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Article

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Luis Rafael Moscote-Salazar[1], Willem Guillermo Calderón-Miranda[2], Nidia Escobar-Hernandez[2], Marticela Cabeza-Morales[1], Andrés M. Rubiano[1], Hernando Raphael Alvis-Miranda[1], Nasly Zabaleta-Churio[1], Amit Agrawal[3]
[1]COLOMBIA, [2]MEXICO, [3]INDIA

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Luis Rafael Moscote-Salazar[1], Willem Guillermo Calderón-Miranda[2], Nidia Escobar-Hernandez[3], Marticela Cabeza-Morales[4], Andrés M. Rubiano[5], Hernando Raphael Alvis-Miranda[6], Nasly Zabaleta-Churio[7], Amit Agrawal[8]

[1]Neurosurgery, Red Latino, Latin American Trauma & Intensive Neuro-Care Organization, Bogotá, COLOMBIA; [2]Radiology, Hospital GEA Gonzalez, Universidad Nacional Autonoma de Mexico, MEXICO; [3]Radiology, Hospital GEA Gonzalez, MEXICO; [4]Faculty of Medicine, Universidad de Cartagena, Cartagena de Indias, COLOMBIA; [5]Neurosurgery, Universidad El Bosque, Bogotá, Red Latino, Latin American Trauma & Intensive Neuro-Care Organization, Bogotá, COLOMBIA; [6]Neurosurgery, Universidad de Cartagena, Cartagena de Indias, COLOMBIA; [7]Red Latino, Latin American Trauma & Intensive Neuro-Care Organization, Bogotá, COLOMBIA; [8]Neurosurgery, MM Institute of Medical Sciences & Research, Maharishi Markandeshwar University, Mullana-Ambala, Haryana, INDIA

Abstract: The dural venous sinus thrombosis is a benign disease, representing about 1% of cerebral vascular events. In some cases the development of the disease increased intracranial pressure or symptomatic epilepsy. The development towards a dural venous sinus thrombosis is rare, but is a condition to be considered before the development of ischemic vascular events and a history of recent head trauma. Intracranial hematomas or skull fractures can lead to the establishment of obstructive pathology of the dural venous sinuses. The knowledge of this entity is necessary for the critical care staff and neurosurgery staff.

Key word: Craniocerebral Trauma, Neurosurgery, Cranial Sinuses, Thrombosis

Introduction

The dural sinus thrombosis (TSD) is a rare (1) event, which occurs most often in young adults and children, with an estimated adult 3-4 incidence for 1,000,000 inhabitants, and 7 per 1,000. 000 for children (2). Approximately 75% of patients are women (3), with dominant 3: 1 compared to men. The incidence when produced as a result of traumatic brain injury (TBI) is unknown; although some believe it is underdiagnosed because usually when the presence of TCE omitted this diagnosis is evaluated. Symptoms to establish a dural venous sinus thrombosis may be nonspecific, from mild headache to the development of severe neurological deficits including coma due to increased intracranial pressure associated with ischemic events and / or bleeding (4).

Risk factors

Once submitted TBI there are factors that act as promoters of the TSD, in fact in 85% of patients with cerebral sinus thrombosis are prothrombotic risk factors or direct causes (5). In the international study of thrombosis of the dural sinuses and cerebral veins (ISCVT), where 624 adults from 89 centers in 21 countries were included, it was found that genetic or acquired thrombophilia, and the use of oral contraceptives were Common risk factors (5), which may explain the female predominance of this entity (3). Other conditions considered TSD promoters are neoplasms, CNS disorders (and dural arteriovenous fistulas), hematologic, nephrotic syndrome, systemic vasculitis, central nervous system infections (bacterial meningitis, cerebral malaria) (6, 7), disease intestinal (5) inflammatory drugs (cisplatin (8), methotrexate, steroids), neurological surgery, lumbar puncture, pregnancy and postpartum period (7). Despite the long list of risk factors for approximately 20% of cases are considered idiopathic (3-5, 9).

Pathogenesis

When a TBI occurs with head injury, skull fractures or intracranial hematomas can generate thrombosis either by direct compression of the breast or endothelial damage in this, generating the activation of the coagulation system resulting in occlusion breast. It is unusual for the TSD occurs in the absence of diastasis of cranial sutures (10), when it occurs in these cases, it means that the associated mechanism is endothelial damage within the venous sinus. The brain also contains an abundance of thromboplastin that is released after injury cause abnormal hypercoagulable state leading to destruction of platelets and erythrocytes followed by thrombus formation (10). Cerebrospinal fluid normally drains into the superior sagittal sinus, through the arachnoid villi. Thrombosis of these sinuses leads to increased venous pressure, which changes the mechanism of absorption of cerebrospinal fluid (CSF), and consequently increases the intracranial pressure (2), added to this can generate cytotoxic and interstitial edema (11) and venous infarction located. This produces dilated and enlarged veins, edema and ischemic neuronal damage, with petechial hemorrhages, which can bind and bruising (2). The most affected areas are in order: transverse sinus, sagittal sinus, sigmoid sinus and straight sinus (11, 12). Often the coexistence of several affected breast (2).

Clinical manifestations

Headache is the main symptom in these patients, the intensity is still not considered pathognomonic of TSD, described intensity from mild to severe (3, 5). Other nonspecific manifestations described are: nausea, vomiting and papilledema (5, 10). The frequency of the appearance of symptoms including headache (70%), followed by convulsions (39.3%), paresis (37.2%), papilledema (28.3%), altered mental status (22%), aphasia (19.1%) , stupor or coma (13.9%), diplopia (13.5%) and visual deficit (13.2%) (3).

Clinical Evaluation

Conducting a thorough neurological examination is vital to ascertain the presence or absence of signs and symptoms, just as should look for the presence of fractures (4), to better approach and early treatment improves the patient's condition (5). Patients with increased intracranial pressure isolated, usually only have severe headache and diplopia if intracranial pressure is very high, the sixth cranial nerve compression. In the fundus papilledema is observed, with temporary visual impairment, which may be permanent (2).

Diagnosis

The diagnosis of this entity is by imaging studies. The first study used is computed tomography (CT) without contrast (12), which reveals: edema, indicating areas hyperdensity hemorrhagic infarction (40% of cases), accompanied by string sign, which is a hyperdense area with cortical vein thrombosed transverse sinus area (13, 14); and the sign of the empty delta (10) which is a triangular area of enhancement with a relatively attenuated in the region of the superior sagittal sinus, considered pathognomonic of this sinus thrombosis occurs in about 28.6% of center these patients and has been found associated with poor prognosis. The appearance of this sign is probably due to the increased flow of the abundant collateral dural venous circulation, the breast surrounding the thrombosed producing central low attenuation (15). However, the absence of these findings does not exclude the diagnosis (5). In more than 25% of cases this study reported no abnormalities (5).

Magnetic resonance imaging (MRI) is considered one of the best methods for diagnosis of this entity, because it allows the visualization of the intracranial vasculature, and can better determine the location and time of the thrombus within the dural sinus (5). On MRI, the thrombus is easily recognizable in the subacute phase (10), primarily to exploration on T1, around the 15th thrombus signal it appears strongly hyperintense on T1 and T2 weighted images, perhaps this is the period of greatest important because it is where most diagnoses (16) are made. In the acute phase, the thrombus is isodense with brain on T1-weighted images and low signal on T2-weighted images. This aspect can be mistaken for blood flow; but by using magnetic resonance venography (MRV) no flow (11, 12) demonstrated. T2 has been proven more sensitive than single T1 or T2 .However, MRI and MRV are likely to flow artifacts techniques and in doubtful cases, particularly if deep venous infarction or cortical venous thrombosis, an endoluminal technique is suspected high resolution as conventional venography or CT is recommended (11).

Gold standard diagnosis is digital subtraction angiography, but because it is invasive and difficult to access, only used in cases where there is doubt about the diagnosis has not been determined by other methods (10-12) or when it is suspected a longstanding TSD with RM images unclear (16).

It has been proposed the use of D-dimer to assess the risk of TSD, in patients in the emergency room, it has reported a sensitivity

of 97.1% and a negative predictive value of 99.6% and specificity of 91.2% with a positive predictive value of 55.7% (5), despite this, this test is not considered routine, and can be performed in all patients, and can be altered in the presence of coexisting conditions (5).

Treatment

The three pillars of treatment are: management of intracranial pressure, convulsions and antithrombotic prevention treatment (1). The goal of this therapy is to stop the underlying thrombotic process and prevent venous thrombosis of other sites that may potentially aggravate the clinical picture.

In antithrombotic therapy, anticoagulation with heparin has long been the traditional treatment, even sometimes is used regardless of the presence of hemorrhage (17, 18). It has further been reported using long-term warfarin (4, 11, 12), of monitored so as to avoid overdose (11).

Care should be taken in patients who undergo surgery immediately in the presence of a TCE, because in them is generally contraindicated postoperative anticoagulation, which can lead to progress TSD therefore requires the suspect Initial of this entity in the presence of TCE and even more if there are risk factors (4).

The use of thrombolytic agents which lyse the clot has quickly emerged as a therapeutic modality, supported interventional neuroradiology techniques to release the agent locally at the site of thrombosis. No randomized, double-blind, placebo-controlled cases, that support thrombolysis as the first-line therapy in patients with thrombosis of the cerebral venous sinuses, compared with unfractionated heparin. Numerous case reports and nonrandomized single study has shown that it is comparatively safe and can rescue patients rapidly deteriorate despite treatment with unfractionated heparin. This practice should be restricted to centers with experience in this therapy (19). It is indicated in patients with progressive deterioration and do not respond to anticoagulant therapy for rapid recanalization of the obstructed sinus (9, 12). When thrombolytic therapy is administered within 72 hours of diagnosis, it has been demonstrated that complete recanalization is obtained in 56.5% and 43.5% partial recanalization in (9). Among the most commonly used thrombolytic agents include: urokinase (73.7%) followed by recombinant plasminogen tissue activator (tPA) (21.5%), and thrombectomy or angioplasty (12.2%) (9).

In a retrospective study of 25 patients with dural sinus thrombosis the role played by cerebral venous congestion and the management of these patients Patients were administered anticoagulation showed a steady course, those who were given thrombolytic therapy had analyzed more outcome of adverse effects, and who underwent thrombectomy presented initial clinical worsening; demonstrating the benefits of antithrombotic therapy as the main management and the role they may have to thrombectomy and thrombolytic therapy in the development of central venous congestion. Apparently the only anticoagulant therapy is not appropriate in these patients when they develop cerebral venous congestion, perhaps due to the loss of collateral flow (20).

Consideration of local or systemic thrombolysis in patients clinically deteriorate despite adequate anticoagulation recommended. There is no consensus to determine the optimal medication, dose, route and method of administration (5).

It is considered a serious event of great associated mortality, that causes a progressive deterioration of the patient or improving the clinical condition, which can lead to serious neurological complications or death if not treated in a timely manner (4, 11). It has reported a lower specific mortality of 10%, but neurologic deficits are present at follow-up 17% of the survivors, and the consequences may require long-term rehabilitation (11). For best results, follow with neuroimaging at least 1 year after the event. Due to increased intracranial pressure, which occurs in some cases, there may be compression of the optic nerve and visual impairments therefore monitoring is recommended together with ophthalmology (11).

Conclusions

Head trauma constitutes a risk factor for developing thrombosis of dural (21) venous sinuses. A correct clinical history and imaging studies allow us to make an accurate diagnosis and timely managing this complication. Especially in young patients with recent head trauma who develop symptoms of intracranial hypertension and which they should discard neurosurgical pathology or intracranial hematomas adjacent to venous structures (22-24). Prognosis depends on the treatment and the other structures involved with head trauma.

Correspondence
Dr. Luis Rafael Moscote-Salazar
E-mail: mineurocirujano@aol.com

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