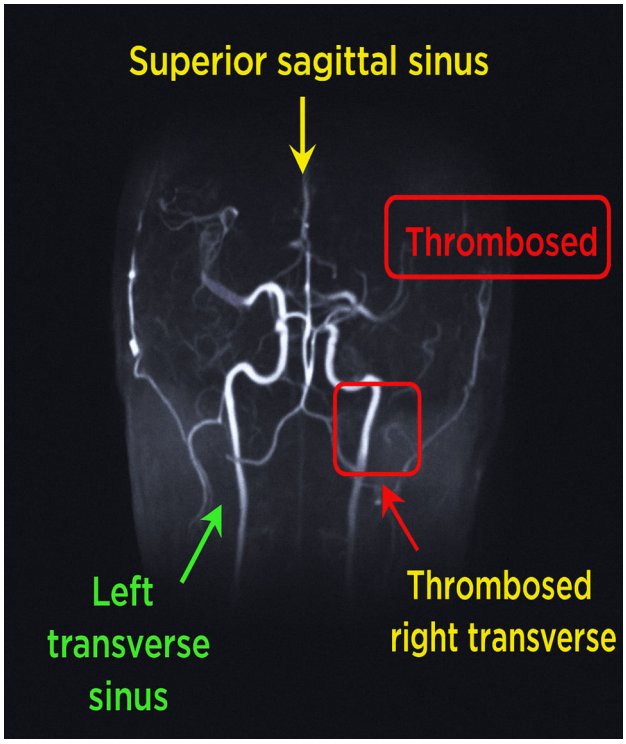
Neuroimaging:

- NCCT Brain was performed immediately after the primary survey, which showed acute cerebral venous sinus thrombosis involving the superior sagittal sinus, inferior sagittal sinus, and right transverse and extending to the sigmoid sinuses. Thrombosis involving few of the cortical veins in bilateral high parietal lobes, subarachnoid haemorrhage noted over the right temporoparietal lobe with mild to moderate cerebral oedema.
- MRI of the brain with MR venography (MRV): was performed within an hour of admission. Revealed cortical venous thrombosis involving the superior sagittal sinus and right transverse sinus, with associated SAH in the right frontoparietal region.
- This axial **non-contrast CT (NCCT) of the brain** shows:

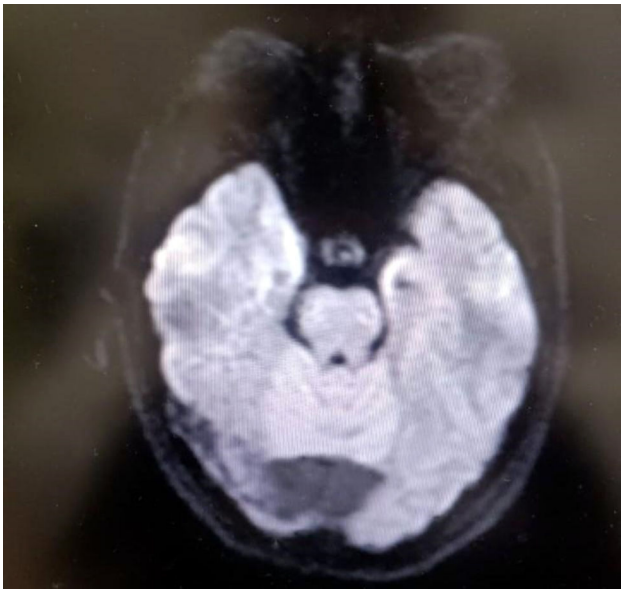


Hyperdensities in the right-sided cortical sulci, particularly over the **right temporal - parietal convexity** - suspicious for **sulcal subarachnoid hemorrhage (SAH)**.



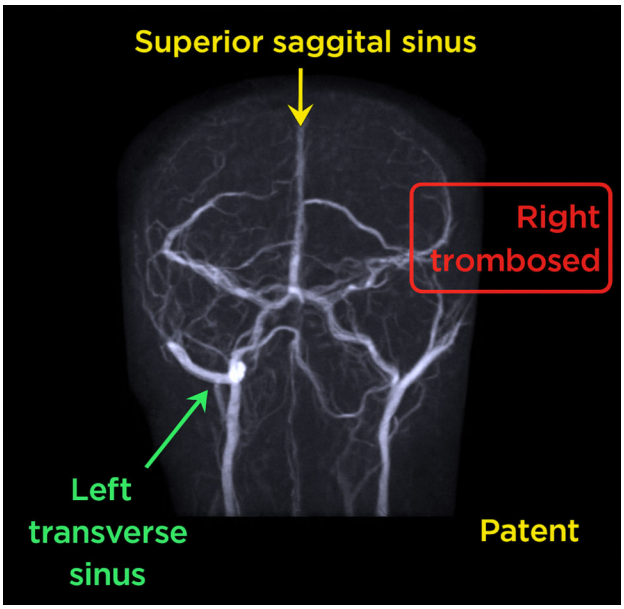


This is an **MRV (Magnetic Resonance Venogram)** image showing **absence of normal flow-related signal in the right transverse and sigmoid sinuses**, indicating **right-sided cerebral venous sinus thrombosis (CVST)**.



This is an axial DWI (Diffusion-Weighted Imaging) MRI brain sequence showing **diffusion restriction** in the **right cerebellar hemisphere**, appearing as hyperintensity on this DWI image.

Likely Acute right cerebellar infarct, likely due to **venous congestion or infarction secondary to cerebral venous sinus thrombosis (CVST)**



This is an MR Venogram (MRV) image, showing **Absence of flow-related signal in the right transverse and sigmoid sinus** suggests **right-sided cerebral venous sinus thrombosis (CVST).**

- The MRV confirmed the diagnosis of CVT by visualizing the filling defects within the affected sinuses. The MRI also helped to characterize the SAH and differentiate it from other causes of intracranial bleeding.
- **CT angiogram** does not show any large arterial stenosis or occlusion; incidentally, an arterial branch from the left vertebral artery is seen joining the right vertebral artery before the formation of the basilar trunk. **Posterior communicating arteries are hypoplastic**.

Blood Investigations:

- **Complete blood count (CBC):** TC-7780, Neutrophils 61.1%

  Hb-10.2gm%, PCV-31,4% Platelets-161000
- **Renal Profile:** Serum Creatinine - 1.10, BUN - 8, Serum sodium: 139, Serum potassium: 4.47, serum chloride: 104, serum bicarbonate: 23.7
- **Coagulation profile APTT:** Test-24, Control: 27; PT: Test-14.9, Control: 13.0; INR-1.17 VITAMIN B12 level-67 was low.
- Hypercoagulable workup: ESR-25, Plasma Homocysteine-10.86 micromol/L, Anti-cardiolipin IgG-negative, Anti-beta-2 glycoprotein-1 IgM and IgG-negative, Antinuclear antibodies-negative. All were within normal limits.
- Serum creatine phosphokinase-249 elevated.
- Other basic metabolic panel: within normal limits. These tests were performed to rule out other potential causes of neurological symptoms and to assess the patient's overall health status.
- **Lumbar puncture (LP):** Lumbar puncture was not performed acutely due to the presence of SAH on the initial MRI and the immediate need to initiate anticoagulation therapy. In the context of confirmed SAH on imaging, performing an LP would not have significantly altered the acute management and carried the theoretical risk of exacerbating bleeding. Furthermore, the presence of CVT itself can sometimes lead to elevated intracranial pressure, making LP potentially risky.
- **Urgency of Treatment:** The immediate priority in acute CVT with SAH is to stabilize the patient, manage any life-threatening complications (like seizures or increased ICP), and initiate

anticoagulation therapy. Delaying treatment to obtain hypercoagulable workup results could worsen the patient's condition.

- **Timing of Tests:** Some hypercoagulable tests are affected by acute thrombosis or anticoagulation therapy, leading to inaccurate results if done too early. It's often preferable to wait until the acute phase has passed and the patient is stable before performing these tests.

Differential Diagnosis:

The differential diagnosis included ischemic stroke, haemorrhagic stroke (including ruptured aneurysm), central nervous system infection (meningitis, encephalitis), demyelinating disease (multiple sclerosis), functional neurological disorder (conversion disorder), posterior reversible encephalopathy syndrome (PRES), and brain tumour. The MRV confirmed the diagnosis of CVT, differentiating it from these other conditions.

Treatment:

The patient received the following treatment in the emergency department:

Anticoagulation: After careful consideration of the risks and benefits in the setting of SAH and consultation with neurosurgery, therapeutic anticoagulation was initiated with enoxaparin 60 mg subcutaneously immediately after neuroimaging. This decision was made based on the understanding that the risk of further thrombosis and neurological deterioration from untreated CVT often outweighs the risk of haemorrhage expansion in this specific scenario. It was later changed to the oral anticoagulant dabigatran 110 mg two times a day on the 7th day of admission when she was discharged from the hospital.

Other medications

Seizure prophylaxis: Levetiracetam 1g IV stat was administered to prevent seizures, which is a common complication of CVT.

Intracranial pressure (ICP) management: Mannitol 300ml IV bolus was given to reduce potential cerebral edema and elevated ICP associated with both the CVT and SAH. Icu management of ICP.

Supportive care: Included strict blood pressure monitoring, intravenous fluids, and close neurological observation in the neurosurgical intensive care unit (ICU).

Hospital Course:

A 24-year-old female with nil comorbidities came to the emergency department on 22/02/2025 with complaints of a sudden onset severe headache for 2 days, a thunderclap type, localized to the right side with neck pain, associated with multiple episodes of vomiting. On 22/2/25, around 7 am, she started having left upper limb weakness, which progressed to left lower limb weakness at 5:30 pm. She had complaints of blurring of vision from the left eye (left homonymous hemianopia?). On arrival to the ED, she was alert and irritable, was obeying commands, and paucity of movements in the left upper limb and lower limb was noted. NCCT BRAIN showed acute cerebral venous sinus thrombosis involving the superior sagittal sinus, inferior sagittal sinus, and right transverse and extending to sigmoid sinuses. Thrombosis involving few of the cortical veins in bilateral high parietal lobes, subarachnoid haemorrhage noted over the right temporoparietal lobe with mild to moderate cerebral oedema. An MRI of the brain with MR venography shows absent flow signals in the superior sagittal sinus and the right transverse and sigmoid sinuses. Findings indicate extensive acute dural sinus and cortical venous thrombosis. Treated with injection of enoxaparin 0.6 mL despite subarachnoid haemorrhage, antiepileptics, anti-oedema measures, and other supportive measures, and shifted to ICU after getting a neurologist's opinion for further evaluation and management. Routine blood investigations showed a serum vitamin B12 level of 78. Other blood work was normal. Serum ANA, ANCA, and APLA profile sent reports were within normal limits. EEG showed generalized epileptiform discharges.

In MICU, she was continued on anticoagulants, antioedema, antiepileptic measures, neuromonitoring, and other supportive measures. The neurology team reviewed during the ICU stay. She had 4 episodes of GTCS on 23/2/25 (left upper limb and lower limb focal seizure with generalization), which lasted for 20-25 seconds with post-ictal confusion. Regained consciousness in between the episodes. She was intubated, and an NCCT brain scan done showed no intraparenchymal hematoma. Diffuse brain swelling was persisting with

an ill-defined area of oedema in the right temporal lobe and subarachnoid haemorrhage over the right temporoparietal lobe. She was started on Inj. Midazolam, Inj. Propofol, and Inj. Ketamine infusions. Antiepileptic drugs were optimized. She improved gradually; there were no further episodes of seizures, and gradual improvement in her sensorium was noted. On 26/2/2025, she was extubated to a facemask. On 27/2/2025, she was weaned to nasal prongs and later to room air. She became hemodynamically stable and was shifted to the ward for further management. In the ward, close neuromonitoring was done. She improved clinically. There was no further recurrence of seizures. Her headache came down. Power in the left upper limb and lower limb improved. Physiotherapy was initiated. She was mobilized out of bed. She made a full neurological recovery. Left-sided upper limb and lower limb weakness improved to (from on day 1) strength by day 7, and GCS returned to 15/15 by day 5. She was transitioned to oral anticoagulation (dabigatran) overlapping with enoxaparin and discharged home with close follow-up with neurology. Discontinuation of OCPs was strongly advised. She was also referred to a haematologist for further evaluation of potential hypercoagulable states, although none were identified.

Follow up imaging:

NCCT Brain done on the next day of admission showed extensive acute dural sinus thrombosis as compared to the previous CT brain, with persisting hyperintensities in the cortical veins, superior sagittal sinus, and right transverse and sigmoid sinuses. Persistent cerebral oedema.

DISCUSSION

Epidemiology

CVST occurs when a blood clot forms in any of the venous sinuses of the brain. Approximately 5 people per million are affected by CVST, and it accounts for approximately 1% of all stroke events.[1] Although cerebral venous sinus thrombosis (CVST) is increasingly encountered in clinical practice and presents with a range of nonspecific symptoms that often overlap with other conditions, its presentation alongside subarachnoid haemorrhage (SAH) or intracerebral haemorrhage (ICH) on CT or MRI remains uncommon. Panda *et al*.[8] reported that 10 (4.3%) of 233 patients with CVST exhibited evidence of cortical SAH. Furthermore, Oda *et al*.[9] found that CVST resulted in 3% of SAH in a retrospective review, indicating that the presence of cortical SAH without involvement of the basal cisterns may be an early indicator of underlying CVST.[8,9] Risk factors:

The risk factors for venous thrombosis in general are linked classically to the Virchow triad of stasis of the blood, changes in the vessel wall, and changes in the composition of the blood.[1] Risk factors are usually divided into acquired risks (e.g., surgery, trauma, pregnancy, puerperium, antiphospholipid syndrome, cancer, exogenous hormones) and genetic risks (inherited thrombophilia).[1] Other known risk factors include systemic conditions like polycystic ovary syndrome (PCOS)7, as seen in our patient.

Table 1: Risk factors of cerebral venous thrombosis[5,12,14-36]

Prothrombotic states	Infection (12%)[5,14,26-28]
Hereditary conditions (34-41%)[5,12,14-18] 1. Prothrombin G20210A mutation (9-21%) 2. Factor V Leiden mutation (9-13%) 3. MTHFR mutation (4.5%) 4. Antithrombin deficiency (3%) 5. Protein C deficiency (2-5%) 6. Protein S deficiency (2-3%)	1. ENT and face infection (8.2-11%) 2. Systemic infectious diseases (4.3) 3. Meningitis (2.1%) **Mechanical causes**[5,12,27-30] 1. Lumbar puncture (1.9%) 2. Head trauma (1.1%) 3. Jugular vein catheterization (1%) 4. Neurosurgical procedures (0.6%) 5. Trauma to cerebral sinuses
Acquired conditions (15.7%)[5,12,14,17-19] 1. Pregnancy and puerperium (11-59%) 2. Antiphospholipid antibody syndrome (6-17%) 3. Nephrotic syndrome (0.6-1%) 4. Hyperhomocysteinemia	**Malignancy (7.4%)**[30-32] 1. CNS tumors 2. Systemic malignancies 3. Myeloproliferative neoplasms

Prothrombotic states	Infection (12%)[5,14,26-28]
Haematology[5,12,14,20] 1. Severe anemia (9-27%) 2. Polycythaemia 3. Thrombotic thrombocytopenic purpura 4. Heperin-induced thrombocytropenia	**Drugs**[5,12,14,30,33,34] 1. Oral contraceptives (54-71%) 2. Hormone replacement therapy (4.3%) 3. Cytotoxic drugs (0.8%) 4. Intravenous immunoglobulin 5. Steroids
Autoimmune and Inflammatory diseases[5,14,21-25] 1. Inflammatory bowel disease (1.6-3%) 2. Systemic lupus erythematosus (1%) 3. Behçet's disease (1%) 4. Sarcoidosis (<1%) 5. Thyrotoxicosis (1.7%) 6. COVID-19 vaccine (<1%)	**Miscellaneous**[12,14,30,35,36] 1. Obesity (23%) 2. Dehydration (1.9%) 3. Dural A-V fistulae (1.6%) 4. Arteriovenous malformations (0.2%) 5. No identifiable reasons (12.5%)

Methylenetetrahydrofolate Reductase (MTHFR); ENT-Ear, Nose, and Throat.

Percentage (%) denotes the prevalence of the risk factors. The data expressed in the table were obtained from original research works and review literature.[5,12,14-36]

Clinical Presentation:

The clinical presentation of CVT is highly varying depending on the area of thrombosis, leading to delays in diagnosis. The superior sagittal sinus is most commonly involved, which may lead to headache, increased intracranial pressure, and papilledema.[15] For lateral sinus thrombosis, symptoms related to an underlying condition (middle ear infection) may be noted, including constitutional symptoms, fever, and ear discharge. Pain in the ear or mastoid region and headache are typical. On examination, increased intracranial pressure and distention of the scalp veins may be noted. Hemianopia, contralateral weakness, and aphasia may sometimes be seen owing to cortical involvement.[16] Headache, generally indicative of an increase in intracranial pressure, is the most common symptom in CVT and was present in nearly 90% of patients in the ISCVT17. In our case, the patient presented with a progressive throbbing headache radiating to the neck, associated with the acute onset of focal neurological deficits and altered mental status features that were suggestive of raised intracranial pressure and focal parenchymal involvement.

Table 2: Clinical presentations according to the effected dural venous sinuses[2-6,11-14,18,29,30,37-39,41-43]

Site of CVT	Clinical presentation
Superior segittal sinus (39-62%)	**Cranial nerve palsies and intracranial hypertension lead to common symptoms:** 1. Headach, nausea, vomiting 2. Blurred vision, occasionally loss of vision 3. Seizaures 4. Aphasia, hemianopia 5. Hemisensory loss and/or hemiparesis 6. Rarely, isolated psychiatric symptoms
Transverse sinus (44-73%)	**Isolated TS involvement without infarction:** 1. Asymptomatic 2. Headach, seizures 3. Left TS involvement with venous infarction: Aphasia. **Involvement of contiguous sinuses:** 1. Intracranial hypertension 2. Cranial nerve IX-XXI palsies
Sigmoid sinus (40-47%)	1. Pain in the mastoid region 2. Cranial nerve VI-VIII palsies
Deep venous system (10.9%)	1. Diminished level of consciousness, or coma 2. Diffuse encephalopatghy 3. Bilateral or fluctuating motor deficites

Cortical veins (3.7-17.1%)	Focal neurological deficits and seizures
Cavernous sinus (1.3-1.7%)	Headache, fever and ocular signs (ocular pain, chemosis, proptosis, ocular nerve palsy)
Inferior sagittal sinus	Motor deficits, seizures
Straight sinus	Motor deficits, mental status changes
Internal jugular vein	Neck pain, tinnitus, and cranial nerve palsies

The data for the construction of this table were obtained from reviews and original studies that evaluate clinical presentation and Dural venous sinuses involvement in the advlt CVT population.[2-6, 11-14,18,29,30,37-39,41-43]

CVT = Cerebral venous thrombosis, **TS** = Transverse sinus.

Pathophysiology:

The exact pathophysiology of CVT is still unclear, but CVT will always present in one of the following clinical syndromes: Intracranial hypertension, focal neurological syndrome, diffuse encephalopathy, and cavernous sinus syndrome.[12] The pathophysiological changes in CVT progress at a slow pace and progress for many weeks before they end up causing symptoms and signs of CVT. Cerebral vein thrombosis increases venous pressure and reduces capillary perfusion pressure, leading to a rise in cerebral blood volume; ultimately, patients end up in intracranial hypertension. However, cortical collateral circulation is engaged, but intracranial hypertension subsequently leads to disruption of the blood-brain barrier and the development of vasogenic edema.[12] This pathophysiology causes failure of the sodium-potassium ATPase-dependent pump, an indirect regulator of intracellular water volume, resulting in cytotoxic edema development. Superficial cortical veins drain into the superior sagittal sinus against the blood flow within the sinus, resulting in blood turbulence, which is further aggravated by the existing fibrous septa at the inferior angle of the sinus. This is the most acceptable explanation of the higher prevalence of thrombosis in the superior sagittal sinus.[12]

Neuroimaging:

Over the past 20 years, neuroimaging has played an increasing role in diagnosing CVT. Since the clinical presentation can be nonspecific and varied, neuroimaging is the cornerstone in detecting CVT in suspected cases. Here are the most commonly used imaging modalities in CVT:

NCCT Brain:

Often the first imaging done is in most of the EDs in suspected cases. Most of the time, an NCCT BRAIN will come with normal findings. Even though it is not a definitive imaging modality to detect CVT, it offers some clues and rules out other acute conditions like large arterial strokes or significant intracranial haemorrhages.

Potential findings on NCCT Brain:

Dense clot sign (dense sinus sign/cord sign): During the initial acute phase, thrombi within a sinus or cortical vein appear brighter than normal brain tissue (hyperdense) due to the presence of deoxygenated blood a direct sign of thrombosis.

Indirect Signs (more common):

Parenchymal changes: Includes oedema, venous infarction, and haemorrhage.

Diffuse brain swelling or decreased ventricular size due to increased intracranial pressure.

CT Venography: Highly sensitive and specific imaging modality for diagnosing CVT, especially in centers where MRI is not feasible.

Key findings in CTV:

Filling defect: The most characteristic sign is a lack of contrast opacification within a sinus or vein the presence of thrombus.

Empty Delta Sign: This particular sign is seen on enhanced contrast-enhanced CT scans of the superior sagittal sinus.

Magnetic Resonance Imaging and Magnetic Resonance Venography:

MRI with MRV is the gold standard imaging modality for diagnosing CVT, particularly thromboses in cortical and deep veins, which can't be picked up in CTV.

MRI sequences and findings:

T1 and T2-weighted images: The appearance of thrombus varies with increasing age.

Acute phase (0-5 days): Thrombus appears iodine in T1 and hypointense in T2 (due to deoxyhaemoglobin).

Subacute phase (5 days to 1 month): The thrombus appears hyperintense on T1 and T2 (due to methaemoglobin).

Chronic phase: (>1 month): variable appearance, often isointense or heterogenous.

FLAIR (fluid attenuated inversion recovery): often can show parenchymal oedema and hyperintense signals within thrombosed sinuses.

Gradient Echo, or GRE/Susceptibility Weighted Imaging (SWI): A highly sensitive imaging modality for detecting acute and subacute haemorrhages.

Diffusion-weighted imaging (DWI): can detect areas of cytotoxic oedema.

Digital Subtraction Angiography:

DSA has been considered the gold standard technique in diagnosing CVT for ages, even though it is less commonly used due to the invasive methods involved, high radiation exposure, and widespread availability of more feasible, highly accurate modalities like CTV and MRV. Hence, it's nowadays rarely used as a definitive diagnostic tool for CVT. Usually there will be filling defects in the dural venous sinuses or cortical veins, delayed venous drainage, and dilated collateral circulation. DSA can identify vascular aneurysm and dural arteriovenous fistula, which might cause the formation of a false "corkscrew" sign due to sluggish venous drainage and vascular congestion.

In this case, imaging revealed extensive thrombosis of the right-sided cerebral venous system and associated sulcal subarachnoid haemorrhage (SAH). SAH is an unusual but recognized complication of CVT, typically resulting from rupture of fragile, dilated cortical veins due to venous hypertension. Unlike aneurysmal SAH, which commonly involves the basal cisterns, venous SAH in CVT tends to localize in the cortical sulci.

Table 3: At a glance merits and demerits of CT, MRI, and DSA techniques[3,5,11,12,27,44-50]

Techniques	Traits	Description
CT Venography	Advantages	1. Good visualization of major venous sinuses 2. Simple, less time consuming, and less motion artifacts 3. Useful in claustrophobic patients, pacemaker, or defibrillator.
	Disadvantages	1. Ionizing radiation exposure 2. Diabetes, and CKD patients may develop contrast nephropathy. 3. Poor resolution for small parenchymal lesion.
	Sensitivity and specificity	1. CT and CTV has 95% sensitivity and 91% specificity. 2. Based on the lesion, overall accuracy is 90% to 100%
	Typical findings	1. Hyperdensity and lack of flow in thrombosed sinuses 2. Dense triangle sign, empty delta sign and Cord sign
MR Venography	Advantages	1. No radiation exposure and good delineation of brain parenchyma. 2. Identify both of cortical and deep venous thrombosis. 3. Early ischemic changes can be detected.
	Disadvantages	1. Time consuming, unavailability and produce motion artifacts. 2. Unavailble for claustrophobic patients, and pacemaker. 3. Risk of gadolinium-induced nephrogenic systemic fibrosis
	Sensitivity and specificity	1. Not known; however, MRV with echoplanar T2 susceptibility-weighted image are considered as the most sensitive sequences.
	Typical findings	1. ≤1 wk: Isointense in T1 and hypointense in T2W images. 2. Up to 2 weeks: Hyperintense on T1 and T2W images 3. >2 wk: Variable appearances; Hypointense in GRE and SWI images; Hyprintensity in DWI enhancement venous wall, and lack of flow in thrombosed sinuses.
DSA	Advantages	1. Precise dynamic information on collateral venous system. 2. Only performed when planned for an endovascular intervention.
	Disadvantages	1. Invasive procedure with associated procedural risks. 2. Skilled person required. 3. Usually, unavailable outside of tertiary hospital.

table cont....

	Sensitivity and specificity	1. Not clearly known
	Typical findings	1. Absence of sinus opacification. 2. Venous congestion with dilated cortical, scalp, and facial veins. 3. Reversal of the flow and enlarged collateral venous drainage.

AV = arteriovenous, **CT** = computed tomography, **CKD** = Chronic kidney disease, **CVT** = Cerebral venous thrombosis, venography, **DSA** = digital subtraction angiography, **MRI** = magnetic resonance imaging, **MRV** = magnetic resonance venography **TOF** = time of flight.

MANAGEMENT

The management of CVT, even in the presence of intracranial hemorrhage, includes prompt initiation of early anticoagulation therapy to prevent thrombus propagation and facilitate recanalization. Studies have shown that anticoagulation with low molecular weight heparin or unfractionated heparin is safe and effective, even in cases with associated hemorrhage. Thrombolysis or mechanical thrombectomy may be considered in refractory or severely deteriorating cases with propagating thrombus. Our patient had multiple risk factors for CVT, including recent OCP use and underlying PCOS, both of which can contribute to a hypercoagulable state. Early recognition and initiation of anticoagulation led to early recovery of neurological status within a week of first symptoms. The incidence of recurrent venous thromboembolism (VTE) after CVT ranges from 1% to 4% per year, with rates of CVT recurrence generally reported as <1% to 2%5,6 per year.[5,6]

This case underscores the importance of maintaining a high index of suspicion for CVT in young women presenting with new-onset headache and focal deficits, especially in the presence of known risk factors. The paradoxical occurrence of clot (CVT) with bleed (SAH) serves as a reminder of the complex pathophysiology and diagnostic challenges associated with this condition.

This case highlights several important clinical points:

Diagnostic challenge: The non-specific presentation of CVT can delay diagnosis. It is crucial to maintain a high level of suspicion, especially in young women with risk factors. MRV remains the best method for diagnosing CVT.

Anticoagulation dilemma: The presence of SAH creates a treatment challenge, as anticoagulation, the main treatment for CVT, is usually not safe in SAH. However, in cases of CVT with SAH, the risk of further blood clots and worsening neurological conditions often exceeds the risk of bleeding. A team approach involving neurologists, neurosurgeons, and haematologists is vital to evaluate the risks and benefits and make informed decisions regarding anticoagulation.

I**mportance of risk factor identification:** Using OCPs is a significant risk factor for CVT. Stopping OCPs is essential for long-term management and preventing recurrence. This case emphasizes the importance of thorough risk assessment in young women with neurological symptoms. Further investigation for other possible hypercoagulable states may also be needed.

This case shows the necessity for more research to develop evidence-based guidelines for managing CVT complicated by SAH.

CONCLUSION

Cerebral venous thrombosis (CVT) with concurrent subarachnoid haemorrhage (SAH) represents a rare and diagnostically challenging entity, particularly in young women with modifiable risk factors such as oral contraceptive use. This case underscores the importance of prompt recognition and initiation of anticoagulation therapy, even in the presence of haemorrhage, when guided by careful multidisciplinary evaluation. Despite severe initial deficits and complications, the patient made a full neurological recovery within a week, highlighting the potential for favourable outcomes with timely intervention. This report contributes to the growing body of evidence supporting individualized management strategies in CVT complicated by haemorrhagic presentations and reinforces the need for further clinical research to inform guidelines.

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