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CLINICAL CASES

Cerebral venous thrombosis in obstetrics: literature review and clinical case reports

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Abstract

The first case of cerebral venous thrombosis, described in the past century, was identified in obstetric patients in 1825. The clinical manifestations represent a real diagnostic challenge due to the few recorded cases, the wide variety of possible clinical symptoms, and the diversity of medical conditions that cause it. In the past century, the etiology was mainly associated with septic processes; however, due to the widespread use of antibiotics, this cause has been considerably reduced. In the context of the puerperium, several conditions make this group of patients more vulnerable, such as dehydration, hypercoagulable state, iron deficiency, puerperal sepsis, and preeclampsia, the latter is particularly known for an endothelial lesion with different degrees of associated severity, according to the clinical phenotype present. This review aims to highlight the most relevant aspects of cerebral thrombosis in the context of a puerperal patient. It will also present clinical cases reports treated successfully in this health unit.

Keywords: *Cerebral. Venous thrombosis. Preeclampsia.*

Introduction

Like the regional systemic circulation, approximately 80% of the brain's blood circulation is in the venous bed, while about 20% is in the arterial circulation. Cerebral venous thrombosis (CVT) accounts for approximately five to ten out of every thousand cases of cerebral vascular disease. The approximate incidence worldwide is three and a half cases per million inhabitants, predominantly found in young people and women, unlike arterial cerebral vascular disease. It is also worth mentioning that cerebral venous disease has significant clinical variability due to the anatomical variants in this system, with constantly changing and sometimes imprecise drainage patterns. Because the venous system at this level lacks valves, it has a substantial capacity for compensation when thrombosis occurs. Regardless of the anatomical variants present, multiple anastomoses between superficial cerebral veins facilitate collateral circulation in thrombosis of this system. These characteristics make this complication have a considerable clinical heterogeneity, dependent on the location, degree of an extension, and functionality of the regional collateral circulation. One of the characteristics of the clinical presentation that is almost invariable is the difference between the involvement of the deep venous system, which generally leads to a worse evolution, with the involvement of the superficial

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system, which is associated with mild and self-limited forms of development. Since it is a disease with a very low incidence, it is hard to perform epidemiological studies and randomized clinical trials (RCT) with high statistical value to clarify better diagnostic, preventive, and therapeutic options. In the present document, we review the bibliography concerning this topic, highlighting its epidemiology, pathological anatomy, diagnostics, and therapeutical options.

Epidemiology

Deep vein thrombosis incidence is estimated to be approximately three cases per million inhabitants. Note that case series in pediatric patients have been described with an incidence up to 10 times higher[1]. This clinical condition is much more frequent in females between the ages of 20-35, inherent to the related obstetric conditions and the hormonal characteristics particular to that sex. It is estimated that it accounts worldwide for five out of every 1000 cases of cerebral vascular events[2].

The ISCVT study "International Study on CVT and Dural Sinus Thrombosis," carried out between May 1998 and May 2001, included 624 adult patients with venous thrombosis. The type of study was prospective observational, multinational (21 countries), and multicenter (89 centers), making it one of the most important concerning this topic. It is worth mentioning that 58% of the cases reported in Mexico were associated with pregnancy or puerperium, as opposed to approximately 8% of the cases reported in other countries[3]. James et al. evaluated more than 9 million pregnant and puerperal patients and found significant results: a total of 2850 cases at a rate of 34.2/100,000 deliveries, a total of 117 deaths correspond to 1.4 deaths per 100,000 deliveries, 22% of the survivors were discharged to another facility, with increasing age, this risk rose with a cutoff point of 35 years or older. The group of patients of African descent had a higher risk with an odds ratio (OR) of 1.5 (95% confidence interval: 0.2-1.9). Medical conditions that were strongly associated with stroke overall included migraine (OR 16.9), thrombophilia (OR 16.0), systemic lupus erythematosus (OR 15.2), previous heart disease (OR 13.2), sickle cell disease (OR 9.1), hypertension (OR 6.1), and thrombocytopenia (OR 6.0). Some obstetric complications were also found to be associated with significantly increased risk, such as postpartum hemorrhage (OR 1.8), preeclampsia (OR 4.4), transfusion (OR 10.3), and puerperal infection (OR 25.0), in all the above, venous thrombosis accounted for 2% of cases[4].

One of the most significant cohort publications in the journal Continuum Neurology was conducted by Fadar et al. It was a retrospective study, which used validated codes to identify all new cases of CVT found (n = 5.567) in the New York and Florida State Inpatient Databases between 2006 and 2016. The standardized annual incidence for CVT by age and sex was 13.9-20.2 cases per 1 million inhabitants (females 20.3-26.9, males 6.8-16.8); by age/sex (age range; females 18-44 years, varying from 24.0% to 32.6%; males 18 to 44 years ranging from 5.3 to 12.8); according to race (Blacks: 18.6-27.2; Caucasians: 14.3-18.5; Asians: 5.1-13.8). The incidence in women aged 18-44 remained unchanged over time[5].

Etiology

It consists of a series of clinical conditions associated with hypercoagulable states, which, in turn, are linked with systemic venous thrombosis and, eventually, with CVT; some of these conditions include adenocarcinoma, polycythemia vera, thrombocytopenia, leukemia, sickle cell anemia, pregnancy, and the puerperal period. Other causes include direct head trauma, venous sinus procedures, bacterial meningitis, and invasive neuromonitoring.

Other predisposing conditions such as antiphospholipid syndrome, factor V Leiden mutation, protein C and S deficiency, prothrombin mutation, and hyperhomocysteinemia should be ruled out. In most cases, thrombophilias represent 22% of cases, antiphospholipid syndrome 6%, associated with the gestational and puerperal period in 10-58% of cases. With current doses of combined contraceptives, the risk of thrombosis has considerably decreased as an independent factor. It has related to a complication of oncology therapies, such as tamoxifen, cisplatin, and l-asparaginase. Reviews have also been found where CVT incidence increased within erythropoietin users[6].

Cerebral venous anatomy

The cerebral venous system drains through a superficial and deep system, draining into the principal sinuses: superior and inferior sagittal, lateral sinuses, cavernous sinus, and straight sinus. Finally, they drain through the internal jugular vein. The superficial venous system drains mainly in the superior sagittal sinus and lateral sinuses due to the large number of anastomoses; occlusion cases at this level are hard to diagnose. The deep system drains venous blood from the deep

white matter of the hemispheres and basal ganglia through the vein of Galen. This system also has multiple anastomoses, which hinders diagnosing small caliber vessel occlusion. However, in the case of thrombosis, it allows venous drainage to have alternative routes. The posterosuperior venous system is formed by the superior and inferior sagittal sinus, lateral sinuses (transverse and sigmoid portions), straight sinus, and occipital sinus. The anteroinferior venous system is made up of the superior and inferior petrosal sinuses and the cavernous sinus. It is crucial to mention that the venous sinuses play a significant role in the reabsorption of cerebrospinal fluid through the arachnoid villi. The superior sagittal system drains venous blood from most of the cerebral cortex and corresponds to the sickle border of the brain. The lateral sinuses drain from the press of the Herophilus to the jugular bulb, with its transverse and sigmoid portion attached to the mastoid process. This portion is susceptible to thrombosis in patients with mastoiditis and otitis media. Drainage from the lateral sinus also comes from the cerebellum, brain stem, and posterior part of the cerebral hemispheres. The cavernous sinuses are located at the cranial base, superolateral to the sphenoidal venous sinuses. There are significant anatomical relationships at this level through its lateral walls run the common ocular motor nerves (III), trochlear (IV), and the ophthalmic and maxillary branches of the trigeminal (V). Medially, the external ocular motor nerve (VI) and the internal carotid artery are accompanied by its sympathetic plexus. The cavernous venous sinuses drain to the internal jugular veins through the petrosal sinuses[1,6].

Physiopathology

CVT is caused by an imbalance between prothrombotic substances and thrombotic processes, leading to the initiation and propagation of the coagulation cascade in the venous sinuses or cerebral veins. Venous blood is forced to remain in the system of small vessels and capillaries provoking an increase in venous and capillary pressure. The specific anatomy of the cerebral venous system and its extensive anastomoses often provides sufficient collateral circulation to compensate for such pressure changes. When this collateral circulation system becomes insufficient, it disrupts the blood-brain barrier and decreases cerebral perfusion pressure, resulting in cerebral edema, ischemia, and often intracerebral hemorrhage. It has been shown that cerebral perfusion is possible in the early stages of venous thrombosis through collateral circulation, as demonstrated in experimental models by laser and Doppler flowmetry. In most cases, parenchymal injury occurs when the thrombus extends to cortical veins; however, in animal models, it has been found that sinus occlusion may be sufficient to cause venous infarcts. Parenchymal lesions occur in approximately 60% of cases, with vasogenic components and cytotoxic edema. As mentioned above, the dural sinuses play a significant role in the absorption of cerebrospinal fluid, mediated by arachnoid villi, known as Pacchionian granulations, found in the walls of the venous sinuses. Dysfunction of these granulations may cause a decrease in cerebrospinal fluid absorption, resulting in intracranial hypertension[7].

Clinical presentation

Symptoms associated with CVT can range from asymptomatic events to life-threatening clinical pictures, depending on the degree of involvement and the venous territory involved. The superior sagittal sinus is the most affected in approximately six out of ten cases, with symptoms ranging from headache due to increased intracranial pressure (ICP) to focal neurological deficits, such as hemiparesis, hemianopsia, and even seizures. Thrombosis of the transverse sinus affected four out of ten cases; if it presents occlusion of the vein of Labbé, it may be related to hemorrhage and is characterized by cephalea, aphasia, and less frequent seizures. Involvement of the sigmoid venous sinus is rare and may cause mastoid pain and, less frequently, cranial nerve neuropathy. Thrombosis of deep veins such as the internal cerebral veins, basal veins of Rosenthal, the vein of Galen, and the sinus rectus can affect up to 18%, causing edema of the thalamus, causing altered alertness, and occasionally paralysis. Isolated intracranial hypertension (typically due to chronically evolving sagittal sinus thrombosis) manifests with papilledema, headache, and visual disturbances. Cavernous sinus thrombosis is the rarest; however, it is easier to diagnose due to its clinical presentation (ocular pain, chemosis, ocular ptosis, and oculomotor nerve palsy associated with previous sinus infection).

Cephalea is present in nine out of ten symptomatic CVT cases. It is a very nonspecific symptom; however, in any patient with disabling intensity, new onset, persistent, worsening with valsalva maneuver, lack of improvement with regular analgesia, and with risk factors for thrombosis or clinical evidence of papilledema, a

complete protocol for a vascular headache should be performed, including contrasted imaging methods.

In the peripartum period, symptoms are non-specific and atypical, with no specific recommendations concerning gestation and puerperium[8]. Post-anesthetic puncture headaches can be confused with cases of CVT, so they should not be underestimated and should be adequately assessed, especially in patients with thrombotic risk factors[9]. After applying an epidural patch due to rupture of the dura mater in post-operative cesarean section patients who present intense and progressive cephalea, imaging studies should be performed quickly to rule out CVT[10].

The "STANDARD GOLD" diagnostic test is a digital subtraction angiography with a sensitivity of 95% and a specificity of 91%. This test demonstrates the absence of flow in the involved venous territory. D-dimer measurements can be performed; however, the evidence level and recommendation strength reported in most guidelines are weak[11].

Treatment

In an acute event of cerebral venous thrombosis, heparin at therapeutic dose should be used, unless there are absolute contraindications. In case of cerebral hemorrhage, there should be an individual evaluation. Evidence: moderate. Recommendation: Strong.

The administration of low-molecular-weight heparins over unfractionated heparins is suggested.

This information does not apply to patients with contraindications for low-molecular-weight heparins (e.g., renal failure) or in situations where rapid reversal of anticoagulation is required. Evidence: Low. Recommendation: Weak.

In the case of thrombolysis as a therapeutic option, no studies with compelling evidence were found. Therefore, the patient's context should be individualized and approached in a multidisciplinary fashion to consider this option.

The use of acetazolamide as part of the treatment protocol is not recommended. Evidence: Low. Recommendation: Weak.

Low-molecular-weight heparin therapy is suggested as a first antithrombotic option in pregnant and postpartum patients. Evidence: Low. Recommendation: Weak[12].

Coumarins are a safe option. However, they should be used as a second line. Direct anticoagulants use is a safe option and an alternative to Warfarin use[13]. The duration of treatment per most of the consulted guidelines should be from 3 to 12 months; it may be shorter if the cause of thrombosis was provoked (traumatic, surgical, and non-surgical) and longer if it was spontaneous[14].

In the context of neurocritical patients, special care is established aimed at maintaining adequate cerebral homeostasis.

In the context of neurocritical patient, special care is established aimed at maintaining adequate brain homeostasis, acronyms such as "THE MANTLE" OR "GHOST-CAP" are used according to publications that arise certain particular neuroprotection goals to favor adequate evolution especially in clinical scenario of trauma; however, they are used in obstetric patients who require neurocritical care[15,16].

Taccone et al., suggest the following goals:

Glucose: target levels between 80 and 180 mg/dL may be reasonable.

Hemoglobin: is an important determinant of oxygen delivery (DO2). No well-designed RCT has addressed ideal transfusion thresholds in patients with acute brain injury, but a 7-9-g/dL threshold seems reasonable.

Oxygen: targeting a SpO2 between 94 and 97% seems reasonable.

Sodium: avoid sodium levels < 135 mEq/L, hypernatremia may occur as a result of ICP-directed therapies, and sodium levels up to 155 mEq/L may be tolerated in such conditions.

Temperature > 38.0°C should be avoided, particularly if associated with neurological deterioration or altered cerebral homeostasis.

Comfort, including control of pain, agitation, anxiety, and shivering, is an important goal, to avoid physical and psychological distress, excessive cerebral stimulation, increased ICP, and secondary tissue.

Arterial blood pressure is the main determinant of CBF. Maintaining a mean arterial pressure (MAP) ≥ 80 mmHg and a CPP ≥ 60 mmHg may be reasonable in unconscious patients; in awake patients, MAP targets can be titrated according to repeated neurological examination.

PaCO2 causes changes in CBF (a 4% change in CBF per mmHg change in PaCO2). If intracranial compliance is reduced, any increase in CBF may increase cerebral blood volume, and thereby ICP. On the other hand, excessive hyperventilation can result in cerebral ischemia, and PaCO2 < 35 mmHg should be avoided.

Godoy et al. suggest the following goals:

Temperature, to avoid hyperthermia is fundamental, goal 36-37°C (core). Hyperthermia can also yield to cerebral hypoxia due to increased metabolism. Therefore, it is desirable to maintain central temperature levels between 36 and 37°C.

Hemoglobin, to keep and maintain good quality and quantity of transporter, is essential. The optimal levels

of Hgb remain unknown; however, it seems reasonable to reach and maintain Hgb values between 7 and 9 g/dL.

Electrolytes and acid basic status: ''Physiological balance is the cornerstone.'' To ensure that Hgb dissociation curve remains within functional ranges (p50 = 26-28 mmHg), to reduce the risk of cerebral ischemia and intracranial hypertension pH: 7.35-7.45 36-37.5°C, and to minimize or treat cerebral edema, it is crucial to maintain a slight hyperosmolar state (serum Na+140-150 mEq/L) and to avoid hypotonic fluids.

Metabolism: ''If metabolism is accelerated, O2 demands increase.'' Brain metabolism is the main determinant of the rate of cerebral O2 consumption. Oxygen pressure of the brain parenchyma locally reflects the balance between the supply and consumption of O2 and should be maintained at values above 18 mmHg. The venous oxygen saturation obtained from the jugular bulb (SvjO2), globally represents the O2 that returns to the general circulation after being consumed by brain cells and should be maintained at values > 55%.

Arterial blood pressure: ''Arterial hypotension is apocalyptic for injured brain.'' Recommended blood pressure targets include systolic blood pressure > 100-110 mmHg; normal volemia, diuresis > 30 mL/h, preserved peripheral perfusion, and central venous pressure: 6-10 cmH2O.

Nutrition and glucose: ''Glucose, essential fuel for the damaged brain.'' Glycemia levels < 110 mg/dL may cause non-ischemic metabolic crises. In contrast, hyperglycemia > 180 mg/dL causes neurotoxic cascades (inflammation, micro thrombosis, and edema) and disturbs the homeostasis of the internal environment (hyperosmolarity and dehydration), compromising the immune status, among other alterations.

Target of oxygenation: ''Both extremes of systemic oxygenation are deleterious.'' Measures must be taken to achieve PaO2 80-120 mmHg, and SaO2 >95%.

Lung protective ventilation: ''Protecting the lungs protects the brain.'' According to available evidence lung protective ventilation with a controlled mode, tidal volumes between 6 and 8 mL/kg, minimum respiratory rates to ensure levels of PaCO2 between 35 and 45 mmHg, and FiO2 and PEEP necessary to achieve systemic oxygenation targets as we mentioned above, to prevent mechanical ventilation-induced lung injury (barotrauma, biotrauma, and volutrauma), plateau pressure should be kept < 2 cm H2O, driving pressure < 13 cm H2O, and mechanical power below 17 J/min. It is recommended not to use routinely hyperventilation and to maintain PaCO2 levels between 35 and 45 mmHg.

Edema and ICP control: ''Brain swollen, brain on the ledge.'' The recommended main targets to be achieved should be the following: (a) ICP< 22 mmHg; (b) CPP: 55-70 mmHg; (c) optic nerve sheath diameter (ONSD)<5.5 mm; d) pulsatility index (PI)<1.2; and e) cerebral CT scan without edema signs.

Tonny et al. suggest based on analysis of recent investigation: "Tissue hypoxia after brain injury is not confined to regions with structural abnormality and can occur in the absence of conventional macrovascular ischemia. This physiologic signature is consistent with microvascular ischemia and is a target for novel neuroprotective strategies" [17]. Godoy et al. suggest a different model on evaluation of brain injury: "For decades, one of the main targets in the management of severe acute brain injury (ABI) has been intracranial hypertension (IH) control. Meanwhile, progress in the understanding of intracranial content (brain, blood, and cerebrospinal fluid) dynamics and recent development in monitoring techniques suggests that targeting intracranial compliance (ICC) could be a more reliable approach rather than guiding actions by predetermined ICP values. Therefore, an intracranial compartmental syndrome (ICCS) can occur with deleterious brain effects, precipitating a reduction in brain perfusion, thereby inducing brain ischemia"[18].

Clinical cases

Case number 1

General characterization

Thirty years old, with no chronic personal or family history of degenerative. Three previous pregnancies with vaginal delivery, history of hospitalization due to urinary tract infection during the second trimester of pregnancy, negative control of culture posterior to treatment with nitrofurantoin. COVID vaccines one dose (Sputnik®), five prenatal medical care, with adequate fetal growth, received aspirin 81 mg from 14 weeks of gestation. Height 1.57 meters, weight 94 kg at the beginning of pregnancy, 108 kg at the end of pregnancy.

Summary of symptoms

During her 36th week of pregnancy without presenting any previous obstetric symptoms, she went (by herself) to the emergency room of this hospital, with a history of 6 h of evolution of pulsatile headache, tinnitus, scotomas, vertigo, facial hemiparesis, nausea, and vomiting on two occasions of gastric content, associated with an episode of loss of consciousness for 3 min

with fall of the same level and subsequent recovery of alertness with drowsiness, no other symptoms related.

Physical examination summary

Height 1.57 meters, weight 94 kg at the beginning of pregnancy, body mass index 38.1 Kg/m^2, body surface area 2.02 m^2.

Blood pressure on admission 200/140 mmHg, MAP 160 mmHg, heart rate 65 beats per minute (BPM), respiratory rate 21 cycles per minute (CPM), treated as severe pre-eclampsia with nifedipine 10 mg and modified Zuspan scheme (four grams in continuous infusion), 15 min after this treatment 151/121 mmHg, MAP 131 mmHg.

Neurological findings

Glasgow Coma Scale (GCS) 14 puntos: verbal response 4 points, motor response 6 points, eye opening 4 points, right pupillary diameter 3 mm, left pupillary diameter 2 mm, brainstem reflexes, no other cranial nerve alterations, unaltered motor sensitivity and response, preserved mental functions, grade III osteotendinous reflexes.

She underwent neurocritical care on the recommendation of the neurology service, with ICP monitoring inferred by optic nerve sheaths diameter measurement ICP 15-16 mmHg, remained under deep sedation after cesarean section for 48 h and subsequently emerged without data of delirium, without any manifestation of neurological deterioration. Tomographic: Areas compatible with subarachnoid hemorrhage of the right frontal region and on the free edge of the cerebellum tent on the right side, both not > 1 cm, rest of the study with cisterns of the base and subarachnoid space of the normal convexity, at the level of cerebral parenchyma basal ganglia with hypodense images in their entirety compatible with edema and subtotal deep venous circulation thrombosis (Fig. 1).

Summary of clinical evolution

Treated in the emergency area with severe hypertension, MAP stabilized to a reduction of 20% during 1st h, then cesarean section was performed due to GCS neurological deterioration of 14 points at admission and 10 points before cesarean delivery, after the surgery she remained in neurocritical care, deep sedation was maintained with propofol at 4 mg/kg/h and midazolam 0.2 mg/kg/h, achieving RASS –5 for 48 h, mechanical ventilation with pressure-controlled mode for 80 h; subsequently, mechanical ventilation progressed without criteria for ventilatory failure, required antihypertensive treatment based on nifedipine 30 mg every 12 h, maintaining MAP between 90 and 100 mmHg, after withdrawal from ventilation required adjustment of antihypertensives required nifedipine 60 mg every 12 h and nebivolol 5 mg every 24 h. Maintained uresis, urea, creatinine, urea nitrogen in normal ranges, adjustment of antihypertensives required nifedipine 60 mg every 12 h, and nebivolol 5 mg every 24 h did not present relevant electrolyte alterations. On admission to hospitalization, her platelet levels were in normal ranges, later she presented moderate thrombocytopenia verified with platelet levels in citrate, at discharge from intensive care with platelet levels in ranges of moderate thrombocytopenia, treatment with intravenous dexamethasone 8 mg twice daily for 48 h (Table 1).

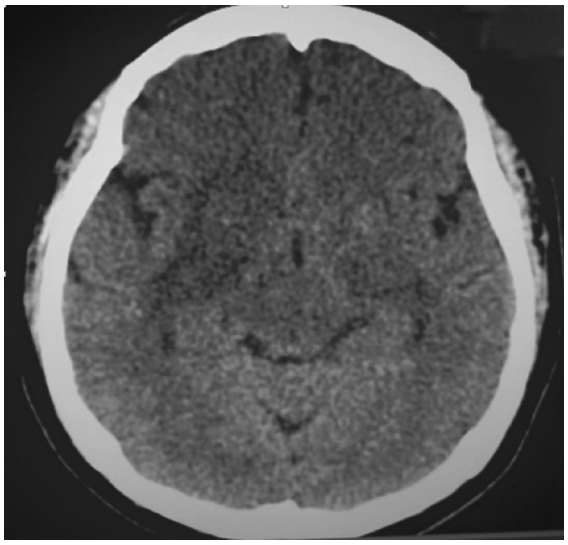

Figure 1. Cranial tomography on axial projection where hypodensity corresponding to the affected area is observed. *Source: Clinical record.*

Treatment received

Received enoxaparin at a therapeutic dose for 7 days and then prophylactically for 3 months, without any complications from treatment, modified Zuspan scheme (four grams in continuous infusion) and then 1 g/h 24 h, in neurocritical care measures for 48 h, including temperature 36-37°C, hemoglobin > 8 g/dL, sodium levels 135-145 meq/L, systolic arterial blood pressure > 110 mmHg, PO2 80-100 mmHg, lung protective ventilation, ICP control optic nerve < 5.5 mm, IP ACM 0.6-1.2, intracerebral pressure < 22 mmHg, and no hypertonic saline solution was necessary.

Table 1. Blood test results.

Day	1	2	3	4	5	6	7	8
Ph (Acidity Index)	7.49	7.38	7.39	7.41	7.39	7.39	7.44	7.42
HCO^{3}- (mmoL/L)	19	21	21	22	22	23	21	20.9
PCO^{2} (mmHg)	27	35	35.2	36	36	29	29	30
PO^{2} (mmHg)	125	92	89	79	78	81	78	71
Base excess (mmoL/L)	8	2	2	1	1.2	1.5	0.5	0.5
Lactate (mmoL/L)	1.9	0.9	1	1	0.9	0.5	0.6	0.8
INR	1	1.33	1.16	1.18	1.2	1.19	1.23	1.25
PT (seconds)	14	15.2	11.6	20.7	20.4	17.8	18.2	17.3
PTT (seconds)	34	60	30	25	32.5	32.3	35.8	49.3
Hematocrit (%)	37	31.5	31	32	31	29	31	28
Platelets ($x10^{3}/mm^{3}$)	159	75	78	81	102	105	110	151
White blood cells ($x10^{3}/mm^{3}$)	12	15.1	5	5.4	8.9	11.5	9.4	8
Glucose (mg/dL)	87	125	92	94	87	85	88	79
Creatinine (mg/dL)	0.8	1.17	1.05	0.95	0.98	1	1	0.98
BUN (mg/dL)	11	16	21	23	24	28	21	18.1
Urea (mg/dL)	19	32	34	45	40	35	32	29
Total Bilirubin (mg/dL)	0.4	0.63	0.8	1	1	0.9	0.9	0.6
AST (UI/L)	29	31	42	60	45	44	40	39
ALT (UI/L)	19	28	32	39	31	32	36	29
LDH (UI/L)	345	450	430	428	390	345	330	290
Sodium (meq/L)	141	144	144	143	140	140	145	140
Potassium (meq/L)	4	4.1	4.4	4.2	4.1	4.1	3.95	3.96
Chlorine (meq/L)	108	110	109	108	104	103	108	106
Calcium (mg/dL)	7.3	8.1	8.2	8.2	8.4	8.4	8.9	8.8

HCO^{3}-: denotes Bicarbonate; PCO^{2} CO^{2}: partial pressure; PO^{2} O^{2}: partial pressure; INR: International Normalized Ratio; PT: prothrombin time; PTT: partial thromboplastin time; BUN: blood urea nitrogen; AST: aspartate aminotransferase; ALT: alanine aminotransferase; LDH: lactate dehydrogenase.
Source: Clinical record.

Obstetric outcome

Cesarean delivery under general anesthesia with estimated bleeding of 450 mL, surgical sterilization, product of gestation with 37 weeks calculated by Capurro, 2900 g, male, with Apgar 7/9, without maternal or neonatal neurological impact. Withdrawal of antihypertensives was achieved at 7 days of puerperium, neurological evaluation without any alteration at discharge, and 1 month later when evaluated by neurology.

5 days of hospitalization in intensive care unit, 3 days in intermediate care room of obstetrics. The newborn was hospitalized for 4 days in intermediate care and subsequently discharged without complications.

Case number 2

General characterization

Twenty-four years old, with no chronic personal or family history of degenerative. Two previous pregnancies

with vaginal delivery, no history of hospitalization during pregnancy. COVID vaccines one dose (Sputnik), six prenatal care, with adequate fetal growth, no intake of aspirin 81 mg. Height 1.59 meters, weight 71 kg at the beginning of pregnancy, 81 kg at the end of pregnancy.

Summary of symptoms

Frontal and temporal headache of moderate to severe intensity of 12 h of evolution, associated with unique episode of convulsion, characterized by generalized tonic and clonic movements approximately 60 s of duration without relaxation of sphincters witnessed by her husband, subsequent recovery of alertness with drowsiness immediately.

Physical examination summary

Height 1.57 meters, weight 94 kg at the beginning of pregnancy, body mass index 28.1 Kg/m^2, and body surface area 1.7 m^2. Blood pressure on admission 143/96 mmHg, MAP 116 mmHg, heart rate 62 BPM, respiratory rate 21 CPM, treated with nifedipine 30 mg, subsequently maintained blood pressure in target ranges MAP 90-95 mmHg, without requiring additional antihypertensives during hospitalization.

Neurological findings

GCS 15 puntos: bilateral pupillary diameter 3 mm, brainstem reflexes and no cranial nerve alterations, unaltered motor sensitivity and response, preserved mental functions. Grade III osteotendinous reflexes. She was provided to neurocritical care on the recommendation of the neurology service, remained under deep sedation after cesarean section for 48 h, with ICP monitoring inferred by optic nerve sheaths diameter measurement ICP 12-14 mmHg, and subsequently emerged without data of delirium, without any manifestation of neurological deterioration. Tomography: Density changes compatible with thrombosis of the superior longitudinal sinus, without any other alteration at the intraparenchymal or ventricular level (Fig. 2).

Summary of clinical evolution

After being treated in the emergency area by history of seizure, diphenylhydantoin was administered initial dose 20 mg per kilogram, then 125 mg every 8 h for 7 days, cesarean section was performed due to initial diagnose of eclampsia. After cesarean delivery, she remained in neurocritical care, deep sedation was maintained with propofol at 3 mg/kg/h and midazolam 0.15 mg/kg/h, achieving RASS –5 for 48 h, mechanical ventilation with pressure-controlled mode with alveolar protection parameters for 60 h, subsequently, mechanical ventilation progressed without criteria for ventilatory failure, required antihypertensive treatment based on nifedipine 30 mg every 24 h, maintaining MAP between 90- and 95 mmHg. Uresis, urea, creatinine, and urea nitrogen in normal ranges did not present relevant electrolyte or metabolic alterations. On admission to hospitalization, her platelet levels were in normal ranges verified with platelet levels in citrate, at discharge from intensive care with platelet levels in normal ranges too (Table 2).

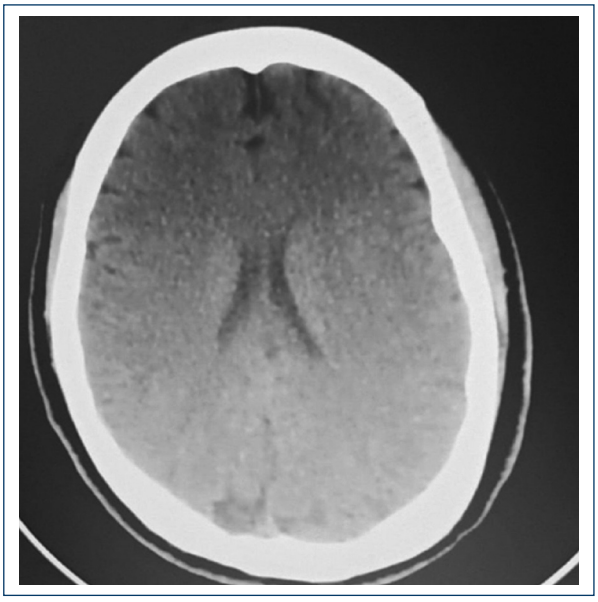

Figure 2. Cranial tomography on axial projection, hypodensity corresponding to the affected area is observed. *Source: Clinical record.*

Treatment received

Received enoxaparin at a therapeutic dose for 7 days and then prophylactically for 3 months, without any complications from treatment, diphenylhydantoin as anticonvulsant, modified Zuspan scheme (four grams in continuous infusion), and then 1 g/h 24 h, in neurocritical care measures for 48 h, including targets of temperature 36-37°C, hemoglobin > 8 g/dL, sodium levels 135-145 meq/L, systolic arterial blood pressure > 110 mmHg, PO2 80-100 mmHg, lung protective ventilation, ICP control optic nerve < 5.5 mm, IP ACM 0.6-1.2, intracerebral pressure < 22 mmHg, and no hypertonic saline solution was necessary.

Table 2. Blood test results.

Day	1	2	3	4	5	6	7	8
Ph (Acidity Index)	7.39	7.40	7.40	7.45	7.44	7.43	7.41	7.44
HCO^{3}- (mmoL/L)	19.1	17.9	17.9	21	21	19	19	20
PCO^{2} (mmHg)	31	28.6	28.6	27	26	26	27	25
PO^{2} (mmHg)	102	70	70	72	71	81	78	71
Base excess (mmoL/L)	-6	-7	-7	-4	-1	-1.5	-1	-0.5
Lactate (mmoL/L)	1.9	0.9	1	1	0.9	0.5	0.6	0.8
INR	0.82	0.94	0.91	1	1.1	1.08	1.05	1
PT (seconds)	10.4	11.8	11.5	12	12.5	12	12.5	12
PTT (seconds)	22.3	28.5	37.5	34	33	34	35	35
Hematocrit (%)	40	40.8	31.7	32	32	32	32.1	32.1
Platelets ($x10^{3}/mm^{3}$)	154	165	165	159	162	165	168	165
White blood cells ($x10^{3}/mm^{3}$)	9.0	15.4	8.5	8.9	7.9	7.9	8	8.1
Glucose (mg/dL)	81	103	84	88	85	92	90	79
Creatinine (mg/dL)	0.64	0.9	0.5	0.6	0.6	0.7	0.78	0.7
BUN (mg/dL)	11	13	9	11	11	10.5	11	12
Urea (mg/dL)	23.5	27.8	19.3	20	22	21	23	22
Total Bilirubin (mg/dL)	0.64	0.9	0.7	1	1.2	0.9	0.8	0.7
AST (UI/L)	118	166	60	65	59	61	60	58
ALT (UI/L)	117	181	99	98	81	88	85	90
LDH (UI/L)	356	424	264	270	220	250	255	245
Sodium (meq/L)	144	141	136	138	139	138	139	138
Potassium (meq/L)	4.39	4.18	4.3	4.2	4.1	4.2	4.1	4.1
Chlorine (meq/L)	113	113	107	106	105	105	104	104
Calcium (mg/dL)	8.4	8.2	8	8.1	8.2	8.3	8.5	8.2

HCO^{3}-: denotes Bicarbonate; PCO^{2} CO^{2}: partial pressure, PO^{2} O^{2}: partial pressure; INR: International Normalized Ratio; PT: prothrombin time; PTT: partial thromboplastin time; BUN: blood urea nitrogen; AST: aspartate aminotransferase; ALT: alanine aminotransferase; LDH: lactate dehydrogenase.
Source: Clinical record.

Obstetric outcome

Delivery by cesarean section, with estimated bleeding of 1200 mL, surgical sterilization, product of gestation with 38 weeks calculated by Capurro 2190 g, masculine with Apgar 8/9, without neonatal neurological impact. Withdrawal of antihypertensives was achieved at 15 days of puerperium, neurological evaluation without any alteration at discharge and 28 days later when evaluated by neurology.

Four days of hospitalization in intensive care unit, 4 days in intermediate care room of obstetrics. The newborn was hospitalized for 2 days in intermediate care and subsequently discharged without complications.

Case number 3

General characterization

Nineteen years old, no chronic degenerative history, no previous symptoms, no prenatal medical care, unknown that she was pregnant, no history of hospitalization during pregnancy. No COVID vaccines, no prenatal care, unknown fetal growth, no intake of aspirin 81 mg. Height 1.47 meters, weight 55 kg at the end of pregnancy.

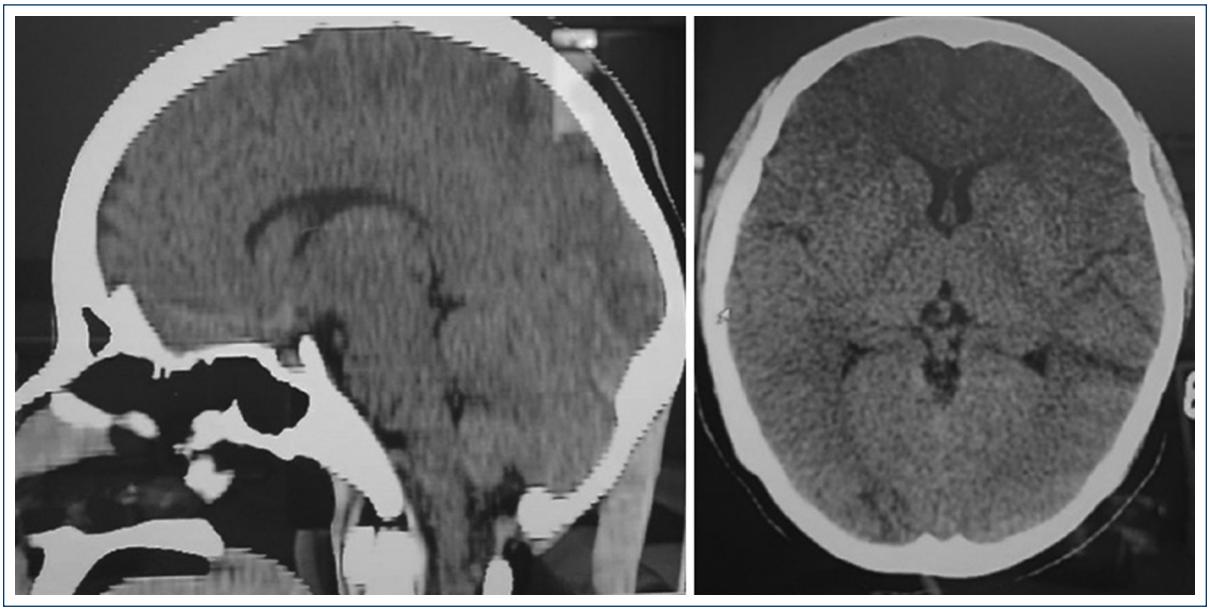

Figure 3. Cranial tomography on axial and sagittal projection hypodensity corresponding to the affected area is observed.
Source: Clinical record.

Summary of symptoms

Frontal and temporal headache of moderate-to-severe intensity of 24 h of evolution, associated with three episodes of convulsion, characterized by generalized, tonic and clonic movements, of approximately 2 min of duration without relaxation of sphincters witnessed by her brother, subsequent recovery of alertness with drowsiness immediately. Initially treated in a private health unit where pregnancy and possible eclampsia were diagnosed, nifedipine 30 mg and magnesium sulfate 4 g were administered and she was referred to this unit urgently.

Physical examination summary

Height 1.47 m, weight 50 kg at the beginning of pregnancy, body mass index 23.1 Kg/m^2, and body surface area 1.43 m^2.

Blood pressure on admission 171/120 mmHg, MAP 137 mmHg, heart rate 110 BPM, respiratory rate 21 CPM, treated with nifedipine 10 mg, 3 dosages, 1 h later 161/123 mmHg, MAP 135 mmHg, heart rate 110 BPM, then it was necessary to administer hydralazine 10 mg to reduce blood pressure to goals (reduction of blood pressure about 20% in the 1st h TAM about 100-110 mmHg), before cesarean delivery maintained blood pressure, but required nitroprusside infusion at doses of 0.5 mcg/kg/min for 12 h, then adjustment of antihypertensives with nebivolol 5 mg daily, nifedipine 30 mg twice a day. A month later no needed of antihypertensives.

Neurological findings

GCS 15 points: bilateral pupillary diameter 3 mm, brainstem reflexes and no cranial nerve alterations, unaltered motor sensitivity and response, and preserved mental functions. Grade III osteotendinous reflexes. She received neurocritical care on the recommendation of the neurology service, remained under deep sedation after cesarean section for 48 h, with ICP monitoring inferred by optic nerve sheaths diameter measurement ICP 10-12 mmHg, and subsequently emerged with delirium treated with haloperidol 5 mg every 6 h for 7 days without any other manifestation of neurological deterioration. Tomography: hypodensities in corticosubcortical areas in the biparietal region, as well as hypodense images of the superior longitudinal sinus, which makes the diagnosis of venous sinus thrombosis highly suggestive (Fig. 3).

Summary of clinical evolution

After stabilization in the shock room by history of seizure, then cesarean delivery was performed due to it. After surgery she remained in neurocritical care, deep sedation was maintained with propofol at 4 mg kg hour and midazolam 0.2 mg kg hour, achieving RASS –5 for

Table 3. Blood test results.

Day	1	2	3	4	5	6	7	8
Ph (Acidity Index)	7.36	7.41	7.39	7.44	7.45	7.39	7.48	7.48
HCO^{3}- (mmoL/L)	12	17.9	18.9	21.5	21.7	19.9	19.9	20.7
PCO^{2} (mmHg)	19	26	27	30	24	27.5	27.9	26.9
PO^{2} (mmHg)	102	95	90	82	80	79	70	74
Base excess (mmoL/L)	-9	-4	-7	-4	-1	-1.5	-1	-0.5
Lactate (mmoL/L)	2.1	0.9	1	1	1	0.4	0.3	0.3
INR	0.82	0.94	0.91	1	1.1	1.08	1.05	1
PT (seconds)	10.4	11.8	11.5	12	12.5	12	12.5	12
PTT (seconds)	22.3	28.5	37.5	34	33	34	35	35
Hematocrit (%)	40.4	40.8	31.7	32	32	32	32.1	32.1
Platelets ($x10^{3}/mm^{3}$)	69	70	92	90	110	120	118	125
White blood cells (x103/mm3)	13	16.4	13	12	12	8	8.5	9
Glucose (mg/dL)	97	95	90	98	99	97	99	78
Creatinine (mg/dL)	1.23	1	1	1	0.98	0.8	0.7	0.49
BUN (mg/dL)	19	21	22	21	18	12	11	12
Urea (mg/dL)	44	43	41	41	39	38	34	26
Total bilirubin (mg/dL)	0.84	1	0.8	0.7	0.8	0.7	0.4	0.21
AST (UI/L)	602	459	458	399	378	359	225	121
ALT (UI/L)	402	300	295	189	126	98	89	62
LDH (UI/L)	957	865	813	785	625	436	318	273
Sodium (meq/L)	138	141	144	144	143	144	142	143
Potassium (meq/L)	4.5	4.8	4.7	4.7	4.9	4	3.9	3.51
Chlorine (meq/L)	109	108	107	109	108	106	105	107
Calcium (mg/dL)	8.6	8.6	8.9	9	9.1	9.1	9	8.9

HCO^{3}-: denotes Bicarbonate; PCO^{2} CO^{2}: partial pressure; PO^{2} O^{2}: partial pressure; INR: International Normalized Ratio; PT: prothrombin time; PTT: partial thromboplastin time; BUN: blood urea nitrogen; AST: aspartate aminotransferase; ALT: alanine aminotransferase; LDH: lactate dehydrogenase.
Source: Clinical record.

48 h, mechanical ventilation with pressure-controlled mode for 56 h; subsequently, mechanical ventilation progressed without criteria for ventilatory failure, required antihypertensive treatment based on sodium nitroprusside (dose of 0.1-0.5 gammas for 12 h), nifedipine 30 mg every 24 h, and nebivolol, maintaining MAP between 90- and 95 mmHg. Maintained uresis, urea, creatinine, and urea nitrogen in normal ranges did not present relevant metabolic nor electrolyte alterations. On admission to hospitalization, her platelet levels were in a normal ranges verified with platelet levels in citrate, at discharge from intensive care with platelet levels in normal ranges (Table 3).

Treatment received

Received enoxaparin at a therapeutic dose for 7 days and then prophylactically for 3 months, without any complications from treatment, modified Zuspan scheme (4 g in continuous infusion) and then 1 g/h 24 h, neurocritical care for 48 h, including targets of temperature 36-37°C, hemoglobin > 8 g/dL, sodium levels 135-145 meq/L, systolic arterial blood pressure > 110 mmHg,

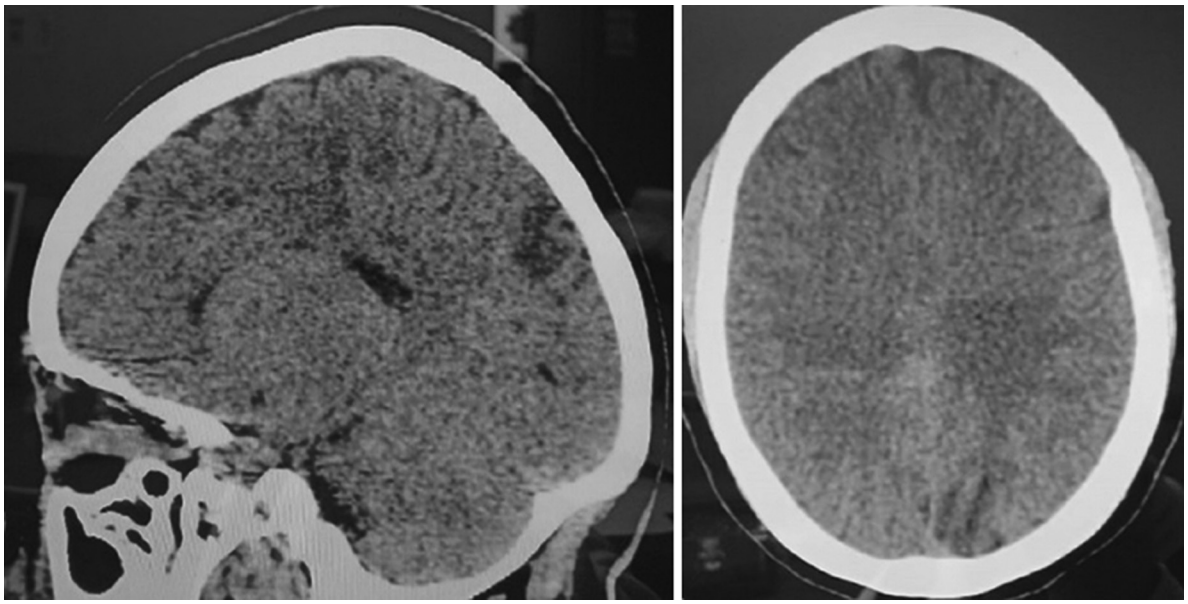

Figure 4. Cranial tomography on axial projection and sagittal hypodensity corresponding to the affected area is observed. *Source: Clinical record.*

PO2 80-100 mmHg, lung protective ventilation, optic nerve < 5.5 mm, IP ACM 0.6-1.2, intracerebral pressure < 22 mmHg, and no hypertonic saline solution was necessary, treated with haloperidol 5 mg every 6 h for 7 days for diagnose of delirium.

Obstetric outcome

Resolution of pregnancy by cesarean section, with estimated bleeding of 350 mL, surgical sterilization, product of gestation with 32 weeks calculated by Capurro, with Apgar 8/9, 1370 g, female, without maternal or neonatal neurological impact. Withdrawal of antihypertensives was achieved at 28 days of puerperium, neurological evaluation without any alteration at discharge and 28 days later when evaluated by neurology.

Five days of hospitalization in intensive care unit, 3 days in intermediate care room of obstetrics. The newborn was hospitalized for 15 days in intensive care neonatal unit by the diagnosis of neonatal sepsis, subsequently discharged without complications.

Case number 4

General characterization

Twenty-two years old, with no chronic personal or family history of degenerative. One previous pregnancy with vaginal delivery, no history of hospitalization during pregnancy. No COVID vaccines, eight prenatal care, with adequate fetal growth, no intake of aspirin 81 mg. Height 1.54 m, weight 60 kg at the beginning of pregnancy, 66 kg at the end of pregnancy.

Summary of symptoms

Frontal and temporal headache of moderate intensity of 36 h of evolution, associated with three episodes of seizures, characterized by generalized, tonic and clonic movements, of approximately 45-60 s duration without relaxation of sphincters witnessed by her mother, subsequent no total recovery of alertness with drowsiness, her parents took her to a first level hospital for initial care, in that health unit she presented a new episode of witnessed seizure with 1 min of generalized duration, tonic and clonic without relaxation of sphincters, 4 g of magnesium sulfate and 10 mg of diazepam were administered, she was referred to this care center by ambulance for care at the third hospital level.

Physical examination summary

Height 1.54 m, weight 60 kg at the beginning of pregnancy, body mass index 25.3 Kg/m^2, body surface area 1.6 m^2.

Blood pressure on admission 150/110 mmHg, MAP 126 mmHg, heart rate 110 BPM, respiratory rate 21

Table 4. Blood test results.

Day	1	2	3	4	5	6	7	8
Ph (Acidity Index)	7.32	7.35	7.41	7.42	7.43	7.44	7.45	7.47
HCO^{3}- (mmoL/L)	17	19	19	21	21	22	23	23.2
PCO^{2} (mmHg)	31	32	30	31	30	29	28	29
PO^{2} (mmHg)	102	95	90	95	79	79	74	69
Base excess (mmoL/L)	-10	-11	-9	-8.5	-7	-4.5	-3	-2.5
Lactate (mmoL/L)	1.9	0.9	1	1	1.1	1.1	1	0.9
INR	0.91	1	1.2	1	1.1	1.2	1.1	1
PT (seconds)	11.5	11.5	12	12	11.5	11.5	11.5	12.5
PTT (seconds)	22	23	23	22	23	23	22	22.9
Hematocrit (%)	42.3	40	39	40	39	38	36	35
Platelets ($x10^{3}/mm^{3}$)	107	110	110	118	125	151	154	230
White blood cells ($x10^{3}/mm^{3}$)	16.8	15	15	19	14	14.5	15	9.4
Glucose (mg/dL)	80	91	90	89	96	82	87	92
Creatinin (mg/dL)	0.67	0.7	0.69	0.66	0.9	0.7	0.65	0.51
BUN (mg/dL)	12	11	13	14	17	15	13	12
Urea (mg/dL)	25.7	29	28	31	31	29	24	25.7
Total Bilirrubin (mg/dL)	0.5	0.8	0.7	0.45	0.5	0.4	0.41	0.3
AST (UI/L)	85	80	78	76	74	53	41	35
ALT (UI/L)	49	44	41	43	42	41	39	44
LDH (UI/L)	423	325	335	495	329	291	300	290
Sodium (meq/L)	137	139	140	141	142	139	140	140
Potassium (meq/L)	3.79	3.9	3.8	3.9	4.1	4.1	4.2	4
Chlorine (meq/L)	110	109	109	108	108	107	108	110
Calcium (mg/dL)	8.4	8.6	8.5	8.7	8.6	8.5	8.8	8.8

HCO^{3}-: denotes Bicarbonate; PCO^{2} CO^{2}: partial pressure; PO^{2} O^{2}: partial pressure; INR: International Normalized Ratio; PT: prothrombin time; PTT: partial thromboplastin time; BUN: blood urea nitrogen; AST: aspartate aminotransferase; ALT: alanine aminotransferase; LDH: lactate dehydrogenase.
Source: Clinical record.

CPM, treated with nifedipine 30 mg, levetiracetam 2 g initially then 1 g each 12 h, subsequently maintained blood pressure in target ranges MAP 100-105 mmHg, blood pressure on admission to Intensive Care Unit 134/81 mmHg, MAP 98 mmHg, heart rate 98 BPM requiring nifedipine (60 mg twice a day), and prazosina (2 mg each 6 horas) during hospitalization.

Neurological findings

GCS 12 points: bilateral pupillary diameter 2 mm, brainstem reflexes and no cranial nerve alterations, unaltered motor sensitivity and response, and altered mental functions. Grade III osteotendinous reflexes. She underwent neurocritical care on the recommendation of the neurology service, remained under deep sedation after cesarean section for 48 h, with ICP monitoring inferred by optic nerve sheaths diameter measurement ICP 10-12 mmHg, and subsequently emerged without data of delirium, without any manifestation of neurological deterioration. Tomography: Density changes compatible with thrombosis of the superior longitudinal sinus, without any other alteration at the intraparenchymal or ventricular level (Fig. 4).

Summary of clinical evolution

Treated in the shock room by history of symptoms suspected diagnose of eclampsia, on admission treated with magnesium sulfate and levetiracetam, then cesarean delivery was performed due to it. After cesarean procedure, she remained in neurocritical care, deep sedation was maintained with propofol at 4 mg/kg/h and midazolam 0.3 mg/kg/h, achieving RASS −5 for 48 h, mechanical ventilation with pressure-controlled mode for 56 h; subsequently, mechanical ventilation progressed without criteria for ventilatory failure, required antihypertensive treatment based on nifedipine 30 mg every 24 h, maintaining MAP between 90- and 95 mmHg. No alterations on uresis, urea, creatinine, and urea nitrogen in normal ranges did not present relevant electrolyte nor metabolic alterations. On admission to hospitalization, her platelet levels were in mild thrombocytopenia verified with platelet levels in citrate (Table 4), at discharge from intensive care with platelet levels in normal ranges too, received dexamethasone 8 mg each 12 h for 4 days.

Obstetric outcome

Completion of pregnancy by cesarean section, with estimated bleeding of 400 mL, product of gestation with 35 weeks calculated by Capurro, male, 1996 g, Apgar 8/9, without maternal or neonatal neurological impact. Withdrawal of antihypertensives was achieved at 21 days of puerperium, neurological evaluation without any alteration at discharge and 28 days later when evaluated by neurology.

Four days of hospitalization in intensive care unit, 4 days in intermediate care room of obstetrics. The newborn was hospitalized for 6 days in intermediate care and subsequently discharged without complications.

Discussion

CVT generally has a limited behavior since the veins of the cerebral system do not have valves and have an extensive system of venous anastomoses. The severity of clinical symptoms and neurological involvement depends on the area involved. In these clinical cases, the disease manifested with symptoms such as horizontal nystagmus, cephalea, and hyperreflexia. The affected venous territory was the left Trolard vein. The patient presented mild cerebral edema without intracranial hemorrhage. One of the most feared complications associated with cerebral edema is hemorrhage in the compromised venous system and the association with life-threatening endocranial hypertension. The most relevant obstetric risk factors are postpartum hemorrhage, preeclampsia, transfusion history, and puerperal infection, the latter being the most significant cause. The symptom reported in the clinical case was preeclampsia, which coincides with the literature reviewed. The treatment described in the different guidelines on venous thrombosis is mainly pharmacological and based on low-molecular-weight heparin, Warfarin, and direct-acting anticoagulants. There are descriptions of a series of cases in which thrombectomy was implemented. In this case, there was an adequate response when using enoxaparin at therapeutic doses, with clinical and imaging resolution of the event. CVT is still a significant diagnostic and therapeutic challenge, due to its high variability of clinical manifestations and its lack of a clear therapeutic consensus[1]. The most affected area in the reported cases was the longitudinal sinus with associated ischemic changes in the occipital region, similar to the cases reported in recent literature, approximately 6-7 of 10 cases[1-4]. The most frequent symptomatology was in relation to frontal and occipital headache of moderate to severe intensity in the four reported cases, similar to the previous case reports that have been reported in the international literature. It is worth mentioning that none of the patients presented data of intracranial hypertension and the highest value of ICP inferred was 16 mmHg; however, three of the cases presented seizures and in one of the cases anisocoria with mild cerebral edema by computed tomography, which suggests that obstetric patients may have less capacity for brain self-regulation. Pérez Lázaro et al. suggest “early, accurate diagnosis can reduce the rate and severity of complications,” Fortunately, in the reported cases, the associated symptoms were detected early[2]. A 28-day follow-up in patients of reported cases found no new evidence of thrombotic events or neurological symptoms, not similar to cases reported by Ferro et al., “patients had a moderate risk of further thrombotic events and seizures”[3].

The incidence, mortality, and disability from pregnancy-related-stroke are higher than previously reported as reported by James et al. where African-American women are at an increased risk, as are women aged 35 years and older[4], similar was found on male patients[5]. In our study, the cases were reported in young patients 30, 24, 22, and 19 years old and mestizo ethnics. In the cases reported, no one of patients developed intracranial hypertension as described by Ropper et al. “CVT is characterized by infarction with focal neurologic deficits and increased ICP,” the level of ICP on the reported cases

was between 10 and 16 mmHg, and only one case with associated cerebral edema[6]. Silvis et al. suggest variety of therapies for CVT, and each should be used in the appropriate setting, preferably guided by data from randomized trials and well-designed cohort studies[7]; however, obstetric patients are very particular due to physiologic conditions related to pregnancy and puerperium, in our experience, 7 days of therapeutic dosage of low molecular heparin is enough to avoid extension of damage related to cerebral thrombosis. The neurocritical care on minimizing injury is based on studies made in patients with traumatic brain injuries because there are no specific recommendations on obstetrics[8-12]. Therapeutic Goals created by Goddoy et al. and Taccone et al., such as the acronyms THE MANTLE and GHOST CAP were provided in the care of the patients reported in our case series.

No specific mechanism has been clearly described to explain how brain tissue damage occurs after CVT[13-18]. It is believed that tissue damage is related to the development of edema and venous congestion that compromises the supply of oxygen, so certain strategies for studying the lesion should be studied with adequate RCT.

Conclusion

CVT is a rarely suspected clinical entity during the puerperal period; however, the prothrombotic state per se of this condition predisposes to this type of thrombosis, as mentioned in this review, as well as at any other level. The clinical manifestations of CVT can be changeable, from a symptom as non-specific as cephalea to seizures and brain death. The appearance of new neurological symptoms in the puerperium should not be underestimated, particularly in patients with additional risk factors such as obesity, preeclampsia, or a history of other thrombotic episodes. Implementing the necessary diagnostic means to clarify the cause of a new-onset neurological clinical manifestation should not be delayed. First-line treatment, despite weak evidence and recommendations, should be initiated with low-molecular-weight heparins, which provided that there are no contraindications. In the obstetric patient, no benefits have been studied with other therapies such as direct anticoagulants or thrombectomy.

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Conflicts of interest

The authors declare no conflicts of interest.

Ethical disclosures

Protection of human and animal subjects. The authors declare that the procedures followed were in accordance with the regulations of the relevant clinical research ethics committee and with those of the Code of Ethics of the World Medical Association (Declaration of Helsinki).

Confidentiality of data. The authors declare that they have followed the protocols of their work center on the publication of patient data.

Right to privacy and informed consent. The authors have obtained the written informed consent of the patients or subjects mentioned in the article. The corresponding author is in possession of this document.

Use of artificial intelligence for generating text. The authors declare that they have not used any type of generative artificial intelligence for the writing of this manuscript nor for the creation of images, graphics, tables, or their corresponding captions.

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