Mixed-Density Subdural Hematoma on CT: Case Report and Review of Subdural Hematoma Classification

BBT'de Mikst Dansite Subdural Hematom: Olgu Sunumu Ve Subdural Hematom Sınıflamasının İrdelenmesi

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Abstract: *Objective:* Predicting the age of subdural hematomas on computed tomography (CT) scans is particularly significant for forensic reasons, and for understanding the lesion's natural history, pathogenesis, and morphology. A case of mixed-density subdural hematoma on CT is discussed, and the classification of subdural hematomas based on hematoma density, morphology, and histopathology is reviewed.

Methods: A 19-year-old male was admitted to the Department of Infectious Diseases in our hospital 20 days after he had sustained head trauma. The presenting symptoms were headache, fever, vomiting, and intermittent agitation, and he was tentatively diagnosed with meningitis. Cranial CT revealed a left frontotemporoparietal subacute hypo-isodense (mixed-density) subdural hematoma. Burr-hole drainage on the same day revealed no neomembranes, and follow-up CT on the fifth day of hospitalization confirmed complete removal of the hematoma.

Results: The lesion was diagnosed as a Yamashima-Type 2 isodense subdural hematoma, and septation, organization, and retraction were identified as the main steps in the pathological process. The mixed density was not considered to reflect a rebleeding-resolution process.

Conclusion: The exact age of subdural hematomas can only be determined by histopathological investigation. Distinct pathogenetic mechanisms occur in the different morphologic types, and these are reflected in the microscopic findings.

Key words: Attenuation coefficient, head injury, hematoma age, hematoma classification, hematoma density, subdural hematoma

Özet: *Amaç.* BT de subdural hematom yaşını tahmin etmek adli açıdan ve doğal gelişimini, patogenez ve morfolojisini anlamada önemlidir. Burada bir mikst dansite subdural hematom olgusu tartışılmakta ve subdural hematom sınıflaması, hematom dansitesi, morfoloji ve histopatolojisine dayanarak irdelenmektedir.

Metodlar: 19 yaşındaki erkek hasta maruz kaldığı kafa travmasından 20 gün sonra hastanemizin Enfeksiyon Kliniği'ne yatırıldı. Geldiğinde başağrısı, ateş, kusma, aralıklı ajitasyon bulguları nedeniyle menenjit öntanısı aldı. Kranial tomografide sol frontotemporalde subakut hipo-izodens (mikst dansite) subdural hematom tespit edildi. Aynı gün yapılan burr-hole drenaj esnasında neomembran izlenmedi. 5. günde kontrol tomografisinde hematomun tamamen boşaldığı gözlendi.

Bulgular: Bu lezyon, patolojik proses olarak "retraksiyon, organizasyon ve septasyon"un rol oynadığı Yamashima-Tip 2 isodense subdural hematom olarak değerlendirildi. Mikst dansitenin "rebleeding-rezolüsyon" işlevine bağlı gelişmediği düşünüldü.

Sonuç: Subdural hematomların gerçek yaşı sadece histopatolojik inceleme ile bulunur. Farklı morfolojik tiplerde, patogenezde farklı mekanizmalar yer alır ve mikroskobik bulgulara yansır.

Anahtar kelimeler: Atenüasyon katsayısı, kafa travması, hematom dansitesi, hematom yaşı, hematom sınıflaması, subdural hematom

INTRODUCTION

Subdural hematomas (SDHs) are classified as hyperdense, isodense, and hypodense according to their density (attenuation coefficient) on computed tomography (CT) scans. Until relatively recently, hematoma age was predicted based on lesion density. Knowing the age of these lesions is particularly significant not only for forensic reasons, but also for understanding the natural history, pathogenesis, and morphology of the SDH. However, it is not always easy to make this estimation. The previous CT characterization of acute SDHs as hyperdense; subacute SDHs as isodense; and chronic SDHs as hypodense is no longer valid. Furthermore, the precise definition of "chronicity" in these cases remains obscure. Munro (29) applied the term "chronic" to cases that showed no evidence of fresh or unhealed brain injury; McKissok (27) defined chronicity as SDHs that were present at 20 days after trauma; and Fogelholm (7) defined well-formed membranes of the hematoma as the criterion for chronicity. Thus, the designation of chronicity covers a range of different pathologic processes.

The exact age of subdural hematomas can be determined by morphology on histopathological examination (26). The histological nature of the outer subdural neomembrane, the features of the hematoma contents, and the presence of an inner neomembrane are all used to classify these lesions (19,39,44). Distinct pathogenetic mechanisms occur in the different morphologic types, and these are reflected in the microscopic findings. However, when burr-hole drainage of a hematoma is performed the specimen is usually insufficient for histological study, and when the history is inconsistent the only evidence for age is lesion density on CT. SDH density on CT images may be misleading regarding age in some circumstances, and is not necessarily related to prognosis (42).

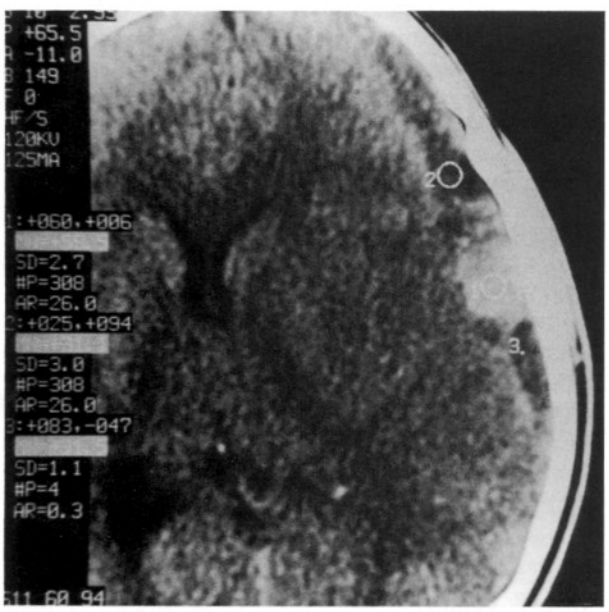


Figure 1: Non-contrast CT showing a mixed-density isodense SDH on the left side, with density ranging from 31.5-59.5 H.

CASE REPORT

A 19-year-old male who had been beaten 20 days earlier presented to the Department of Infectious Diseases. He had developed headache, fever, vomiting, and intermittent agitation, and had been tentatively diagnosed with meningitis. Cranial CT revealed a left frontotemporoparietal subacute hypo-isodense (mixed-density) SDH, (Figures 1-5) and the patient was transferred to our clinic on 1 September 2000. The patient's neurological examination was normal, and the hematoma was removed the same day by two-burr hole drainage. No outer or inner neomembranes were observed during the procedure. The hematoma was relatively fresh and dense. It was possible to aspirate all the

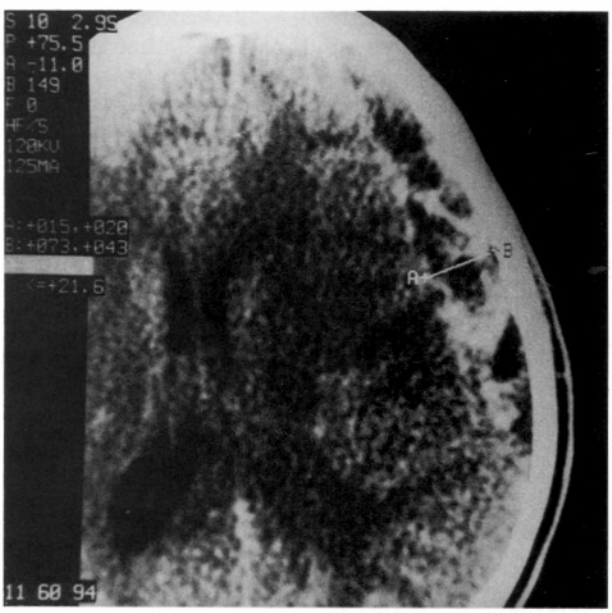


Figure 2: Non-contrast CT reveals a septated, organized, 18.1 mm-thick hematoma.

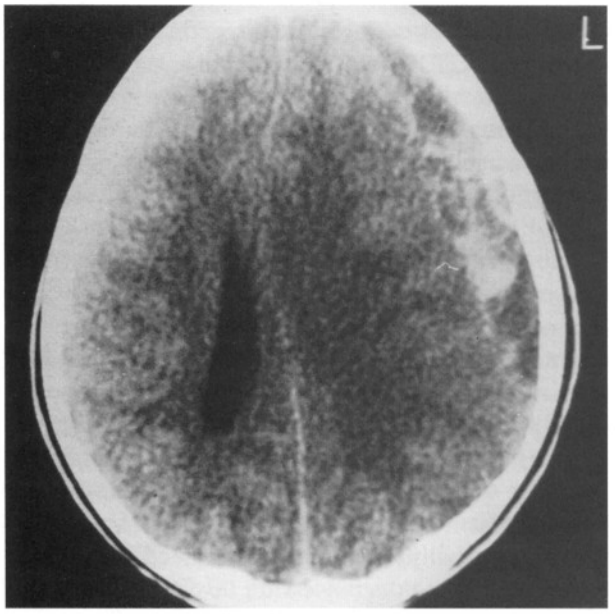
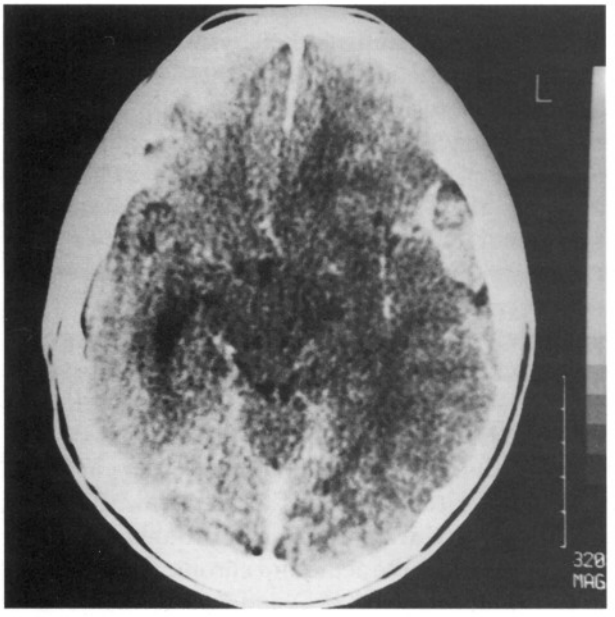


Figure 3: Non-contrast CT shows the mixed-isodense, septated hematoma causing a midline shift to the right.

DISCUSSION

In 1977, Scotti et al. (36) evaluated the attenuation coefficients of various SDHs on CT. Their report stated that acute SDHs (<7 days) are 100% hyperdense, subacute SDHs (7-22 days) are 70% isodense, and chronic SDHs (>22 days) are 76% hypodense. Likewise, Bergstroem et al. (2) reported that density of extra-axial hematomas tends to decrease at a predictable rate over time, and that subdural hematomas reach isodensity within 2 weeks to 1 month. Some authors have consistently reported that isodensity is more common in subacute SDHs than in the other age categories (14,36). In other words, numerous studies have indicated that SDH density decreases gradually, with hyperdensity in the acute phase, isodensity in the subacute phase, and hypodensity in the chronic phase.

In contrast, Lipper and Kishore (24) claimed that classification of SDHs as "acute" or "chronic" cannot be established solely on CT features. Also, many



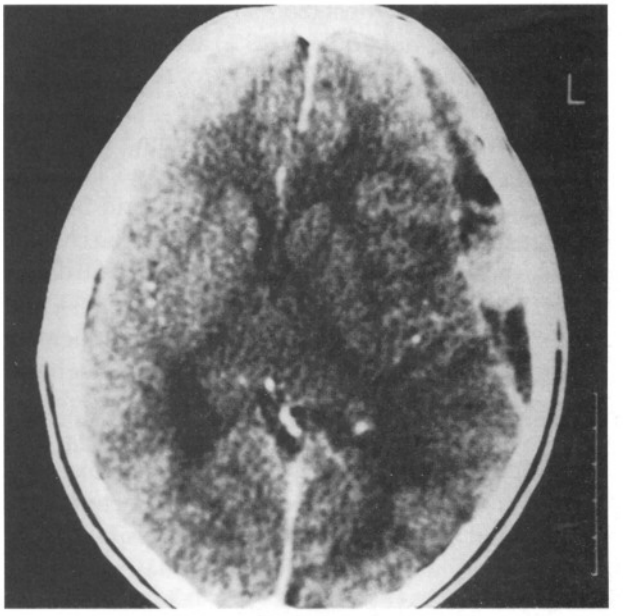


Figure 4, 5: CT scans with contrast show the septation and organization of the hematoma clearly.

material without having to perform a craniotomy. Follow-up CT on 5 September 2000 confirmed complete removal of the hematoma (Figure 6). The patient did well postoperatively. No hematological disorders were identified before or after the operation.

reports have supported their claim that isodensity is more common in chronic than in subacute cases (3,4,8,21,40). In 1997, Lee (22) reviewed 446 cases and found that 98.6% of the acute SDHs were hyperdense. However, regarding the subacute SDHs, 45.7% were hypodense, 42.9% were isodense, and 11.4% were

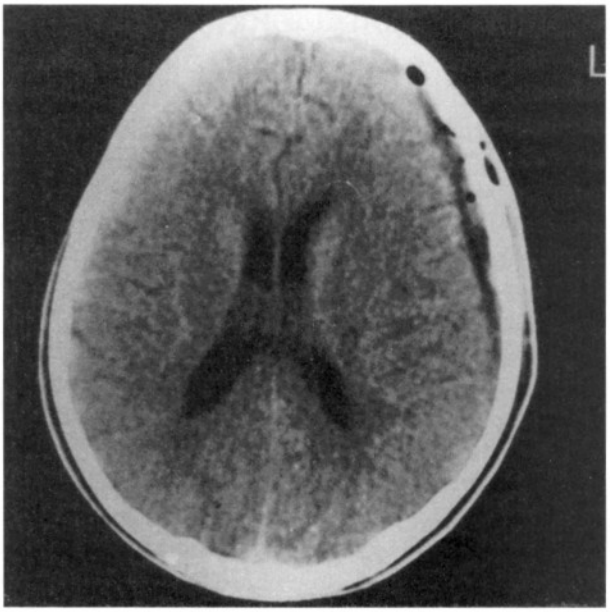


Figure 6: The postoperative CT scan confirmed total removal of the SDH.

hyperdense on CT. Most (86.7%) of the chronic SDHs were isodense, but some (13.3%) were hypodense. Overall, the author noted that 64% of the hypodense SDHs were subacute, and 73.2% of the isodense SDHs were chronic. Interval studies confirmed the mean ages of the hyperdense, hypodense, and isodense SDHs as 0.5±1.6 days, 20.9±20.7 days, and 54.9±44 days, respectively. The mean densities of the chronic SDHs were 38±6.9 Hounsfield units (H) (20-30 days), 43.8±12.8 H (31-60 days), 51.8±5.1 H (1-90 days), and 44.2±8.3 H (>90days). The data showed that density gradually increases up to 90 days and then begins to decrease.

These changes in density result from rebleeding and resolution processes, which are characteristic of "traditional" (Yamashima-Type 1) SDHs (44). SDH hyperdensity usually decreases gradually (2,24,36), but there are exceptions (12,17,30,34,38) because brain tissue is rich in thromboplastin (1). After trauma, active fibrinolytic systems in the cerebrospinal fluid (11,18,31,32) accelerate the resolution process so that it occurs faster than in epidural hematomas. The outer neomembrane is usually established in the first week, and the inner neomembrane in the third week after lesion formation (19,43). Neocapillaries that originate from the outer membrane (19) at 2-4 weeks are fragile. This fragility leads to repeated microhemorrhaging (15,25), which is reflected as a gradual increase in density on CT. The outer membrane is a site of erythrocyte production. (5,6,37). The greater the surface area and thickness of the outer membrane, the more neovascularization and fibrinolytic activity occurs, leading to more rebleeding (5,25,28). This probably explains why hematomas with thick membranes more often persist or recur (28). If the rate of rebleeding exceeds that of absorption, the chronic SDH will expand over time (25). This is in contrast to Gardner (9) and Gudeman's (13) "osmotic pressure" theory, and the "effusion" theory proposed by Giltin (10), Rabe (33), and Sato (35). Both these theories were put forward to explain SDH expansion. As the neomembrane matures and the neovasculature stabilizes, the microhemorrhaging decreases, resolution overcomes rebleeding, and CT density begins to decrease after 90 days.

It is noteworthy that isodensity observed for up to 90 days is not always homogeneous, and changes occur due to the rebleeding process. In 1979, Tsai (41) classified "isodensity" as homogeneous (43.9%), mixed (35.2%), and layering types (15.5%). Lee (22) supported Tsai's study with his own interval studies that revealed the mean ages of these distinct SDHs as 54.6±33.1 days, 59.5±57.7 days, and 42.8±37.8 days, respectively. According to Tsai, rebleeding, resolution, and local differentiation of fibrinolytic activity all play roles in the pathogenesis of traditional Yamashima-Type 1 isodense SDHs. In rare cases, homogeneous lesions may transform to the layering type if the patient is in recumbent position for an extended period (16). In this position, the gravitational separation of blood components (16,41) leads to layering-type isodensity, which is characteristic of Yamashima-Type 3 isodense SDHs.

As our case exemplifies, isodense SDHs of mixed density are sometimes encountered in the interval between the acute and chronic phases. These are Yamashima-Type 2 isodense SDHs (44). According to Bergstroem (2), this type is formed by septation of the hematoma cavity, fibrous organization, and retraction of the hematoma. This was the likely pathogenesis of our patient's lesion, since we found no neomembrane as a potential source of rebleeding. The resolving SDH exhibits non-homogeneous attenuation between 4 and 22 days due to intermingled, irregularly contoured areas with different proportions of high- (35 H) or low attenuation (15 H). Mixed density reflects blood clot

retraction, rather than local attenuation differences of non-coagulated venous blood (2). Attenuation is defined by the hemoglobin protein fraction, and the only cases in which retracted clot attenuation may be altered are those in which hemoglobin levels are low (30). Our patient had normal hemoglobin levels.

The existence of membranes determines the histopathological classification and chronicity of these lesions. In Yamashima-Type I SDH, blood degradation products induce the inflammatory process (20), which, in turn, forms the outer membrane responsible for repeated microhemorrhaging. The outer membrane is clearly separated from the hematoma, with a maximum thickness of 500-800 µm. This membrane is responsible for the expansion of the hematoma, but plays no role in organization. Any local hyperfibrinolysis (15) will aggravate the rebleeding process. The inner membrane develops at a later stage. The time to hematoma formation is approximately 71 days. Trauma is a factor in 82% of SDHs with inner membranes, but coexisting skull fracture is uncommon (2%). Yamashima-Type I SHDs show homogeneous or mixed isodensity on CT.

Yamashima-Type 2 SDHs are seen at the beginning of the late stage of acute SDH, and have no outer or inner membrane. Instead, a thick layer of granulation tissue forms and becomes firmly attached to the hematoma. This firm fibrous tissue is almost devoid of neocapillaries. Fibroblast columns form within it, and these invade and gradually replace the hematoma. Septation then occurs and hematoma organization begins at the outermost part of the lesion. Any neocapillaries that form are secondary. The time to formation of this type of SDH is 22 days. Trauma is a factor in 88% of cases, and fracture in 71% (44). These lesions show mixed isodensity on CT.

Yamashima-Type 3 SDHs (5%) are the layering type of isodense lesion (16). Trauma is an issue in only 50% of cases, but coexisting fracture is present in 25% (44). The interval to formation of this type of hematoma is 33 days. These lesions have a very thin outer membrane and no inner membrane. They resemble Type 1 SDHs but contain no macrocapillaries or inflammatory cells. It is the atypical form of chronic subdural effusion mixed with recent hemorrhage (23,31) that usually tends to undergo sedimentation. If fresh blood and cerebrospinal fluid leak into the subdural space through a small tear in the arachnoid membrane (40), the SDH transforms to a Type 1 and an inner membrane eventually forms. Sometimes surgical trauma facilitates this transformation (44).

In summary, the existence of an inner membrane determines the chronicity of the SDH (that is, identifies it clearly as a Type 1 lesion). If this membrane is not present, then the lesion is either a Type 2 or Type 3 SDH. Microscopic study of the neomembrane and the hematoma contents determines the exact age of the lesion. The mixed-density SDH in our case was assessed as a Type 2 lesion. Retraction, septation, and organization were the main pathologic processes thought to be occurring, in contrast to the rebleeding-resolution process characteristic of Type 1. The combination of the absence of both membranes, the short time interval of 22 days, and the history of trauma confirmed the diagnosis.

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Factors in the natural history of chronic subdural hematomas that influence their postoperative recurrence.

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Based on the internal architecture and density of each hematoma, the CSDHs were classified into four types, including homogeneous, laminar, separated, and trabecular tyes. The recurrence rate associated with the separated type was high, whereas that associated with the trabecular type was low. Chronic subdural hematomas are believed to develop initially as the homogeneous type, after which they sometimes progress to the laminar type. A mature CSDH is represented by the separated stage and the hematoma eventually passes through the trabecular stage during absorption. Based on the intracranial extension of each hematoma, CSDHs were classified into three types, including convexity, cranial base, and interhemispheric types. The recurrence rate of cranial base CSDHs was high and that of convexity CSDHs was slow.