

The Pathogenesis of Chronic Subdural Hematomas (CSDH): A Study on the Formation of Chronic Subdural Hematomas and Analysis of CT Findings

Neda Jafari; Lyle Gesner; Joseph M. Koziol MD, FACS; Otakar R. Hubschmann MD
Saint Barnabas Medical Center
Livingston, New Jersey

■ Saint Barnabas Medical Center
■ Barnabas Health

Learning Objectives

By the conclusion of this session, participants should be able to:

- 1) Identify a few theories regarding the pathogenisis of formation of CSDH
- 2) Discuss the multifactorial relationship of the origin of CSDH

Introduction

The pathophysiology of CSDH remains unknown. The most accepted theory suggests rebleeding from immature vessels in neomembranes. Another theory suggests CSDH originate from subdural hygromas (SH). Since chronic rebleeding would be reflected in changes in chemical composition of CSDH while SH would require the presence of CSF, these theories could be tested.

Methods

We studied the chemical composition and the presence of CSF in CSDH in 41 patients. The relationship of these components to the appearance of CSDH on CT scans was studied. In this prospective study, 58 samples (41 patients) treated surgically were analyzed. CSDH were evaluated for presence of CSF using beta-2 transferrin (B2T) and substances related to red blood cell hemolysis. They were compared to normal serum values and CT appearance of CSDH.

| Table 1 | Positive β2T | Negative β2T | Unilateral | 4 | 25 | Bilateral | 10 | 19 | Total (%) | 14 (24%) | 44 (76%) | | Presence of Beta-2 Transferrin in CSDH

Results

Twenty four percent of the samples contained B2T. Total protein, LDH, total bilirubin, and RBC count in CSDH were statistically different when compared to normal serum, indicating an active process of rebleeding and hemolysis rather than plasma ultrafiltration. Concentrations did not correlate with specific CT scan appearance.

Conclusions

The absence of CSF in CSDH in 76% of cases suggests most CSDH do not originate from SH. The presence of hemolysis/cell breakdown byproducts in all samples supports the hypothesis that the primary enlargement of CSDH develops through neomembrane and neovascular formation, rebleeding, and inhibition of blood coagulation. Our study confirms that the origin and enlargement of CSDH is multifactorial. CT scan findings do not correlate with the chemical composition or presence of CSF in CSDH.

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