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CEREBRAL VENOUS SINUS THROMBOSIS IN A YOUNG FEMALE FOLLOWING SHORT-TERM ORAL CONTRACEPTIVE USE: A RARE CASE REPORT

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ABSTRACT Cerebral venous sinus thrombosis (CVST) is an uncommon cerebrovascular disorder that disproportionately affects young women, in whom oestrogen-containing contraceptives are a recognised precipitant. We report a 21-year-old woman who developed CVST after only ten days of combined oral contraceptive use. She had a four-to-five-month history of oligomenorrhoea and an ultrasonographically detected simple follicular cyst, for which combined norgestrel and ethinylestradiol had been prescribed. She presented with an acute left fronto-temporal headache radiating to the ipsilateral hemiface, with nausea and photopsia, followed by a single generalised tonic–clonic seizure without focal deficit; neurological examination was unremarkable. Magnetic resonance imaging with an epilepsy protocol showed a gyriform diffusion abnormality in the left high frontal region with adjacent oedema and susceptibility blooming, together with loss of flow signal in the mid and posterior superior sagittal sinus. Magnetic resonance venography confirmed a long-segment superior sagittal sinus thrombosis with left frontal venous infarction. The suspected contraceptive was withdrawn and the patient was anticoagulated with low-molecular-weight heparin bridged to an oral anticoagulant, with an antiepileptic drug added. Even brief oestrogen exposure can precipitate CVST; a low threshold for venographic evaluation and prompt anticoagulation are essential.

KEYWORDS : Cerebral venous sinus thrombosis; Superior sagittal sinus; Oral contraceptives; Venous infarction.

INTRODUCTION

Cerebral venous sinus thrombosis (CVST) is an uncommon form of stroke, with an estimated incidence of approximately 13 to 16 per million per year, and it accounts for only a small proportion of all cerebrovascular events (Coutinho et al., 2012) –(Silvis et al., 2017). Unlike arterial stroke, it predominantly affects children and young to middle-aged adults, with a striking female preponderance in the reproductive age group that is largely attributable to sex-specific prothrombotic exposures such as pregnancy, the puerperium and hormonal contraception(Bousser & Ferro, 2007) –(Silvis et al., 2017). The superior sagittal and transverse sinuses are the most frequently affected (Stam, 2005). Combined oral contraceptives containing ethinylestradiol induce a hypercoagulable state and are among the commonest precipitants of CVST in young women, with the risk amplified when an underlying thrombophilia coexists(de Bruijn et al., 1998)(Martinelli et al., 1998) ––(Amoozegar et al., 2015). This risk is generally considered to accrue with duration of use and to be highest during the initial months of exposure. We describe a young woman who developed a symptomatic superior sagittal sinus thrombosis with venous infarction after only ten days of a combined oral contraceptive — an unusually short exposure that reinforces the importance of considering CVST in any young woman presenting with new headache or seizure while using oestrogen-containing contraception.

Case Report

Presentation. A 21-year-old woman presented to the emergency department with an acute left fronto-temporal headache radiating to the ipsilateral hemiface. The pain was moderate to severe in intensity and was accompanied by nausea and photopsia (flashes of light), without vomiting or any preceding flu-like illness. The headache was followed by a single episode of generalised tonic–clonic seizure at home, with no loss of consciousness and no post-ictal neurological deficit. She had no previous history of seizures. Her gynaecological history was notable for oligomenorrhoea of four to five months' duration, and pelvic ultrasonography had demonstrated a simple follicular cyst. For these complaints she had been prescribed a combination of norgestrel and ethinylestradiol (norgestrel 0.5 mg with ethinylestradiol 0.05 mg once daily), which she had taken for ten days before the onset of the seizure. There was no history of smoking, prior venous thromboembolism, or a family history of thrombosis.

Examination. On admission the patient was afebrile and haemodynamically stable. She was conscious and well oriented. There were no signs of meningeal irritation, no cranial nerve involvement and no focal neurological deficit or lateralising signs. Fundoscopy showed no papilloedema.

Investigations. Persistent headache and giddiness followed by a seizure prompted neuroimaging. Plain magnetic resonance imaging (MRI) of the brain with an epilepsy protocol demonstrated a focal gyriform area of restricted diffusion involving the left high frontal gyrus and adjacent juxtacortical region, hyperintense on diffusion-weighted imaging without a corresponding marked reduction on apparent diffusion coefficient maps, with surrounding T2/FLAIR hyperintense oedema. Minimal susceptibility blooming along the adjacent cortical sulcus suggested a small haemorrhagic component or cortical venous congestion. There was patchy loss of the normal flow signal within the mid and posterior thirds of the superior sagittal sinus, raising suspicion of CVST. Subsequent contrast-enhanced MRI with magnetic resonance venography (MRV) revealed a long-segment central non-enhancing filling defect involving the mid and posterior superior sagittal sinus with near-complete luminal occlusion, preserved peripheral wall enhancement, and reconstitution of flow in the posterior segment through collateral cortical venous channels. The findings were consistent with left frontal venous infarction secondary to superior sagittal sinus thrombosis (Figure 1). Routine haematological and coagulation parameters together with a thrombophilia screen were obtained; the results are summarised in Table 1.

Treatment. The suspected offending drug was discontinued

immediately. The patient was commenced on therapeutic anticoagulation with low-molecular-weight heparin (enoxaparin 60 mg subcutaneously every 12 hours), which was subsequently bridged to an oral anticoagulant (warfarin titrated to a target international normalised ratio of 2.0–3.0). An antiepileptic drug (levetiracetam 500 mg twice daily) was added for seizure control.

Outcome and follow-up. The patient's headache and giddiness improved and she remained seizure-free during her admission. She was discharged after seven days in a stable neurological condition (modified Rankin Scale score 0–1). She was counselled to permanently avoid oestrogen-containing contraception, oral anticoagulation was planned for six months, and follow-up MR venography at three months was arranged to document recanalisation.

Table 1. Laboratory and thrombophilia evaluation

Parameter	Result (illustrative)	Reference range
Haemoglobin	*12.9 g/dL*	12.0–15.0
Total leucocyte count	*8.3 ×10³/µL*	4.0–11.0
Platelet count	*256 ×10³/µL*	150–410
Erythrocyte sedimentation rate	*16 mm/h*	0–20
Prothrombin time	*13.0 s*	11.0–13.5
INR (pre-anticoagulation)	*0.98*	0.8–1.2
Activated partial thromboplastin time	*30 s*	26–36
D-dimer	*3.03 µg/mL FEU*	< 0.50
Protein C activity	*107 %*	70–140
Protein S activity	*86 %*	60–130
Antithrombin III activity	*99 %*	80–120
Lupus anticoagulant	*Negative*	Negative
Anticardiolipin antibody (IgG/IgM)	*Negative*	Negative
Anti-β2-glycoprotein I antibody	*Negative*	Negative
Factor V Leiden mutation	*Not detected*	Not detected
Prothrombin G20210A mutation	*Not detected*	Not detected
Serum homocysteine	*11.4 µmol/L*	5.0–15.0
JAK2 V617F mutation	*Not detected*	Not detected

DISCUSSION

Cerebral venous sinus thrombosis is an uncommon but clinically important cause of stroke in the young, with an estimated incidence of approximately 13 to 16 per million per year (Coutinho et al., 2012). It shows a marked predilection for women of reproductive age, in whom sex-specific prothrombotic states — pregnancy, the puerperium and hormonal contraception — account for much of the excess risk (Bousser & Ferro, 2007) –(Silvis et al., 2017). The superior sagittal sinus, involved in the present case, is among the most frequently thrombosed sinuses (Stam, 2005). Combined oral contraceptives containing ethinylestradiol shift the haemostatic balance towards a procoagulant state, and multiple studies have established them as an independent risk factor for CVST: a case–control study reported substantially increased odds of cerebral sinus thrombosis among users (de Bruijn et al., 1998), and a systematic review and meta-analysis confirmed the association across hormonal contraceptive preparations ––(Amoozegar et al., 2015). In large Indian registries, CVST likewise affects predominantly young adults, with identifiable prothrombotic risk factors in the majority (Narayan et al., 2012).

The distinctive feature of this case is the remarkably short interval — approximately ten days — between initiation of the contraceptive and the thrombotic event. Contraceptive-associated venous thrombotic risk is generally considered to be highest during the early months of use, and a symptomatic event within days is unusual. Such a presentation raises the possibility of a previously silent thrombophilia unmasked by even brief oestrogen exposure, as the coexistence of an inherited or acquired thrombophilia with oral contraceptive use is known to multiply the risk of cerebral vein thrombosis(Martinelli et al., 1998). In the present patient the thrombophilia screen was unremarkable (Table 1), which favours the contraceptive acting on an individual predisposition rather than a defined heritable defect; had the screen shown an abnormality such as factor V Leiden, the prothrombin G20210A variant or antiphospholipid antibodies, a combined "second-hit" mechanism would be implicated. The oligomenorrhoea for which the contraceptive was prescribed may reflect a polycystic ovary–spectrum disorder, although any independent contribution of this to thrombotic risk remains uncertain and should be interpreted cautiously.

Headache is the commonest and often earliest symptom of CVST, and seizures occur in roughly two-fifths of patients, more frequently when the thrombosis produces cortical or parasagittal venous infarction with a haemorrhagic component — a distribution that explains the generalised tonic–clonic seizure in this patient (Ferro et al., 2004). Magnetic resonance imaging combined with MR venography is the diagnostic modality of choice (Saposnik et al., 2024). The gyriform diffusion abnormality without a marked reduction in the apparent diffusion coefficient, the susceptibility blooming and the surrounding vasogenic oedema are characteristic of venous congestion and infarction, whereas the long-segment non-enhancing filling defect on contrast venography confirms the thrombus and helps distinguish it from a flow-related artefact or a hypoplastic sinus segment. Reconstitution of flow through cortical collaterals, as seen here, is a recognised compensatory response.

Anticoagulation is the cornerstone of treatment and is recommended even in the presence of haemorrhagic venous infarction, because preventing thrombus propagation outweighs the risk of aggravating haemorrhage (Einhäupl et al., 1991) (de Bruijn & Stam, 1999) –(Ferro et al., 2017) (Saposnik et al., 2024). Acute treatment with low-molecular-weight heparin followed by an oral anticoagulant is standard; the direct oral anticoagulant dabigatran has shown efficacy and safety comparable with dose-adjusted warfarin in a randomised trial and offers an alternative to vitamin K antagonists (Ferro et al., 2019). For thrombosis provoked by a transient risk factor such as oral contraceptive use, a limited course of anticoagulation of three to six months is generally appropriate, and oestrogen-containing contraception must be permanently discontinued with counselling on non-hormonal or progestogen-only alternatives. With prompt recognition and treatment the prognosis is generally favourable, most patients achieving functional independence (Ferro et al., 2004); follow-up venography is advised to document recanalisation.

CONCLUSION

Cerebral venous sinus thrombosis should be considered in any young woman presenting with new-onset headache, with or without seizures, who is using oestrogen-containing contraception — even after only brief exposure. A low threshold for MR venography, evaluation for an underlying thrombophilia, and prompt anticoagulation, which is safe even when venous infarction is haemorrhagic, are central to management. Oestrogen-containing contraception should be permanently discontinued and safer alternatives counselled. This case reinforces that short-term contraceptive use is not necessarily a benign exposure and can precipitate potentially serious cerebral venous thrombosis in susceptible individuals.

DECLARATIONS

Consent for publication: Written informed consent was obtained from the patient for publication of this case report and any accompanying images.

Ethical approval: Case reports at the authors' institution are handled per institutional policy.

Conflicts of interest: The authors declare that they have no conflicts of interest.

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Author contributions: [K.T. — concept, patient care, manuscript drafting; A.T., V.S., A.D., C.G. — patient care, literature review, critical revision.]

Abbreviations: CVST, cerebral venous sinus thrombosis; MRI, magnetic resonance imaging; MRV, magnetic resonance venography; DWI, diffusion-weighted imaging; FLAIR, fluid-attenuated inversion recovery; INR, international normalised ratio; LMWH, low-molecular-weight heparin; mRS, modified Rankin Scale; OCP, oral contraceptive pill.

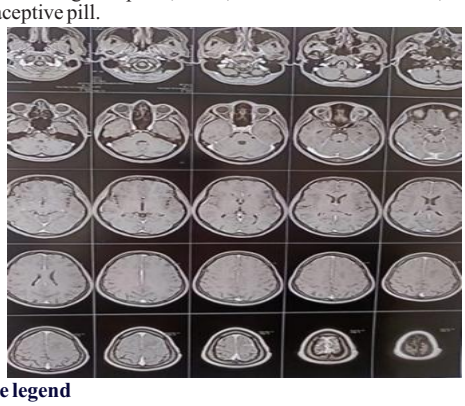

Figure legend

Figure 1. Axial T1-weighted magnetic resonance images of the brain

(contiguous sections from the skull base to the vertex) in the index patient. Magnetic resonance venography confirmed superior sagittal sinus thrombosis with left frontal venous infarction.

REFERENCES

1. Amoozegar, F., Ronksley, P. E., Sauve, R., & Menon, B. K. (2015). Hormonal Contraceptives and Cerebral Venous Thrombosis Risk: A Systematic Review and Meta-Analysis. Frontiers in Neurology, 6, 7. https://doi.org/10.3389/fneur.2015.00007
2. Bousser, M.-G., & Ferro, J. M. (2007). Cerebral venous thrombosis: An update. The Lancet. Neurology, 6(2), 162–170. https://doi.org/10.1016/S1474-4422(07)70029-7
3. Coutinho, J. M., Zuurbier, S. M., Aramideh, M., & Stam, J. (2012). The incidence of cerebral venous thrombosis: A cross-sectional study. Stroke, 43(12), 3375–3377. https://doi.org/10.1161/STROKEAHA.112.671453
4. de Bruijn, S. F., & Stam, J. (1999). Randomized, placebo-controlled trial of anticoagulant treatment with low-molecular-weight heparin for cerebral sinus thrombosis. Stroke, 30(3), 484–488. https://doi.org/10.1161/01.str.30.3.484
5. de Bruijn, S. F., Stam, J., Koopman, M. M., & Vandenbroucke, J. P. (1998). Case-control study of risk of cerebral sinus thrombosis in oral contraceptive users and in [correction of who are] carriers of hereditary prothrombotic conditions. The Cerebral Venous Sinus Thrombosis Study Group. BMJ, 316(7131), 589–592.https://doi.org/10.1136/bmj.316.7131.589
6. Einhäupl, K. M., Villringer, A., Meister, W., Mehraein, S., Garner, C., Pellkofer, M., Haberl, R. L., Pfister, H. W., & Schmiedek, P. (1991). Heparin treatment in sinus venous thrombosis. Lancet, 338(8767), 597–600. https://doi.org/10.1016/0140-6736(91)90607-q
7. Ferro, J. M., Bousser, M.-G., Canhão, P., Coutinho, J. M., Crassard, I., Dentali, F., di Minno, M., Maino, A., Martinelli, I., Masuhr, F., de Sousa, D. A., & Stam, J. (2017). European Stroke Organization guideline for the diagnosis and treatment of cerebral venous thrombosis – Endorsed by the European Academy of Neurology. European Stroke Journal, 2(3), 195–221. https://doi.org/10.1177/2396987317719364
8. Ferro, J. M., Canhão, P., Stam, J., Bousser, M.-G., Barinagarrementeria, F., & ISCVT Investigators. (2004). Prognosis of cerebral vein and dural sinus thrombosis: Results of the International Study on Cerebral Vein and Dural Sinus Thrombosis (ISCVT). Stroke, 35(3), 664–670. https://doi.org/10.1161/01.STR.0000117571.76197.26
9. Ferro, J. M., Coutinho, J. M., Dentali, F., Kobayashi, A., Alasheev, A., Canhão, P., Karpov, D., Nagel, S., Posthuma, L., Roriz, J. M., Caria, J., Frässdorf, M., Huisman, H., Reilly, P., Diener, H.-C., & RE-SPECT CVT Study Group. (2019). Safety and Efficacy of Dabigatran Etexilate vs Dose-Adjusted Warfarin in Patients With Cerebral Venous Thrombosis: A Randomized Clinical Trial. JAMA Neurology, 76(12), 1457–1465. https://doi.org/10.1001/jamaneurol.2019.2764
10. Martinelli, I., Sacchi, E., Landi, G., Taioli, E., Duca, F., & Mannucci, P. M. (1998). High risk of cerebral-vein thrombosis in carriers of a prothrombin-gene mutation and in users of oral contraceptives. The New England Journal of Medicine, 338(25), 1793–1797. https://doi.org/10.1056/NEJM199806183382502
11. Narayan, D., Kaul, S., Ravishankar, K., Suryaprabha, T., Bandaru, V. C. S. S., Mridula, K. R., Jabeen, S. A., Alladi, S., Meena, A. K., & Borgohain, R. (2012). Risk factors, clinical profile, and long-term outcome of 428 patients of cerebral sinus venous thrombosis: Insights from Nizam's Institute Venous Stroke Registry, Hyderabad (India). Neurology India, 60(2), 154–159. https://doi.org/10.4103/0028-3886.96388
12. Saposnik, G., Bushnell, C., Coutinho, J. M., Field, T. S., Furie, K. L., Galadanci, N., Kam, W., Kirkham, F. C., McNair, N. D., Singhal, A. B., Thijs, V., Yang, V. X. D., & American Heart Association Stroke Council; Council on Cardiopulmonary, Critical Care, Perioperative and Resuscitation; Council on Cardiovascular and Stroke Nursing; and Council on Hypertension. (2024). Diagnosis and Management of Cerebral Venous Thrombosis: A Scientific Statement From the American Heart Association. Stroke, 55(3), e77–e90. https://doi.org/10.1161/STR.0000000000000456
13. Silvis, S. M., de Sousa, D. A., Ferro, J. M., & Coutinho, J. M. (2017). Cerebral venous thrombosis. Nature Reviews Neurology, 13(9), 555–565.https://doi.org/10.1038/nrneurol.2017.104
14. Stam, J. (2005). Thrombosis of the cerebral veins and sinuses. The New England Journal of Medicine, 352(17), 1791–1798. https://doi.org/10.1056/NEJMra042354