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Cerebral Venous Thrombosis in Slovenia: A Case Series Analysis Showing a Significant Decrease in Fatal Outcomes

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SUMMARY

Cerebral venous thrombosis (CVT) is a rare cerebrovascular disease with a highly variable presentation and a variety of predisposing/provoking factors. Mortality is generally low, between 5% and 2–3%, and in some studies it was zero.

To find out the mortality rate at the Department of Neurology, University Medical Centre Ljubljana, we analyzed the medical records of patients treated at our department from January 2008 to December 2022. There were 98 CVT patients hospitalized during this time.

In 4 of the patients (3 women), CVT ended fatally, which accounts for a mortality rate of 4.08%. Each patient presented different predisposing/predicting factors ranging from pregnancy/hormonal contraception to interventional surgery and COVID-19 vaccination. The common clinical features of the patients were aggravating headache, bilateral neurological signs, and an altered mental status leading to coma. An elevated D-dimer concentration was a common laboratory finding, and a hemorrhagic venous infarction was found radiologically in all the patients.

The mortality rate lowered during the observation time, dropping from 12% in the years 2008–2012 to 1.37% in the years 2013–2022 ($P = 0.022$). We concluded that follow up, learning from previous mistakes, and a closer collaboration between the neurology and neuroradiology wards led to increased vigilance of the attending specialists and helped to improve the treatment of CVT.

KEYWORDS

Cerebral venous thrombosis; Increased D-dimer; Hemorrhagic venous infarction; Lowering fatal outcomes

Introduction

Cerebral venous thrombosis (CVT) is a relatively uncommon cerebrovascular disorder, constituting only a small fraction of all strokes. According to a Dutch multicenter study, the incidence of CVT is 1.3 cases per 100 000 adults[1]. The presentation and underlying causes of CVT exhibit considerable variability, both clinically and radiologically[2]. Despite advancements in imaging techniques facilitating the identification of less overt cases, recognizing the symptoms and etiology of CVT remains challenging[3]. Mortality associated with CVT is generally low, although some reports indicate varying rates[4,5].

During the COVID-19 pandemic, there was speculation about an increase in CVT cases, but limited reports focused on CVT in COVID-19 patients[6,7]. Notably, a more evident increase in CVT cases was observed in connection with post-vaccination immune responses, such as vaccine-induced immune thrombotic thrombocytopenia (VITT). Cerebral venous thrombosis emerged as the most common thrombotic complication in individuals with VITT after SARS-CoV-2 vaccination, often resulting in a high mortality rate. Adenoviral vector COVID-19 vaccines were frequently implicated in these cases[8]. Typically, VITT syndrome manifested 4 to 28 days after vaccination with adenoviral vector vaccines[9]. Additionally, well-established predisposing or provoking factors for CVT, such as pregnancy, sex, hypercoagulability, and surgery (particularly interventional procedures) were identified in our patients[10].

The multifaceted etiology of CVT demands heightened vigilance from healthcare providers in order to provide an accurate diagnosis. The prognosis for patients with CVT depends on the presenting symptoms and a prompt administration of appropriate therapy[11]. Hemorrhagic infarction is considered a particularly ominous prognostic sign, with an increased likelihood of fatal outcomes; a pattern consistent with the pre-COVID pandemic era[12].

To gain a comprehensive understanding of the clinical characteristics of patients with CVT experiencing fatal outcomes between 2008 and 2022, we conducted a retrospective search of our clinical and radiological databases at University Medical Centre (UMC) Ljubljana. We manually examined records to identify relevant clinical and radiological data associated with fatal cases treated in the neurological ward over the past 15 years. Our objective was to elucidate the clinical and radiological features of CVT patients with lethal outcomes, aiming to enhance awareness and potentially prevent or mitigate such events in the future.

Patients and methods

The medical records of patients admitted to UMC Ljubljana from January 2008 to December 2022 were meticulously reviewed for this study. Only patients who were diagnosed and treated at this hospital, and in whom CVT was clinically and radiologically confirmed, were included in the study. The focus was specifically on patients with fatal outcomes during their hospitalization. Data collection was retrospective and observational, encompassing a thorough examination of all accessible clinical and radiological records.

Patients presenting with additional systemic pathologies capable of causing hemorrhagic lesions, such as amyloidosis or posterior reversible encephalopathy syndrome (PRES), were deliberately excluded from the study to ensure the homogeneity of CVT cases[13]. Data were collected from hospital admission records, the hospital data system, and the AGFA radiological database, adhering strictly to the principles outlined in the Declaration of Helsinki. Approval for the study was obtained from the Slovenian National Medical Ethics Committee (163/02/09).

The diagnosis of CVT was established through a comprehensive approach involving a clinical examination, brain computer tomography (CT), CT

venography (CTV), brain magnetic resonance imaging (MRI), and MR venography (MRV) whenever deemed necessary. Laboratory findings, including D-dimer, C-reactive protein, coagulation screening, and complete blood count were also considered in the diagnostic process. Environmental precipitating factors for CVT — such as surgery or trauma — were investigated, along with known intrinsic and acquired predisposing or precipitating factors (e.g., infections, contraceptive use, malignant diseases, hematologic conditions, chronic non-infective inflammatory diseases, intracranial hypotension, cerebral amyloid angiopathy, and acquired or genetic prothrombotic states)[14].

Statistical analysis of the data was conducted using the Mann–Whitney rank sum test, providing a rigorous and objective method for evaluating the collected information.

Case series

Patient 1

A 29-year-old pregnant woman was admitted to the neurological ward of a Slovenian general hospital with complaints of headaches and transient neurological signs. Initially suspected to be a complicated migraine, as the patient had a history of migraines, she was admitted for further diagnostics. Considering her pregnancy, radiological examinations were initially avoided. However, two days after admission, the patient's focal signs became permanent, prompting the performance of radiological examinations.

A head CT revealed a parasagittal hemorrhagic infarction, and CTV indicated an occluded superior sagittal sinus. Recognizing the severity of the situation, the patient was promptly transferred to our hospital in a deteriorated condition. Upon arrival, she was drowsy and exhibited bilateral neurological signs, including sluggish pupils and bilateral Babinski signs. Further diagnostics revealed bilateral parasagittal hemorrhagic infarctions in addition to the superior sagittal sinus thrombosis.

Immediate anticoagulant treatment with intravenous heparin was initiated; however, the patient's condition continued to deteriorate overnight, progressing to an irreversible coma. The patient succumbed within 24 hours after admission. This unfortunate turn of events underscores the complexity and urgency associated with managing CVT during pregnancy, necessitating timely and decisive interventions to mitigate potential complications.

Patient 2

A 42-year-old man was admitted to the Department of Neurology at UMC Ljubljana for an elective closure of a dural arterial-venous fistula (DAVF). Three years prior, he had experienced a mesencephalic brain hemorrhage, recovering well with no subsequent neurological symptoms. Initial CT angiography and digital subtraction angiography revealed no abnormalities. However, during a follow-up digital subtraction angiography two years later, a DAVF was identified, feeding the posterior cerebral arteries and draining into the left transverse sinus, while the right transverse sinus, sigmoid sinus, and upper part of the right jugular vein were thrombosed.

A year later, the patient returned for DAVF closure due to disease progression. A mild elevation of D-dimer (611 µg/L), an increased platelet count (403 x 10ˆ9/L) and lower serum fibrinogen (1.77 g/L) — considered reactive by the consulting hematologist — were noted. Angiography revealed an 8 mm malformation draining into Galen's vein. The DAVF was successfully closed using coils and a liquid embolic agent (Onyx). The draining veins remained patent. Post-procedure, the patient experienced

dysarthria and slight right-sided hemiparesis, but was generally stable and was admitted to the stroke unit for recovery.

However, about 4 hours later, the patient abruptly lapsed into a coma, displaying a skew deviation of the eyes, divergent strabismus, nonreactive pupils (2 mm in diameter), an absent right corneal reflex, and bilateral pyramidal signs. A follow-up CT revealed a brainstem hemorrhage (Figure 1a-e), hematocephalus, hydrocephalus, and an evolving ischemic stroke in the territory of the left posterior cerebral artery (Figure 1b, e), occluded with Onyx (Figure 1c upper arrow). Ventricular drainage was initiated (Figure 1d) and patent basal veins and the vein of Galen were observed (Figure 1d). However, they were partially filled with Onyx (Figure 1b, c lower arrow, f). Although CTV indicated mostly patent venous paths, the patient's condition deteriorated over the following 10 days. Nevertheless, the radiologist's opinion was that a hemorrhagic infarction occurred in the venous part of DAVF due to partially closed drainage veins. Further lab tests revealed elevated D-dimer (up to 3 225 µg/L), WBC, and CRP, which might have been partially caused by a respiratory infection. Transferred from intensive care to the stroke unit, the patient succumbed to subsequent pulmonary infections without neurological improvement.

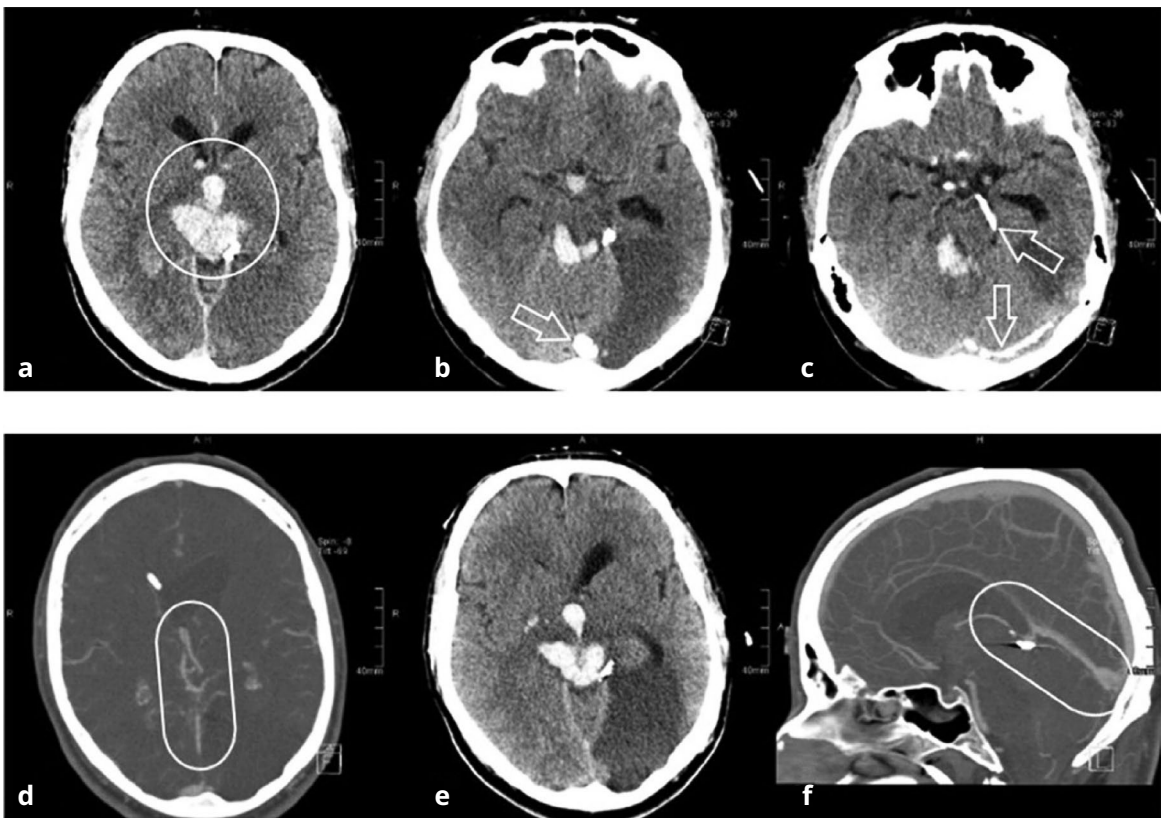


Figure 1. CT/CTV images of Patient 2. a: brain hemorrhage with hematocephalus; b/e: ischemic stroke in the territory of the left posterior cerebral artery, initial and demarcated; c: left posterior cerebral artery filled with the embolic agent (upper arrow); d: ventricular drain inserted (right lateral ventricle), deep veins are patent (encircled); b/c: traces of the embolic agent in the confluence of the sinuses (arrow) and left transverse sinus (lower arrow); f: traces of the embolic agent in the distal vein of Galen (encircled).

Patient 3

A 23-year-old woman presented to the neurological emergency department with a one-week history of progressive headaches, right-sided paresthesia, and slurred speech. She had been feeling unwell and experienced fluctuating speech difficulties. The patient was taking oral contraceptives and had a recent history of a tick bite. Family history revealed pulmonary embolisms in two relatives on her father's side. During her clinical examination, she appeared agitated with inadequate verbal contact and vomiting. Divergent strabismus was observed, although no obvious motor paresis was noted.

An initial brain MR (Figure 2a), CT, and CTV did not reveal an arterial/venous obstruction, but a lumbar puncture indicated elevated cerebrospinal fluid (CSF) protein content (0.95 g/L), WBC 117, RBC 7, and the presence of hemoglobin. Due to the recent tick bite, the patient was initially admitted to the infectious disease ward for suspected tick-borne or herpetic meningoencephalitis. However, all tests for brain infectious diseases were negative, and the patient deteriorated despite Acyclovir treatment.

A follow-up MR/MRV (Figure 2b, c) showed a thrombosis of the left transverse and sigmoid sinus and jugular vein bulb (Figure 2b, c arrows), with

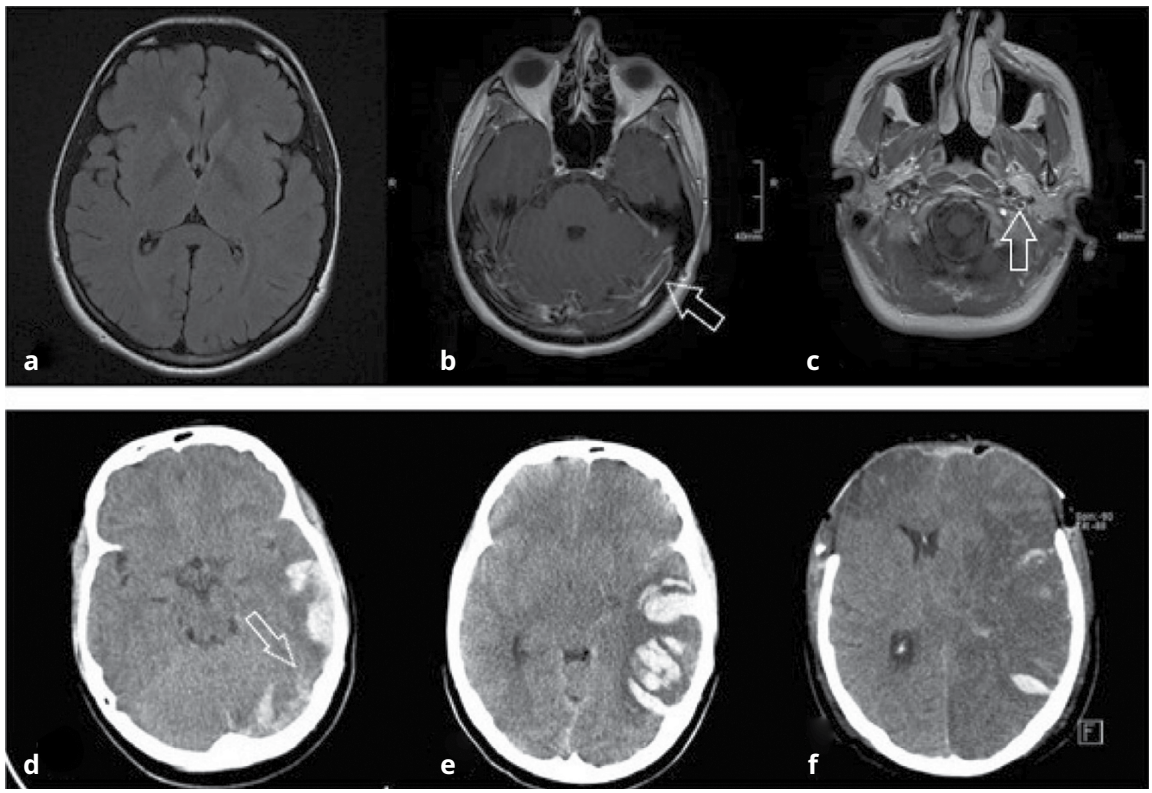


Figure 2. CT/ MR/MRV images of Patient 3. a: normal initial MR; b/c: control MR showing CVT of the left transverse, sigmoid sinus and jugular vein bulb (arrows); d: hemorrhagic infarction with suspected Labbe vein thrombosis (arrow); e: enlargement of the hemorrhagic infarction; f: edema enlargement, decompressive craniotomy.

a CT revealing a hemorrhagic venous infarction (Figure 2d).

After five days, the patient was transferred to the division of vascular neurology. She was awake but unresponsive, with narrow and nonreactive pupils, divergent strabismus, and Bell's phenomenon upon eyelid opening. Anticoagulant treatment with low molecular weight heparin was initiated. The following day, an increase in venous hematoma and extensive cerebral edema was observed (Figure 2e), suggestive of Labbe vein thrombosis (Figure 2d arrow). Brain herniation occurred, leading to a decompressive craniotomy (Figure 2f). Despite all interventions, intracranial pressure remained elevated at 38 mmHg, and brain death was established. The patient was subsequently disconnected from the respirator. This case underscores the challenges associated with diagnosing and managing cerebral venous thrombosis, especially in young patients with atypical presentations.

Patient 4

A 20-year-old woman was urgently evaluated in the neurological emergency department due to the onset of progressive headaches occurring approximately 6 hours before presentation. She described frontal headaches, nausea, transient blindness, and paresthesia in her upper extremities. Notably, her

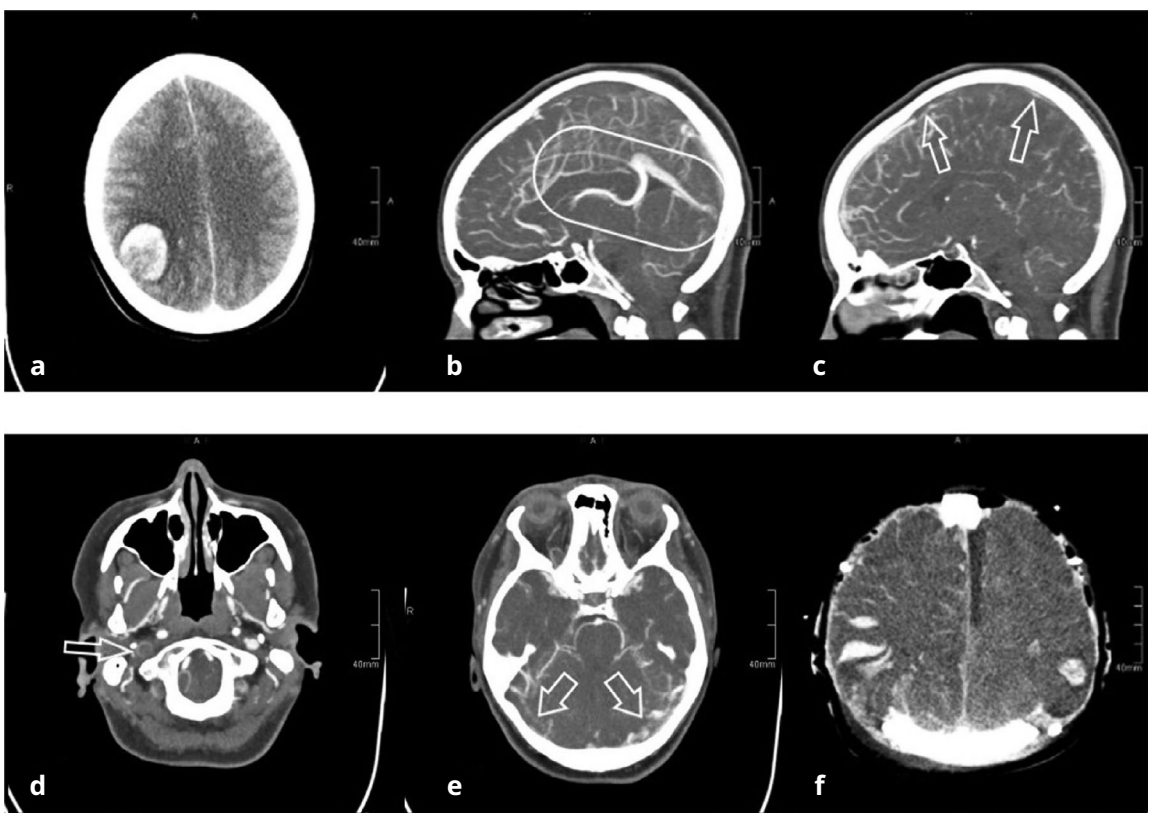


Figure 3. CT/CTV images of Patient 4. a: intracerebral hematoma and mild diffuse brain edema; b: patent basal cerebral veins, vein of Galen and straight sinus; c: thrombosis of the superior sagittal sinus (arrows); d: thrombosis of the right sigmoid sinus and jugular bulb (arrow); e: right transverse sinus thrombosis and suspected left transverse sinus thrombosis (arrows); f: decompressive craniotomy due to brain edema.

sister mentioned a mild headache the day before. Two weeks prior, the patient had received the Ad26.COV2.S COVID-19 vaccine. During the examination, she displayed psychomotor impatience and reported visual field loss in the left lower quadrants, poor gaze fixation, and left gaze paresis. Additionally, her left upper extremity exhibited apparent weakness without clear lateralization or pathological reflexes. A head CT revealed an intracerebral hematoma with subarachnoid hemorrhage in the right parietal lobe and diffuse brain edema (Figure 3a).

Contrast-enhanced CTV indicated patent basal cerebral veins, the vein of Galen, and straight sinus (Figure 3b). However, thrombosis was observed in the superior sagittal sinus (Figure 3c, arrows), right transverse sinus, left suspected transverse sinus (Figure 3e, arrows), right sigmoid sinus, and the jugular bulb (Figure 3d, arrow). Over the next 3 hours her condition deteriorated, leading to a comatose state with a dilated right pupil and bilateral pyramidal signs. A follow-up CT demonstrated worsening cerebral edema, bilateral sulcal effacement, compressed ventricles, and subfalcine and transtentorial herniations. Given the possibility of VITT, intravenous immunoglobulin (IVIg) treatment, antiedematous therapy (mannitol and thiopental), and steroids for low thrombocyte levels were initiated. The diagnosis of VITT was subsequently confirmed by the presence of heparin-induced positive antiplatelet factor 4 (PF4) antibodies, despite negative SARS-CoV-2 tests.

Although initial measures provided temporary relief, her right pupil dilated again, prompting an extensive decompressive craniotomy after thrombocyte plasma infusion (Figure 3f). Unfortunately, this intervention did not yield a lasting effect, and brain death was confirmed the following day. The post-mortem examination revealed respirator brain with severe edema, and bilateral transtentorial and tonsillar herniation. Thrombi were also identified in pulmonary arteries and liver veins. This tragic case underscores the critical importance of recognizing and managing rare complications associated with COVID-19 vaccination, particularly VITT, in a timely and vigilant manner.

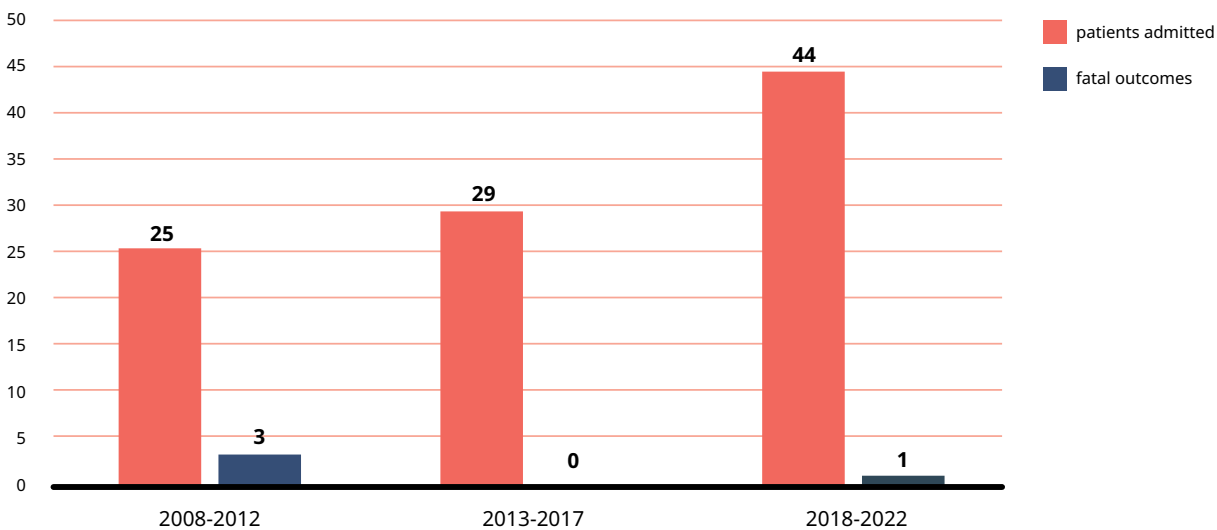


Figure 4. A flow chart showing the number of hospitalized patients and fatal outcomes due to CVT over a period of 15 years (2008–2022).

Table 1. Summary of laboratory outcomes

Parameters	Ref. range.	Patient 1	Patient 2	Patient 3	Patient 4
Hemoglobin	120–160 g/L	162*	127–141	95–133	87–135
Hematocrit	0.37–0.47 %	0.43*	0.369–0.395	0.303–0387	0.263–0.378
WBC	4–10 10^9/L	18.6*	12.9–16.2	11.7–20.5	21.4–39.5
Platelet	140–340 10^9/L	319*	403–475	131–150	16–78
CRP	Up to 5 mg/L	23.6*	< 5–47	16–184	16–56
Prothrombin time	Sec (0.70–1.20)	Nd**	0.73–0.96	0.65–0.96	0.53–0.65
aPTT	24.0–35.5 sec	Nd**	26.5–31.7	22.7–78.1	28.6–32.3
Thrombin time	14–21 sec	Nd**	15.6–21.8	13.3 to > 150	18.3–21.4
Fibrinogen	1.8–3.5 g/L	Nd**	1.52–5.50	2.66–3.52	1.1–2.3
D-dimer	Up to 500 µg/L	Nd**	611–3252	2914–7043	14 376–30 578

WBC – white blood cells, CRP – C reactive protein, aPTT – partial thromboplastin time
* Findings are from the hospital where Patient 1 was first admitted
** In Patient 1, laboratory data are not complete due to changes in the information system in the Department of Neurology

Results

Over a 15-year observational period, 98 patients were admitted due to CVT. A specific focus was placed on patients with fatal outcomes during hospitalization, revealing 4 cases (3 women) aged 20 to 42. Figure 4 illustrates admission and mortality rates from 2008 to 2022 in 5-year increments. An increase in the number of cases is probably due to improved diagnostics in the years 2013–2017, as well as the COVID-19 epidemic and/or vaccination in the years 2018–2022.

All female patients reported headaches upon admission, while the male patient did not. Except for Patient 2 (admitted for elective dural arteriovenous fistula closure), all patients presented with bilateral focal deficits upon admission or shortly thereafter. Altered mental status/confusion was universal at admission, progressing rapidly to stupor and coma. Notably, no seizures were observed. All the patients exhibited a hemorrhagic venous infarction attributed to CVT on radiology screening.

Predisposing/provocative factors for CVT included pregnancy in Patient 1, a positive family history of pulmonary embolism and hormonal contraception in Patient 3, interventional surgery in Patient 2, and Ad26.COV2.S COVID-19 vaccination in Patient 4. Key laboratory findings are listed in Table 1.

Statistical analysis revealed a decline in fatal outcomes from 12% in the first 5 years (2008–2012) to 1.37% in the last 10 years (2013–2022) ($P = 0.022$).

Discussion

This retrospective observational study spanning the last 15 years underscored significant advancements in the treatment of CVT, leading to a notable reduction in mortality rates. The shift from a mortality rate of 12% in the years 2008–2012 (3 fatal outcomes out of 25 patients) to a mortality rate of 1.37% in the years 2013–2022 (1 fatal outcome out of 73 patients) is attributed to improved diagnostic capabilities and increased vigilance among attending neurologists and neuroradiologists.

The symptoms of CVT are intricate and diverse, often making early diagnosis challenging. However, all 4 patients in this study were diagnosed within a few days, showcasing an improvement in diagnostic efficiency over time. It took the longest time to the right diagnosis in Patients 1 and 3, since they were misdiagnosed initially as a complex migraine and viral meningitis; 2 and 6 days were needed for the right diagnosis, respectively.

Patient 1 was admitted in 2008 and Patient 3 in 2012. In the meantime, our information system changed and no detailed records for the case of Patient 1 are still available.

Both cases revealed clinical predisposing/provocative factors for CVT, including pregnancy and a history of spontaneous abortion in Patient 1. The majority of CVTs related to pregnancy occur in the third trimester of pregnancy and in the puerperium 6–8 weeks after birth[15]. This was, however, not the case with Patient 1, who was in the first trimester of pregnancy. Unfortunately, the patient's history of migraine and pregnancy led to delays in diagnostics, particularly radiological ones.

Patient 3 had a family history of potential thrombophilia and used hormonal contraception. This combination could dramatically increase the risk of CVT[16,17]; in a review analysis, oral contraceptive users were found to have an increased risk[5]. In general, most women who use oral contraceptives are in the prime period of their life, i.e. under the age of 51. It is widely accepted that the use of oral contraceptives is associated with an increased risk of CVT and that the risk with oral contraceptive use in women is greater among those with a hereditary prothrombotic component[15]. Patient 3 was not confirmed as having a presumed prothrombotic condition. Furthermore, the possibility of viral (tick-borne or possibly herpetic) meningoencephalitis seemed reasonable due to anamnestic and CSF data. Even the initial CT/CTV was negative, which postponed the right diagnosis. An early CVT diagnosis contributes to a better outcome[17], reducing fatal outcomes to under 5% as reported by some multicenter studies, or even to 0 as reported by some smaller studies[18]. The authors believe that awareness of a greater possibility of CVT pathology in women, especially younger women, is an important diagnostic factor. Moreover, it is also recommended for such patients to be treated in an institution with special experience in CVT[18].

There were no laboratory results available for Patient 1, and the results of the initial lumbar puncture for Patient 3, combined with anamnestic data, were unfortunately more indicative of viral (herpetic or tick-borne) meningoencephalitis than to CVT (increased CSF proteins, WBC, and RBC). Later on, however, the tests for brain infectious diseases were negative. The only possible initial hint seemed to have been the moderately raised D-dimer value (881 µg/L). However, even though the attending neurologist suspected a thrombotic etiology, the initial radiological examinations were negative. It seems, therefore, that a preceding examination of a possible prothrombotic condition was the missing link.

Despite non-significant effects in multicenter studies, mechanical cleaning or local thrombolysis of venous sinuses might help in the progression of symptoms in addition to anticoagulant therapy/decompressive craniotomy, particularly in the case of space-occupying lesions (e.g., hemorrhagic infarctions) and occluded superficial communicating veins[11,15].

The etiology of DAVF in Patient 2 emphasized the possibility of cerebral sinus thrombosis as a predisposing factor. Due to CVT, the pre-existing micro-A-V communications gradually distend to form a DAVF[20,21]. It is possible that our patient was also suspected of experiencing such a course of events.

An early multicenter study in 2013 revealed a liquid embolic agent (Onyx) appropriate for the treatment of AVMs with embolization; however, a fatal case occurred in the study due to an extensive venous thrombosis[22]. The mortality rate was 4.3%, but patients with brainstem AVMs/DAVFs were not included. A variety of therapies for DAVFs have been suggested by newer studies. Detachable coils are the preferred interventional method, despite the inclusion of classical/stereotactic surgery, embolization, and radiotherapy[23]. Detachable coils were used successfully to reduce flow through a brainstem DAVF in a recent report[24]. When our patient was admitted for treatment in 2012, these facts were largely unknown. Onyx embolization was a fairly new technique that seemed safe and effective in early reports[22,25]. After about a decade of its use however, a higher number of hemorrhagic complications became evident, most often due to pial artery supply, a giant venous part of DAVF, and higher Onyx volume (≥ 6 mL)[26]. We propose that hemorrhage to the venous part of DAVF in our patient might have happened due to pial artery supply, as the venous part of DAVF was not giant (8 mm). In addition, the volume of Onyx that was used could not be precisely determined, but was estimated to be less than 6 mL.

Thrombocytopenia after adenovirus-based DNA vector vaccination has long been recognized as a possible side effect of adenovirus-based DNA vector vaccination. Fortunately, the complication has not appeared frequently so far[27]. In Norway, 5 middle-aged women suffered CVT with thrombocytopenia within 14 days of vaccination with ChAd-Ox1 vaccines, and 4 of them had a fatal outcome[28]. All but one of the patients succumbed within days after admission, despite maximum treatment efforts. This was also the case with Patient 4 in our study, who was admitted to the Division of Vascular Neurology, UMC Ljubljana. The course of the thrombotic event was probably too fast for IVIg to have any effect. Anticoagulant treatment was not administered. According to a recent review, treatment with non-heparin anticoagulants is not associated with lower mortality[29].

Despite the belief that a COVID-19 infection could lead to thrombotic complications, there were significantly more reports of CVT after COVID-19 vaccination[7,29]. Although uncommon (3.83 per million cases), VITT syndrome caused by the Ad26.COV2.S COVID-19 vaccine is a serious adverse event[31].

We emphasize that, despite the increase in numbers of CVT patients during the last 5 years, we observed a single lethal outcome after vaccination with Ad26.COV2.S COVID-19 vaccine, and none due to COVID 19 infection. By adding new laboratory techniques, we might even improve our results in the future[32].

This case series had several limitations. First, the sample was presented in detail, but was small in size, and the data on Patient 1 were limited due to a change in the hospital's information system. Also, the study was observational, therefore the examination techniques/methods were not planned in advance. Nevertheless, we believe that our analysis of mismatches in reasoning and overenthusiasm with new, unproved materials helped to clarify these difficult cases and make them instructive.

Conclusion

Despite a small number of detailed cases being presented, this case series illustrated the progress made in CVT treatment over 15 years. Elevated

D-dimer emerged as a consistent finding across cases, suggesting its potential as an early screening tool when radiology is inconclusive. The use of genetic/acquired thrombophilia test improvements in radiological diagnostics and close collaboration between emergency neurologists and neuroradiologists contributed to improved early detection and treatment.

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SAŽETAK

Cerebralna vaskularna tromboza u Sloveniji: analiza serije slučajeva ukazuje na znatan pad smrtonosnih ishoda

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Cerebralna venska tromboza (CVT) rijetka je cerebrovaskularna bolest s varijabilnom prezentacijom i različitim čimbenicima rizika. Smrtnost je niska, između 5 % i 2–3 %, a u nekim studijama uopće je nema. Da bismo ustanovili smrtnost bolesnika od CVT-a na Odjelu za neurologiju Kliničkog bolničkog centra Ljubljana analizirali smo medicinske podatke bolesnika hospitaliziranih od početka siječnja 2008. do kraja prosinca 2022. Bilo je ukupno 98 bolesnika. U 4 bolesnika (3 žene) bolest je završila smrtnim ishodom, što iznosi 4,08 % od ukupnog broja. Svaki bolesnik imao je drugačije čimbenike rizika za CVT, od trudnoće/hormonske kontracepcije do intervencijske kirurgije i cijepljenja protiv COVID-a 19. Uobičajene kliničke poteškoće bile su glavobolja, bilateralni neurološki znakovi i promjene mentalnog statusa koje vode u komu. Laboratorijski, najčešće je bio povišen D-dimer, dok smo radiološki kod svih pacijenata pronašli hemoragijski moždani udar. Smrtnost tijekom opservacijskog vremena značajno se snizila: od 12 % u razdoblju 2008.–2012. do 1,37 % u razdoblju 2013.–2022. godine ($P = 0.022$). Zaključujemo da su promatranje, učenje iz pogrešaka i bolja suradnja između neurologa i neuroradiologa pripomogle boljem liječenju bolesnika s CVT-om i nižoj smrtnosti.

KLJUČNE RIJEČI

Cerebralna venska tromboza; Povišen D-dimer; Hemoragijski moždani udar; Smanjenje smrtonosnog ishoda