RMMG
Revista Médica de Minas Gerais

CASE REPORT

Extensive cerebral venous thrombosis in late puerperium: a case report

Extensa trombose venosa cerebral em puérpera tardia: relato de caso

Marina Siqueira Teixeira[1], Marcelo Paes Menezes Filho[2]

ABSTRACT

Introduction: Cerebral venous thrombosis (CVT) is characterized by partial or complete occlusion of dural venous sinuses or cortical cerebral veins. It is more common in women, likely due to associations with oral contraceptive use, pregnancy and the postpartum period, typically occurring 2 to 3 weeks after delivery. **Case Report:** J.V.C. had left occipital headache that started 17 days prior, with worsening over the last 3 days, becoming refractory to regular dipyrone and associated with nausea, vomiting, visual disturbances and right superior temporal and left nasal quadrantanopsia. The patient had undergone vaginal delivery 34 days prior, with a single fetus at 38 weeks of gestation, without complications. The cranial angio-MRI revealed extensive thrombosis of cortical veins and venous sinus. Full anticoagulation with enoxaparin was initiated upon diagnosis of central venous thrombosis, along with optimized analgesia and administration of dexamethasone 40 mg/day for 3 days, resulting in complete resolution of headache and associated symptoms after 7 days of therapy. **Discussion and Conclusion:** CVT is an important cause of stroke in women during pregnancy and the postpartum period, accounting for 5-20% of cases and is more frequent in the puerperium and in patients aged 20-35 years. The patient in this case had CVT at 32 years old, 34 days after delivery. However, there was no report of the presence of other risk factors commonly associated with CVT. The absence of risk factors associated with the postpartum period is a notable characteristic of this case, as is the extent of cerebral venous involvement.

Keywords: Intracranial thrombosis; Sinus thrombosis, intracranial; Postpartum period.

[1] Hospital Escola Álvaro Alvim (HEAA), Campos dos Goytacazes, Rio de Janeiro, Brazil.

[2] Faculdade de Medicina de Campos (FMC), Hospital das Clínicas da Faculdade de Medicina da Universidade de São Paulo (HCFMUSP), Campos dos Goytacazes, Rio de Janeiro, Brazil.

Associate Editor Responsible:
Henrique Vitor Leite
Faculdade de Medicina da Universidade Federal de Minas Gerais.
Belo Horizonte/MG, Brazil.

Corresponding Author:
Marina Siqueira Teixeira
E-mail: marina.siqueira.teixeira@gmail.com

Supporting Sources:
None

Conflicts of Interests:
None

Ethics Committee:
Número do Parecer - 81914324.1.0000.5244

Received on: November 12nd, 2024
Approved on: August 17th, 2025
Publication Date: January 9th, 2026.

DOI: 10.5935/2238-3182.2025e35409-en

RESUMO

Introdução: A trombose venosa cerebral (TVC) é uma apresentação rara de acidente vascular encefálico (AVE), na qual ocorre a oclusão parcial ou incompleta de seios venosos durais ou de veias cerebrais corticais. É mais frequente em mulheres, com mediana de idade de 34 anos, provavelmente pela associação da doença com contraceptivos orais, gestação e puerpério, ocorrendo habitualmente 2-3 semanas após o parto. Outros fatores de risco associados incluem trombofilias, obesidade, malignidade, infecção e trauma cranioencefálico. **Relato de Caso:** J.V.C. procurou atendimento com queixa de cefaleia occipital esquerda, de intensidade progressiva iniciada há 17 dias e com piora nos últimos 3 dias, quando se tornou refratária à dipirona regular e se associou a náuseas, vômitos, turvação visual e quadrantonopsia superior temporal direita e nasal esquerda. Há 34 dias, foi submetida a parto por via vaginal, de feto único de 38 semanas, sem intercorrências. Apresentou angioressonância de crânio com extensa trombose venosa cortical na região parieto-têmporo-occipital esquerda e trombose dos seios transverso e sigmoide e do bulbo jugular ipsilaterais; congestão venosa (edema) na transição têmporo-occipital direita; e acentuada dilatação e trombose de veias corticais na transição têmporo-occipital esquerda. Foi iniciada anticoagulação plena com enoxaparina ao diagnóstico de trombose venosa central, analgesia otimizada, além de dexametasona 40mg/dia por 3 dias, com melhora completa da cefaleia e dos sintomas associados após 7 dias de terapia. **Discussão e Conclusão:** A TVC é uma importante causa de AVE em mulheres durante a gestação e no pós-parto, correspondendo de 5%-20% dos casos e sendo mais frequente no puerpério e em pacientes de 20-35 anos. Obedecendo à prevalência geral da doença, a paciente do caso acima apresentou TVC aos 32 anos e 34 dias após o parto. No entanto, não houve relato da presença de fatores de risco frequentemente associados à TVC, tais como trombofilias, obesidade, infecções durante a gestação, anemia e trauma cranioencefálico. A ausência de fatores de risco associados ao puerpério se mostra como característica importante do relato acima, tal como a extensão do acometimento venoso cerebral. A TVC tem incidência estimada de 1,16-2,02 a cada 100.000 pessoas, tratando-se, portanto, de uma doença incomum.

Palavras-chave: Trombose cerebral; Trombose dos seios intracranianos; Período pós-parto.

Introduction

Cerebral venous thrombosis (CVT) is characterized by partial or incomplete occlusion of dural venous sinuses or cortical brain veins. Thrombi commonly originate in the dural sinuses, later propagating to cortical brain veins and may also affect deep venous structures, leading to specific neurological syndromes[1]. It is more prevalent in women, with a median age of 34 years, showing a ratio of 3:1 compared to men, likely due to the association of the condition with oral contraceptives, pregnancy, and the puerperium, typically occurring 2-3 weeks postpartum. However, there is a low risk of recurrence of the condition[2,3]. Other associated risk factors include thrombophilias, obesity, malignancy, infection, and traumatic brain injury.

CVT has two pathophysiological mechanisms, including the emergence of parenchymal lesions due to ischemia and increased venous and capillary pressure, leading to the disruption

of the blood-brain barrier, resulting in decreased cerebrospinal fluid absorption and increased intracranial pressure (ICP). As ICP progresses, cerebral edema and hemorrhage may occur due to vessel rupture[1,2]. Pregnancy and puerperium are accompanied by physiological changes that favor the onset of cerebral venous thrombosis, such as increased coagulation factors and fibrinogen, elevated estrogen levels, hemodynamic stress, resistance to protein C and decreased protein S[4].

The following case report had as objective describing na extensive CVT, affecting various cerebral venous sites, in a late postpartum woman without prior risk factors. The report's relevance is due to the presentation of a case that, although uncommon, has typical characteristics, seeking to familiarize the medical team to the diagnosis and management of the disease. This case was approved by the Ethics Committee under number 81914324.1.0000.5244.

Case Report

J.V.C., female, 32-year-old, of African descent, married, pharmacy technician, native and resident of Campos dos Goytacazes (RJ), sought medical attention complaining of progressive left occipital headache that started 17 days prior, worsening over the last 3 days, becoming refractory to regular dipyrone and associated with nausea, vomiting, visual blurring and right superior temporal and left nasal quadrantanopsia. She reported a history of bilateral temporal headaches, approximately 1 episode per week, of mild intensity, relieved by 1g of dipyrone, which worsened after an attempt to use combined oral contraceptives, leading to discontinuation of the method.

Thirty-four days prior, she underwent vaginal delivery of a single fetus at 38 weeks, with first-degree lacerations, without other complications. During prenatal care, she had no complications during pregnancy. This was her first pregnancy, with no history of previous miscarriages. The patient had no comorbidities and denied allergies, alcohol or tobacco use, personal or family history of thrombotic disease and continuous medication use.

The patient received initial care at a reference obstetric unit and was subsequently transferred to the Hospital Escola Álvaro Alvim. Upon admission to the current unit,

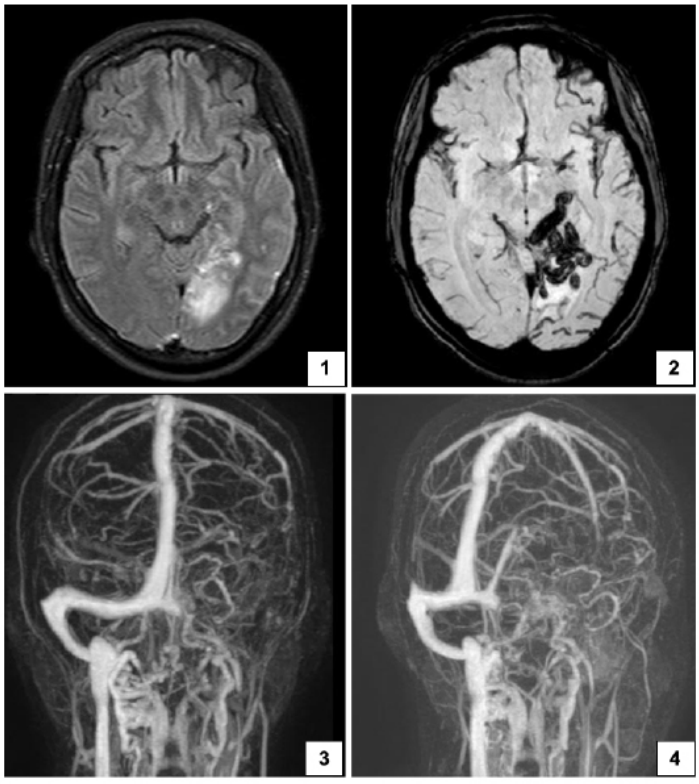

Figure 1-4. Cranial Angio-MRI. Marked dilation and thrombosis of cortical veins on the medial surface of the left temporal and occipital lobes, draining into the internal cerebral veins and ipsilateral transverse sinus, characterized by the presence of hyperintensity on T1-weighted images, hypointensity on SWI/T2* sequences, and lack of contrast enhancement; the presence of T1 hyperintensity suggests methemoglobin, indicating a subacute phase; slight ectasia and thrombosis of cortical veins at the left parieto-temporal junction, as well as thrombosis of the sigmoid and transverse sinuses and the ipsilateral jugular bulb; contrast enhancement of the thrombus within the left sigmoid sinus and jugular bulb indicates reorganization and suggests a later subacute phase; an area of hyperintensity on FLAIR and T2-weighted images on the medial surface of the left temporo-occipital junction, with no restriction on diffusion or hemorrhagic changes consistent with edema, secondary to venous congestion; hypoplasia of the P1 segment of the left posterior cerebral artery with vascularization of the P2 segment via a prominent posterior communicating artery, representing a fetal vascular pattern and an anatomical variation.

the physical examination revealed her to be awake, alert, oriented in time and space, in good general condition, normal-colored mucosae, hydrated, non-icteric, acyanotic and eupneic in ambient air; moving all four extremities freely, with muscle strength graded as V/V, preserved sensitivity, isocoric and photoreactive pupils; regular heart rhythm, two heart sounds, normal heart tones, no murmurs, present and symmetric pulses; universally audible vesicular murmur without adventitious sounds; abdomen soft, depressible, non-tender on palpation, without palpable masses or visceromegaly; lower limbs without edema, with free calves.

Among the complementary exams, cranial angio-MRI (Images 1-4) revealed extensive cortical venous thrombosis in the left parieto-temporo-occipital region and thrombosis of the transverse and sigmoid sinuses and ipsilateral jugular bulb; venous congestion (edema) in the right temporo-occipital transition; and marked dilation and thrombosis of cortical veins in the left temporo-occipital transition, necessitating the exclusion of a dural arteriovenous fistula; however, no enlarged feeding arteries were characterized in the current examination. Additionally, the cerebral arteriography showed venous drainage from the left hemisphere occurring through the deep venous system and extracranial venous collaterals; absence of filling of the left transverse venous sinus in the venous phase; and the arterial system showed no evidence of fistulas or arteriovenous malformations (AVMs). The requested laboratory tests are summarized in Table 1.

After the angio-MRI results, full anticoagulation with enoxaparin was initiated upon diagnosis of central venous thrombosis, along with optimized analgesia and dexamethasone 40mg/day for 3 days, resulting in complete resolution of headache and associated symptoms after 7 days of therapy.

In an ophthalmological evaluation after improvement of visual symptoms, the patient presented with visual acuity of 20/20 in both the right and left eyes; biomicroscopy showed clear corneas, isochoric and photoreactive pupils; tonometry revealed 10mmHg in the right eye and 12mmHg in the left eye; fundoscopy of the right eye showed a pink optic nerve with sharp borders, physiological cupping, mild peripapillary venous engorgement, a macula with preserved shine, vascular pattern appropriate for age, intact periphery, and attached retina; fundoscopy of the left eye showed a pink optic nerve with sharp borders, physiological cupping, a macula with preserved shine, vascular pattern appropriate for age, intact periphery and attached retina; intrinsic ocular

Table 1. Laboratory exams performed during hospitalization.

Laboratorial Exam	Results	Reference Values
Hemoglobin	12,2 g/dL	12-17 g/dL
Hematocrit	34,3%	37-50%
White Blood Cells	8200 mm^3	4000-11000 mm^3
Platelets	189000/ mm^3	140000-400000/ mm^3
ESR	28 mm	Até 10 mm
I.N.R.	1,18	-
aPTT	26,1"	28-43"
Sodium	139 mEq/L	136-147 mEq/L
Potassium	4,1 mEq/L	3,7-5,1 mEq/L
Magnesium	2,2 mg/dL	1,6-2,6 mg/dL
Calcium	11,7 mg/dL	8,8-11 mg/dL
ANA	Non Reagent	Non Reagent
Lupus Anticoagulant	0,92	≤ 1,2
Antithrombin III	88%	75-125%
IgG Anticardiolipin	9,4 GPL	< 15 GPL
IgM Anticardiolipin	9,4 MPL	< 12,5 MPL
Factor V Leiden	Negative	Negative
Protein C	138%	70-140%
Protein S	55%	60-130%
TSH	1,25 µUI/mL	0,48-5,6 µUI/mL
Free T4	1,21 ng/dL	0,85-1,5 ng/dL

Caption: ESR = Erythocyte Sedimentation Rate; INR = International Normalized Ratio; aPTT = Activated Partial Thromboplastin Time; ANA = Antinuclear Antibody; TSH = Thyroid-Stimulating Hormone.

musculature was preserved; keratometry of the right eye K1 43.65 / K2 44.20 and left eye K1 43.65 / K2 44.05.

The patient was discharged after clinical improvement, prescribed rivaroxaban 20mg/day and referred for follow-up in Internal Medicine, to repeat cranial angio-MRI and thrombophilia investigation within 6 months to 1 year, and in Ophthalmology, for retinography and visual field testing. The patient was advised to avoid hormonal contraceptives and placement of an IUD was suggested, for which she was referred to the Gynecology clinic.

Discussion

Cerebral venous thrombosis is a significant cause of CVA in women during pregnancy and the postpartum period, accounting for 5%-20% of cases and being more frequent in the puerperium and in patients aged 20-35 years[4]. In accordance with the general prevalence of the disease, the patient in the above case presented with CVT at 32 years of age, 34 days after delivery. However, there was no report of the presence of other risk factors commonly associated with CVT, such as thrombophilias, obesity, infections during pregnancy, anemia and traumatic brain injury[2,5].

Among the clinical manifestations of CVT, the most common is acute or subacute headache, occurring in 70-90% of patients and progressing to severe intensity hours or days after onset[1,2]. In cases of cortical infarcts, seizures may occur in up to 60% of cases, according to Bajko et al. (2021)[6], along with focal neurological deficits. Papilledema, visual disturbances and other signs of increased intracranial pressure may also be present. J.V.C.'s initial clinical presentation coincided with the most prevalent symptoms, including progressively intense headache, nausea, vomiting and visual disturbances; however, the patient did not experience seizure episodes.

In suspected CVT, it is necessary to perform cranial angio-MRI or CT angiography if the former is not available[2]. In this case, the former was performed, revealing thromboses in multiple cortical veins and in the left sigmoid and transverse sinuses, which were also the most affected venous sinuses (58.3% and 54.2%, respectively) in a retrospective study by Gazioglu et al. (2021)[5]. However, the patient's angio-MRI suggested an anatomical variation, leading to the performance of arteriography, which didn't confirm it.

The investigation of underlying thrombophilias was initiated during anticoagulation with enoxaparin, which can skew results and thus it cannot be arbitrarily asserted that the deficiency of protein S is related to the patient's CVT[7]. Therefore, the reevaluation of these markers was postponed until after recovery from central lesions, in order to assess possible thrombophilia without ongoing anticoagulation.

The treatment of this condition in the acute phase consists of anticoagulation with low molecular weight heparin or unfractionated heparin, with a preference for the former. The presence of associated hemorrhage does not contraindicate the use of anticoagulants due to its underlying pathophysiology. In patients with neurological deterioration despite anticoagulation, venous thrombolytics or mechanical thrombectomy may be considered. After the acute phase, anticoagulation can be maintained with warfarin or direct oral anticoagulants (DOACs)[1,7,8]. In the above case, the patient responded well to enoxaparin in the acute phase, combined with dexamethasone to reduce the existing cerebral edema. In the maintenance phase, rivaroxaban was chosen for convenience, considering the patient's young age.

Conclusion

The absence of associated risk factors in the puerperium is a significant characteristic of the above case, as is the extent of cerebral venous involvement. CVT has an estimated incidence of 1.16-2.02 per 100,000 people, making it an uncommon disease[1]. Therefore, it is important to report cases where its typical characteristics are demonstrated to familiarize the medical team with the diagnosis and management of this condition.

Authors' Contributions

We describe contributions to the papers using the taxanomy (CRediT) provide above:

Conceptualization, Investigation, Methodology, Visualization & Writing – original draft: MS Teixeira. *Project administration, Supervision & Writing – review & editing*: MS Teixeira; MP Menezes Filho. *Validation & Software*: doesn't apply. *Resources & Funding acquisition*: doesn't apply. *Data curation & Formal Analysis*: doesn't apply.

Copyright

References

1. Ropper AH, Klein JP. Cerebral Venous Thrombosis. Longo DL, editor. N Engl J Med [Internet]. 2021 Jul; [cited 2024 Jun 01]; 385(1):59-64. Available from: http://www.nejm.org/doi/10.1056/NEJMra2106545.

2. Coutinho JM. Cerebral venous thrombosis. J Thromb Haem. 2015;13:S238-44. Available from: https://www.jthjournal.org/article/S1538-7836(22)04238-6/fulltext.

3. Algahtani H, Bazaid A, Shirah B, Bouges RN. Cerebral venous sinus thrombosis in pregnancy and puerperium. Brain Circ [Internet]. 2022; [cited 2024 Jun 01]; 8(4):180-7. Available from: https://journals.lww.com/brci/fulltext/2022/08040/cerebral_venous_sinus_thrombosis_in_pregnancy_and.3.aspx.

4. Gazioglu S, Dinc G. Cerebral venous sinus thrombosis in pregnancy and puerperium. Acta Neurol Belg. 2021;121(4):967-72. DOI: https://doi.org/10.1007/s13760-020-01459-3.

5. Khan M, Arauz A, Uluduz D, Barboza MA, Duman T, Cano-Nigenda V, et al. Predictors of Mortality and Functional Outcome in Pregnancy and Puerperium-Related Cerebral Venous Thrombosis [Internet]. Cerebrovasc Dis [Internet]. 2023; [cited 2024 Jun 01]; 52(4):393-400. Available from: https://karger.com/ced/article-abstract/52/4/393/836333/Predictors-of-Mortality-and-Functional-Outcome-in?redirectedFrom=fulltext.

6. Bajko Z, Motataianu A, Stoian A, Barcutean L, Andone S, Maier S, et al. Postpartum cerebral venous thrombosis—a single-center experience. Brain Sci [Internet]. 2021; [cited 2024 Jun 01]; 11(3):1-9. Available from: https://www.mdpi.com/2076-3425/11/3/327.

7. Linnemann B, Hart C. Laboratory Diagnostics in Thrombophilia. Hamostaseologie [Internet]. 2019; [cited 2024 Jun 01]; 39(1):49-61. Available from: https://www.thieme-connect.com/products/ejournals/abstract/10.1055/s-0039-1677840.

8. Ferro JM, Bousser MG, Canhão P, Coutinho JM, Crassard I, Dentali F, et al. European Stroke Organization guideline for the diagnosis and treatment of cerebral venous thrombosis – endorsed by the European Academy of Neurology. Eur J Neurol [Internet]. 2017 Oct 1; [cited 2024 Jun 01]; 24(10):1203-13. Available from: https://onlinelibrary.wiley.com/doi/epdf/10.1111/ene.13381.