Chronic Subdural Hematoma: Past, Present, and Future

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Introduction

Chronic subdural hematoma (CSDH) is a common neurosurgical condition characterized by abnormal collection of blood products in the subdural space with indolent course of progression. Its pathophysiology is complex and many theories have been put forward over time. Its presentation varies from a minimally symptomatic event to potentially serious neurosurgical emergencies. The surgical evacuation is the mainstay of its management although there has been some interest in pharmacological and minimally invasive endovascular options in this millennium. The prognosis depends on a number of factors, some of which are interdependent.

History

The first case of a CSDH was reported by a German physician Johan J. Wepfer in 1657 following a necropsy on a stroke patient.[1] Two centuries later, Rudolph Virchow in 1857 described this condition as "pachymeningitis haemorrhagica interna."[2] Later Trotter in 1914 put forward the theory of trauma to the bridging vein as a cause of what he called a "subdural hemorrhagic cyst." The official name of CSDH emerged after contributions form Putman and Cushing in 1925.[3] Since then, our understanding of its pathophysiology is based on the theory of osmotic gradient across the semipermeable membrane as proposed by Gardner in 1932[4] and recurrent bleed from the hematoma capsule as supported by Apfelbaum et al in 1974.[5]

Epidemiology

The incidence of CSDH in the general population is estimated to range from 1.72 to 20.6/100,000 per year.[6] However, some studies have shown incidence of up to 58.1/100,000 per year for those over the age of 65 years and up to 127.1/100,000 per year for those over the age of 80 years.[7] This is likely to rise further with the aging population on the one hand and increasing use of antiplatelet/anticoagulant medication on the other.[8] A large epidemiological study of 63,358 patients has shown that 78.2% were over the age of 70 years and 93% were over the age of 60 years.[9]

Pathophysiology

CSDH commonly occurs following a head injury that is usually trivial especially in the elderly although history of trauma could be absent in about 30 to 50% cases.[10] Risk factors include age-related cerebral atrophy with resultant increase in the subdural space from 6 to 11%.[11] Tendency to fall is also contributing to the development of CSDH in the elderly population. Chronic alcoholism, long-term anticoagulation, and intracranial hypotension have also been implemented in the development of CSDH. A variety of other mechanisms including angiogenesis, vascular permeability factor release, and other growth factor release are also considered in its evolution.[12]

Presentation

In most patients, the clinical progression can be generally categorized into three phases: initial trauma, latent period, and clinical manifestation.[13]

CSDH presents with varying symptoms and hence is so aptly considered "the great imitator."[14] Fifty percent to 70% of them present with altered mental state, while other common presentations include headache (14–80%), focal neurological deficit (58%), seizures (6%), and falls (74%). Atypical presentations include extrapyramidal manifestations and other rare neurological syndromes, for example, Gerstmann's

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syndrome.[15] Bilateral CSDH occurs in approximately 10 to 25% of patients.

Patients can be categorized according to Markwalder grading scale in to five grades depending on their clinical condition, and this can help decide the management plan.[16]

Diagnosis

Diagnosis is often confused with other possibilities like brain tumor (27%), subarachnoid hemorrhage (10%), or stroke (6%) due to a variety of presentations discussed earlier.[10] Computed tomography (CT) scan is a preferred modality of imaging to confirm the diagnosis due to its easy access and cost-effectiveness; however, it is less sensitive than magnetic resonance imaging (MRI) in identifying membranes—27 versus 60% according to one study. MRI is also required to identify isodense collections without midline shift or those at the vertex and in the posterior fossa.[17,18]

Management

The current treatment of choice is surgical evacuation although the technique varies greatly including twist drill craniotomy (TDC), burr hole craniostomy, and standard craniotomy (SC), which is usually reserved for those with solid clots or recurrence. The study by Santarius et al[19] has confirmed that routine use of subdural drains reduces the risk of recurrence. According to the study by Peters et al, there was no significant difference in post-op complications, outcomes, or late recurrences (30–90 days) in any of the three surgical techniques; however, early recurrence up to 30 days was higher for TDC (37.3 vs. 2.9 vs. 16.7%), while SC had higher risk of stroke and prolonged stay especially in those older than 80 years.[20] The risk of recurrence in a surgical series is reported to be 3 to 20% in different studies focusing on burr hole evacuations.[21,22]

A study by Jones and Kafetz has also shown that 23% of patients did not warrant surgery as the volume was small.[23] Since the turn of the century, attempts have been made to treat CSDH with medical measures including administration of steroids[24] or minimally invasive endovascular options like middle meningeal artery embolization (MMAE).[24]

Prognosis

The outcome varies following surgical management with morbidity and mortality around 16 and 6.5%, respectively, as reported by Rozzelle et al in a large series with 157 patients.[25] Among various prognostic factors, neurological status at the time of diagnosis is the most important. On the other hand, advanced age is the most controversial factor, although the majority would argue that the presence of multiple comorbidities may lead to poor outcome.[26] Recurrence is known to occur in 10 to 25% cases, perhaps influenced by risk factors including anticoagulation, post-op seizure, bilateral SDH, and large initial hematoma volume.[27]

Conflict of Interest
None declared.

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