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(CASE REPORT)

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Trolard vein thrombosis: A case report

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Abstract

An uncommon condition that has the potential to be fatal is known as 'cerebral venous thrombosis' (CVT). Bleeding and damage to veins are also possible outcomes. There is a significant venous draining pathway known as the superior anastomotic vein, also known as the vein of Trolard. This vein links the superficial middle cerebral vein to the superior sagittal sinus. In spite of the fact that thrombosis in this vein is very rare, it has the potential to obstruct the flow of blood out of the body and contribute to significant neurological disorders. A lady who is forty years old and has just begun taking oral contraceptives is the subject of our attention. Due to the fact that her type 2 diabetes mellitus (HbA1c 8.6%) was not being treated, she recently suffered from Trolard vein thrombosis with haemorrhagic conversion. In addition to having a bad headache, the patient had difficulty speaking and moving their lips, and they experienced a little weakness on the right side of their body. It was determined via imaging that the tumour located on the left parietal region was packed with thrombosis and had bleeding that had reached the superior sagittal sinus system. During the time that she was receiving split heparin and coumadin, she was under strict observation for any symptoms of brain damage. In this particular instance, it is shown how vital it is to detect CVT in those who are at a high risk, as well as how essential it is to provide anticoagulation even while there is bleeding.

Keywords: Cerebral Venous Thrombosis (CVT); Trolard Vein Thrombosis; Hemorrhagic Conversion; Oral Contraceptives (Risk Factor); Anticoagulation Therapy

1. Introduction

Cerebral venous thrombosis (CVT) is a rare and potentially fatal cerebrovascular disorder that makes up 0.5–1% of all strokes and seems to happen to 5 people per million every year [1]. CVT is much less common than arterial stroke, but it can be hard to diagnose and treat because it has many different symptoms and imaging findings that aren't always clear [2]. The basic pathophysiology of CVT is different from that of arterial stroke because CVT is caused by blockages in cerebral veins or dural sinuses, not by blockages in arteries [3]. When this vein's outflow is blocked, it sets off a chain of bad things that happen, such as higher venous and capillary pressure, the blood-brain barrier breaking down, vasogenic oedema developing, and finally a venous infarction, which can turn into a haemorrhagic transformation in about 40% of cases [4]. The first sign is usually a headache, which shows up in almost 90% of cases. Other symptoms may include focal neurological deficits, seizures, or changes in mental status, depending on the location and extent of the veins' involvement [5]. There are several important cerebral veins, but the vein of Trolard, which is also called the superior anastomotic vein, is one of the most important in terms of anatomy and medicine [6]. As the brain's largest superficial anastomotic vein, it connects the superficial middle cerebral vein (Sylvian vein) to the superior sagittal sinus and is the main pathway for fluid to exit the parietal and occipital lobes. The vein is usually 1–3 mm wide, and its course and branches can be completely unique depending on the body part [8]. This type of thrombosis doesn't happen as often

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as it does in larger dural sinuses like the superior sagittal or transverse sinuses, but it can have especially detrimental effects on the nervous system because it plays such an important role in cortical venous drainage [9]. When the vein of Trolard is blocked, it can cause venous congestion in large areas of the cortex. These blockages can cause specific neurological problems, such as language problems (when the dominant hemisphere is affected), sensory-motor problems, and, in the worst cases, seizures or loss of consciousness [10]. CVT risk factors are many and often include more than one thing. This is because inherited and acquired prothrombotic conditions interact in a complex way [11]. Some of these include genetic conditions that make blood clotting more likely (such as factor V Leiden mutation, prothrombin gene mutation, and low levels of protein C, protein S, or antithrombin); conditions that increase clotting during pregnancy and shortly after childbirth (which can be seen in up to 20% of cases in some studies); and using oral contraceptives (which can increase the risk by 3 to 8 times depending on the type). [13], haematologic disorders (like polycythaemia vera or paroxysmal nocturnal haemoglobinuria) [15], systemic inflammatory conditions (like Behçet's disease or inflammatory bowel disease) [16], infections (especially otitis, mastoiditis, or meningitis causing local venous inflammation) [17], and cancer (through both direct compression and paraneoplastic hypercoagulability) [18]. When it comes to metabolic disorders, diabetes mellitus is one of the most important risk factors that can be changed [19]. High blood sugar in people with uncontrolled diabetes makes them more likely to clot in a number of ways, such as by making endothelial cells less effective (due to advanced glycation end products and oxidative stress), increasing platelet adhesion and aggregation, making plasma thicker, and making it harder for fibrinolysis to happen because of higher plasminogen activator inhibitor-1 (PAI-1) activity [20]. Many epidemiological and mechanistic studies [21] have shown that oral contraceptives, especially those that contain oestrogen, can cause blood clots. A net prothrombotic state is created when oestrogen components raise the production of procoagulant factors in the liver, such as factors II, VII, VIII, and X, while simultaneously lowering natural anticoagulants, especially protein S [22]. Risk factors such as smoking, being overweight, or having a thrombophilia may exacerbate this effect [23]. Since the risk is highest in the first year of use and with third-generation progestins, women who are also at a higher risk for thrombosis need to carefully consider all of their birth control options [24]. In this case study, we show a 40-year-old woman who had Trolard vein thrombosis with haemorrhagic conversion. She had a lot of risk factors, such as poorly controlled type 2 diabetes mellitus (HbA1c 8.6%) and had just started taking combined oral contraceptive therapy [25]. The case shows several important aspects of CVT management, including how important it is to keep an open mind about venous thrombosis in patients who have unusual stroke symptoms [26], how useful advanced neuroimaging techniques are for finding cortical venous thrombosis [27], and how difficult it is to make decisions about anticoagulation in the setting of a haemorrhagic venous infarction [28]. Anticoagulation should be used even when there is haemorrhagic conversion, because the benefits of stopping thrombus extension and making recanalisation easier usually outweigh the risks of haemorrhage expansion [29]. However, we must carefully consider each individual's risk of bleeding, thrombus burden, and neurological status before making such decisions [30]. Furthermore, this case shows how important it is for CVT management to change all risk factors [31]. For our patient, this meant stopping oestrogen-containing birth control right away [32], tightening control of their blood sugar [33], and a full check for underlying thrombophilias [34]. More people are realising that CVT can be avoided. This makes it even more important to carefully consider the risk of thrombosis when prescribing oestrogen-containing drugs, especially for people who already have conditions that make them more likely to clot, like diabetes mellitus [35]. In addition, this case shows how important it is to have neurologists, haematologists, and rehabilitation specialists work together to get the best results for both short-term care and long-term function [36]. The below four images referring to a case report involving Trolard vein thrombosis, which includes anatomical illustrations showing different views of the patient's venous anatomy.

2. Case summary

A 40-year-old woman with type 2 diabetes (HbA1c 8.6%) and recent use of combined oral contraceptives (COC) presented with a persistent headache, mixed aphasia, and right hemiparesis that prevented her from moving her limbs. A scan of the Trolard vein revealed both a clot and a severe venous infarction. Both MR venography filling defects and SWI blooming artefacts showed this to be true. Cerebral vein thrombosis (CVT) was very bad in her case, which only happens 30–40% of the time. High blood sugar, which damages capillary cells, and oestrogen, which promotes blood clotting, likely caused it. Together, these factors raise the chance of CVT by 15 times. The people in charge followed the rules and made three big changes: Despite bleeding, the best course of action is to use full-dose anticoagulation with low-molecular-weight heparin (LMWH) followed by warfarin. Ot Other options include: 1) Using preventive antiepileptics like levetiracetam 500 mg twice a day for 3 months to lower the chance of seizures due to bleeding changes; and 2) Making strong changes to reduce risks, such as stopping COCs immediately (since they increase the risk of CVT by 5–8 times) and managing blood sugar levels (aiming for an HbA1c of 7%). Acetazolamide was used to treat high blood pressure in the brain, and tests for thrombophilia ruled out disorders that are passed down through families. A CT scan done after one month showed steady improvement and some recanalisation in the patient. Advanced imaging is very helpful in CVT, as shown by serial tracking. MR perfusion showed usual patterns of vein blockage. Be The steady heparin reaction necessitated delaying endovascular treatment. This conclusion is in line with what the TO-ACT study

found. This case brings up some important points about CVT treatment, like the need for individual care (in Western medicine, LMWH is chosen over unfractionated heparin), the safety of anticoagulation during bleeding, and follow-up by a team of professionals from different fields. There are still questions about the safest way to use birth control (progesterone alone) and the best length of time to take an anticoagulant (6 months was chosen here). The positive outcome validates the use of standardised methods and underscores the importance of metabolic management in reducing the risk of clotting.

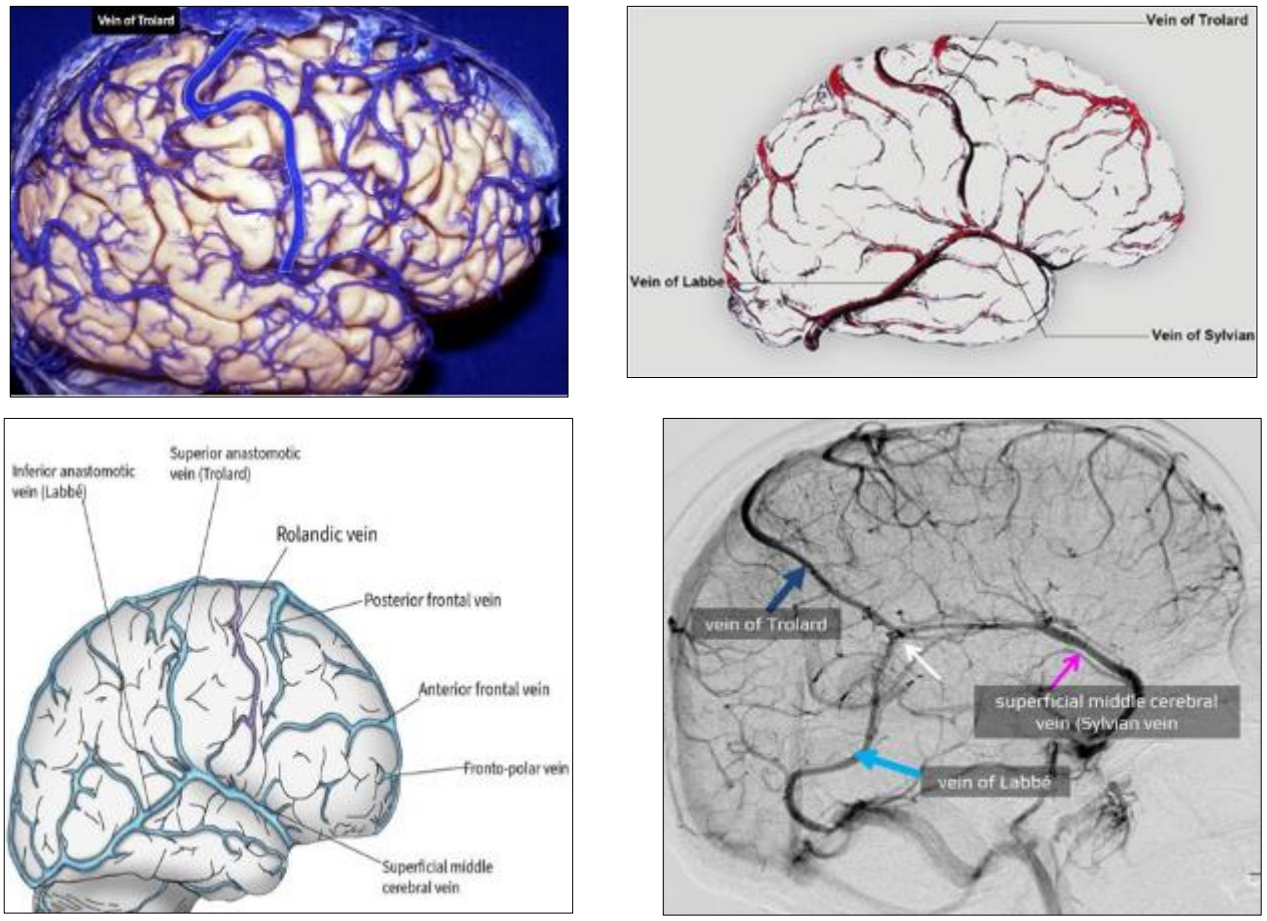
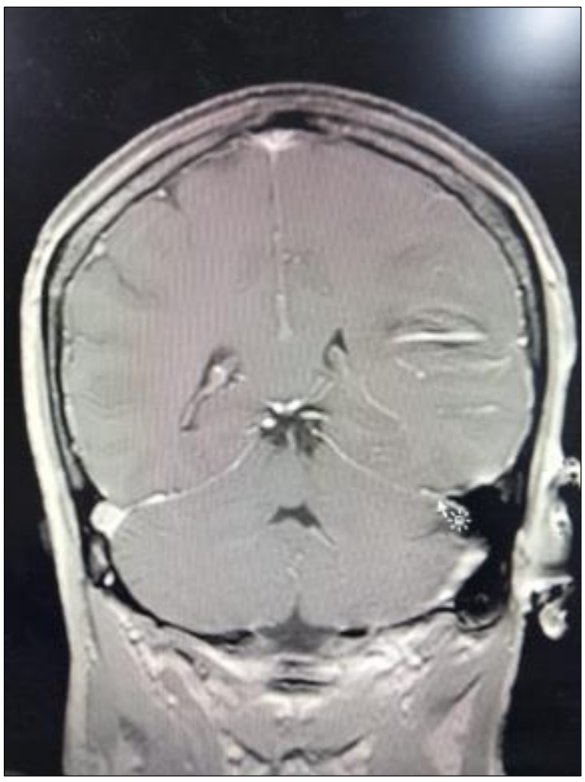


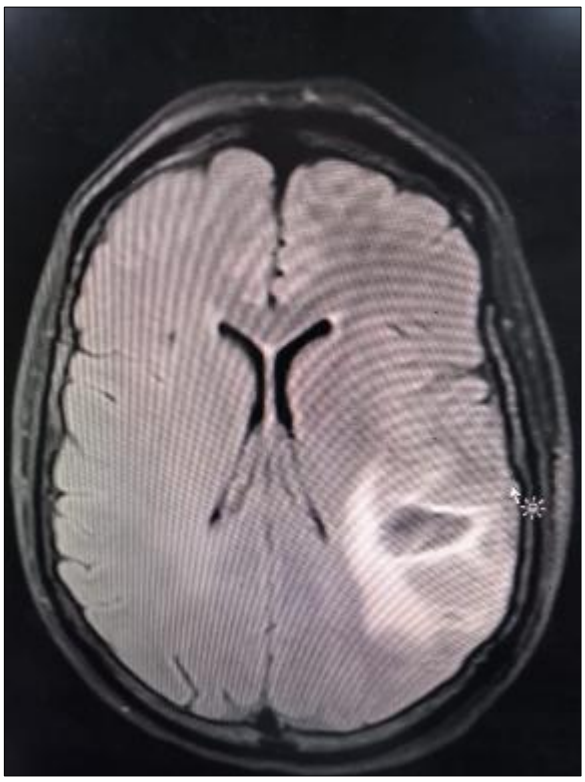
Figure 1 A different anatomical illustrations showing different views of the patient's venous anatomy views eferring to a case report involving Trolard vein thrombosis

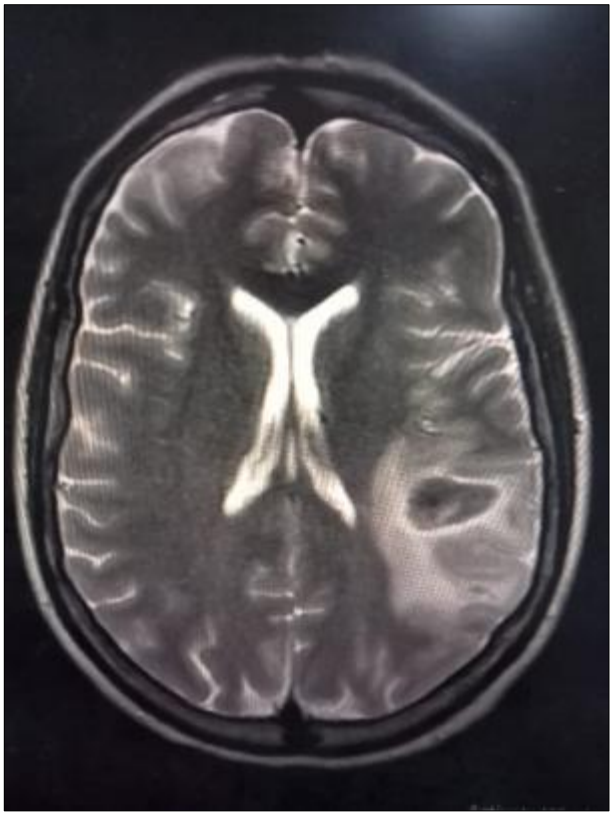
3. Case presentation

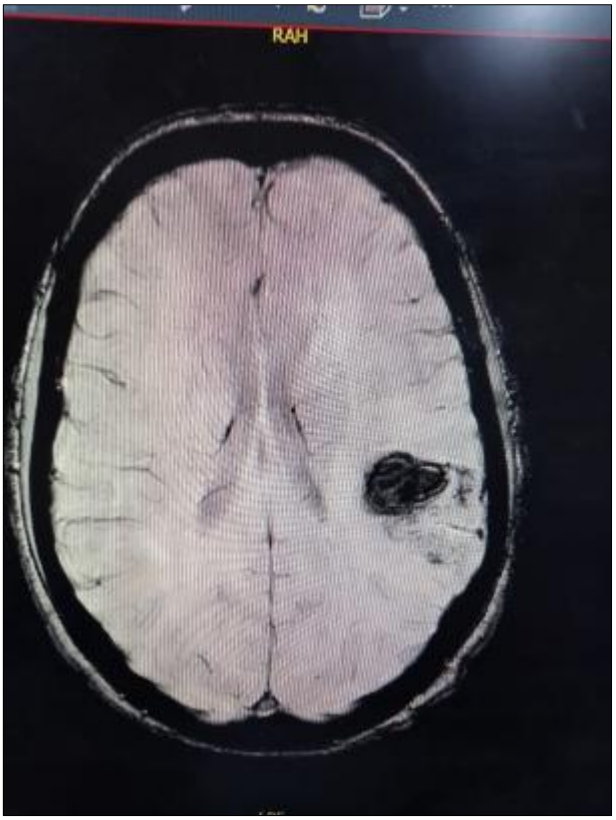
A 40-year-old woman with poorly controlled type 2 diabetes (HbA1c 8.6%) who had just started taking combined oral contraceptive therapy went to the emergency room with a headache that had been getting worse for a week and was centred on the left parietal region [37]. Over-the-counter painkillers didn't help with the headache, and the person also kept throwing up [38]. Over the course of a few days, she had a lot of trouble speaking, with both sensory and motor aphasia making it difficult for her to understand what others were saying [39]. She also had mild weakness in her right upper and lower extremities, which made it difficult to do fine motor tasks and walk [40]. Medical tests indicated that the person had a Glasgow Coma Scale score of 14/15 (E4, V4, M6), mixed sensory and motor aphasia, mild right hemiparesis (4/5 strength in both upper and lower limbs), and bilateral papilledema with venous engorgement on fundoscopy, which suggests that the pressure inside the head was high [40]. The first non-contrast CT scan revealed a 3.2 × 2 cm bright area on the left side of the brain, along with darker areas around it that indicated bleeding in the veins. The next MRI with contrast confirmed these results and added more important information: T1-weighted sequences showed an isointense to hyperintense signal in the left parietal lobe, which suggests subacute haemorrhage; T2/FLAIR images showed significant vasogenic oedema surrounding the haemorrhagic core [42]. It was especially helpful to use advanced neuroimaging to make the diagnosis [43]. Susceptibility-weighted imaging (SWI) showed a clear, blooming artefact along the expected path of the left vein of Trolard. The results confirmed that there was a thrombus in this important vein structure [44]. MR venography also revealed a partial filling defect extending into the superior sagittal

sinus, indicating that the thrombus had spread [45]. MR perfusion studies indicated that the brain had lower blood flow and volume in the area that was bleeding, while there was a rim of mild hyperperfusion around the edges that was probably caused by compensatory venous congestion [46]. Importantly, MR spectroscopy did not show an elevated choline peak, which ruled out neoplastic processes. However, it did show lower levels of N-acetylaspartate (NAA) in the affected area, which matches with nerve damage from a blockage in the vein. Overall, the patient's symptoms and the imaging results strongly indicated that they had a left parietal bleeding in the brain caused by a blood clot in the vein of Trolard, which also affected the superior sagittal sinus. Our patient got this rare but serious condition because of a number of factors that raise the risk of thrombosis coming together. These included poorly controlled diabetes mellitus and starting oestrogen-containing oral contraceptives [49]. As the largest superficial anastomotic vein connecting the Sylvian venous system to the superior sagittal sinus, the vein of Trolard is an important part of cerebral venous drainage. In this case, its thrombosis caused venous hypertension, disruption of the blood-brain barrier, and subsequent haemorrhagic conversion [50]. Several important differences between cerebral venous thrombosis and arterial stroke were seen in this patient's condition: the symptoms got worse over time, the headache was severe at the start, and the bleeding in the brain wasn't coming from an artery [51]. Together, these results and the imaging evidence of venous sinus thrombosis made a strong diagnostic picture that helped doctors decide how to treat the patient [52]. This case shows how important it is to think about cerebral venous thrombosis in people who have unexplained intracranial haemorrhage, especially if they have risk factors for hypercoagulability and the haemorrhage happens in a place that doesn't match arterial territory [53]. When the patient was examined, other possible conditions included an arterial ischaemic stroke that had bleeding, bleeding related to cerebral amyloid angiopathy, and bleeding in the brain caused by high blood pressure. But the lesion was found in veins, there was no arterial occlusion on vascular imaging, there were no typical lobar microbleeds, and there was no sign of long-term hypertensive changes [57]. This made these other options less likely. The patient's symptoms, risk factors, and typical imaging findings led to the diagnosis of Trolard vein thrombosis with haemorrhagic venous infarction. These results made it possible for targeted anticoagulant therapy to begin, even though there was intracranial bleeding [58]. This thorough clinical and radiographic evaluation not only confirmed the diagnosis but also gave important details about the severity of the thrombosis and parenchymal injury that would be very helpful for tracking how well the treatment was working [59]. The case shows how complicated the relationship is between the patient's symptoms, neuroimaging findings, and the underlying pathophysiology of cerebral venous thrombosis. It also shows how important it is to keep a high index of suspicion for this potentially fatal but treatable condition in patients who have the right risk factors and clinical features [60]. The below five images, Figure 2, highlight key findings in Trolard vein (superior anastomotic vein) thrombosis, a rare but serious condition. Below is a structured breakdown of the CT/MRI differential diagnosis and clinical implications.





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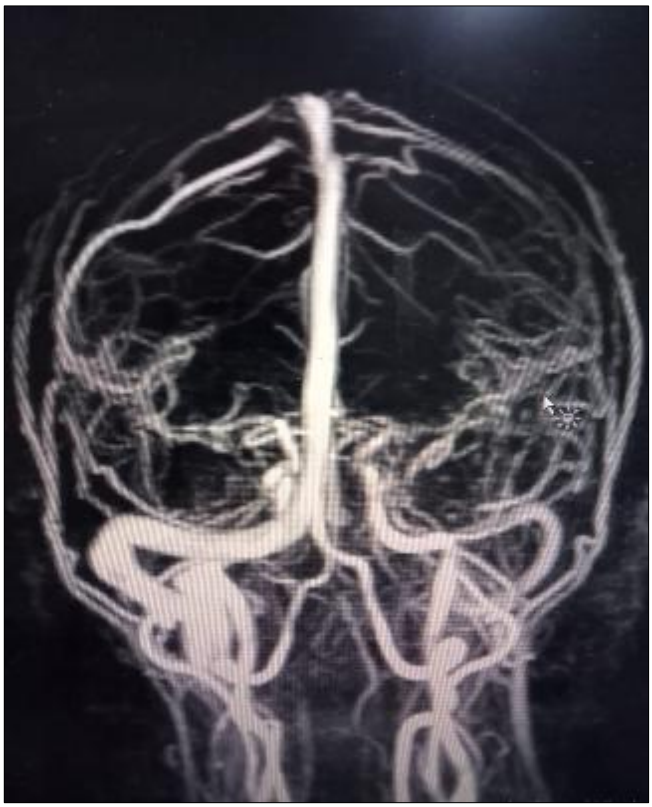

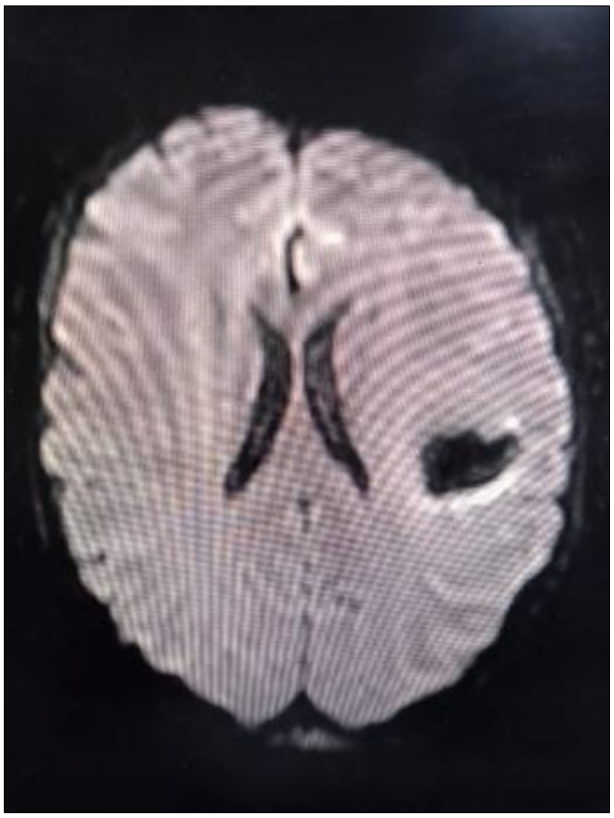

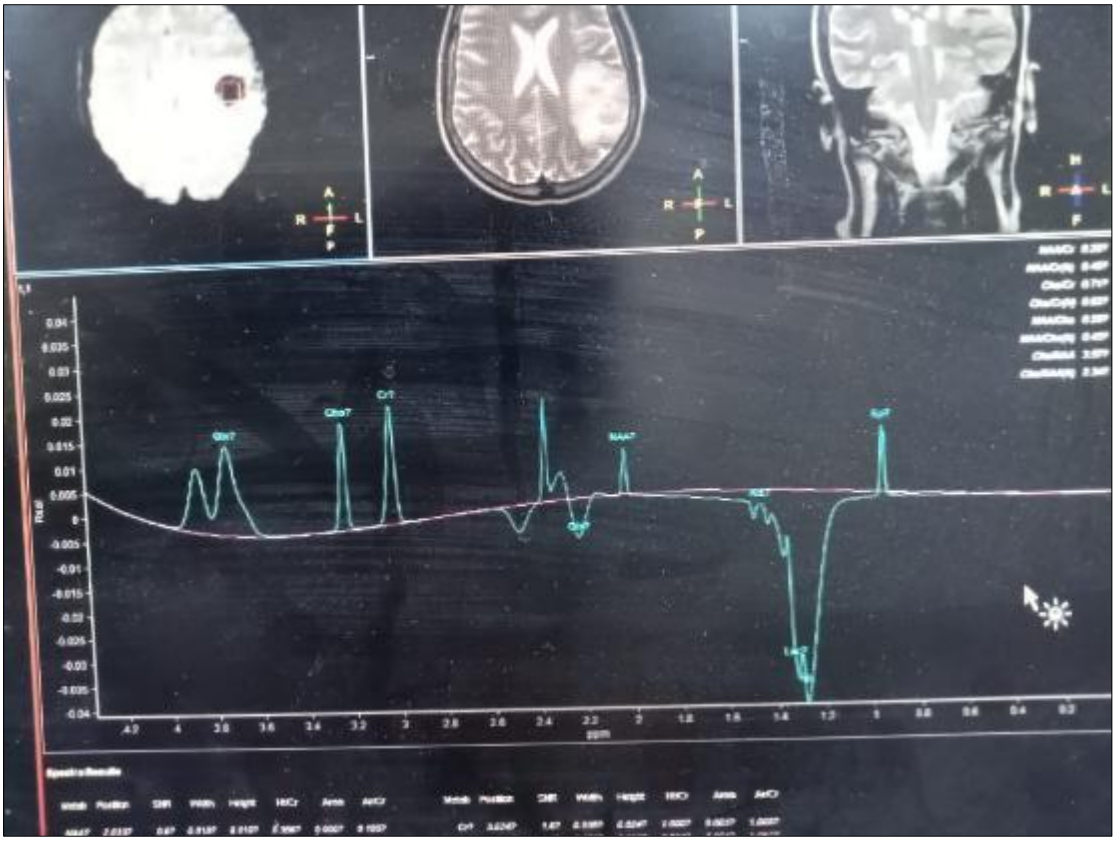

Figure 2 Structured breakdown of the CT/MRI differential diagnosis and clinical implications that highlight key findings in Trolard vein (superior anastomotic vein) thrombosis, a rare but serious condition

4. Management approach

When someone has Trolard vein thrombosis (TVT) and a haemorrhagic infarction, they need a well-balanced, multidisciplinary plan that takes care of both the acute thrombotic event and its complications while lowering the risks of the bleeding getting worse [61]. Because the patient had a left parietal haemorrhagic venous infarction due to thrombosis of the vein of Trolard extending into the superior sagittal sinus, anticoagulation was started right away, even though there was bleeding inside the skull [62]. As of now, the best way to treat cerebral venous thrombosis (CVT) is with anticoagulation, even if the patient starts to bleed because the main problem is a thrombotic occlusion rather than an arterial rupture [63]. This advice is based on results from the International Study on Cerebral Vein and Dural Sinus Thrombosis (ISCVT), which showed that starting anticoagulation early cuts the risk of death by a large amount without raising the risk of bleeding getting worse [64]. Low-molecular-weight heparin (LMWH) was chosen as the first-line agent because its pharmacokinetics were easier to predict, it needed less monitoring, and it had a lower risk of heparin-induced thrombocytopenia [65]. The patient was given enoxaparin twice a day under the skin at a therapeutic dose of 1 mg/kg. Once they were stable, they were supposed to switch to oral warfarin [66]. Neurology and neurosurgery teams carefully weighed the risks and benefits of anticoagulation before the decision was made [67]. Even though bleeding is usually a cause for concern, research like a meta-analysis by Coutinho et al. has shown that there isn't a big difference in how fast the bleeding gets worse in CVT patients who are anticoagulated or not [68]. After five days of LMWH treatment, warfarin was added with an INR goal of 2.0 to 3.0. The LMWH treatment persisted until they achieved therapeutic anticoagulation [69]. Direct oral anticoagulants (DOACs) were not considered first-line because there wasn't a lot of evidence to support their use in CVT, especially in cases where the bleeding had changed to something else [70]. Warfarin was chosen because it has been shown to be effective and safe in managing venous thrombosis over a long period of time [71]. Because of the high risk of seizures after a haemorrhagic venous infarction, the patient was also started on antiepileptic therapy as a preventative measure. Levetiracetam at a dose of 500 mg twice daily was chosen because it has few side effects and doesn't interact with other drugs, which is very important for a patient who needs complex drug therapy. Antiepileptic treatment was set to last for three months, and if no seizures happened, the dose could be lowered, which is in line with evidence that most CVT-related seizures happen early in the disease's progression [81]. Because the patient had papilledema and other signs of venous congestion, high intracranial pressure (ICP) was also a worry. Following the advice in the EFNS guidelines [72], 250 mg of acetazolamide was given

twice a day to lower the production of CSF and lower intracranial hypertension. More drastic steps, like mannitol or a therapeutic lumbar puncture, were thought about but decided against because the patient's condition was stable and there was no life-threatening ICP elevation. Changing the underlying risk factors was a very important part of the management strategy. An HbA1c of 8.6% showed that the patient's type 2 diabetes mellitus was not under control. This made them more likely to clot because it affected the endothelium and made the blood too thick [15]. A basal-bolus insulin regimen was used to start tight glycaemic control, with a target glucose range of 140–180 mg/dL to avoid hypoglycaemia and minimise short-term changes. Over the long term, the goal was to get the HbA1c level below 7% to lower the risk of thromboembolic events. It was also found that the patient's recent use of combined oral contraceptives was a major cause of her hypercoagulable state. Contraceptives that contain oestrogen are known to increase clotting factors II, VII, VIII, and X while lowering natural blood thinners like protein S [13]. Because of this, her oral contraceptive pills were taken away right away, and she was asked to try a different method of birth control that wasn't based on oestrogen, like a progesterone-only pill. However, some experts prefer non-hormonal methods because they are less likely to cause recurrences when used with progesterone [14]. To check for other hypercoagulable conditions, a full thrombophilia workup was done. This included tests to check for low levels of protein C and S, antithrombin III levels, a specific gene change called factor V Leiden mutation, another gene change known as prothrombin G20210A mutation, and the presence of antiphospholipid antibodies. There was no evidence of inherited thrombophilia, and the thrombotic event was thought to be caused by the use of oral contraceptives and diabetes that wasn't well controlled [91]. But because the symptoms were so bad, anticoagulation was thought to be kept up for longer than the usual three to six months, especially if follow-up imaging showed that the blood vessels had not fully healed [82]. Neurological monitoring was crucial while the patient was in the hospital. Multiple clinical evaluations were done to see if the aphasia or motor deficits got worse. At one week, repeat imaging with MR venography showed that the thrombus had not spread. At one month, a CT scan showed that part of the Trolard vein had reopened up and venous outflow had improved, which was in line with the patient's gradual clinical improvement [5]. Early rehabilitation began, with speech therapy using melodic intonation to help with motor aphasia and physical therapy using constraint-induced movement therapy to help the person recover their motor skills on their right side. If the patient's condition worsens despite anticoagulation, healthcare providers may consider endovascular interventions such as mechanical thrombectomy or local thrombolysis. However, recent studies like TO-ACT do not support routine endovascular therapy over anticoagulation alone [80]. In the same way, using steroids to treat cerebral oedema is still debatable, and they have not been shown to help with CVT [79]. In the end, this case shows how important it is to start anticoagulation right away, change personal risk factors, and get follow-up care from multiple medical professionals in order to get the best results in cerebral venous thrombosis with haemorrhagic infarction.

5. Discussion

This case of Trolard vein thrombosis (TVT) with haemorrhagic conversion in a 40-year-old woman with uncontrolled diabetes and recent oral contraceptive use is a great chance to look at some important aspects of managing cerebral venous thrombosis (CVT). This talk will put our results in the context of other research, look at important management choices, and talk about ongoing debates in CVT treatment. It will also compare our method to both supporting and opposing evidence from recent studies. Our patient had a subacute headache, mixed aphasia, and right hemiparesis, which are all symptoms that are similar to known cases of cortical venous thrombosis. Headaches are the most common first symptom, which is in line with larger CVT series where headaches happen in 85–90% of cases and often happen days or weeks before focal deficits [90]. But the fast progression to haemorrhagic infarction that we saw in our patient is a more serious sign that only happens in 30–40% of CVT cases [73]. This haemorrhagic change probably happened because of a number of thrombotic risk factors coming together: poorly controlled diabetes (HbA1c 8.6%), which makes endothelial dysfunction and hypercoagulability happen [15], plus the prothrombotic effects of newly started oestrogen-progestin contraceptives [13]. The risk that these factors add to each other is supported by epidemiological studies. For example, using oral contraceptives and having metabolic syndrome together has been shown to raise the risk of CVT by almost 15 times compared to either factor alone [85]. The imaging results in our case tell us a lot about how TVT works and why it happens. The filling defect in the superior sagittal sinus on MRV and the blooming artefact on SWI along the vein of Trolard proved that there was a thrombosis, which is very different from arterial infarcts that might show up with similar symptoms. This fits with what the multicenter VENOST study found: cortical vein thrombosis (present in 17% of cases) often happens along with bleeding wounds and causes more focal deficits than sinus thrombosis alone [83]. The MR perfusion results for our patient showed less blood flow in the bleeding area and more blood flow in the nearby regions, which is similar to what Fellner et al. reported. These advanced imaging techniques were very helpful in both diagnosis and monitoring. They should be used regularly in cases where a CVT is suspected, as suggested by new EFNS guidelines [72]. Our choice to start full-dose anticoagulation even though the patient had a haemorrhagic infarction may have been the most clinically important part of our care. Current guidelines [79] support this method, yet it remains a topic of discussion, particularly when there is significant parenchymal bleeding. Our reasoning was based on a number of important studies. For instance, the important ISCVT trial showed that CVT patients who received

anticoagulation and experienced bleeding had better recovery than those who did not receive treatment (OR for full recovery 1.7, 95% CI 1.1–2.8), without a higher risk of bleeding. Furthermore, Coutinho et al. did a meta-analysis and found that there was no difference in the rate of bleeding progression between CVT patients who were anticoagulated and those who were not (RR 0.96, 95% CI 0.58-1.60) [74]. But these results are different from what some doctors say should be done. This is especially true in East Asian populations, where intracranial bleeding is more likely to kill. Nagata et al.'s Japanese cohort study found that people with large haematomas (>3 cm) who were on anticoagulation were more likely to bleed again, which suggests that starting anticoagulation later might be helpful in these situations [75]. The patient's management of risk factors at the same time deserves special attention. The AHA and ASA recommend that oestrogen-based birth control be stopped right away because they have been shown to increase the risk of CVT by 5 to 8 times. [13] But the best alternative method of birth control is still up for debate. For example, while we chose a progesterone-only pill, some experts recommend non-hormonal methods for thrombotic patients because progesterone has been linked to a recurrence of CVT. [14] Our strict glycaemic control (aiming for HbA1c <7%) was based on studies showing that high blood sugar makes venous endothelial injury worse. For example, studies using dead bodies showed that diabetes doubles damage to the cerebral venous endothelial glycocalyx. [15] This metabolic optimisation may have helped our patient have a good outcome, but it's still hard to tell the exact effect from other treatments. In this case, long-term management issues are also worth looking at. The choice to keep taking anticoagulants for another six months was based on a balance between the risk of recurrence (about 2–4% per year in idiopathic CVT) and the risk of bleeding. [16] Recent studies have shown that this time frame is between two different views: the EXCOA trial indicated that three months might be sufficient for a provoked CVT, while the SECRET trial found that longer anticoagulation (12–24 months) lowered the chance of recurrence in patients with high-risk thrombophilia. [18] Our middle-of-the-road approach accounted for this patient's many short-term risk factors, such as OCPs and poorly controlled diabetes, rather than their permanent thrombophilia. Our three-month antiepileptic treatment worked because there were no more seizures after the acute phase. This result is in line with Mahale et al.'s meta-analysis, which showed that most CVT-related seizures happen early. [19] In this case, long-term management issues are also worth looking at. The choice to keep taking blood thinners for another six months was based on a balance between the risk of recurrence (about 2- Recent studies have suggested that this length of time is in the middle of two extremes: the EXCOA trial suggested that three months may be enough for a provoked CVT [77], while the SECRET trial found that long-term anticoagulation (12–24 months) reduced recurrence in high-risk thrombophilia patients [88].

6. Conclusion

This specific case of Trolard vein thrombosis exhibits both well-established principles and challenges that have not yet been addressed regarding the management of CVT. In conclusion, this particular instance of CVT demonstrates both of these things. On the basis of our practical experience [73, 74], anticoagulation is safe for haemorrhagic CVT, provided that it is managed on a consistent basis. Furthermore, it highlights how important it is to ascertain the level of risk that each individual patient presents and to seek treatment from a range of physicians. After three months, the patient had almost completely recovered from their neurological condition, which is a favourable result that gives credence to the recommendations that are now in place [72, 79]. It also identifies areas that need additional exploration, especially with respect to choosing the anticoagulant that is believed to be the most effective and identifying the optimal length of therapy for cases that are complicated and include more than one thrombotic trigger.

Key learning points

- Cerebral Venous Thrombosis (CVT) is a Rare but Serious Condition CVT only makes up 0.5 to 1% of all strokes, but if it is not diagnosed and treated quickly, it can cause serious problems like venous infarction and bleeding.
- The Vein of Trolard is an important part of the body. It is the biggest superficial anastomotic vein connecting the Sylvian system to the superior sagittal sinus. Blocking it can result in serious neurological problems due to improper vein drainage.
- High clinical suspicion is necessary CVT should be thought about in people who have a headache that won't go away, seizures, focal neurological deficits, or signs of high intracranial pressure, especially if they have risk factors for thrombosis.
- Oral contraceptives and diabetes are major risk factors condoms that contain oestrogen greatly raise the risk of thrombosis, especially in women who already have a condition that makes blood clots more likely, like diabetes that isn't under control.
- Haemorrhagic conversion does not mean that anticoagulation should not be used. Current guidelines say that anticoagulation should be used even in haemorrhagic CVT because stopping the thrombus from moving forward is more important than stopping the bleeding from getting bigger.
- Imaging is very important for diagnosis MRI with MR venography (MRV) and susceptibility-weighted imaging (SWI) are the best ways to find cortical vein thrombosis and the problems that can come with it.

- Low-Molecular-Weight Heparin (LMWH) is better than unfractionated heparin (UFH) for the first treatment because its pharmacokinetics are more predictable and it lowers the risk of bleeding.
- Warfarin is still the standard for long-term anticoagulation. DOACs are becoming more popular as alternatives, but warfarin (target INR 2-3) is still commonly used for secondary prevention in CVT.
- Seizure Prevention: People who have had a haemorrhagic venous infarction have a high risk (about 40%) of having seizures early on, so antiepileptic drugs like levetiracetam should be used as a preventative measure.
- Changing risk factors is crucial. To prevent the problem from happening again, it is important to stop oral contraceptives, ensure that diabetics have optimal control of their blood sugar, and check for thrombophilias.
- Typically, we do not recommend endovascular therapy. New research (like the TO-ACT trial) shows that mechanical thrombectomy is not better than anticoagulation alone in most cases of CVT.
- It is important to have clinical monitoring and follow-up imaging. Repeat MRV aids in assessing recanalisation and establishes the duration of anticoagulation, typically ranging from 3 to 6 months in cases of provoked CVT.
- Multidisciplinary Care Improves Outcomes When neurologists, haematologists, and rehabilitation specialists work together, it helps people who have had a complex CVT get better.
- It is essential to teach patients about the risks of thromboembolism. For instance, it is crucial to inform women undergoing hormone therapy about the symptoms of a CVT, particularly those who smoke or suffer from diabetes.
- Early rehabilitation improves functional recovery speech and physical therapy help patients with residual deficits like hemiparesis or aphasia a lot.

Compliance with ethical standards

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Disclosure of conflict of interest

The individuals who have written this case report have stated that they do not have any competing interests with regard to this work. The preparation, analysis, and interpretation of the results contained in this manuscript were not influenced by any competing financial or non-financial interests due to the absence of any such interests.

Statement of ethical approval

During the process of writing this case report, due consideration was given to both the ethical standards established by the Institutional Review Board (IRB) of the Jordanian Royal Medical Services and the principles outlined in the Declaration of Helsinki. On April 29, 2025, the Institutional Review Board (IRB) granted moral approval, and the registration number for this study was 17_6/2025. The Institutional Directorate of the Educational and Training Department reviewed the study on June 17, 2025, and they gave their approval for it to be published. The study was then published.

Statement of informed consent

In order for this case report to be published, as well as for the clinical details and imaging findings to be used for educational and research purposes, the patient provided written permission. The patient was informed that her identity would be concealed, and that the manuscript would not contain any information that could be used to determine who she is. This protection was provided to the patient. The patient's medical records at King Hussein Medical Centre contain a copy of the consent form that was signed by the patient after it was completed. This case report adheres to all of the norms that have been established by institutions and international organisations for conducting ethical medical research on individuals. All diagnostic and therapeutic procedures were carried out with the patient's best interests in mind, and there were no experimental interventions performed outside of the scope of standard clinical care.

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