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ABSTRACT

Bilateral chronic subdural hematoma is a neurosurgical pathology whose incidence in older adults has been increasing, as a consequence of the ageing of the population, added to the factors that are linked to it. Neurosurgical diseases with chronic evolution generate a high burden of disease due to morbidity, disability, mortality and health costs associated with reinterventions and rehabilitation. For this reason, the interest in this disease has been increasing, also justified by the little information there is about it, unlike unilateral chronic subdural hematomas, although it has been described that both may have pathophysiological similarities that help to understand them.

Introduction

The average age of the population has been increasing and with it are associated several pathologies to which the elderly population is exposed, as well as environmental factors [1]. Chronic subdural hematomas (CSDH) are one of the most frequent conditions seen in the population over 65 years of age, however, there are several factors that may predispose to this disease [2,3].

Bilateral CSDH are collections of blood degradation products located between the dura mater and arachnoid, and the denomination of chronicity is given by the amount of time elapsed, which is between 2 or 3 weeks [2,4,5,6,7,8]. It has been described as a pathology that differs from unilateral hematomas in the form of clinical presentation and the rapidity with which it can progress and lead patients to marked deterioration if timely treatment is not carried out [9]. Pathophysiologically, bilateral CSDHs have no clear difference from their unilateral counterparts [9]. The treatment of choice is surgical and ranges from craniotomies and endoscopic treatment to YL-1 type hematoma needle aspiration [4]. Based on the above, the objective of this review is to present practical concepts on the identification and approach to chronic bilateral subdural hematoma.

Epidemiological focus of the disease

The incidence rate of bilateral CSDH ranges from 1-5 cases per 100,000 person-years to 7.4 per 100,000 in the elderly population [10–13]. The most frequent location of CSDH is supratentorial, as opposed to the posterior fossa, whose diagnosis and pathogenesis are poorly understood [14]. Likewise, the average age of patients diagnosed with CSDH has been found to range from approximately 62 to 74 years, and, in the case of bilateral CSDH, a study by Cheng et al found that the average age of patients was 79 years [2].

Predisposing factors of CSDH

Unlike acute subdural hematomas (Acute-SDH), CSDH are poorly associated with head trauma [2,15]. Despite this, trauma is considered to be the primary cause, as it has been reported to be associated with CSDH in 50-80% of cases [2]. On the other hand, antiplatelet and anticoagulant therapies, in addition to alcohol abuse, liver disease, renal disease in hemodialysis patients, and intracranial hypotension associated with ventricular shunt valves have been linked to the development of hematoma [2,3,5].

Pathophysiology of CSDH

In contrast to unilateral CSDH, the pathophysiology of bilateral CSDH is still unknown [9]. However, hypotheses and premises have been established as to what may be occurring, such as the involvement of the bridging veins [9,10]. When bilateral CSDH is present in the posterior cerebral region, blood deposition may be involved with the occipital, sigmoid and transverse sinuses [14]. The development and progression of the hematoma is gradual and slow, initially constituting a thick outer membrane around the hematoma that eventually results in a complete capsule about two weeks later [15], which requires cell proliferation for the formation of a collagen-rich granular tissue [5].

On the other hand, the increase in hematoma size may be related to the increased permeability of capillaries in the outer membrane of the hematoma, which consequently lead to high blood extravasation and plasma exudation [11]. In addition, there is localized inflammation characterized by increased fibrinolysis, release of tissue plasminogen activator and elaboration of angiogenic factors that promote neovascularization [5].

Clinical presentation of CSDH and its diagnosis

The form in which CSDH manifests itself is very variable and can even be confused with other more frequent entities such as stroke, Parkinson's disease, dementia, among others [3,5]. In general, cognitive impairment, headache, and motor deficits are the most common presenting symptoms [3,5,13], although patients have also been shown to manifest nausea, vomiting, mental changes, and convulsions. In a study by Huang Y. et al. in which 25 of the 98 people evaluated had bilateral CSDH, it was observed that headache and mental changes were the most common presenting symptoms, followed by nausea and finally seizures [16].

However, because CSDHs present in most cases with unilateral convexity [16], many studies have relied on the comparison of several clinical features between the two presentations in order to analyze the point differences in the frequency of presentation of a symptom, or other feature, between unilateral and bilateral CSDHs [13,16,17].

In a study by Agawa et al. it was observed that

symptoms such as headache, pupil abnormalities, and acute impairment of consciousness occurred more frequently in patients with bilateral CSDH, while hemiparesis occurred more frequently in patients with unilateral CSDH [13]. Likewise, in the study by Huang Y. et al. it was observed that hemiparesis is more frequently seen in patients with unilateral CSDH since, in the case of patients with bilateral CSDH, there is a lower possibility of the central brain structures being deflected due to the counterbalance of the mass effect on both sides, which is reflected in the radiological findings that showed a lower incidence of midline changes with respect to unilateral [16].

On the other hand, as mentioned above, CSDH are often confused with other diseases, so it is of utmost importance to be really informed about the symptomatology of pathologies with similar clinical presentation to CSDH, in order to be able to make the differential diagnosis together with other complementary diagnostic tests [5,16]. Over the years, cases have been reported in the literature on patients with bilateral CSDH presenting with isolated oculomotor nerve palsy, which is a rare symptom in the atypical clinical presentation of this pathology, as it is commonly associated with vascular disorders, posterior circulation aneurysms or with traumatic, inflammatory or neoplastic diseases [7,18]. Corrivetti et al, by 2016, published two case reports, one of them was about an 81-year-old patient with diabetes and on anticoagulant management who was referred to the hospital with headache and left palpebral ptosis, on physical examination he showed complete oculomotor nerve palsy and mydriasis, and his computed tomography (CT) scan showed bilateral CSDH. He subsequently underwent bilateral surgical evacuation and then, 24 hours after surgery, the ptosis had disappeared and the mydriasis gradually improved [7]. Based on the above, the authors refer that having at least one predisposing factor (diabetes mellitus, hypertension, among others) generates a nervous vulnerability, which is necessary but not sufficient to develop this symptom, in this way, It is suggested that the bilateral pressure exerted on both cerebral hemispheres causes a displacement of the posterior part of the brain causing a compression of the encephalic trunk and this, added to the cisternal compression and the narrow vascular corridor through which the oculomotor nerve passes, stretches and flattens the nerve leading to a nervous alteration [7,18].

On the other hand, Guppy et al, by 2017, reported a rare case of a patient with an abnormal gait, where it was initially thought to be cervical myelopathy due to stenosis, however, he also presented Parkinson's-like symptoms (decreased facial expression, random gait and stooped posture) so a CT scan was requested which subsequently showed bilateral CSDH. After drainage of the CSDHs the symptoms improved, with bilateral CSDH and cervical stenosis being the final diagnosis [19]. Finally, in order to confirm CSDH and to make the definitive diagnosis, patients usually undergo CT or MRI [10,11].

Therapeutic approach

Because bilateral CSDH usually occur in the elderly population with other comorbidities such as coagulation disorders, and because of their rapid clinical evolution compared to unilateral CSDH, it is of vital importance that they are detected early in order to be treated as efficiently and promptly as possible [3,5]. Therefore, two types of treatments have been proposed, which are classified into non-surgical procedures, such as the use of steroids or mannitol, which require a longer recovery time for symptoms and a prolonged hospital stay [3], and surgical procedures (surgical drainage), the latter being the best therapeutic alternative due to its wide range of procedures and the low morbimortality it represents [3,11].

To evaluate the patient's neurological status, the clinical severity of CSDH and the post-surgical evolution, the Markwalder classification system is used, with a scale ranging from grade 0 to 4, the higher the grade, the worse the patient's clinical situation [3,20]. The decision to surgically treat a bilateral CSDH, should be subject to two circumstances which are the clinical manifestations and the radiological findings which include the presence of a mass effect or a midline shift [5,16]. It is generally accepted that symptomatic patients should undergo surgery because surgical evacuation produces an immediate recovery and a favorable evolution in a high percentage of patients [5]. For surgery, neurosurgeons have proposed several surgical techniques such as small craniotomy with endoscopic removal, burr craniotomy with or without continuous drainage of the closed system, large craniotomy followed by hematoma removal

with membranectomy, cranial drilling (twist-drill), and others [5,11,21].

Indications for craniotomy include the presence of any clot causing symptomatology with or without failed conservative or less invasive management, evidence of radiographic progression during a time of conservative observation, and, in addition, craniotomy should be considered in cases of recurrence despite previous surgery or when there are clots with evidence of membranes that may inhibit evacuation [20].

On the other hand, it has been shown that some patients are treated surgically by auger punctures on the side of the subdural hematoma along with irrigation of the subdural space with normal saline, in addition to performing drainage of the closed system by silicone tubes that are removed after the drainage has slowed or stopped, or removal of the hematoma has been confirmed by postoperative CT scanning [10,11,16]. In addition, some patients with coagulation disorders who use anticoagulants as therapy have been shown to have vitamin K, fresh frozen plasma or prothrombin complex administered prior to surgery as a preoperative indication [5,13].

Complications

Recurrence is the most common complication of CSDH with an incidence that can vary from 0% to 31.6% depending on the surgical technique [22,23]. Recurrence is defined as re-accumulation of the hematoma with progressive neurological deficit and represents the main problem after initial trephination because about 20% of patients require at least one re-intervention [22]. A higher recurrence rate has been reported in patients with bilateral CSDH compared to patients with unilateral CSDH [23]. In some cases of bilateral CSDH, when the contralateral hematoma is asymptomatic and thin, trephination surgery is only performed in one side [24]. Up to 20% of these cases may require evacuation of the contralateral hematoma due to subsequent enlargement and appearance of symptoms after a certain period of time [24,25].

The pathophysiology of this complication is not fully understood [22], but in conjunction with bilateral hematoma, many factors have been proposed as predictive of postsurgical recurrence of CSDH [24], such as use and reinitiation of antiplatelet or anticoagulant therapy [22], postoperative midline shift, preoperative hematoma size, mean hematoma density, diabetes mellitus, and postoperative air harvesting [24]. Hematomas that are hypointense or isointense on preoperative MRI on the T1 sequence correlate with a high recurrence rate, because it may reflect intracellular deoxyhemoglobin, signifying relatively recent bleeding [24].

Craniostomy with trephination is a simple surgical procedure; however, postoperative bleeding is another well-known and devastating complication. In a study of 303 patients with CSDH, the incidence of acute postoperative intracranial hemorrhage was estimated to be approximately 4.57% [23]. It was initially attributed to hypertension and anticoagulation therapy in some patients, but later other mechanisms were discussed [22]. It was mainly attributed to a loss of autoregulation of blood vessels with subsequent hypertension and cortical hyperaemia following rapid brain shift after surgery with excessive drainage of the subdural space and hypoperfusion syndrome [22,23]. More studies are needed to be certain about the treatment of acute postoperative bleeding, but many authors recommend slow brain decompression to prevent rapid intracranial changes [22].

Infections are a rare complication of CSDH that can lead to subdural empyema [22,26]. A preexisting subdural hematoma can become infected through hematogenous infection, although the actual incidence rate of this complication is less than 1% of cases [26]. If suspected, it should be investigated with computed tomography as first-line imaging and as treatment, trephination lavage and craniotomy with complete evacuation of the hematoma have been proposed as surgical adjuncts to systemic antibiotic therapy [22,26].

The development of seizures or status epilepticus following CSDH evacuation is a complication with an overall incidence ranging from 1% to 23% [22,27]. Risk factors associated with the development of chronic hematomas are alcohol abuse, change of mental status, previous stroke, and hematoma density on CT scan [27,28].

In this order of ideas, it is necessary to produce more evidence of better quality on the global epidemiology, pathophysiology and management of bilateral chronic subdural hematoma, considering that it is a pathological condition that generates neurological disease burden. Similarly, training in their suspicion and identification, to promote

professionalism and practice in neurosurgery at the different levels of care. It is necessary to know data from low- and middle-income countries, where there are difficulties in the management of pathologies such as these, due to the absence of specialized centers in rural regions [29,34]. Especially in the pandemic and post-pandemic period where funding for research in lines other than emerging infectious diseases has been lost [35-37].

Conclusions

It is important to recognize the differences in presentation between unilateral and bilateral CSDH, as well as with other pathologies of similar presentation that do not require urgent imaging methods, as this may confuse the diagnosis and delay treatment. Bilateral hematoma tends to involve greater mass displacement than unilateral hematoma and reduces the autonomic capacity to buffer increased intracranial pressure, making it even more important to implement early surgical decompression of bilateral mass lesions in order to prevent rapid neurological deterioration. Healthcare professionals should be aware of the higher incidence of recurrence in patients with bilateral treatment, the risk factors and signs for early detection of recurrence, as well as other complications that, although of lower incidence, represent a risk for patient survival.

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