

# Traumatic Low Attenuation Subdural Fluid Collections in Children Younger Than 3 Years

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**Objective:** To determine the time course for the development of posttraumatic nonhemic subdural fluid collections in infants and young children.

**Design:** Retrospective consecutive case series during 16 years.

**Patients:** Fifty-five head trauma patients younger than 3 years with low attenuation subdural fluid on computed tomography.

**Main Outcome Measure:** Time after head trauma when low attenuation fluid first becomes visible.

**Setting:** Regional pediatric medical center.

**Results:** The initial visualization of low attenuation sub-

dural fluid was within 4 days of the trauma for 44 of the patients. The mean  $\pm$  SD size of the subdural fluid collections when first identified was  $4.6 \pm 2.0$  mm (range, 2-12 mm), and the maximum observed size was  $7.7 \pm 3.5$  mm (range, 3-21 mm). The mean  $\pm$  SD time after injury until the maximum observed size was  $16 \pm 18$  days (range, 0-87 days). Low attenuation subdural fluid and high attenuation intracranial hemorrhage coexisted on at least 1 computed tomographic study during the first week after the trauma in 42 (81%) of the 52 patients with hemorrhage.

**Conclusion:** Low attenuation subdural fluid collections (distinct from clotted blood) in infants and young children with head injuries most often develop during the first week after the traumatic event.

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**T**RAUMA-RELATED subdural pathologic abnormalities can include the accumulation of liquid blood, clotted blood, and nonhemic fluid. On computed tomography (CT), nonhemic subdural fluid produces attenuation values that are low in comparison to those of clotted blood or normal brain. The CT demonstration of a low attenuation subdural fluid collection in an infant or toddler is particularly important because it may represent a chronic hematoma, indicating that remote trauma has occurred. Likewise, the identification of acute intracranial hemorrhage in association with a low attenuation subdural fluid collection raises the possibility that more than 1 episode of significant head trauma has occurred; in the appropriate setting, this pattern is pathognomonic of child abuse.<sup>1</sup> However, a low attenuation subdural fluid collection can occur during the acute phase of head trauma, and, when accompanied by areas of hemorrhage, a mistaken diagnosis of an acute and a chronic subdural hematoma may be made. When the clinical history is uncer-

tain, appropriate interpretation of the neuroimaging findings is essential in attempting to estimate the time at which the trauma occurred.

Although trauma-related low attenuation subdural fluid collections are commonly encountered on neuroimaging studies of infants and young children, little specific information is available in the medical literature concerning the time course for their development. Most reports of head injury in infants and young children mention these subdural fluid collections only as indicators of remote trauma, ie, chronic subdural hematomas and hygromas.<sup>2-4</sup> We describe 55 children younger than 3 years with CT examinations demonstrating low attenuation subdural fluid collections after a single traumatic event. The main objective of this retrospective study was to determine the time course for the development of these fluid collections.

## METHODS

The study population was derived from all children younger than 3 years undergoing cranial

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**Table 1. Number of Neuroimaging Studies for the Study Patients**

No. of Studies	No. of Patients
1	2*
2	8
3	9
4	8
5	8
6	5
7	5
≥8	10

\*Died within 24 hours of the injury.

**Table 2. Number of Patients for Time Intervals Between Injury and Formation of Subdural Fluid Collections**

Time Range, d	No. of Patients
<1	19
1	12
1-2	3
1-3	5
1-17	1
1-19	1
1-30	1
1-49	1
1-80	1
2	1
2-3	1
2-4	2
2-8	1
2-9	1
3-6	1
3-14	1
3-38	1
3-47	1
4-9	1

**Table 3. Number of Patients With and Without Subdural Fluid in Grouped Time Ranges\***

Time Range	Yes	Indeterminate	No
<24 h	19 (35)	0	36 (65)
24-28 h	12 (22)	13 (24)	30 (55)
<1 wk	44 (80)	11 (20)	0
<2 wk	48 (87)	7 (13)	0
<3 wk	50 (91)	5 (9)	0

\*Data are given as number (row percentage).

CT examinations at a regional pediatric hospital between January 1, 1986, and December 31, 2001. We reviewed the imaging reports for these children and selected all those demonstrating low attenuation subdural fluid collections. Blinded to the neuroimaging findings, we evaluated the medical records for these patients and excluded those with conditions of medical or surgical origin. We also excluded children for whom the date of the head injury could not be determined. The resulting group of children with trauma-related subdural fluid collections constituted the study population. We determined the most likely date, time, and mechanism of injury from the medical records.

We retrospectively reviewed all available CT scans for the study patients and recorded the size, location, and character (including the attenuation values) of the low attenuation subdural fluid collections, and the type, location, and character of coexisting or preexisting intracranial hemorrhage. To be included in the study population, both of us (pediatric radiologists) had to independently determine that at least 1 of the patient's CT examinations demonstrated fluid that was of lower attenuation than brain in the subdural space. Each of us reviewed the imaging study results blinded to the clinical information and to each other's interpretation. As criteria for a subdural location of the fluid, we included a crescent shape, effacement of adjacent sulci, displacement of cortical veins, and lack of extension into sulci. We used the appearance on sequential CT studies to confirm the diagnosis and reviewed all available magnetic resonance imaging findings.

We assigned each neuroimaging examination a time in days equal to the date of imaging minus the date of injury. We determined the time after the injury when the low attenuation fluid was first visible for each patient and assumed that the lesion could have developed anytime between the day of that neuroimaging study and the day after the previous study that had not shown subdural fluid. For example, if CT scans were available for days 1 and 4 after the injury and the subdural fluid was first visible on the day 4 study, we assigned a time range of origin of 2 to 4 days for that patient. Because the imaging times were not standardized in this retrospective study, the resultant time ranges were not uniform.

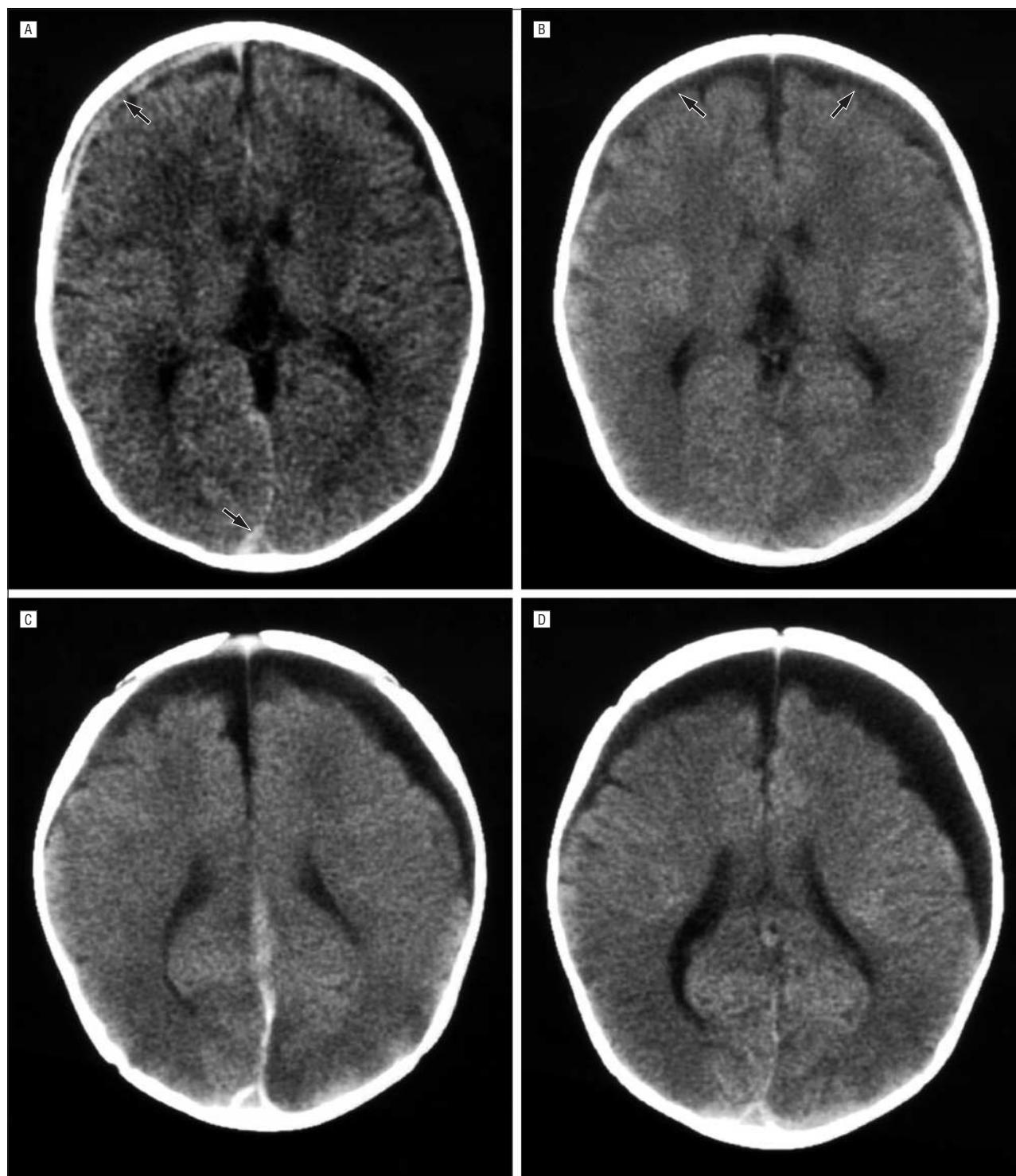
Data are given as mean ± SD.

## RESULTS

Eighty-six patients with trauma-related subdural fluid collections were identified. The date of injury could be determined for 55 (64%) of these children. The resultant study population of 55 children included 33 boys (60%) and 22 girls (40%). The mean age was  $8.4 \pm 13.1$  months (median, 4.0 months). The mechanism of injury was intentional trauma in 41 patients (75%), unintentional trauma in 10 (18%), and of uncertain intent in 4 (7%).

Two hundred sixty-seven neuroimaging examination findings for the 55 children were reviewed. The number of imaging studies available for each of the patients is given in **Table 1**. The time ranges during which the low attenuation subdural fluid collections first became visible on CT are reported in **Table 2** and **Table 3**. The initial visualization of the subdural fluid definitely occurred during the first week in 44 (80%) of the 55 study patients (**Figure**). The remaining 11 patients did not have sufficient imaging during the first week to determine if the origin was within or beyond this period, although all had imaging studies within 3 days of the trauma that showed no subdural fluid. The potential time ranges for the initial subdural fluid accumulation in these 11 children were: 1 to 17, 1 to 19, 1 to 30, 1 to 49, 1 to 80, 2 to 8, 2 to 9, 3 to 14, 3 to 38, 3 to 47, and 4 to 9 days. Therefore, the time of origin could be determined to be less than 2 weeks for 4 of these 11 patients and less than 3 weeks for 6. There were no patients for whom CT showed definite initial appearance of subdural fluid more than 4 days after the trauma.

The mean attenuation of the traumatic low attenuation subdural fluid collections was  $8.8 \pm 5.5$  Hounsfield



Computed tomographic scans of a 6-month-old male child abuse victim, who had been struck in the face and violently shaken by his father. A, Two hours after the injury. The arrows indicate minimal high attenuation clotted blood in the right convexity and posterior interhemispheric subdural spaces. Note the normal generous subarachnoid spaces in the frontal regions, with accompanying normal adjacent sulci. B, Two days after the injury. The arrows indicate small bilateral low attenuation convexity subdural fluid collections. The subdural fluid produces slightly higher attenuation than does the adjacent clear cerebrospinal fluid in the subarachnoid space. C, Six days after the injury, there are larger bilateral subdural fluid collections that are indistinguishable from chronic hygromas. The adjacent sulci are effaced, and cerebral edema is present. There is an increase in high attenuation clotted blood in the posterior interhemispheric fissure. D, Eleven days after the injury, there are increased sizes of the subdural fluid collections, with compression of the adjacent portions of the cerebrum. The posterior interhemispheric subdural hematoma has almost disappeared.

units (range, 0-22 Hounsfield units). The mean size of the fluid collections when first identified was  $4.6 \pm 2.0$  mm (range, 2-12 mm). The mean maximum observed size was  $7.7 \pm 3.5$  mm (range, 3-21 mm). The mean time after in-

jury until the maximum observed size was  $16 \pm 18$  days (range, 0-87 days).

The location of the low attenuation fluid was in the frontal region in all but 1 patient (98%), and parietal in-

involvement was present in 47 (85%). The specific locations were frontal only in 7 patients; frontal and parietal in 33; frontal, parietal, and temporal in 9; frontal, parietal, temporal, and occipital in 3; frontal, parietal, temporal, occipital, and posterior fossa in 2; and posterior fossa only in 1. The fluid collections were unilateral in 9 (16%) and bilateral in 46 (84%). Forty-one (89%) of the bilateral fluid collections were symmetrical (ie, identical locations and maximum thicknesses within 3 mm).

The most common associated hemorrhage was in the subdural space, occurring in 48 (87%) of the patients. Epidural hemorrhage occurred in 3 (6%) of the patients and subarachnoid hemorrhage in 1 (2%). Three patients (6%) had no visible intracranial hemorrhage. In 27 (52%) of the 52 patients with intracranial hemorrhage, the clotted blood and the subdural fluid were noted to be in separate locations (eg, epidural vs subdural spaces, or posterior interhemispheric subdural space vs frontal convexity subdural space). In 4 (8%) of the 52 patients, the clotted blood and the subdural fluid were mixed together. In 21 (40%) of the 52, the subdural fluid and clotted blood were adjacent to each other, but did not appear mixed; the pattern of hemorrhage in these patients was linear or laminated in 11 (52%), patchy in 6 (29%), and focal in 4 (19%). Low attenuation subdural fluid and high attenuation intracranial hemorrhage coexisted on at least 1 CT study during the first week after the trauma in 42 (81%) of the 52 patients with hemorrhage (Figure 1C). Only 1 patient showed conversion of a low or mixed attenuation subdural fluid collection on the initial CT study to a dense uniform clot on subsequent imaging. At the time of first visualization of the low attenuation fluid, the ventricles and cerebral sulci were prominent in 2 patients, small in 33 patients, and normal in 19 patients; prominent ventricles and small sulci were present in 1 patient.

Eight (15%) of the 55 study patients died within 30 days of the injury. Imaging follow-up of the remaining patients showed that subdural fluid collections persisted for at least 1 month in 10 children (18% of the 55 patients), spontaneous resolution occurred in 14 (25%), and subdural shunts were placed in 8 (15%). Imaging follow-up was inadequate to determine the outcome beyond 30 days in 15 patients (27%). The mean time between injury and the last imaging study for those patients with persistent fluid collections was  $89 \pm 66$  days. Imaging signs of atrophy (ventriculomegaly and prominent sulci) developed subsequent to visualization of the low attenuation subdural fluid in 16 (48%) of the 33 patients for whom follow-up imaging of at least 2 weeks after the injury was available. Twelve of these 16 patients had imaging studies that allowed evaluation beyond 30 days, with persistent subdural fluid in 4, spontaneous resolution in 2, and subdural shunt placement in 6.

#### COMMENT

Our findings indicate that traumatic low attenuation subdural fluid collections in children younger than 3 years most often develop within 1 week of the traumatic event. In fact, imaging studies in our 55 patients failed to dem-

onstrate any instances of definite initial appearance of low attenuation subdural fluid beyond a 4-day posttrauma interval.

The major practical implication of our findings relates to timing of the trauma for medicolegal purposes. Our data indicate not only that low attenuation subdural fluid collections can be identified within hours or days after the injury but also that combinations of clotted intracranial blood and subdural fluid can occur after a single traumatic insult, potentially mimicking an acute and a chronic subdural hematoma. Forty-two of our study patients had at least 1 CT examination on which high attenuation intracranial hemorrhage and low attenuation subdural fluid coexisted. This does not mean that hemorrhage into a preexisting nonhemic subdural fluid collection does not occur or that a chronic subdural fluid collection is not an important marker for child abuse. Many of the 31 patients excluded from the study population because of lack of information concerning the date of injury had large nonhemic subdural fluid collections that were likely chronic. The identification of membranes and a large size may help to distinguish these chronic lesions from acute fluid collections. Signs of brain atrophy, when present, also suggest a remote insult.

Many health care professionals caring for infants and young children consider the imaging demonstration of trauma-related low attenuation subdural fluid to indicate that several weeks have passed since the injury. This concept has largely developed from observations of adults with large acute subdural hematomas that progress to chronic subdural hematomas. A true chronic subdural hematoma shows a temporal evolution in attenuation values on CT.<sup>5</sup> During the "subacute" phase of the injury, clot lysis causes the high attenuation of the hematoma to slowly decrease until it is isodense to brain after 2 to 3 weeks. Eventually, the attenuation values fall until they are equal to, or slightly higher than, those of clear cerebrospinal fluid. Oozing of blood or serum from the neovascular membrane of a subacute or chronic hematoma may alter the attenuation values.

Several observations in our patients suggest that clot lysis is an uncommon mechanism for the formation of nonhemic subdural fluid in infants and young children, or that it occurs as part of a multifactorial process. These observations include the appearance of post-traumatic low attenuation subdural fluid much more quickly than should occur with clot lysis (days rather than weeks in at least 44 of the 55 patients), the occurrence in patients without intracranial hemorrhage (3 patients), and the occurrence in anatomic locations remote to the hemorrhage (27 patients). Other potential pathogenic mechanisms for posttraumatic low attenuation subdural fluid include a hygroma due to a tear in the arachnoid membrane,<sup>6</sup> an effusion from traumatized meninges,<sup>7,8</sup> and a hyperacute hematoma with fresh unclotted blood or areas of unretracted semiliquid clot.<sup>9,10</sup> A hyperacute hematoma pattern, with sequential CT examinations showing conversion of liquid extravasated blood to a hyperdense clot, was demonstrated in only 1 of our patients.

We did not find atrophy to be an important cause for the initial formation of the subdural fluid collections



### What This Study Adds

The presence of low attenuation subdural fluid on computed tomography in infants and young children who have head injuries is an important marker for prior trauma and raises the suspicion for child abuse. However, there is sparse information in the medical literature concerning the time course for the development of these fluid collections in children of this age. Our findings indicate that the initial appearance of low attenuation subdural fluid most often occurs within a few days of the traumatic event. A small amount of low attenuation subdural fluid accompanied by high attenuation intracranial hemorrhage is not pathognomonic of acute and remote trauma.

in our patients. Most of the study patients showed accumulation of low attenuation subdural fluid earlier after the trauma than brain volume loss due to atrophy would be expected. Also, the first visualization of low attenuation subdural fluid coincided with signs of brain swelling or edema (small ventricles and compressed sulci) in 60% (33/55) of the patients. Atrophy may play a role in the persistence of a nonhemic subdural fluid collection. Signs of atrophy eventually developed in 16 (48%) of the 33 study patients for whom follow-up imaging was available; only 2 of the 12 patients with atrophy who underwent imaging at least 30 days after the injury had spontaneous resolution of the low attenuation subdural fluid.

There is sparse information in the medical literature concerning the time course for the development of traumatic low attenuation subdural fluid collections. Most of the available studies evaluate adult patients. Interpretation of published studies is complicated by a lack of uniformity in the use of crucial terminology, such as *hygroma*, *traumatic effusion*, and *chronic subdural hematoma*.

Lee et al<sup>11</sup> performed a review of serial CT scans in 58 patients, including 10 children younger than 10 years with "traumatic subdural hygroma" and found the mean time at diagnosis to be 11.6 days after the injury. Fobben et al<sup>12</sup> described the magnetic resonance imaging detection of traumatic subdural hygromas in 2 children. The lesions were identified 3 days after the injury in a 7-week-old infant and 15 days after the injury in a 4-year-old child. French et al<sup>13</sup> noted subdural hygromas in 6.6% of 196 head injury patients who had follow-up CT images available for review; the hygromas were detected a mean of 22 days (range, 6-46 days) after the trauma. Hwang and Kim<sup>14</sup> described the development of hygromas within 2 weeks of the injury in 3 infants who had acute subdural hematomas. Dias et al<sup>15</sup> detected low attenuation subdural fluid collections at 3, 7, 11, and 12 days after presentation in 4 infants with acute subdural hemorrhage. There are reports of hyperacute subdural hematomas in infants, with low attenuation areas due to unclotted blood occurring in association with subdural hemorrhage seen on CT performed soon after the trauma.<sup>10,16</sup>

In the imaging evaluation of an infant with suspected abnormal low attenuation extra-axial fluid, it is

essential to differentiate between fluid in the subarachnoid and subdural spaces, because the former is usually of little significance and the latter indicates important pathologic abnormalities. The subarachnoid spaces in normal infants are frequently slightly prominent, and greater prominence occurs with the self-limited entity of "benign extra-axial fluid collections of infancy." If the CT signs used in distinguishing between fluid in the subdural vs subarachnoid spaces are inconclusive (see the "Methods" section), further evaluation with magnetic resonance imaging is mandatory.

Several deficiencies of our study deserve comment. The lack of uniformity in imaging times after the traumatic events in this retrospective case series resulted in varying ranges for the observed intervals between the injury and the initial appearance of subdural fluid. In a few of the patients, the lack of sufficient imaging studies precluded distinction between an acute hygroma or effusion and a true chronic subdural hematoma. None of the study patients had surgical or autopsy confirmation of the imaging findings during the acute posttraumatic period.

An additional deficiency is that the determination of the date and time of the head injury often relied on subjective information provided by caretakers. In 38 of our patients, the absence of preexisting low attenuation subdural fluid was confirmed on the first CT examination. The 17 patients who demonstrated low attenuation subdural fluid on the first CT examination present a particular problem in avoiding the mistaken inclusion of children with preexisting abnormalities. However, 7 of these patients had well-documented accidental injuries, including 6 who were involved in motor vehicle crashes. Formal multidisciplinary investigations of the circumstances of the injury were carried out for many of the study subjects, including all of the children with a subsequent determination of child abuse. Several children suspected of being abused were excluded from the study population because of unreliable clinical histories; those who were included had information that allowed estimation of the time of injury with a reasonable degree of certainty, based on a confession by a perpetrator or reports from a witness to the event.

### CONCLUSIONS

Our findings indicate that most trauma-related low attenuation subdural fluid collections in children younger than 3 years develop within the first week after the head injury. Coexistent clotted subdural blood is often present during the acute phase of the injury, and the imaging appearance may mimic that of acute hemorrhage into a chronic subdural fluid collection. Therefore, although low attenuation subdural fluid collections in association with acute intracranial hemorrhage may represent multiple traumatic events, they are not diagnostic. The pathogenesis of these fluid collections is likely multifactorial.

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