Acute-on-Chronic Subdural Hematoma: Not Uncommon Events

Kyeong-Seok Lee, M.D., Jae-Jun Shim, M.D., Seok-Man Yoon, M.D., Jae-Won Doh, M.D., Il-Gyu Yun, M.D., Hack-Gun Bae, M.D.
Department of Neurosurgery, Soonchunhyang University Cheonan Hospital, Cheonan, Korea

Objective: Patients with asymptomatic chronic subdural hematoma (SDH) are prone to fall or slip. Acute trauma on these patients may develop acute subdural bleeding over the chronic SDH. We recently experienced 9 patients with acute-on-chronic SDH. We report the clinical and radiological features of this lesion.

Methods: We retrospectively examined the computed tomographic (CT) scans of 107 consecutive patients who diagnosed as chronic SDH from January 2008 to December 2010. All cases of CSDH were diagnosed on CT with or without MRI scan.

Results: Acute-on-chronic SDH is not rare, being 8% of chronic SDH. The most common cause of trauma was a slip in drunken state. Alcoholism with multiple episodes of trauma was one of the prominent histories. Acute-on-chronic SDH appeared as a hyperdense layer of clot with irregular blurred margin or lumps in liquefied hematoma. Single or two burr holes was usually effective to remove the hematoma.

Conclusion: Repeated trauma may cause acute bleeding over the chronic SDH. It will be helpful to understand the role of repeated trauma as a mechanism of hematoma enlargement.

Key Words: Chronic subdural hematoma · Computed tomography · Craniocerebral trauma · Diagnosis.
**Table 1. Clinical features of patients with acute-on-chronic subdural hematoma**

| No. | Sex | Age | GCS | Cause | Symptom                  | Past History                               | PT(INR) | Anticoagulants |
|-----|-----|-----|-----|-------|--------------------------|---------------------------------------------|---------|---------------|
| 1   | F   | 66  | 9   | ?     | Discovered on the road   | Craniotomy 10 yr pta                        | 1.12    | No            |
| 2   | M   | 48  | 3   | Slip  | Discovered on the road   | HI 10 D, 1 M pta; warfarin for atrial fibrillation | 2.44    | Aspirin, warfarin |
| 3   | M   | 52  | 10  | ?     | Discovered on the road   | Traffic accident 15 yr pta                  | 0.94    | No            |
| 4   | M   | 52  | 15  | ?     | Headache for 9d          | Aspirin                                    | 1.01    | Aspirin       |
| 5   | M   | 63  | 15  | Slip  | Hemiparesis for 3d       | Alcoholic LC; slip 2 M pta                  | 0.95    | No            |
| 6   | M   | 65  | 15  | Slip  | Hemiparesis for 3d       | Craniotomy 12 yr pta                        | 0.97    | No            |
| 7   | M   | 69  | 15  | Slip  | Hemiparesis for 1d       | Slip 3 M pta                               | 0.99    | No            |
| 8   | M   | 80  | 11  | ?     | Hemiparesis for 4d       | Diabetes, COPD, alcoholic LC                | 1.12    | Aspirin       |
| 9   | M   | 83  | 15  | Slip  | Hemiparesis for 2w       | Stomach CA 7 yr pta; femur fracture 5 yr pta| 1.06    | No            |

GCS : preoperative Glasgow Coma Score, pta : prior to admission, HI : head injury, LC : liver cirrhosis, COPD : chronic obstructive pulmonary disease, CA : cancer, INR : international normalized ration

Three patients received anticoagulation therapy. However, results of the laboratory coagulation test was marginally abnormal in six patients on admission except case 2.

**Radiological features**

The chronic SDH were unilateral in eight cases and bilateral in one case. The acute bleeding was usually hyperdense clot with irregular blurred margin (Fig. 1, 2). It was often lumps in liquefied hematoma as in case 5 and 6. The degree of midline shift was usually more than 8 mm corresponding to the thick hematomas (Table 2). In case 2, the CT scan of the brain revealed a large SDH with three different features in the right hemisphere (Fig. 3). He used warfarin due to atrial fibrillation for 2 years. Detailed history taking revealed three episodes of head injuries; in-car accident on 49 days prior to admission (PTA), falling on 9 days PTA, and slipping down on the day of admission with corresponding subdural lesions. We presumed the layered oval hematoma resulted from the in-car accident, hypodense crescentric hematoma was developed by the falling, and the hyperdense hematoma was made by the slipping.

**Treatment and outcome**

Although the hematomas were a mixture of semisolid clot and liquefied hematoma, we could remove the hematoma by single or two burr holes in seven patients. We did not try to remove the clot vigorously (Table 2). We removed some clot with gentle irrigation and suction. On the immediate postoperative CT scans, we could find remained subdural hematomas (Fig. 4).

We placed a soft silicon drain in all cases. Semisolid clot was usually drained out or resolved within several days. In case 4, we had to perform craniotomy to remove the clot under the neomembrane. In case 7, we used an endoscopy to suck out the semisolid clot around the corner of the hematoma cavity. In case 2, his relatives refused surgical treatment. All except one patients were improved after surgery. One patient died of cardiac failure on the third hospital day. One another patient died of metastasis from the gall bladder cancer, within about 3 months after the operation.

---

**Table 2. Radiologic features of patients with acute-on-chronic subdural hematoma**

| No. | Side | Type                | Density* | Size (mm) | Shift (mm) |
|-----|------|---------------------|----------|-----------|------------|
| 1   | Left | Two layered         | 67       | 20.5      | 8.8        |
| 2   | Right| Mixed pattern       | 70       | 28.1      | 26.5       |
| 3   | Right| Hyperdense thick layer | 70       | 24.6      | 20.7       |
| 4   | Left | Homogeneous hyperdensity | 53       | 16.4      | 13.0       |
| 5   | Left | Lumps in low density | 55       | 24.8      | 10.3       |
| 6   | Right| Lumps in isodensity | 47       | 20.2      | 13.0       |
| 7   | Left | Hyperdense layer    | 60       | 19.9      | 14.9       |
| 8   | Right| Scattered lumps in low density | 51   | 20.2      | 8.8        |
| 9   | Bilateral | Hyperdense thin layer | 65   | 22.0+11.8 | 9.6        |

*Hounsfield Unit. Size : maximum thickness of hematoma, Shift : degree of midline shift

---

**Table 3. Treatment and outcome of patients with acute-on-chronic subdural hematoma**

| No. | Surgery     | Operative findings | Outcome               |
|-----|-------------|--------------------|-----------------------|
| 1   | Burr hole   | Liquefied hematoma & clot | Died of GB CA 3 M later |
| 2   | Refused     | Not operated       | Died of HI on HD 5    |
| 3   | Burr hole   | Liquefied hematoma & clot | Recovered             |
| 4   | Craniotomy  | Membrane within hematoma, liquefied hematoma | Recovered             |
| 5   | Burr hole   | Liquefied hematoma & clot | Recovered             |
| 6   | Burr hole   | Liquefied hematoma & clot | Recovered             |
| 7   | BH & endoscope | Membrane within hematoma, liquefied hematoma & clot | Recovered             |
| 8   | Burr hole   | Liquefied hematoma & clot | Died of MI on HD 3    |
| 9   | Burr hole   | Liquefied hematoma & clot | Recovered             |

BH : burr hole, GB : gall bladder, CA : cancer, HD : hospital day, MI : myocardial infarction
DISCUSSION

Acute-on-chronic SDH is not rare. In this study, we found 8% of chronic SDH were actually acute-on-chronic SDHs. There are a few cases designated as acute-on-chronic SDH in the literature. Curiously, it is hard to find any comments on the acute-on-chronic SDH, even in clinical series reporting more than a thousand cases of chronic SDH. Age and sex distribution were identical with the typical chronic SDH. Alcoholism with multiple episodes of trauma was one of the prominent features. Patients with chronic SDH are prone to the trauma since they are usually aged and enjoy drinking. The bridge veins in the potential subdural space are thin-walled, and might be under significant tension by the hematoma. Since the chronic SDH usually show excessive activation of both the coagulation and fibrinolytic systems, acute bleeding into the hematoma cavity may not make a solid clot.

Although there was an acute subdural bleeding, the patients visited our hospital several days after the trauma. The reason was that the bleeder was usually venous or capillary, either from bridging veins or fragile new vessels in the neomembrane. The outer membrane contains many fragile sinusoidal vessels that are often the source of repeated multifocal bleeding. Like the repeated hemorrhages from the outer membrane, repeated trauma may cause acute bleeding over the chronic SDH as a mechanism of hematoma enlargement. Even though the chronic SDH continue to enlarge, brain atrophy in the elderly may allow the hematoma to accumulate before symptoms become obvious. The patients become symptomatic after compression of the pyramidal tract with significant midline shift. Bleeding from the bridging veins may produce clot, while bleeding from the neomembrane may diffuse into the pre-existing hematoma cavity. Diffusion without clot formation may produce homogeneous hyperdense chronic SDH, as in case 4.

CT remains the preferred diagnostic procedure for chronic SDH. Acute SDH is usually hyperdense in the CT, whereas chronic one is iso- or hypodense. Rebleeding into a chronic SDH with admixture of fresh blood and lucent fluid can lead to isodensity. Insidious repeated microhemorrhage from the neo-
Acute-on-Chronic Subdural Hematoma

A. S. Lee, T. J. Kim, M. J. Kim, J. C. Park, K. H. Park, J. H. Park, J. H. Park, J. S. Park, S. K. Park, J. H. Shin, M. S. Kim, and Y. W. Park

Acute-on-chronic subdural hematoma (SDH) is not rare, being 8% of chronic SDHs. Wilensky et al. (1955) suggested that acute-on-chronic SDH is not rare, being 8% of chronic SDHs. Acute-on-chronic SDHs may be difficult to diagnose, as the presentation and clinical signs may be similar to those of chronic SDH. However, acute-on-chronic SDHs have a more insidious presentation compared to the hyperacute one (20). In case 2, we presumed the layered oval hematoma result from the in-car accident, and the hyperdense crescentic hematoma was developed by the fall in, and the hyperdense hematoma was made by the slipping.

Warfarin or other oral anticoagulants may increase intracranial hemorrhagic complications (16, 20). The risk of SDH with anticoagulation therapy is 4- to 15-fold (20). However, anticoagulation therapy is usually safe with international normalized ration (INR) from 2.0 to 3.0, since the incidence of hemorrhagic complications increase with INR >4.8 (16).

Even though the hematomas had a solid clot, removal of the liquefied hematoma only by a burr hole was possible to relieve the displacement. Endoscopy may be helpful to remove a semisolid clot clearly with INR >4.8. In case 4, there is a thin acute hematoma after a craniotomy. In case 7, we could remove the semisolid clot clearly with endoscopy.

**CONCLUSION**

Acute-on-chronic SDH is not rare, being 8% of chronic SDHs. Repeated trauma may cause acute bleeding over the chronic SDH. It will be helpful to understand the role of repeated trauma as a mechanism of hematoma enlargement.

**References**

1. Chen JC, Levy ML: Causes, epidemiology, and risk factors of chronic subdural hematoma. Neurosurg Clin N Am 11: 399-406, 2000

2. Fernando S, Obalado RE, Walsh IR, Lowe LH: Neuroimaging of nonaccidental head trauma: pitfalls and controversies. Pediatr Radiol 38: 827-838, 2008

3. Gelabert-González M, Iglesias-Pais M, García-Allut A, Martínez-Rumbo R: Chronic subdural haematoma: surgical treatment and outcome in 1000 cases. Clin Neurol Neurosurg 107: 223-229, 2005

4. Haines DE, Harkey HL, al-Mefty O: The “subdural” space: a new look at an outdated concept. Neurosurgery 32: 111-120, 1993

5. Kloss BT, Lagace RE: Acute-on-chronic subdural hematoma. Int J Emerg Med 3: 511-512, 2010

6. Lee KS: Natural history of chronic subdural hematoma. Brain Inj 18: 351-358, 2004

7. Lee KS, Bae WK, Bae HG, Doh JW, Yun IG: The computed tomographic attenuation and the age of subdural hematomas. J Korean Med Sci 12: 353-359, 1997

8. Lasique JC, Monsalve G: Suspended brain in a degraded bottom: bilateral subdural chronic hematomas with acute rebleeding. J Trauma 69: 240, 2010

9. Miele VJ, Carson L, Carr A, Bailes JE: Acute on chronic subdural hematoma in a female boxer: a case report. Med Sci Sports Exerc 36: 1852-1855, 2004

10. Park SH, Kang DH, Park J, Hwang JH, Hwang SK, Sung JK, et al.: Fibrinogen and D-dimer analysis of chronic subdural hematomas and computed tomography findings: a prospective study. Clin Neurol Neurosurg 113: 272-276, 2011

11. Ramachandran R, Hegde T: Chronic subdural hematomas—causes of morbidity and mortality. Surg Neurol 67: 367-372; discussion 372-373, 2007

12. Sambasivan M: An overview of chronic subdural hematoma: experience with 2300 cases. Surg Neurol 47: 418-422, 1997

13. Sargent S, Kennedy JG, Kaplan JA: “Hyperacute” subdural hematoma: CT mimic of recurrent episodes of bleeding in the setting of child abuse. J Forensic Sci 41: 314-316, 1996

14. Sato S, Suzuki J: Ultrastructural observations of the capsule of chronic subdural hematoma in various clinical stages. J Neurosurg 43: 569-578, 1975

15. Stein PD, Grandison D, Hua TA, Sletetlahguh PM, Henry JW, Turlapaty
16. Takahashi N, Kimura H, Kitai R, Sato M, Yoneda M, Yamamoto C, et al.: Acute on chronic subdural hematoma as a rare complication in a microscopic polyangiitis patient receiving antithrombotic treatment. Clin Nephrol 72: 211-215, 2009
17. Tan ST, Hung CT: Acute-on-chronic subdural hematoma: a rare complication after spinal anaesthesia. Hong Kong Med J 9: 384-386, 2003
18. Tsai FY, Huprich JE, Segall HD, Teal JS: The contrast-enhanced CT scan in the diagnosis of isodense subdural hematoma. J Neurosurg 50: 64-69, 1979
19. Wilms G, Marchal G, Geusens E, Raaijmakers C, Van Calenbergh F, Goffin J, et al.: Isodense subdural haematomas on CT: MRI findings. Neuroradiology 34: 497-499, 1992
20. Wintzen AR, Tijssen JG: Subdural hematoma and oral anticoagulant therapy. Arch Neurol 39: 69-72, 1982