Chronic Subdural Hematoma
Epidemiology and Natural History

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KEYWORDS

• Chronic subdural hematoma • Epidemiology • Natural history • Pathogenesis

KEY POINTS

- Chronic subdural hematoma (CSDH) is a common disease that is prevalent predominantly in elderly patients.
- The incidence of CSDH ranges from 1.72 to 20.6 per 100,000 persons per year.
- Clinical progression of CSDH has 3 stages: initial formation, latency, and clinical presentation.
- Risk factors for CSDH include advancing age, male gender, and antiplatelet or anticoagulant use.

INTRODUCTION AND OVERVIEW

Chronic subdural hematoma (CSDH) is a common disease characterized by the abnormal collection of blood products in the subdural space with a relatively indolent course of disease progression.[1,2] The overall incidence of CSDH was reported to range from 1.72 to 20.6 per 100,000 persons per year,[3–8] with an incidence that is significantly higher in the elderly.[1,6,7,9] A trend toward an increase in incidence has been observed, and may be attributed to the overall aging population resulting from an increase in life expectancy.[10–12] The formation of CSDH remains unclear, and the pathophysiology has been hypothesized to be triggered by inflammatory responses,[13] transformation from acute subdural hematoma,[14] or an increased osmotic oncotic pressure gap between the hematoma and blood vessels.[15–17] Additionally, subclinical brain injury resulting in minor trauma to bridging veins may also facilitate the chronic accumulation of blood within the hematoma encapsulated by neomembranes.[11–14,18,19]

Manifestations of CSDH are variable, and mainly caused by immediate intracranial compression through expansion of the hematoma. Presenting symptoms include headaches, seizure, mental status changes, weakness, sensory disturbance, dysarthria, gait disturbance, nausea and vomiting, stroke, and coma.[20–22] In patients presenting with CSDH, 3% to 20% present with comatose status,[20,21,23,24] and severe impairment with brain herniation may occur in 2% of cases.[21] Of note, 10% to 30% of patients were also on anticoagulation or antiplatelet therapy.[10,12,23,25,26] CSDH can be diagnosed quickly using computed tomography, showing a crescent-shaped mass with a slight hypodensity representing the fluid sac of hematoma encased by the neomembranes.[27] Increased density and heterogeneous density may be noted as a natural progression of the disease or if recent bleeding is present.[28]

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Surgical evacuation of the hematoma remains the mainstay of treatment for symptomatic patients with diagnosed CSDH. Access to the hematoma capsule is approached through twist drill openings or burr hole openings; craniotomy is reserved generally for extensive hematoma, and is considered as a second-tier surgical option for most cases.[12,21,29,30] For asymptomatic patients, nonsurgical management is preferred, which includes eliminating precipitant factors such as anticoagulation or antiplatelet medications, correction of underlying coagulopathy, and symptom management.[12] Prognosis after surgical treatment has been favorable in most reported series, with 80% to 90% of treated patients achieving a satisfactory outcome at follow-up,[21] with mortality reported to be low at 0.5% to 4.3%.[20,21,23] Noticeably, the recurrence of CSDH may be as high as 70%, but only 10% to 20% of recurred CSDH require reoperation.[12,21,23,29,31–35]

EPIDEMIOLOGY

The incidence of CSDH has only been reported sporadically throughout the years. In a Finnish study conducted by Foelholm and Waltimo[4] in 1967, 64 residents of the city of Helsinki diagnosed with CSDH were included over a 7-year period, rendering an overall incidence of 1.72 per 100,000 persons per year. The authors highlighted a significant increase of incidence of up to 7.35 in the elderly population of 70 to 79 years of age. After this study, Kudo and colleagues[6] examined the incidence of CSDH in the Awaji island of Japan from 1986 to 1988, and concluded an overall incidence rate of 13.1 per 100,000 persons per year, with an incidence of 3.4 in the population under 65 years old and 58.1 in the elderly. A North Wales study by Asghar and colleagues[7] including patients between 1996 and 1999 revealed a lower incidence rate of 8.2 per 100,000 persons per year in patients greater than 65 years of age. In contrast, another Japanese study examining incidence of CSDH between 2005 and 2007 used data from a national registry. The authors reported an increased incidence of CSDH of 20.6 per 100,000 persons per year, with 76.5 in the 70 to 79 age group and 127.1 in the 80 years old age group.[3] Additionally, in a recent study by Balser and colleagues[8] focusing on the veteran population in United States, an overall incidence rate of 79.4 per 100,000 persons per year was observed, with age-standardized rate at 39.4 per 100,000 persons per year.

From these studies, it seems that the incidence of CSDH has been increasing over the years, with the increasing risk of CSDH attributable to the aging population and the increasing prevalence of anticoagulation/antiplatelet medication use in general populations.[3,4,6–9,12,13,24,25,27,28,33,35–38] However, the interpretation of the reported incidence rate should be managed with caution. To date, there are only 2 epidemiologic studies examining overall incidence of CSDH in a relatively closed population with low migration rates—the Finnish study and the Awaji island study in Japan—and a considerable difference in incidence rate has been noted between these 2 studies. Although the difference has been generally explained by increasing incidence of CSDH over the 20-year interval between these 2 studies,[8] the examined populations are heterogeneous, and confounding factors associated with population differences may also modify significantly the risk of CSDH. For instance, the incidence of traumatic brain injury, which is considered one of the precipitating factors for development of CSDH, is generally low in Finland, and may account for the lower rate of CSDH in the Finnish population. According to a nationwide, population-based study in Finland examining total traumatic brain injury from 1991 to 2005, the overall incidence was 101 per 100,000 persons per year, significantly lower than that reported in other countries, including the Netherlands, Estonia, and New Zealand.[39–42] In the veteran study where traumatic brain injury within the included population is considered highly prevalent, the projected incidence rate of CSDH was reported to be 121.4, which is significantly higher than that of the general population in other studies; conversely, in the same study, the prediction of CSDH incidence was only 17.4 in civilian-based models.[8] Additionally, in a study reported by Baechli and colleagues[37] examining the prevalence of CSDH in a large single-center study in Switzerland during a 7-year interval (1996–2002), the annual incidence remained relatively constant. Therefore, whether the global incidence of CSDH is truly increasing warrants cautious consideration, and the trend of an increased incidence for CSDH should be adjusted for population and environmental differences for accurate interpretation.

NATURAL HISTORY AND RISK FACTORS

Origin of Chronic Subdural Hematoma

Numerous theories have been proposed regarding the pathogenesis of CSDH. Virchow first related the origin of CSDH to an inflammatory response of the brain in 1857, in which he described a case of "pachymeningitis hemorrhagic interna," and noted a membranous structure in the inner surface of the dura believed to be the inflammatory origin for hematoma formation.[43,44] In contrast,