ISSN 1220-8841 (Print)
ISSN 2344-4959 (Online)

ROMANIAN
NEUROSURGERY

Vol. XXXVIII | No. 1 March 2024

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DOI: 10.33962/roneuro-2024-020

Romanian Neurosurgery (2024) XXXVIII (1): pp. 121-126
DOI: 10.33962/roneuro-2024-020
www.journals.lapub.co.uk/index.php/roneurosurgery

Decompressive craniectomy for dural venous sinus thrombosis. A neurosurgical approach

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Keywords
intracranial embolism and thrombosis, intracranial sinus thrombosis, decompressive craniectomy, neurosurgery

ABSTRACT

A rare event, dural sinus thrombosis occurs more frequently in young adults and children. Generally, medical treatment is the preferred option for this condition; however, if no improvement is observed with medical treatment, decompressive craniectomy is suggested as the preferred surgical intervention. A non-systematic literature search was conducted in PubMed and SCOPUS databases until June 2023, using keywords such as "Decompressive craniectomy," "Dural venous sinus thrombosis," and "Traumatic Brain Injury," along with their synonyms in both English and Spanish. The search revealed that genetic or acquired thrombophilia and the use of oral contraceptives were the most common risk factors, explaining the female predominance of this condition. Patients with dural sinus thrombosis commonly experience headaches, the intensity of which is not yet considered pathognomonic for the condition, ranging from mild to severe. Other nonspecific symptoms include nausea, vomiting, and papilledema. Thrombolytic agents are utilized to rapidly dissolve the clot, supported by interventional neuroradiology techniques to administer the agent directly at the thrombosis site. Studies have reported the effectiveness of emergent decompressive craniectomy in patients with recent onset of dural sinus thrombosis, leading to good results, especially in cases where cerebral hernia is present.

INTRODUCTION

Dural sinuses thrombosis (DST) remains a rare event [1], more frequently afflicting young adults and children, with an estimated incidence of 3 to 4 cases per 1,000,000 inhabitants for adults and 7 per 1,000,000 for children [2]. Approximately 75% of patients affected are women, outnumbering men with a ratio of 3:1 [3]. The incidence

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ISSN online 2344-4959
© Romanian Society of Neurosurgery

First published
March 2024 by
London Academic Publishing
www.lapub.co.uk

resulting from traumatic brain injury (TBI) remains unknown; some experts suggest it is underdiagnosed because clinicians frequently omit this diagnosis during TBI evaluations. Symptoms associated with dural venous sinus thrombosis vary, ranging from mild headaches to severe neurological deficits, including coma, due to elevated intracranial pressure associated with ischemic and/or hemorrhagic events [4]. Risk factors exacerbating the prognosis of DST patients include age over 37 years, altered mental status, brain hemorrhage, and coma, among others. Presence of seizures, altered consciousness, and coma heightens mortality rates in patients [5]. Typically, medical treatment is the first choice for this condition; however, when patients do not show improvement with medical treatment, experts suggest decompressive craniectomy as the preferred surgical intervention. The prognosis relies heavily on clinical presentation and imaging studies [6]. This article outlines the risk factors, pathogenesis, clinical manifestations, evaluation, diagnosis, treatment, and prognosis of dural sinus thrombosis, along with the application of decompressive craniectomy.

A non-systematic literature search explored PubMed and SCOPUS databases until June 2023, utilizing keywords such as "Decompressive craniectomy," "Dural venous sinus thrombosis," and "Traumatic Brain Injury," in addition to their synonyms in both English and Spanish. All types of publications containing evidence or data related to dural venous sinus thrombosis were included. Exclusion criteria involved articles lacking full text. Grey literature sources were also consulted, culminating in the consideration of 48 articles.

Risk factors

Certain factors promote DST when a TBI occurs; in fact, prothrombotic risk factors or direct causes are found in 85% of patients with sinus thrombosis [7]. In the international study of dural sinus and cerebral vein thrombosis, involving 624 adults from 21 countries, researchers identified genetic or acquired thrombophilia and the use of oral contraceptives as the most common risk factors, potentially explaining the female predominance of this condition [3,7].

Other conditions that contribute to DST include neoplasms, central nervous system (CNS) alterations (arteriovenous and dural fistulas), hematological conditions, nephrotic syndrome, systemic vasculitis, infections of the central nervous system (bacterial meningitis, cerebral malaria) [8,9], inflammatory bowel disease [7], medications (cisplatin methotrexate, steroids) [10], neurological surgery, lumbar puncture, pregnancy, and the puerperium [9]. Despite the extensive list of risk factors, approximately 20% of cases are considered idiopathic [3,4,7,11]. Moreover, the use of oral contraceptives and coagulopathies contribute to the increased occurrence of this condition [5].

Pathogenesis

In cases of TBI involving skull fractures or intracranial hematomas, thrombosis can occur either due to direct compression of the sinus or endothelial damage within it. This endothelial damage activates the coagulation system, leading to sinus occlusion. DST rarely occurs in the absence of cranial suture diastasis; when it does, it implies that the associated mechanism involves endothelial damage within the venous sinus [12]. The brain contains abundant thromboplastin, released after injury, creating an abnormal hypercoagulable state that results in platelet and erythrocyte destruction followed by thrombus formation [12].

Normally, cerebrospinal fluid (CSF) drains into the superior sagittal sinus through arachnoid villi. Thrombosis in these sinuses increases venous pressure, disrupting the CSF absorption mechanism, consequently raising intracranial pressure [2]. This condition can lead to cytotoxic and interstitial edema [13] and localized venous infarction. The outcome includes dilated veins, edema, ischemic neuronal damage, petechial hemorrhages, which can merge to form bruises [2].

Two theories have been proposed: Cerebral vein thrombosis (CVT) causing local effects due to venous obstruction and venous sinus thrombosis (VST). These events are often considered to occur simultaneously. CVT results in cytotoxic and vasogenic edema around venous vessels, accompanied by venous infarcts, while VST leads to increased venous pressure and decreased CSF absorption, elevating intracranial pressure (ICP) [5]. The most frequently affected sinuses, in order, are the transverse sinus, sagittal sinus, sigmoid sinus, and straight sinus [13,14]. Coexisting involvement of multiple sinuses is common [2].

Clinical manifestations

Patients primarily experience headaches, which vary in intensity from mild to severe and are not yet considered pathognomonic for DST [3,7]. Other nonspecific symptoms include nausea, vomiting, and papilledema [7,12]. The frequency of these symptoms is as follows: headaches (70-95%) [3,5], seizures (39.3%), paresis (37.2%) [3], papilledema (28.3-41%) [3,5], altered mental state (22%), aphasia (19.1%), stupor or coma (13.9%), diplopia (13.5%), and visual deficit (13.2%) [3]. Involvement of the deep venous system results in altered consciousness, abnormal pupillary reactions, and eye movement abnormalities [5].

Clinical evaluation

Conducting a comprehensive neurological examination is crucial to detect signs and symptoms as well as to identify the presence of fractures, which should be actively investigated [4]. This approach enables a more accurate and timely diagnosis, leading to improved patient outcomes [7]. Patients with elevated intracranial pressure often experience isolated symptoms, typically manifested as severe headaches and diplopia, especially when intracranial pressure is significantly elevated due to compression of the sixth cranial nerve. Examination of the fundus reveals papilledema and transient visual impairment, which can become permanent if the underlying condition is not promptly addressed [2].

Diagnosis

The diagnosis of this condition relies on imaging studies. Initially, computed tomography (CT) without contrast is employed [5,15]. This scan reveals edema and areas of hyperdensity indicating a hemorrhagic infarction (present in 40% of cases). Additionally, it identifies specific signs, such as the 'cord sign,' an area of hyperdensity with a thrombosed cortical vein in the transverse sinus region [14,16], and the 'empty delta sign,' a triangular area of enhancement with a relatively attenuated center, seen in the superior sagittal sinus. This sign, observed in about 28.6% of patients, is considered pathognomonic for sinus thrombosis and is associated with a poor prognosis [12]. Its appearance is likely due to increased flow in the large collateral dural venous circulation surrounding the thrombosed sinus, resulting in a central region of low attenuation [17]. However, the absence of these findings does not rule out the diagnosis; abnormalities are not reported in over 25% of cases [7]

Magnetic resonance imaging (MRI) is considered one of the most effective methods for diagnosing this condition as it allows visualization of intracranial vasculature, providing precise information about the location and timing of the thrombus within the dural sinus [7]. In the subacute phase, the thrombus is easily identifiable, particularly on T1-weighted images, with the signal becoming intensely hyperintense around day 15 on both T1 and T2-weighted images, a critical period for diagnosis [17]. In the acute phase, the thrombus appears isodense with the brain on T1-weighted images and low-signal on T2-weighted images. This appearance can be confused with blood flow; however, magnetic resonance imaging with venography (MRV) can confirm the absence of flow [13,15]. Contrasted T2 images have demonstrated higher sensitivity compared to T1 or regular T2. Yet, due to the potential for flow artifacts in MRI and MRV, particularly in cases where deep venous infarction or cortical venous thrombosis is suspected, high-resolution endoluminal techniques such as phlebography or conventional CT are recommended [13].

Digital subtraction angiography, the gold standard in diagnosis, is invasive and challenging to access, making it a last resort used only when there are doubts about the diagnosis that cannot be resolved through other methods [5,12,13,15]. It is also employed in cases of suspected long-standing DST with unclear MRI images [18].

Although the D-dimer test has been proposed to assess DST risk in emergency department patients, with reported sensitivity of 97.1%, negative predictive value of 99.6%, specificity of 91.2%, and positive predictive value of 55.7%, it is not a routine procedure. Its results can be influenced by coexisting pathologies, and it is not universally applicable [7].

Treatment

The treatment strategy revolves around three key aspects: managing intracranial pressure, preventing seizures, and administering antithrombotic treatment. The goal of this therapy is to halt the underlying thrombotic process and prevent venous thrombosis from other sites that could potentially worsen the clinical condition [1].

Traditionally, heparin anticoagulation has been the primary treatment, occasionally used irrespective of the presence of bleeding [5,19,20]. Additionally, monitored long-term use of warfarin has been reported [4,13,15] to prevent overdose [21]. Caution is necessary for patients immediately post-surgery following a TBI, as anticoagulation is generally contraindicated during the postoperative period. This caution is crucial to prevent DST progression. Hence, it is imperative to consider the possibility of DST in the presence of a TBI, especially if there are associated risk factors [4].

The use of thrombolytic agents for rapid clot dissolution, supported by interventional neuroradiology techniques delivering the agent locally at the thrombosis site, has emerged as a therapeutic option. Although there are no randomized, double-blind, placebo-controlled studies supporting thrombolysis as the first-line therapy for cerebral venous sinus thrombosis in comparison to unfractionated heparin, numerous case reports and a single non-randomized study have demonstrated its comparative safety and effectiveness, particularly in rescuing patients who deteriorate rapidly despite unfractionated heparin treatment. This practice should be limited to experienced centers [22]. It is indicated for patients experiencing progressive deterioration unresponsive to anticoagulant therapy, aiming for rapid recanalization of the obstructed sinus [11,15]. When administered within 72 hours of diagnosis, thrombolytic therapy achieves complete recanalization in 56.5% of cases and partial recanalization in 43.5%. Commonly used thrombolytic agents include urokinase (73.7%), tissue activator of recombinant plasminogen (tPA) (21.5%), and thrombectomy or angioplasty (12.2%) [11].

In a retrospective study analyzing 25 patients with DST leading to venous congestion in the brain, anticoagulation therapy alone resulted in a stable course. Patients who received thrombolytic therapy experienced more adverse effects, and those initially undergoing thrombectomy showed clinical deterioration. This highlights the benefits of antithrombotic therapy as the primary approach and the potential role of thrombectomy and thrombolytic therapy in central venous congestion development [23]. Anticoagulant therapy alone does not seem appropriate in these patients when cerebral venous congestion occurs, possibly due to collateral flow loss [23]. Therefore, in cases where patients deteriorate clinically despite adequate anticoagulation, consideration of local or systemic thrombolysis is recommended. There is no consensus on the optimal medication, dosage, route, or method of administration [7].

Prognostic aspects

This condition is regarded as a severe event with high associated mortality. It leads to progressive patient deterioration or a lack of improvement in the clinical condition, potentially resulting in severe neurological complications or death if not promptly treated [4,7,8,9,10,11,12,13]. Although specific mortality rates below 10% have been reported, 17% of survivors exhibit neurological deficits, and these individuals may require long-term rehabilitation due to lasting sequelae. It is advisable to conduct neuroimaging follow-ups at least one year after the event. Some cases experience increased intracranial pressure, leading to optic nerve compression and visual disorders. Hence, regular ophthalmology follow-ups are recommended [13].

Decompressive craniectomy

Decompressive craniectomy (DC) is a surgical technique that involves removing a portion of the skull to facilitate brain expansion, thus mitigating increased ICP and edema while controlling this pathological process [24,25]. DC enhances oxygen supply and perfusion pressure, proving beneficial in reducing ICP in various conditions such as severe head injury, stroke, and subarachnoid hemorrhage [24,25]. It has also demonstrated usefulness in DST cases unresponsive to medical interventions [26]. While randomized studies confirming the benefits of DC in DST management are lacking [5], Stefani et al [27] reported its efficacy in patients with cerebral hernia due to DST, especially in recent clinical onsets, yielding positive outcomes [27]. Patients who underwent DC in this study experienced favorable recoveries and positive functional results. Additionally, they noted improved outcomes when DC was combined with full doses of heparin administered 24 hours post-surgery [28]. Similar results have been reported in other cases, although these findings are not conclusive [29-31].

Despite the existing gap in understanding the utility of such techniques in cerebrovascular

disorders and traumatic brain injuries, these methods have the potential to enhance morbidity rates, reduce mortality, decrease disability, and alleviate the burden of neurological diseases [32-35]. Furthermore, advancements in science and technology, particularly in robotic neurosurgery, offer promising avenues to maintain and restore the functional capacity and quality of life for affected individuals [36-38].

Conclusions

Traumatic brain injury constitutes a risk factor for dural venous sinus thrombosis development. Accurate diagnosis and timely management of this complication can be achieved through a thorough clinical history and imaging studies. This is particularly crucial in young patients with recent head trauma who exhibit symptoms of intracranial hypertension. In such cases, it is essential to rule out neurosurgical pathology or intracranial hematomas adjacent to venous structures. The prognosis is contingent upon the treatment provided and the involvement of other structures in the traumatic brain injury.

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