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*Editorial*

# Chronic subdural hematoma needs to be named differently

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Our understanding of the pathophysiology of chronic subdural hematoma (CSDH) has evolved through time. It was considered a stroke in the 17th century, an inflammatory disease (pachymeningitis chronica hemorrhagica) in the 19th century, and a trauma-induced lesion in the 20th century.[3] Whereas the etiology and natural course of an acute subdural hematoma (SDH) is pretty straightforward, a CSDH is not merely a consequence of an acute SDH. Even though the primary cause of single-compartment chronic SDH is trauma or acute subdural hematoma, there is definitely much more to it in cases of CSDH with multiple layers. Similarly, the single compartment or monolayer CSDH responds excellently to simple burr-hole evacuation, but once multiple layers are formed, surgical treatment becomes increasingly complex. However, both are called CSDH, and therefore, especially for the junior resident on call receiving these patients, the distinction is not important, even though it is critical for patient management. We, therefore, advocate that both the current definition and the nomenclature appear over simplistic and need revision.

Several theories have been proposed to explain the mechanism of CSDH. Oncotic pressure theory and effusion theory were proposed in 1934 and 1955, respectively, to explain that expansion of an original subdural clot occurred through the osmotic attraction of cerebrospinal fluid (CSF) by blood within the semipermeable hematoma neomembranes.[8] These theories were disproved when the osmolality and oncotic pressure of SDH fluid, venous blood, and CSF were compared. Markwalder supported that the mechanism of hematoma enlargement was repeated micro-hemorrhage from the membrane of chronic SDH by reviewing the literature in 1981.[4]

CSDH has been previously described as a heterogenous pathology with not only variable radiological features but also requiring a variety of surgical options and recurrence rates. Nakaguchi *et al*., studied 116 patients harboring 126 CSDHs and, based on the internal architecture and density of each hematoma, classified them into four types. These included homogeneous, laminar, separated, and trabecular types. The recurrence rate was found to correlate with the internal architecture as described on plain computed tomography (CT) scans.[4] Shimizu *et al*., in a similar study, described 388 patients with CSDH and classified them into four groups according to CT findings: low-density hematoma, gradation-density hematoma (GDH), iso-density-hematoma, and mixed-density hematoma. The rate of symptomatic CSDH recurrence correlated with CT findings and was higher in cases with GDH (23.5%).[6] In this specific type, the pathological findings showed more inflammatory cells and blood vessels within the outer membrane. It was shown previously that inflammation leads to the creation

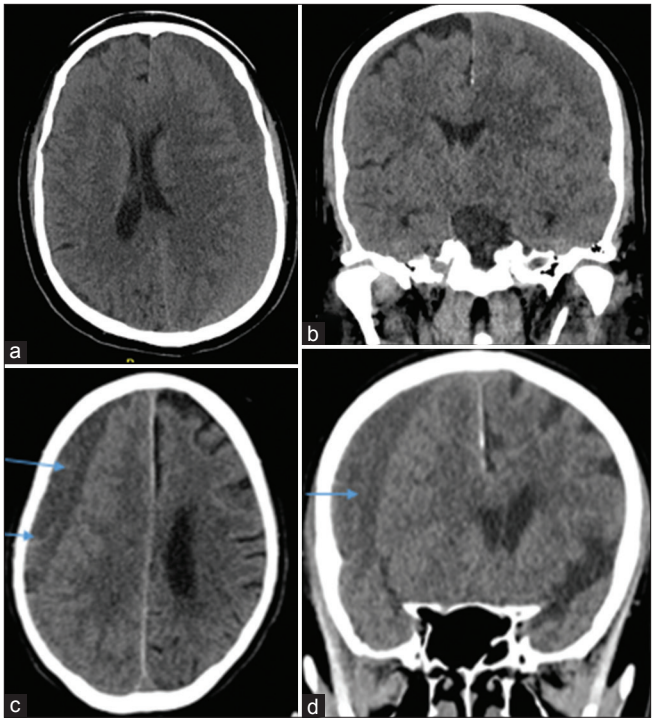


**Figure 1:** (a and b) Computed tomography (CT) scan axial and coronal images of a middle-aged patient with a single compartment left chronic subdural hematoma. (c and d) CT scan axial and coronal images of another middle-aged patient with a multi-compartmental right chronic subdural hematoma (arrows highlight membranes).

of fragile blood vessels within the outer membrane wall, which prevents fibrinolytic processes, resulting in continued bleeding. Therefore, the high CSDH recurrence rate of GDH was related to outer membrane inflammation. The same theory brought some interest in the role of steroids in the treatment of this variant of CSDH, although it was soon debunked.[6]

Along with pathophysiology and radiology, the treatment options for these hematomas are also different. Tanikawa *et al*.,[7] classified all cases according to the intrahematomal membrane structure of CSDH on T2-weighted magnetic resonance (MR) imaging. The first group, labeled type B, included hematomas that had no intrahematomal membrane and were monolayer multilobule. The second group, labeled type C, consisted of hematomas, which were divided into multiple layers by the intrahematomal membrane. The outcome of type C patients treated with burr holes was significantly inferior to that of those who underwent a small craniotomy. The authors concluded that a considerable number of cases appeared to need craniotomy and resection of intrahematomal membrane for complete recovery in CSDH and that T2-weighted MR imaging could be used as a basis for selecting the operative procedure for CSDH.[7] The recurrence rates were similarly reported to be up to 80.6% by Kim *et al*., in patients with multiple membranes.[2]

These membranes are a hallmark of this CSDH variant. Since the blood supply of these neomembranes is derived, in large part, from the external carotid artery through the middle meningeal artery (MMA), it was hypothesized that embolizing the MMA may break the repetitive cycle of microbleeding and inflammation.[5] Over the past decade, multiple centers have reported successful endovascular embolization of the MMA as an adjunct or replacement to surgical drainage. Embolic material penetrates the neovascular membranes, and the rate of hematoma reabsorption appears to be accelerated with MMA embolization, likely due to the reduction in membrane bleeding. In multiple recent large series, only 4–8% of primarily MMA-embolized CSDHs required surgical rescue.[1,5]

In summary, CSDH is not merely a chronic form of acute SDH but rather encompasses a spectrum of pathologies with diverse causes and treatment approaches. While a simple CSDH that does not show layers or compartments on imaging may result from ASDH and can be managed with a burr-hole procedure, a chronic multilayered SDH presents a more intricate pathophysiology necessitating a different therapeutic strategy, including a mini-craniotomy, removal of membranes with or without MMA embolization [Figure 1]. Therefore, the authors advocate for a change in terminology that clearly differentiates between the two types and does not confuse the junior resident on call trying to make the best plan for their patient.

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