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## CLINICAL INFORMATION

### Cerebral venous thrombosis after spinal anesthesia: case report<sup>☆</sup>



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#### Abstract

**Introduction:** Cerebral venous thrombosis (CVT) is a rare but serious complication after spinal anesthesia. It is often related to the presence of predisposing factors, such as pregnancy, puerperium, oral contraceptive use, and malignancies. Headache is the most common symptom. We describe a case of a patient who underwent spinal anesthesia and had postoperative headache complicated with CVT.

**Case report:** Male patient, 30 years old, ASA 1, who underwent uneventful arthroscopic knee surgery under spinal anesthesia. Forty-eight hours after the procedure, the patient showed frontal, orthostatic headache that improved when positioned supine. Diagnosis of sinusitis was made in the general emergency room, and he received symptomatic medication. In subsequent days, the headache worsened with holocranial location and with little improvement in the supine position. The patient presented with left hemiplegia followed by tonic-clonic seizures. He underwent magnetic resonance venography; diagnosed with CVT. Analysis of pro-coagulant factors identified the presence of lupus anticoagulant antibody. The patient received anticonvulsants and anticoagulants and was discharged on the eighth day without sequelae.

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**PALAVRAS-CHAVE**

Raquianestesia;  
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Trombose venosa  
cerebral

*Discussion:* Any patient presenting with postural headache after spinal anesthesia, which intensifies after a plateau, loses its orthostatic characteristic or become too long, should undergo imaging tests to rule out more serious complications, such as CVT. The loss of cerebrospinal fluid leads to dilation and venous stasis that, coupled with the traction caused by the upright position, can lead to CVT in some patients with prothrombotic conditions.

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**Trombose venosa cerebral após raquianestesia: relato de caso****Resumo**

*Introdução:* A trombose venosa cerebral (TVC) é uma complicação rara, mas grave, após raquianestesia. Está frequentemente relacionada com a presença de fatores predisponentes, como gestação, puerpério, uso de contraceptivos orais e doenças malignas. O sintoma mais frequente é a cefaleia. Descrevemos um caso de um paciente submetido à raquianestesia que apresentou cefaleia no período pós-operatório complicada com TVC.

*Relato de caso:* Paciente de 30 anos, ASA 1, submetido à cirurgia de artroscopia de joelho sob raquianestesia, sem intercorrências. Quarenta e oito horas após o procedimento apresentou cefaleia frontal, ortostática, que melhorava com o decúbito. Foi feito diagnóstico de sinusite em pronto socorro geral e recebeu medicação sintomática. Nos dias subsequentes teve pioria da cefaleia, que passou a ter localização holocraniana e mais intensa e com pequena melhora com o decúbito dorsal. Evoluiu com hemiplegia esquerda seguida de convulsões tônico-clônicas generalizadas. Foi submetido à ressonância magnética com venografia que fez o diagnóstico de TVC. A pesquisa para fatores pró-coagulantes identificou a presença de anticorpo lúpico. Recebeu como medicamentos anticonvulsivantes e anticoagulantes e teve alta hospitalar em oito dias, sem sequelas.

*Discussão:* Qualquer paciente que apresente cefaleia postural após uma raquianestesia, e que intensifica após um platô, perca sua característica ortostática ou se torne muito prolongada, deve ser submetido a exames de imagem para excluir complicações mais sérias como a TVC. A perda de líquido cefalorraquidiano leva à dilatação e à estase venosa, que, associadas à tração provocada pela posição ereta, podem, em alguns pacientes com estados protrombóticos, levar à TVC.

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**Introduction**

Since the first case reported by August Bier in 1898,<sup>1</sup> post-puncture headache has been a problem for patients undergoing dural puncture. In the classical description, the post-dural puncture headache (PDPH) has frontal or occipital location, gets worse with upright position and essentially improves or disappears with the supine position. The onset and duration of PDPH symptoms may be extremely variable, but in most cases they occur within the first 48 h after the puncture and have a self-limiting character, lasting only a few days. In some cases, it may be associated with nausea and vomiting.<sup>1</sup> Various causes have been associated with the onset of PDPH, particularly the needle gauge and tip design. But even with small gauge needles and in experienced hands, PDPH still has an incidence of 0.16–1.3%.<sup>2</sup>

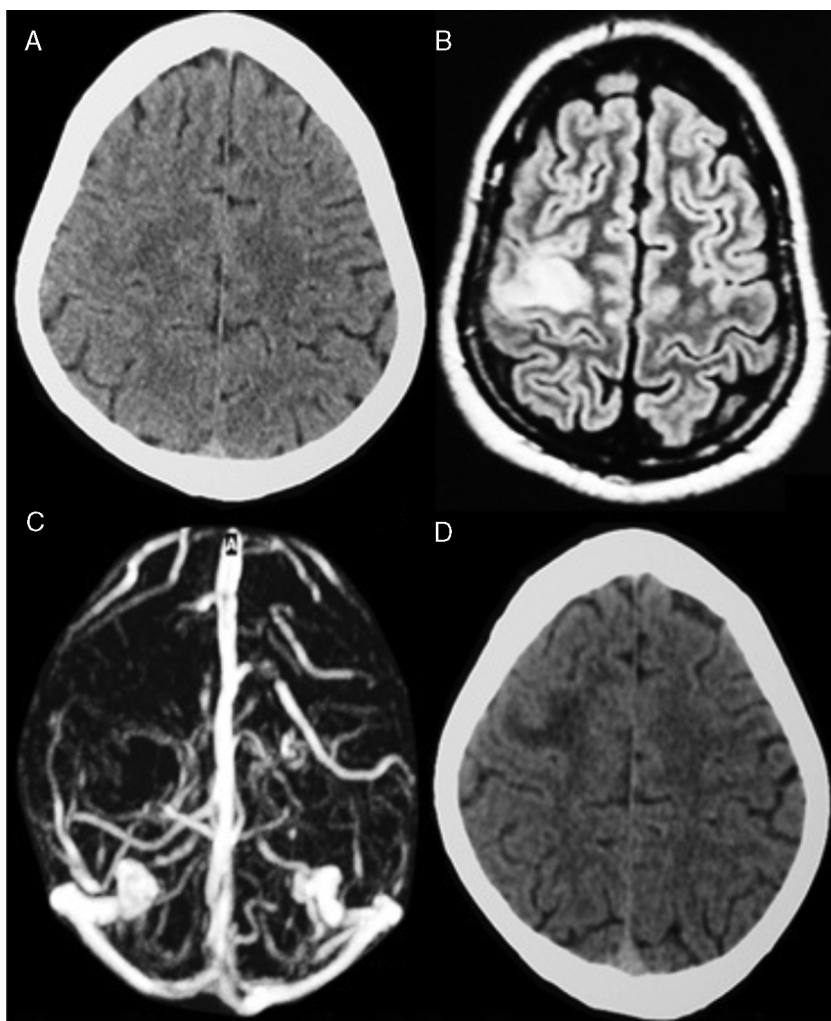
Although the classic description of PDPH has a benign course, it does not always have this favorable outcome, as it may be a symptom associated with more severe complications, although rare. Among these complications, cerebral venous thrombosis (CVT) is a major concern and

can be a diagnostic challenge when associated with lumbar puncture.

The objective of this paper is to report the case of a patient who presented with a clinical picture of CVT after spinal anesthesia for orthopedic surgery.

**Case report**

Male patient, aged 30 years, 82 kg, 1.71 m, fireman, previously healthy, proposed surgery of unilateral knee arthroscopy. The patient had a history of surgery for appendicitis at age 14 and ENT procedure three years ago, with general anesthesia and without complications. He had no comorbidity and the physical examination was normal. The patient was classified as ASA I and subjected to spinal anesthesia applied in L3-4 with Quincke needle tip 27G. Bupivacaine 0.5% hyperbaric (15 mg) was administered. The knee arthroscopy was performed with a tourniquet on the lower limb at the thigh level, with an inflation pressure of 380 mmHg for 40 min (min). The procedure lasted about 1 hour (h), uneventfully. The patient received midazolam



**Figure 1** Imaging study of the case. A, B, C are tests performed on the first day of symptoms and D is the control examination performed after 10 days. A, cranial CT without contrast to discrete hypodense in right frontal lobe; B, brain MRI Flair showing vasogenic edema in the topography of the pre-central gyrus law; C, MRI angiography demonstrating acute thrombosis of superficial cortical veins in the right frontal convexity; D, head CT without contrast with hypodense in right frontal lobe, featuring better definition of the lesion relative to the initial cranial CT.

(5 mg), cefazolin (1 g), ondansetron (4 mg), dipyron (2 g), and cetroprofeno (100 mg). After surgery, the patient was taken to the post-anesthesia recovery unit and then to the ward and was discharged the same day.

Forty-eight hours after the procedure, the patient developed severe right frontal headache, orthostatic in nature, which improved with rest. In a period of 12 h, the headache evolved to a holocranial headache, severity of 10 on a scale of 0–10 (0 = no pain and 10 = unbearable pain), particularly in the upright position. In the supine position, the patient still complained of pain, severity of five on the scale mentioned above. In addition to the headache, the patient also reported a clogged ear sensation. He sought medical care in the emergency department, where he was diagnosed with sinusitis. The patient was treated with antibiotics, anti-inflammatory and antiallergic drugs. On the fourth post-operative day, the patient had paresthesia in the left arm, which evolved into grade III hemiparesis around the left side of the body. The next day (fifth postoperative day), he had

an episode of generalized tonic–clonic seizure, received initial care by the emergency medical system (Samu) and was taken to the hospital where, on arrival, he presented new generalized convulsive episode, with myoclonus. He was admitted to the intensive care unit, where he was treated with anticonvulsants and subjected to imaging test. Computed tomography (CT) of the head revealed a small right frontal hypodensity and magnetic resonance imaging (MRI) with contrast venography showed vasogenic edema associated with acute thrombosis of the superficial cortical veins in the right frontal convexity (Fig. 1A–C). With this diagnosis, specific treatment was started with full anticoagulation.

After diagnosis and treatment, a detailed family history revealed thrombotic events. The patient's father had an episode of cerebral ischemia and two uncles had deep vein thrombosis. Laboratory tests showed hyperlipidemia (total cholesterol of 331 mg dL<sup>-1</sup> and triglycerides of 414 mg dL<sup>-1</sup>) and screening for procoagulant factors revealed increased lupus anticoagulant—screening test 1.24 (nl < 1.15) and

confirmatory test 1.46 ( $VN < 1.21$ ), with confirmation of the presence of nonspecific inhibitor—lupus anticoagulant.

The patient was discharged after eight days of admission, taken anticonvulsant (diphenylhydantoin) and oral anticoagulant drugs. After 10 days of the ictal event, a control head CT was performed, which showed a right frontal hypodense area, better defining the subacute ischemic lesion with regression of the edema (Fig. 1D).

Three months after the event, the patient reported cyclothymic behavioral changes, with episodes of euphoria alternating with periods of depression. No motor deficit remained.

## Discussion

The number of published reviews reporting the simultaneous occurrence of PDPH and CVT is limited, which prevents the knowledge of the true incidence of this complication. Furthermore, there is the fact that many cases are not reported or even diagnosed. Greater awareness of CVT from case reports like this can help increase the identification of patients at high risk and earlier treatment.

This paper describes a case of CVT after lumbar puncture for spinal anesthesia in a previously healthy patient. The development of signs and symptoms in the second postoperative day, characterized by orthostatic headache, leads to the diagnosis of PDPH. However, two features draw attention in this patient: the headache severity and the location change from frontal to holocranial. The previous history of sinusitis masked clinical suspicion of other complication and only the appearance of warning signs, such as motor deficits and seizures, led to the suspicion of a more serious involvement.

Our patient had no predisposing conditions that could help in the diagnosis of CVT. This is a rare condition with multiple causes or risk factors, such as use of oral contraceptives and other drugs, infections, malignant and inflammatory diseases, postpartum period, and congenital thrombophilia.<sup>3,4</sup> Few cases of CVT have been described after post-dural puncture for spinal or epidural anesthesia,<sup>5</sup> myelography, intrathecal administration of drugs or related to diagnosis.<sup>6</sup> Cases of CVT after regional anesthesia reported in the literature are rare and usually associated with the postpartum period.<sup>6</sup>

Of course, there is a dilemma for the physician who attends a patient with a headache after a spinal anesthesia, as undoubtedly the first diagnosis will be PDPH. In this case, we can mention as a complicating factor the overlapping of another diagnosis, of sinusitis, as the causal factor of the headache, although acute sinusitis is an uncommon cause of headache with the manifested characteristics.<sup>7</sup>

CVT has a wide and remarkable semiological variety. Continuous headache related to the standing position, dizziness, nausea, vomiting, blurred vision, motor signs, seizures, reduced mental awareness, and coma may be present. Headache is the most common symptom<sup>8,9</sup> and can simulate the PDPH itself,<sup>10</sup> cerebral hemorrhage, or migraine-type headache.<sup>11,12</sup> The specific presentation depends on the location and extent of thrombosis, degree of collateral venous circulation around thrombosis, and presence of cortical lesions associated.<sup>13</sup> Thrombosis of a single cortical vein

may cause focal motor or sensory deficits, while an extensive thrombus in a large venous sinus will cause more generalized neurological symptomatology, which include headache, signs of increased intracranial pressure, convulsions, and coma.<sup>14</sup> Furthermore, signs and symptoms may be intermittent when thrombosis and fibrinolysis occur simultaneously, leading to fluctuations in the circulation around the thrombosed vessels and intracranial pressure.<sup>14</sup>

In epidemiological terms, CVT is more common in women, aged between 20 and 35 years, and it seems that there is no ethnic predominance. It is widely accepted that the frequency of CVT is much higher in pregnant women, compared to the general population, and accounts for 34% of reported cases in the literature.<sup>14</sup> Usually, it has an acute onset during pregnancy and in most cases it occurs in the postpartum period. When related to the postpartum period, CVT may have an acute or longer onset. The venous congestion and damage to the vascular endothelium, which may be secondary to labor and expulsion period, combined with the typical state of postpartum hypercoagulability could contribute to the increased risk after birth.<sup>15-17</sup>

Although it has prevalence in pregnant women, CVT can also affect other patients. In a review by Mahesh et al.,<sup>18</sup> 52 cases of CVT that occurred after lumbar puncture were analyzed. The cases were allocated into a group of obstetric patients (34.06% of cases), a second group of patients who underwent diagnostic lumbar puncture, and a third group of patients who underwent the puncture for anesthesia or injection of drugs. In the obstetric patients, 72.2% had postural headache as the first symptom and changes in the headache pattern were seen in about 50% of patients. Most of them had prothrombotic predisposition or previous history of oral contraceptive use. Patients in both non-obstetric groups had postural headache in almost 100% of cases, with standard change in 77% of the group that underwent diagnostic lumbar puncture and in 40% of those who underwent the puncture for anesthesia or injection of drugs. Demyelinating diseases were seen in 82% of the group with diagnostic lumbar puncture and prothrombotic status in 66% of patients undergoing anesthesia.

Since the first description of the association between lumbar puncture and CVT by Schou and Scherb<sup>19</sup> in 1986, there has been a constant debate of the causal relationship between lumbar puncture and CVT: whether there is an association or a mere coincidence between the two events. However, over the past two decades, there is good evidence to suggest causality, according to which lumbar puncture alone can trigger CVT.<sup>20</sup> Nevertheless, in most reported cases, there are other risk factors for CVT that puts in doubt the true unique role of lumbar puncture in the genesis of CVT.

The CVT pathogenesis induced by lumbar puncture can be explained by the Monro-Kellie doctrine. This theory suggests the skull as a rigid structure in which the intracranial components, brain tissue, blood, and cerebrospinal fluid (CSF) are in a state of pressure balance. In pathological conditions, a decrease or increase of one of those elements will lead to a compensation change in the volume of the other two, so that the intracranial contents remain constant. In the specific case of lumbar puncture, when CSF hypotension occurs, the CSF volume and pressure are significantly reduced. As a consequence, there will be a blood volume increase, mainly

in the venous compartment, at the expense of stasis and dilation of the dural venous sinuses and cortical veins. This change occurs sharply in tough fibrous meninx (dura mater), and as it has no blood-brain barrier, such fact would explain the contrast agent extravasation on a diagnostic imaging test.<sup>19</sup> With the reduced CSF volume, there will be a relative decline and traction of the brain as a whole, together with the distortion and elongation of dural and cortical veins. These changes will eventually damage the vascular wall. All these changes are aggravated by the standing position due to acute dilation of the veins, as well as the stretching of its walls.<sup>19</sup> The described phenomenon meets the Virchow's theory, according to which the three main causes for thrombosis occurrence would be blood stasis and the change in the vessel wall and blood composition.<sup>19,20</sup>

Such pathophysiological phenomena described above end up creating a vicious cycle because thrombosis of cortical veins or superior sagittal sinus leads to a decrease in venous drainage, which reduces the absorption of CSF by arachnoid villi, further increasing the intracranial pressure. Simultaneously, venous stasis leads to blood stasis and focal cerebral infarction. This triggers signs and symptoms such as severe headache, nausea, focal neurological signs, seizures, and altered consciousness. The occurrence of subdural hematoma and intracranial hemorrhage after lumbar puncture is related to the same pathophysiological mechanism.<sup>2,21</sup>

The CSF volume escaping through the hole made by the puncture needle is responsible for secondary venodilation. However, this dilation is not directly correlated with the volume lost. In this context, Grant et al.,<sup>22</sup> in a study with magnetic resonance, showed that even small volumes of CSF (about 1.8 mL) that are lost after a lumbar puncture are sufficient to lead to PDPH. Moreover, Ghaleb et al.<sup>23</sup> reported that a loss of 10% of the CSF volume can trigger headache.

To reinforce the hypothesis of the relationship between lumbar puncture and the occurrence of cerebral thrombosis, Canhão et al.<sup>20</sup> used the transcranial Doppler in 13 patients and recorded the mean blood flow velocity in the straight sinus before, during, and after a lumbar puncture. The study demonstrated a reduction in blood flow velocity of approximately 50% in the straight sinus after the lumbar puncture. This reduction in blood flow velocity would be secondary to reduced intracranial pressure induced by decreased CSF.<sup>20</sup>

It is well accepted that multiple risk factors are often seen in the same patient with CVT. Indeed, it has been demonstrated in the literature the association with malignancy, thrombophilia, postpartum status, use of oral contraceptives, and intrathecal injection of steroids or cytostatics. In such patients, lumbar puncture has been one of the precipitating factors of CVT by the mechanism described by Canhão et al.<sup>24</sup> Consequently, faced with a case of CVT after lumbar puncture, other risk factors for venous thrombosis should be investigated, as diagnosed in the patient described herein, a prothrombotic status by the lupus antibody presence.

Neuroimaging studies are needed to confirm the diagnosis of CVT. Computed tomography closes the diagnosis in only 30% of cases.<sup>11</sup> Magnetic resonance imaging associated with venography is the gold standard method for final diagnosis, with sensitivity close to 100%.<sup>24</sup>

CVT treatment is primarily non-invasive, although endovascular thrombolysis and surgical thrombectomy are considered in severe cases.<sup>25,26</sup> Anticoagulation is the treatment of choice, but the indications for its use remain somewhat controversial, as approximately 50% of cases are associated with hemorrhagic cerebral infarction.<sup>27</sup>

As for the prognosis, the clinical course of CVT is unpredictable and often there is worsening of symptoms after the diagnosis. Changes in consciousness, coma, and intracranial hemorrhage are important predictors of adverse clinical course.<sup>28</sup>

Thus, we concluded that major complications after regional anesthesia are rare, but can be devastating to the anesthesiologist, and especially to the patient. Although most cases of PDPH evolve satisfactorily, it should not be neglected, as in the case of CVT, such semiological finding is present in about 90% of cases and may be the only manifestation in 10%. At such times, there is a considerable potential for morbidity and even death. Therefore, attention must be paid when the headache changes its postural characteristic and when the patient has risk factors for venous thrombosis.

## Conflicts of interest

The authors declare no conflicts of interest.

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