The Computed Tomographic Attenuation and the Age of Subdural Hematomas

The sequential change in density (attenuation coefficient) of subdural hematomas (SDHs) in computed tomography (CT) is important in understanding the pathogenesis and evolution of SDHs. We retrospectively investigated the age of SDHs by CT in 446 cases. We included 30 cases of chronic SDHs, in whom the density was directly measured in the CT. The density of acute (within 7 days) SDH was hyperdense in 98.6%, isodense in 1.1%, and hypodense in 0.3% of the cases. In subacute (8-22days) SDHs, it was hypodense in 45.7%, isodense in 42.9%, and hyperdense in 11.4%. In chronic (over 22 days) SDHs, 86.7% was isodense and only 13.3% was hypodense. In hypodense SDHs, 64.0% was the subacute, and 73.2% of the isodense SDHs was the chronic one. The mean interval from injury to CT was 0.5±1.6 days in hyperdense SDHs, 20.9±20.7 days in hypodense SDHs, and 54.9±44.0 days in isodense SDHs. In 30 cases of chronic SDH, the average density was 38.0±6.9 Hounsfield number(H) in 20~30 days, 43.8±12.8 H in 31~60 days, 51.8±5.1 H in 61~90 days, and 44.2±8.3 H in over 90 days. The density of acute SDH is usually hyperdense. It becomes hypodense within 3 weeks. Then the density progressively increases by the repeated microhemorrhage, which is the mechanism of enlargement of chronic SDH. The density of chronic SDH increases with time up to 90 days, then decreases again after maturation of the neomembrane, which is the mechanism of spontaneous resolution.
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Key Words : *Subdural hematoma, Attenuation coefficient, Computed tomography, Head injury*

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INTRODUCTION

In 1977, Scotti et al. reported that the density (attenuation coefficient) of subdural hematomas (SDHs) in computed tomography (CT) was hyperdense in 100% of acute (within 7 days) patients, isodense in 70% of subacute (8~22 days) group, and hypodense in 76% of the chronic (over 22 days) group (1). Bergstroem et al. observed that the density of extra-axial hematomas decreased at a predictable rate with time, and isodensity would be reached between 2 weeks and 1 month after bleeding in a subdural collection (2). Some reported that the isodensity was common in the subacute SDHs (1, 3). From this, it seems to be popularly understood that the density of SDH will decrease with time, i.e., hyperdense in acute, isodense in subacute, and hypodense in chronic SDHs.

However, Lipper and Kishore said that absolute reliance on classification of hematomas as acute or chronic based on CT density can lead to inaccuracies (4). There are several reports that the isodensity was more common in the chronic SDHs than subacute SDHs (5~8). Recently, we also found that most of the isodense SDH were chronic rather than subacute (9).

The relationship between the density and the age (time interval from the head injury to the CT) of SDH is important to understand the natural history and pathogenesis of SDHs. The age of subdural hematoma is also important to assume the time of injury, especially in forensic cases (10). We investigated the age of SDH by CT scan.

MATERIALS AND METHODS

We re-evaluated 446 CT scans from 429 patients with SDH during a seven-year-period, from 1988 to 1994. We had treated 497 patients with SDH during study period. We excluded 68 cases, because they could not remember the trauma at all or there was no history of trauma. In 14 patients, we performed 17 follow-up CT

scans before surgery, and we included those CT scans. In addition, we included 18 patients, in whom the date of the injury was not accurate, but clearly more than one month. The age of SDH was within 7 days in 351 cases (78.7%), 8~22 days in 35 cases (7.8%), over 22 days in 60 cases (13.5%). Since Scotti et al. started to investigate the relationship between the density and age of SDH (1), we divided these 446 cases into acute (within 7 days), subacute (8~22 days), and chronic (over 22 days) groups as their division.

The density of SDH was divided into hyperdense (Group I), isodense (Group II), and hypodense (Group III) groups. Isodense hematomas were subclassified into homogeneous (IIa), mixed (IIb), and layering (IIc) types.

In thirty cases of chronic SDH, we collected data prospectively from January 1993 to December 1994. We treated 68 patients with chronic SDH during those period. In 30 patients (44.1%), we were able to identify the date and the cause of head injury by detailed history taking. The cause of head injury was road traffic accident in 18 (60%), industrial accident in 4 (13.3%), and falling or slipping in 8 (26.7%) patients. The density of chronic SDH was measured directly in the CT scan. In three cases of mixed density and four cases of layering type, the density was calculated by averaging 2 to 4 attenuation coefficients.

RESULTS

1. Densities of the Subdural Hematomas

The density of acute SDH was hyperdense in 98.6%, isodense in 1.1%, and hypodense in 0.3% (Table 1). In the subacute group, it was hypodense in 45.7%, isodense in 42.9%, and hyperdense in 11.4%. In chronic SDHs, 86.7% were isodense and only 13.3% were hypodense. Of the hyperdense SDHs, 98.9% were acute, and 64.0% of hypodense SDHs were subacute. Of the isodense SDHs, 73.2% were chronic and 21.1% were subacute (Fig. 1).

Table 1. Distribution of density in CT according to the interval

Interval*	Group I	Group II				Group III
		IIa	IIb	IIc	IIt	
0- 7	346	0	2	2	4	1
8-22	4	3	11	0	14	17
> 22	0	32	12	9	53	7
Total	350	35	25	11	71	25

* in days ; I=hyperdense ; II=isodense ; IIa=homogeneous ; IIb=mixed ; IIc=layering ; IIt=isodense in total ; III=hypodense

We calculated the mean age of SDH from the 428 cases in whom the date of the injury was accurate. It was 0.5±1.6 days in the hyperdense SDHs (Group I), 20.9±20.7 days in the hypodense SDHs (Group III), and 54.9±44.0 days in the isodense SDHs (Group II) (Table 2). These differences in interval were statistically significant ($P<0.01$).

In isodense SDHs, 49.3% were homogeneous, 35.2% were mixed, and 15.5% were the layering type. The mean interval from injury to CT was 54.6±33.1 days in the homogeneous, 59.5±57.7 days in mixed, and 42.8±37.8 days in layering isodense SDHs (Table 2). These differences between subtypes were statistically not significant ($P>0.2$).

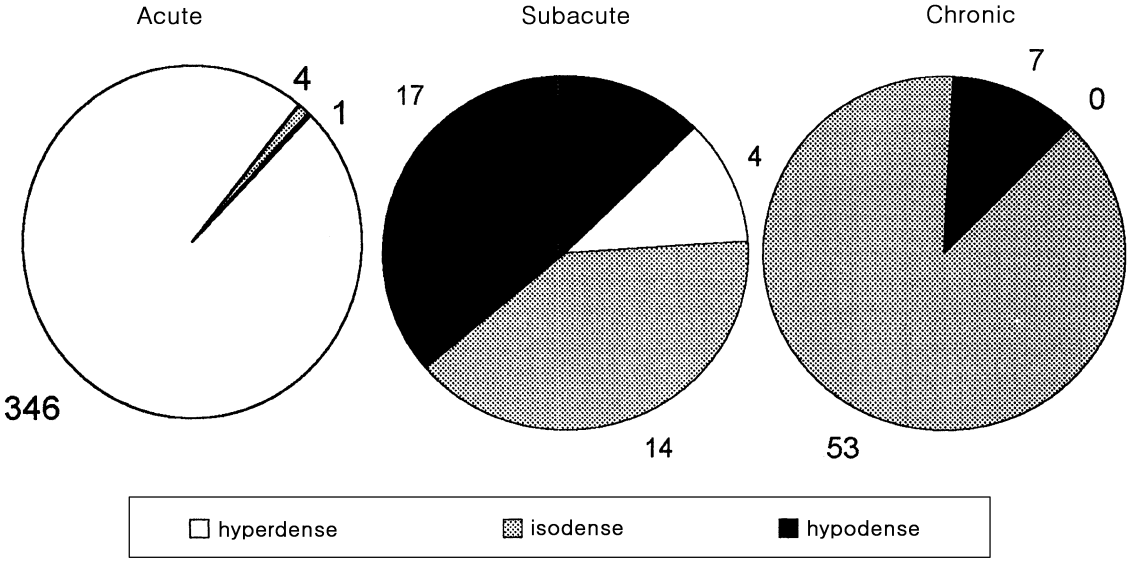

Fig. 1. Percentage ratio of acute, subacute, and chronic subdural hematomas in each density.

2. Densities of the Prospectively Collected Chronic Subdural Hematomas

In prospectively collected thirty cases of chronic SDH, the age of chronic SDH was 20~30 days in 5, 31~60 days in 10, 61~90 days in 9, and over 90 days in 6 (range from 20 to 121 days). The average density was 38.0±6.9 Hounsfield number(H) in 20~30 days, 43.8 ±12.8 H in 31~60 days, 51.8±5.1 H in 61~90 days, and 44.2±8.3 in over 90 days (Table 3).

3. Density Change in Conservatively Managed Acute Subdural Hematomas.

In 14 cases, we obtained 31 CT scans before surgery. The interval from the injury to CT was within 3 days in 17 scans. The density was hyperdense in all these 17 scans. The interval was 4~7 days in 5 scans. The density was mixed-type isodense in 4 (Fig. 2, 3), and hypodense in one. In 7 scans of 8~22 days interval, the density was hypodense in 5 scans, mixed-type isodense in one, and homogeneous isodense in one (Fig. 4). The density was hypodense in 2 scans of over 22 days interval (Table 4).

DISCUSSION

Most literature concerned with the density of SDH in CT were reported in the 1970s. Before thorough investigation on the density and time interval, isodense SDHs

Table 2. Density and mean interval from injury to CT

Density	N	Mean Interval*	SD	Range
Group I	350	0.5	1.6	0- 12
Group IIa	26	54.6	33.1	11-140
IIb	19	59.5	57.7	4-216
IIc	10	42.8	37.8	6-115
IIt	56	54.9	44.0	4-216
Group III	23	20.9	20.7	4- 81

*in days ; SD=standard deviation ; I=hyperdense ; II=isodense ; IIa=homogeneous ; IIb=mixed ; IIc=layering ; IIt=isodense in total ; III=hypodense (I : III ; P<0.05, IIt : III ; P<0.01)

Table 3. Mean density of prospectively collected 30 cases of CSDH

Interval(days)	N	Hounsfield No.	SD
20-30	5	38.0	6.9
31-60	10	43.8	12.8
61-90	9	51.8	5.1
over 90	6	44.2	8.3

Table 4. Density change in conservatively managed acute subdural hematomas

Interval(days)	N	hyperdense	isodense	hypodense
0 - 3	17	17	0	0
4 - 7	5	0	4	1
8 - 22	7	0	2	5
over 22	2	0	0	2

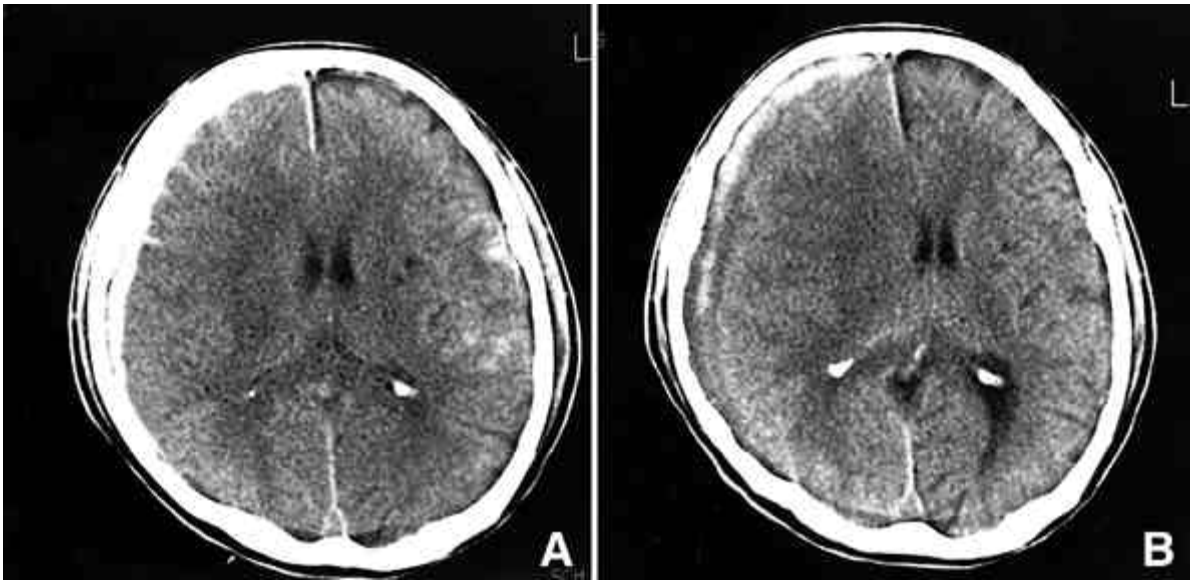

Fig. 2. Mixed-type isodensity was observed in a 53-year-old man on the 8th day after the injury (B). Peripheral part of the hematoma was hypodense, while the central part was hyperdense. The density was hyperdense on the date of the injury (A). He underwent surgical evacuation on the 10th day after the injury.

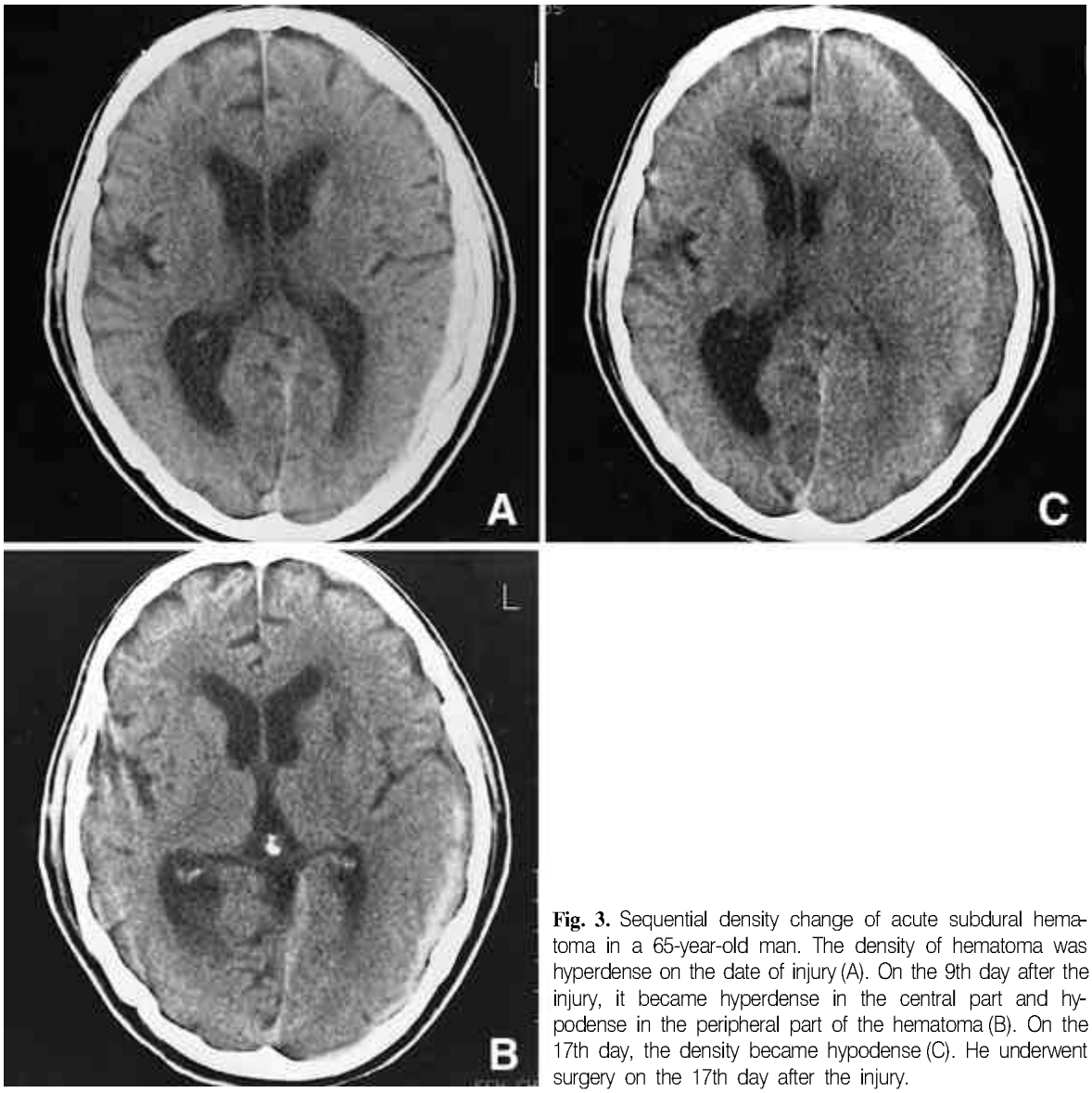

Fig. 3. Sequential density change of acute subdural hematoma in a 65-year-old man. The density of hematoma was hyperdense on the date of injury (A). On the 9th day after the injury, it became hyperdense in the central part and hypodense in the peripheral part of the hematoma (B). On the 17th day, the density became hypodense (C). He underwent surgery on the 17th day after the injury.

became the subject of interest. Then, most literature dealt with the diagnostic methods and correct interpretation of isodense SDHs. With advances in CT technology and knowledge, the diagnosis of isodense SDHs became easier to treat. After this, there were few reports on the density of SDH and time interval in CT.

In this study, hypodensity was more common in the subacute group. Not only the distribution, but also the mean age of isodense SDHs was longer than that of hypodense SDHs. These results could have been expected due to the following reasons.

Presumptive Sequential Change of Density in Subdural Hematomas

The pathogenic mechanism of enlargement of chronic SDHs was a matter of debate (11~19). Various hypotheses, such as osmotic pressure (13, 15) or effusion (14, 18, 20), were assumed as a mechanism. In 1981, Markwalder denied such theories and concluded that repeated microhemorrhages were responsible for the enlargement of chronic SDHs (17). According to this theory, we presumed the sequential change of density in SDHs as Fig. 5.

The density of acute SDH is usually hyperdense (1, 2, 4, 11, 12), except for a few unusual situations (21~25). Thereafter the density of extra-axial hematomas will decrease with time (2). Because the brain is a very concentrated source of tissue thromboplastin (26), cerebrospinal fluid contains very active fibrinolytic systems after trauma (27~30). So, resolution is more rapid in SDHs than in epidural hematomas. Most of the acute

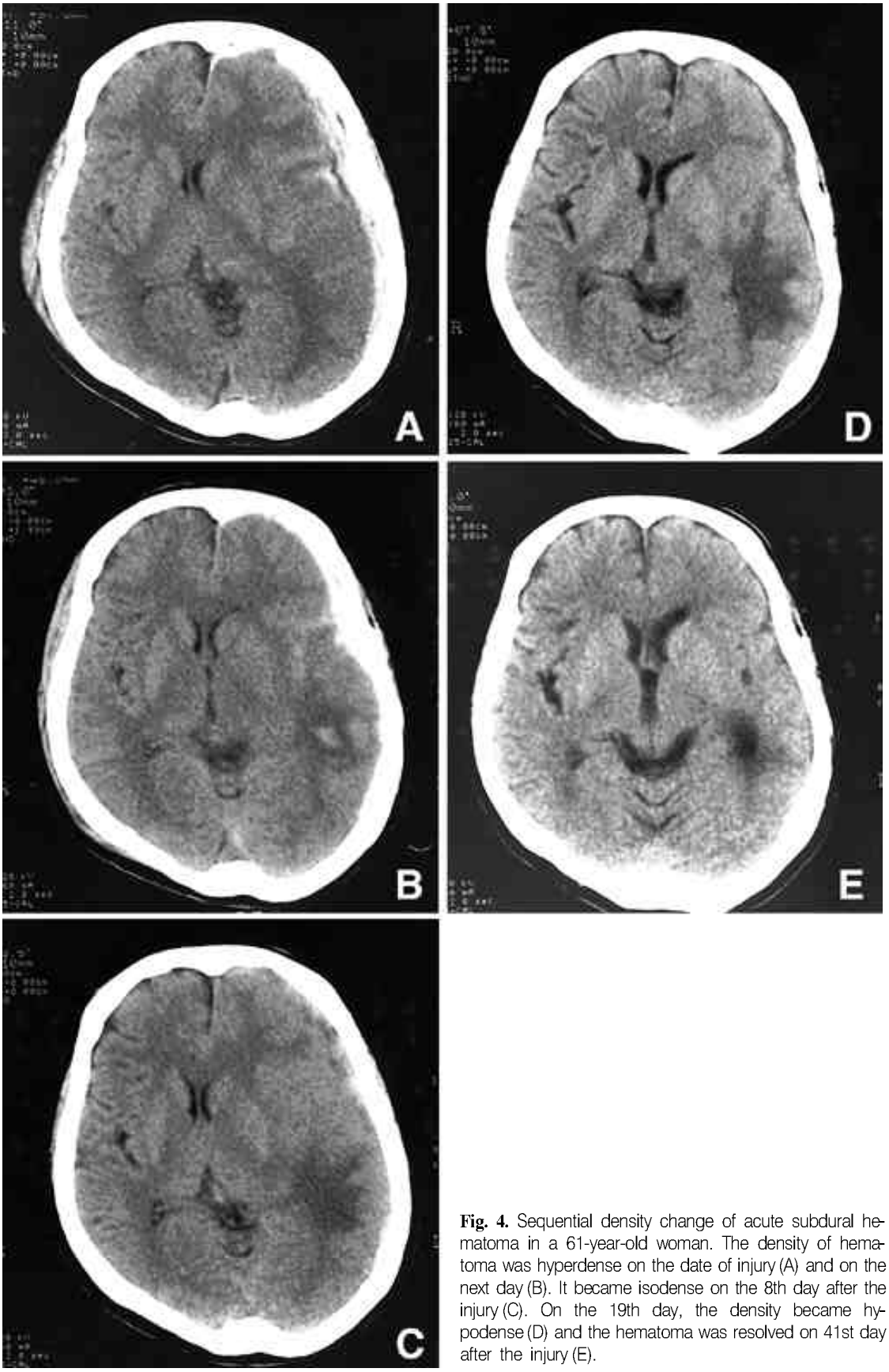

Fig. 4. Sequential density change of acute subdural hematoma in a 61-year-old woman. The density of hematoma was hyperdense on the date of injury (A) and on the next day (B). It became isodense on the 8th day after the injury (C). On the 19th day, the density became hypodense (D) and the hematoma was resolved on 41st day after the injury (E).

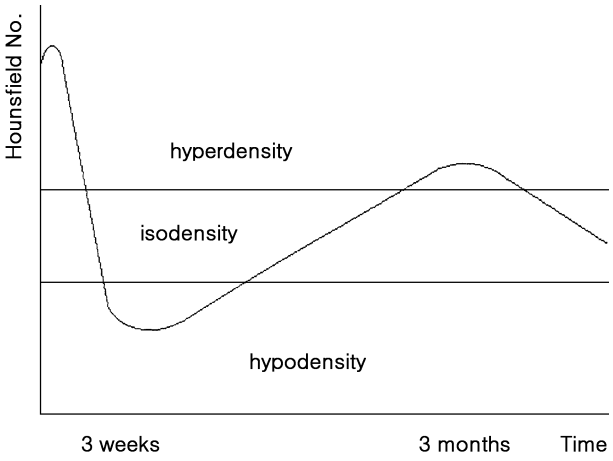

Fig. 5. Presumed sequential change of density and its mechanisms of subdural hematomas in computed tomography.

SDH resolved into hypodensity within 3 weeks. On occasion, a mixed type of isodense SDH can be found during this transitional period. The density becomes hypodense from the peripheral part, while it is still hyperdense in the central part. This change is just same as in the case of intracerebral hemorrhage. In 14 cases of acute SDH, we were able to obtain sequential CT scans by conservative management. The density of all these acute SDHs became hypodense within 3 weeks. In 4 patients, mixed type of isodense SDH was found between 1 to 3 weeks after the injury. Outer membrane of the SDH is formed within 1 week, and thin inner membrane and thick outer membrane will be formed within 3 weeks (10). Two to four weeks after the injury, neocapillaries are developed (10). During this period, SDH would be hypodense. Then, repeated microhemorrhages will follow from the fragile neocapillaries in the outer membrane (16, 17). The density of the SDH will increase again slowly and continuously. The wider the surface area of outer membrane, the more extensive the neovascularity, and the higher the fibrinolytic activity within the hematoma, the more blood will be mixed. The density value of the subdural accumulation becomes isodense with time, and often over 46 Houndsfield number. Therefore, we can expect that the older the age of SDH, the higher the density.

The average density of chronic SDH was decreased again after 90 days. This decrease might be related to the maturation of the neomembrane and stabilization of the neovasculature. Chronic SDH will enlarge when repeated microhemorrhage exceeds absorption. When the neomembrane is matured, the neocapillary is no longer fragile. Maturation of the neomembrane and stabilization of the neovasculature would result in progressive decrease of the microhemorrhage and the density. This mechanism is responsible for the spontaneous resolution of chronic SDH.

In 1979, Tsai et al. classified the isodensity into homogeneous, mixed, and layering types (31). They proposed resolution, rebleeding and separation of components as corresponding pathogenic mechanisms of isodense SDH. They thought resolution of acute SDH might be the most common origin of isodense SDH, although they suggested rebleeding probably played a more important role than was previously suspected. Although the three types of isodense SDH clearly differs in morphology, each type of isodense SDH is not different in the interval. Local difference in fibrinolytic activity, septation of hematoma cavity and fresh hemorrhage may produce mixed type. Gravitational separation of the blood component will produce layering type (31, 32). Homogeneous type might become layering type, when the patients lie down in a brow-up position for a long time (32).

It is incorrect that the density of SDH is hyperdense in acute, isodense in subacute, and hypodense in chronic cases. The age of SDH can be assumed by histopathological examinations of the subdural membranes (10). However, for chronic SDHs, twisted drill hole drainage or burr hole drainage is the most common mode of treatment (11, 12), by which it is hard to obtain a specimen for pathologic examinations. In such cases, density of the hematoma in the CT scan would be a unique clue for assuming the time of injury except history. Correct knowledge on the density of the SDH in CT scan will be important in assuming the time of injury.

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