A Standardized Classification for Subdural Hematomas

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Abstract: Subdural hematomas are a frequent and highly heterogeneous traumatic disorder, with significant clinical and socioeconomic consequences. In clinical and medicolegal practice, subdural hematomas are classified according to its apparent age, which significantly influences its intrinsic pathogenic behavior, forensic implications, clinical management, and outcome. Although practical, this empirical classification is somewhat arbitrary and scarcely informative, considering the remarkable heterogeneity of this entity. The current research project aims at implementing a comprehensive multifactorial classification of subdural hematomas, allowing a more standardized and coherent assessment and management of this condition. This new method of classification of subdural hematomas takes into account its intrinsic and extrinsic features, using imaging data and histopathological elements, to provide an easily apprehensible and intuitive nomenclature. The proposed classification unifies and organizes all relevant details concerning subdural hematomas, hopefully improving surgical care and forensic systematization.

Key Words: subdural hematoma, traumatic brain injury, classification, trauma

(*Am J Forensic Med Pathol* 2016;00: 00–00)

Subdural hematomas are one of the most common clinical entities in neurosurgical practice and a frequent consequence of trauma, a major occurrence in forensic medicine. Upon diagnosis, distinct clinical pictures are possible, varying from a potentially lethal acute subdural hemorrhage to the more insidious chronic subdural hematoma (CSDH) or even a rare calcified subtype. After acute traumatic brain injury or other traumatic events (as in falls without direct head trauma), small subdural hemorrhages are frequently found on computerized tomography (CT) scans and, in most cases, do not require any specific treatment. Spontaneous (nontraumatic) subdural bleedings are seldom diagnosed, namely, after aneurysmal hemorrhage[1] (associated with poor outcome), other medical conditions (hematological, infectious diseases, metastatic lymphoid nodule),[2] or even as an iatrogenic event (after lumbar puncture).[3]

The pathophysiology of subdural hemorrhages is fairly described, with its time-dependent heterogeneous nature. Approximately 1% to 6% of untreated acute subdural hematomas experience transformation into CSDHs,[4] on average 3 weeks after initial trauma. However, these 2 entities display significantly different behaviors and dynamics.[5] Initially, blood within the subdural space evokes an inflammatory response. Later, as activated fibroblasts and capillaries proliferate, an organized neomembrane of granulation tissue develops[6,7] and achieves structural integrity within subdural space, while dural adhesions originate internal septations and loculations with fluid levels. These newly formed fragile structures eventually originate new bleeds, resulting in hemosiderin deposits, and allow exudation from outer membranes,[8,9] reinforcing persistent protein-rich chronic SDHs. Rebleeding is not uncommon, namely, in patients sustaining a second injury and/or clotting disorders, culminating in a heterogeneous mixture of fresh blood and partially liquefied hematoma, frequently with a sediment level.

In a period of hours over days or weeks, SDH's intrinsic characteristics may vary due to growth (representing failure of conservative management), chronicization, reabsorption, or rebleeding in chronic or mixed type SDHs.

Computerized tomography scanning remains the most important diagnostic test for this disorder. Imaging appearance of SDHs has been used as sole criteria in different systematizations systems (see Discussion). However, as these proposed classifications lack clinical applicability, the persistent terminology in our daily routine (classical division in chronic, subacute, and acute)[10] remains simplistic, imprecise,[11] and, above all, not effective in providing concise and clear information. Expressions as "chronic with an acute component" are vague, prone to underestimation, and represent an obvious lack of uniformity in nomenclature.[12]

In the subacute phase, typically between 72 hours to 3 weeks after brain injury, SDHs are found to be isodense on CT scan (compared with brain parenchyma). This setting is obviously influenced by patient's hematocrit level, clotting capability, or an eventual rebleeding. Posteriorly, as protein degradation takes place, subdural fluid accumulation commonly evolves to a so-called chronic state, hypodense on CT scan, susceptible of maturing into dural adhesions, septations, and capsule formation. The subacute category is highly subjective, as it includes distinct macroscopic appearances and different definitions of its respective timings, depending on the author (for classic medicolegal studies with histomorphological analysis, see Walter et al[13]).

Current literature includes well-established surgical guidelines for decompression of acute subdural hematomas: subdural hemorrhages with a thickness greater than 10 mm and/or midline shift greater than 5 mm on CT scan should be surgically evacuated regardless of patient's Glasgow Coma Scale score.[14] There is also clinical consensus on draining chronic subdural hemorrhages if symptomatic and/or presenting with thickness greater than 1 cm. The broad consensus regarding these surgical criteria reinforces the feasibility of a suitable classification with well-established pathophysiological parameters and tested clinical correlation. A more standardized approach to SDH's grading should not only be expected but is also accomplishable and necessary.

METHODS

In this proposed classification (Tables 1, 2), the authors use 4 well-known and routinely used parameters; these are as follows:

1. Maximum thickness
2. Midline shift at the level of the foramina of Monro
3. Macroscopic structure
4. Density compared with brain parenchyma.

The origin of initial bleeding is not taken into account. Whether it is due to tearing of bridging veins draining cortical areas directly into dural sinuses, or whether it is originated (less

Manuscript received August 10, 2015; accepted March 25, 2016.
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The authors report no conflict of interest.
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ISSN: 0195-7910/16/0000–0000
DOI: 10.1097/PAF.0000000000000255

TABLE 1. Proposed Subclassification of Subdural Hematomas According to Its Intrinsic Characteristics

Type	Imaging	Setting
I	Hyperdense, relatively homogeneous	Acute setting
II	Isodense, relatively homogeneous	Subacute setting (1–3 wk after TBI)
III	Hypodense, relatively homogeneous	Chronic setting
IV	Isodense to hypodense, relatively heterogeneous	Evidence of recent rebleeding
V	Hypodense in its liquefied component, relatively heterogeneous; internal septations and loculations	Chronic setting
VI	Calcified hyperdense, relatively homogeneous	Chronic setting

TBI indicates traumatic brain injury.

commonly) from other structures (as superficial cortical vessels or lacerated cerebral parenchyma), this distinction is frequently difficult to achieve and of no clear clinical/forensic relevance on itself on most cases.

Based on an easily apprehensible codification, one is able to reach a succinct SDH's description regarding clearly defined subgroups (Table 3), with distinct expected behaviors and severity, medical/surgical management, and prognosis.

Ideally, concomitant relevant details (including side and associated lesions) should be mentioned properly. A good example of this would be "left frontal-temporal-parietal SDH type I.C with 17 mm and associated contusion" or "right frontal SDH type III. B," which will probably be well tolerated in an elderly patient and amenable to conservative treatment (Figs. 1–6).

Types 1, 2, and 3 (Table 1) refer to the most commonly described SDHs with a relatively homogenous appearance—type 1 describes acute subdural hemorrhages soon after traumatic brain injury; type 2 indicates the so-called subacute phase (typically between 3 and 21 days), as the clot ages, protein degradation occurs and the SDH progressively becomes isodense with brain parenchyma on CT scan; type 3 depicts the typical chronic presentation. Types 4 and 5 refer to heterogeneous SDHs: type 4 presupposes evidence of recent rebleeding; type 5 is associated with septations and loculations, implying a bigger probability of recurrence after surgical treatment. Type 6 is reserved for rare cases of SDHs with a significant calcified component.

Systematization depicted in Table 2 is based on SDHs volume/mass effect and its effect on other intracranial contents, indirectly establishing a clinically relevant distinction between possibly symptomatic and nonsymptomatic SDHs. Type A and B SDHs lack significant mass effect, despite subdural hematoma thickness over 1 cm in type B. Type A SDHs most likely won't be of clinical relevance and shouldn't be a candidate for surgery. Both types C and D imply midline shift greater than 5 mm, usually after significant mass effect due to a large hemorrhage (namely, in type C). Subgroup type D (SDH thickness <1 cm, midline shift >5 mm), more rare, is normally associated with subjacent contusion and/or brain edema or even to a smaller hemorrhage in a younger nonatrophied brain. Types C and D are both probable candidates for surgical treatment, as neurological deficits are expected with this degree of brain herniation.

TABLE 2. Proposed Classification of Subdural Hematomas According to Its Extrinsic Characteristics

Type	Thickness	Middle Shift
A	≤1 cm	≤5 mm
B	>1 cm	≤5 mm
C	>1 cm	>5 mm
D	≤1 cm	>5 mm

When 2 different findings are combined, as in SDHs with both septations and rebleeding, the most prominent factor should define the categorization (1 example would be "SDH type V.C with 15 mm and discrete rebleeding").

Although rare, calcified hematomas (subgroup VI) are a known entity,[15] estimated to represent 0.3% to 2.7% of chronic SDHs,[16] and are therefore assigned with a distinct subgroup.

The information contained in this systematic codification should always be integrated with the patient's age, clinical status, and concomitant imaging findings. This classification does not take into account the presence of neurological signs or symptoms, which should obviously be mentioned upon initial patient evaluation and may influence treatment decision regarding surgical versus conservative management.

This classification is intended to be employed on convexity SDHs, the most frequent and likely to require surgical intervention. Interhemispheric SDHs are different regarding location and clinical behavior,[17] rarely evolving into chronicity. Therefore, as midline shift is not observed in this context, it is preferable to simply mention its thickness. Cranial base SDHs or "burst lobe"-related SDHs are also not amenable to classification according to this methodology.

DISCUSSION

In 1974, Wilfrido et al[18] gathered data from 23 SDHs patients and described 4 major discernible patterns (on brain scans plus angiographic studies): curvilinear band, biconvex, diffuse (or hemispheric), and combined (type 1/2 or type 2/3). This classification, based solely on imaging criteria, is of mere historical value nowadays. Alternatively, Yamashima and Yamamoto[19] proposed a classification for CSDHs based on their pathological macroscopic aspect. Subdural hematomas were divided into the following: type I, typical CSDH with visible inner membrane, acting as an expanding lesion; type II, acute subdural hematoma in

TABLE 3. Proposed Combined Classification of Subdural Hematomas—24 Possible Subtypes

	I	II	III	IV	V	VI	
A	I.A	II.A	III.A	IV.A	V.A	VI.A	Probably amenable to conservative treatment
B	I.B	II.B	III.B	IV.B	V.B	VI.B	
C	I.C	II.C	III.C	IV.C	V.C	VI.C	Probable candidates for surgical treatment
D	I.D	II.D	III.D	IV.D	V.D	VI.D	
	Purely Acute setting	Subacute setting	Chronic/mixed setting				

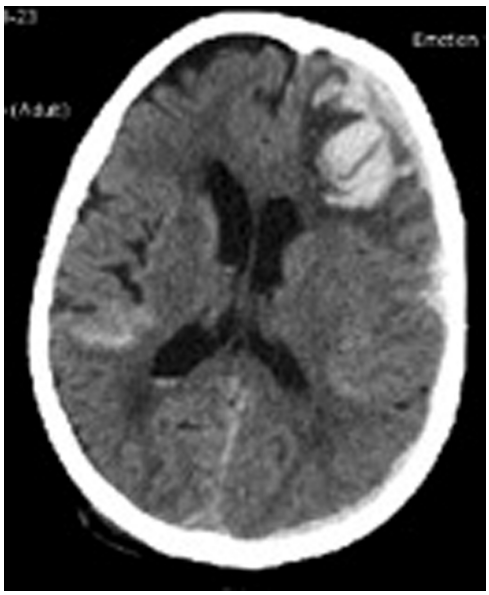

FIGURE 1. Type I.A, left frontal SDH with 5 mm and associated contusion.

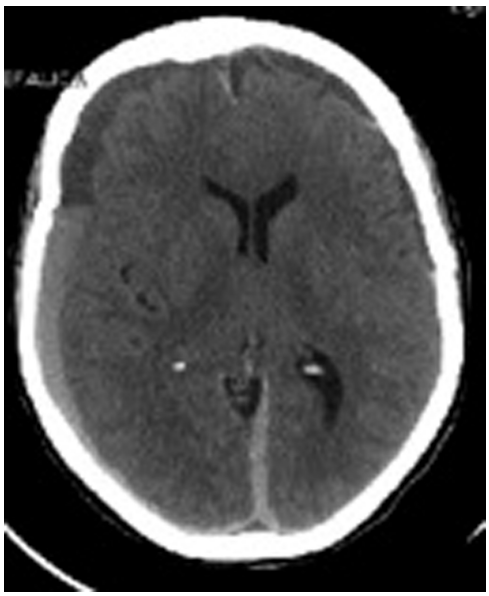

FIGURE 3. Type IV.B, right frontaltemporalparietal SDH and interhemispheric SDH.

chronic healing stage and no visible inner membrane, as a result of previously unrecognized or unoperated acute SDH; and type III, chronic subdural effusion of hemorrhagic type.

Wintzen[20] attempted a more embracing classification on SDHs. His series included 154 cases with and 50 cases without preceding trauma. The traumatic cases were categorized according to duration of symptoms (or its absence). The nontraumatic cases were subdivided in acute and nonacute, the latter (from 24 hours to 6 months after onset of symptoms) presenting with better prognosis. The author concludes that "Combining the chronological criterion with the criteria 'outcome' and 'associated brain injury' did not improve perspectives for a useful classification."[20] Of notice is the fact that this classification, as well many others,[21] does not take into account relevant data from imaging examinations, namely, SDH's extension and thickness.

More recently, Nakaguchi et al[22] classified chronic SDHs into 4 types (homogeneous, laminar, separated, and trabecular type) and correlated them with fluctuations in hematoma contents (white and red blood cells, fibrinogen, and platelets, among others). This classification is therefore solely based on temporal changes in SDH's internal architecture and density on CT scans.[23] Subdural hematomas were also divided in convexity, cranial base, and interhemispheric types, based on intracranial extension. Attempts were made to correlate these data with postoperative recurrence rate. Tosaka et al[24] classified SDHs according to its intraoperative condition regarding craniostomy, with having a

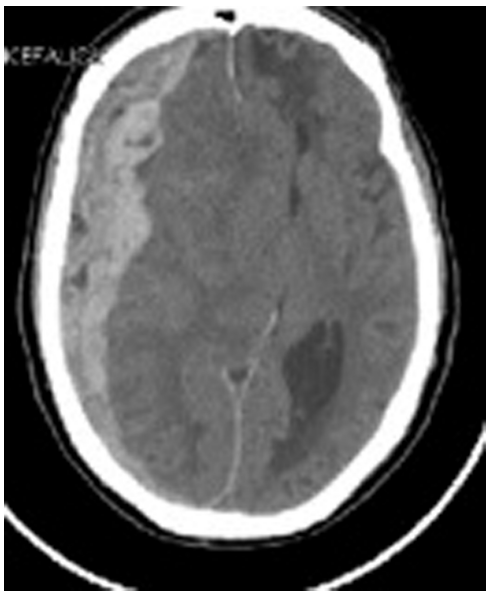

FIGURE 2. Type I.C, right frontotemporoparietal SDH. Emergent surgical decompression is indicated.

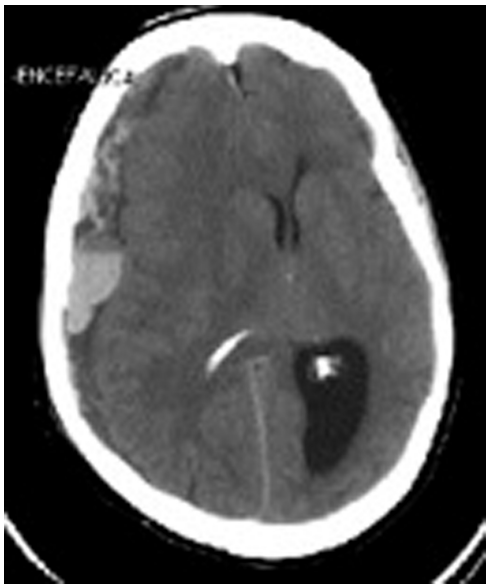

FIGURE 4. Type IV.C, right frontaltemporalparietal SDH—evidence of recent bleeding imposed on a chronic SDH.

"closed" or "open condition," in relation to routes for freely passing of air, saline, or hematoma fluid. Fujisawa et al[25] divided SDH's MRI appearance in low, high, and mixed intensity, isointensity, and layered. Nomura et al[26] and Naganuma et al[27] also elaborated similar morphological classifications for SDHs. Again, although of academic value, these classifications lack a true utility and purpose, failing to provide a practical and effective way to articulate imaging data and relevant clinical criteria.

None of these proposed classifications stood the test of time and clinical practice, as they were unpractical or basically useless, which explains the persistent use of an incomplete classification as in acute, subacute, and chronic. This somewhat practical approach on SDHs has been sufficient so far and one might question the need to address this particular issue. However, from a purely scientific perspective, the use of this empirical classification raises valid questions. First of all, it divides a complex pathology into 3 oversimplified categories. Hematomas with similar time of progression may behave and progress differently to chronicity.[28] Second, these oversimplified categories are ultimately defined by a combination of possibly nonrelated radiological and clinical criteria, including anamnesis subject to flaws and incoherencies. Frequently, the patient cannot identify time of initial traumatic injury, adding the fact that it is not even a necessary precedent. There is also an obvious difficulty and intrinsic ambiguity when describing specific findings, for example, chronic hematoma with acute component opposed to acute hematoma superimposed on a previous fluid accumulation.

The comprehensive classification we propose takes into account specific variables (thickness, midline shift, density on CT scan, macroscopic structure), with obvious clinical relevance and already in use in current daily practice. It does not introduce new concepts or change disease management, it simply groups tested and validated parameters—morphometric, histopathological, imaging (postmortem CT scan is increasingly relevant in forensic medicine)[29,30]—into stratified categories. There is an obvious and intentional correlation between subgroups, probability of causing neurological deficits and criteria for surgical procedures. Case presentation and discussion should become more objective and prognosis may even be amenable to stratification, eliminating most of the subjectivity associated with clinical discussions and forensic conclusions on difficult matters (eg, abusive head trauma).[31,32] On the long term, the physician is able to ascertain patient's progress and SDH's evolution, compare examinations and their reports, as well as verify and compare notes from other admissions or clinical appointments (when frequently the images are not available).

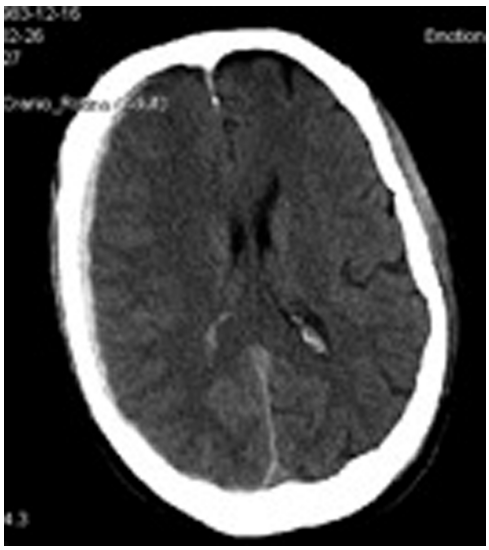

FIGURE 5. Type I.A, right frontaltemporalparietal SDH. No surgical treatment is indicated.

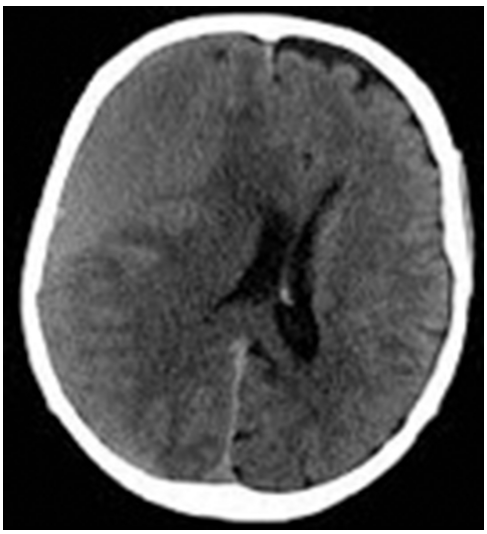

FIGURE 6. Type II.C right frontalparietal SDH.

It also provides a more systematic method for data clustering and outcomes measurement,[33] comparing different procedures and surgical criteria, management and clinical progression (including disability and death), enabling different Departments (namely Forensics and Neurosurgery) to develop databases with categorized and detailed information concerning neurosurgical and medicolegal practice.

At the moment, this new method of SDH's classification is being put to the test in the clinical practice. The authors will publish and discuss their results in the second part of this work, hoping to demonstrate the validity and utility of this new systematic approach.

CONCLUSIONS

Such a heterogeneous entity should be reported with proper categorization, capable of providing a correct evaluation of risk, pathogenesis, management, and respective prognosis, allowing a more accurate patient discussion among clinicians. Concerning medicolegal issues, a systematized and accurate classification will certainly facilitate a thorough evaluation of inflicted lesions, its relationship to final outcome, and all related forensic practice questions.

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