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Original Article

Computed tomography findings in multiple simultaneous intracerebral hemorrhages

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Abstract: Objective: In the present study, computed tomography (CT) findings of Multiple Simultaneous Intracerebral Hemorrhages (MSICHs) are presented. Methods: Of 139 patients who had cranial CT in March 2010-June 2015 period and who had been found to have spontaneous ICH, 20 patients (14 males and 6 females) with MSICHs were included in the present study at the Gaziosmanpaşa University School of Medicine Research and Training Hospital. All patients in this study were subjected to 5 mm, non-contrast CT examination on admission. Number, localization and volume of hematomas were studied in CT examinations. Results: Age of the patients with MSICH ranged from 1 to 83 (mean 59.26±18.94). MSICH incidence in all ICHs was 14.4%. Seven patients had primary MSICHs due to hypertension or amyloid angiopathy, while 13 patients had secondary MSICHs. A total of 84 hematomas were detected in all patients, and 70 of them (83.3%) were of lobar, 7 (8.3%) deep, 4 (4.8%) of cerebellar and 3 (3.6%) of brain stem location. Hematoma volumes varied from 0.01 to 111.15 ml (median 0.50 ml). No statistical difference was observed between the volumes of primary and secondary MSICHs (P=0.32). Conclusion: An increase has been observed in MSICH detection incidence by practicing thin-slice cranial CT examinations. Number and sizes of MSICHs could vary independent of etiological factors.

Keywords: Computed tomography, hemorrhagic metastasis, hypertension, intracerebral hemorrhage, multiple simultaneous hemorrhages

Introduction

Occurrence of multiple simultaneous hemorrhages in different arterial irrigation areas of the brain is a relatively rare clinical event compared to solitary spontaneous intracerebral hemorrhages (ICHs) [1]. Non-traumatic multiple simultaneous intracerebral hemorrhages (MSICHs) accounts for 2.0-5.6% of all intracerebral hemorrhages, and their etiologies may include uncontrolled hypertension, vasculitides, sympathomimetic drugs, primary and metastatic brain tumors, amyloid angiopathy, sinus thromboses, thrombolytic treatments, coagulopathies and multiple infarcts with hemorrhagic transformations [2-6]. Cranial computed tomography (CT) findings of MSICHs developed due to etiologies other than traumas are discussed in the present paper.

Methods

We reviewed all medical records of patients with MSICHs who were admitted to the Gaziosmanpaşa University School of Medicine Research and Training Hospital Tokat, Turkey between March 2010 and June 2015. A total of 20 patients (14 males and 6 females) with MSICHs were included in the present study. These were the ones with MSICHs among 139 ICH patients who were found to have spontaneous intraparenchymal hemorrhage in their cranial CT examinations performed due to various neurological complaints such as headache and clouding of consciousness and other symptoms. All patients with spontaneous ICH hematomas except for hypertensive SICH cases were evaluated with Brain MRI included T2-weighted gradient echo sequence.

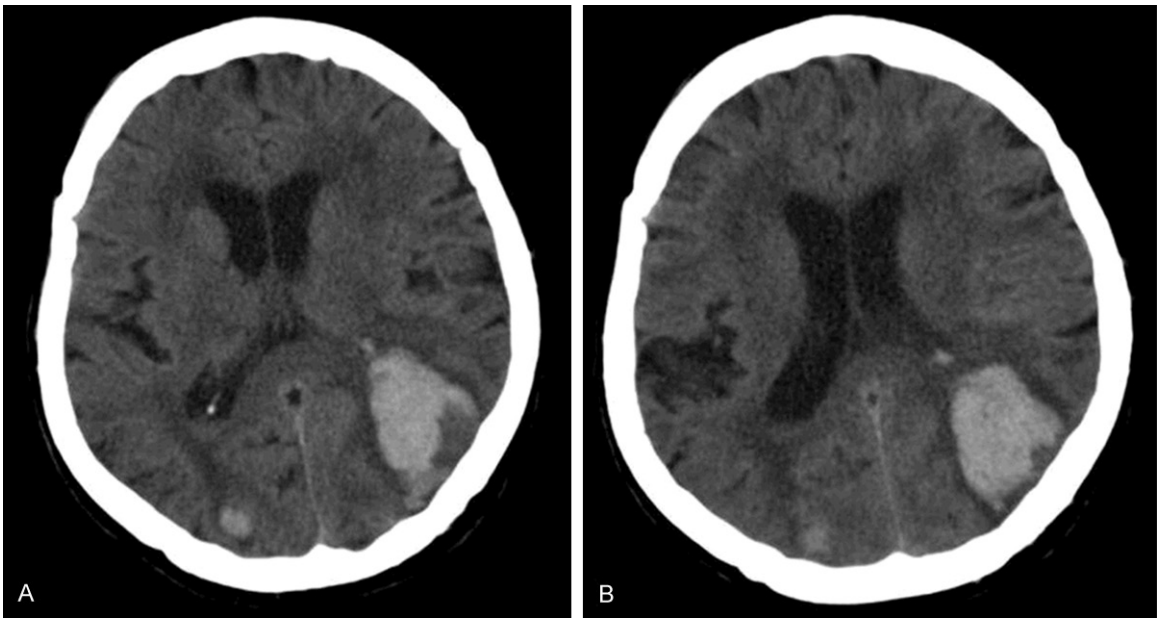

Figure 1. CT imaging of an 83 years old female patient who had hypertension and developed intense headache. A and B. Simultaneous hematomas are observed in both occipital lobes, left one being larger.

Hypertension and amyloid angiopathy dependent hemorrhages were considered primary MSICHs, while others were considered secondary MSICHs. After taking the approval of local ethic committee (Gaziosmanpaşa University School of Medicine, No: 14-KAEK-158), CT scanning images of the patients in Picture Archiving and Communication System (PACS, GE) were studied retrospectively. All patients included in this study were subjected to 5 mm, non-contrast head CT examination on admission. All CT examinations were performed at 2- or 8-slice multidetector CT scanners (Somatom Sprit; Siemens, Erlangen, Germany and LightSpeed Ultra; GE, Milwaukee, USA).

We defined MSICH as spontaneous hemorrhage in two or more discrete intra-parenchymal locations with CT density profiles within the established hemorrhage range based on Hounsfield units [5]. Number, localization and volume of hematomas were studied in CT examinations. Cerebral hematoma locations were classified as deep when they were in basal ganglia, thalamus and in the neighborhood, and as lobar when they were in cerebral parenchyma outside these areas. The maximum hemorrhage width (W, transverse diameter), length (L, anteroposterior diameter), and height (H, craniocaudal diameter) were measured, and the hemorrhage volume was estimated using the following formula: Hemorrhage volume = W × L × H × 0.5. Statistical differences between primary and secondary MSICH volumes were studied. Examinations and measurements were carried out simultaneously by two radiologists (E.G. and M.B.).

All statistical analyses were performed using Statistical Package for the Social Sciences (SPSS) version 15.0. Data was presented as percentages (%), median or mean ± standard deviations (SD). Mann Whitney-U test was used to test the continuous variables.

Results

Of all 139 patients with intraparenchymal hemorrhage, forty-seven had primary etiological factors while 86 had secondary ones and 6 had no evident reason for hemorrhage. Hypertension was the etiological factor in 6 patients with MSICH, lung cancer in four, malign mesothelioma in two, and colon neuroendocrin carcinoma, signet ring cell carcinoma of omentum, HELLP (hemolysis, elevated liver enzyme levels, and low platelet levels) syndrome, warfarin use, sinus thrombosis, factor X deficiency, acute myeloblastic leukemia and amyloid angiopathy each in one patient. Thus, seven of the patients (35%) had primary MSICH while thirteen (65%) had secondary MSICH. The age of the patients

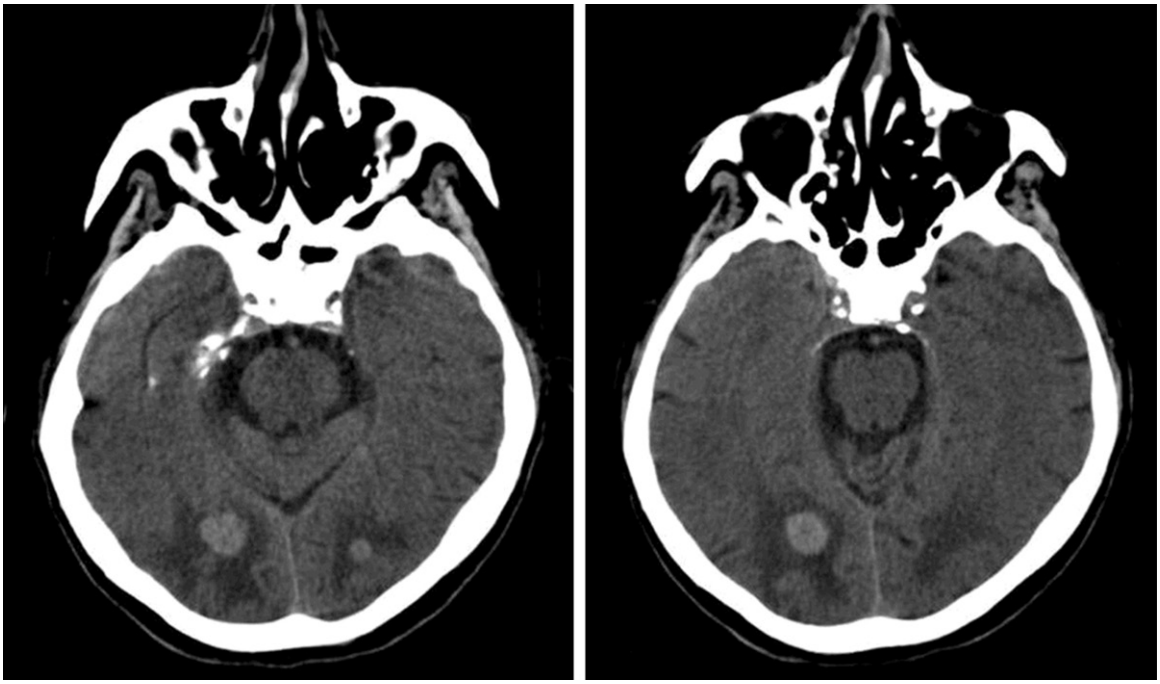

Figure 2. CT imaging of a 65 years old male patient who had lung cancer and developed headache. A and B. Simultaneous hemorrhagic bleedings secondary to metastasis in both occipital lobes with clear vasogenic edema around which was not proportional to the size.

with MSICH varied from 1 to 83 (mean 59.26±18.94). Incidence of MSICH in all ICH cases was 14.4%. In cranial CT of 20 MSICH patients, a total of 84 hematomas were detected. Seventy of them (83.3%) were of lobar origin, while 7 (8.3%) were of deep, 4 (4.8%) of cerebellar and 3 (3.6%) of brain stem origin. Nine patients had simultaneous hematomas in two parenchymal areas, four patients in four areas, two patients in seven areas, and one patient each in 3, 5, 6, 9 and 13 areas (**Figures 1**-**5**). Forty one of the hematomas were located on the left and thirty nine on the right. Seven of the MSICHs (8.3%) had infratentorial locations and 77 had supratentorial ones (91.7%). One patient had infratentorial hemorrhage in two different foci, whereas five patients had both infra- and supratentorial hemorrhages. All MSICHs in other patients were of supratentorial location. Hematoma volumes varied from 0.01 to 111.15 (median 0.50). The difference between average volumes of hematomas in primary and secondary MSICHs was not significant (P=0.32). Demographic features, etiological factors, hematoma localizations and localities and volumes were given in **Table 1**.

Discussion

ICH accounts for 8-14% of all strokes in Europe and the United States, but they are more common in many Asian countries [5]. Compared to solitary ICHs, MSICHs are considered extremely unusual [5]. It has been mentioned that rare frequency reports of MSICH could be due to the fact that most data were obtained before the introduction of CT and that the early data with CT examinations came from cranial examinations by old generation CT machines using thick slices of 10-15 mm [3, 5]. It is possible that the condition might have been under-detected and under-reported if small lesions had been outside the limits of CT slice thickness. With increased use of multidetector CT in modern day, standard brain CT slices became 4-5 mm thick [5]. In a relatively recent study on 522 spontaneous ICH patients carried out in 2006-2009 period, though slice thickness was not mentioned, Stemmer et al. [5] found a relatively high MSICH incidence rate of 5.6%, which is higher than the earlier studies in literature [6]. A high MSICH incidence of 14.4% in the present study could be due to 5 mm slice thickness in CT, inclusion of both primary and secondary

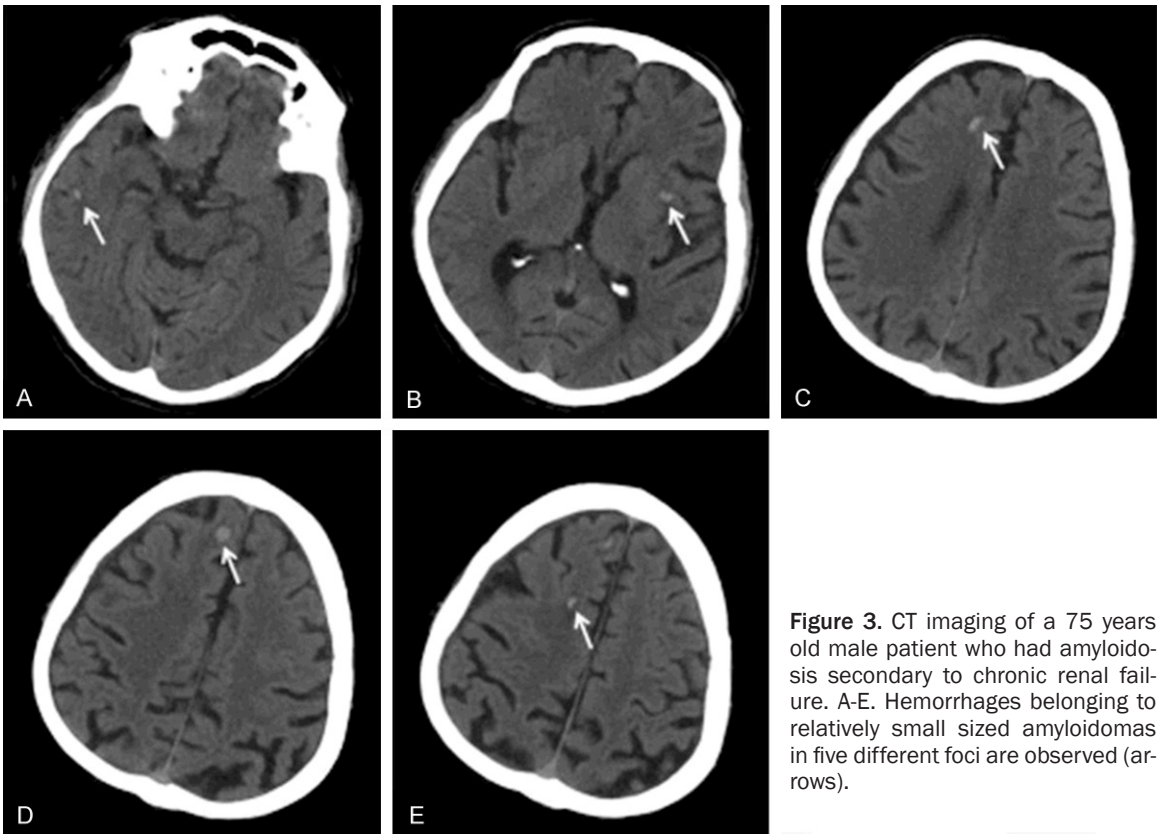

Figure 3. CT imaging of a 75 years old male patient who had amyloidosis secondary to chronic renal failure. A-E. Hemorrhages belonging to relatively small sized amyloidomas in five different foci are observed (arrows).

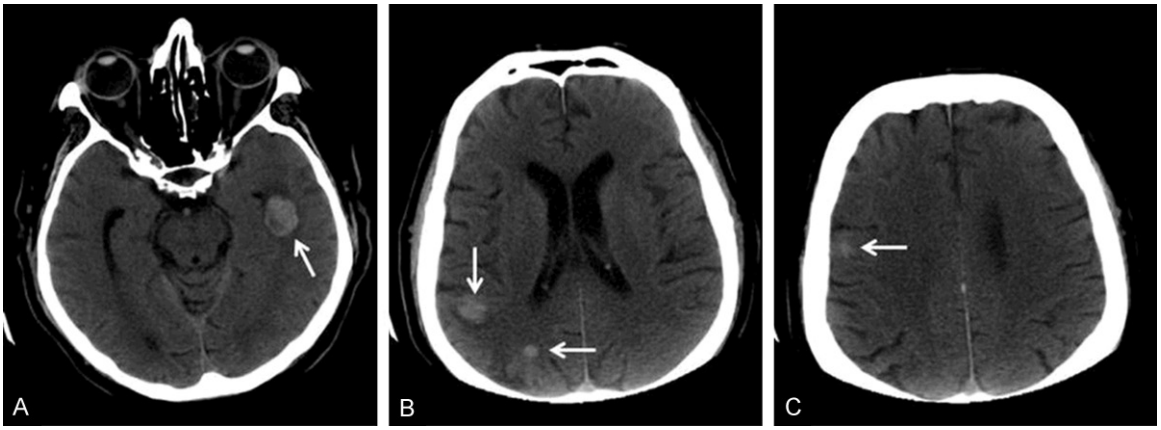

Figure 4. CT imaging of a 60 years old male patient who had been using Warfarin and had developed sudden headache. Simultaneous hematomas of different sizes, (A) One in left temporal lobe, (B) Two in right occipital lobe, and (C) One in left frontal lobe.

MSICHs in the study and population structure. The smallest MSICH volume detected by CT examinations in the literature is 0.2 ml [5]. Thinner slice thickness employed in CT examinations in the present study allowed detecting the hemorrhages of as small as 0.01 ml volume. In terms of the association between hematoma volumes and etiological factors, no significant difference was observed for the volumes of MSICHs between primary and secondary MSICHs (P=0.32).

A few studies have found the incidence of primary MSICHs in primary intracerebral hemorrhages to vary from 0.7% to 3.4% of [4-8]. Localities of the 94 hematomas in 48 primary

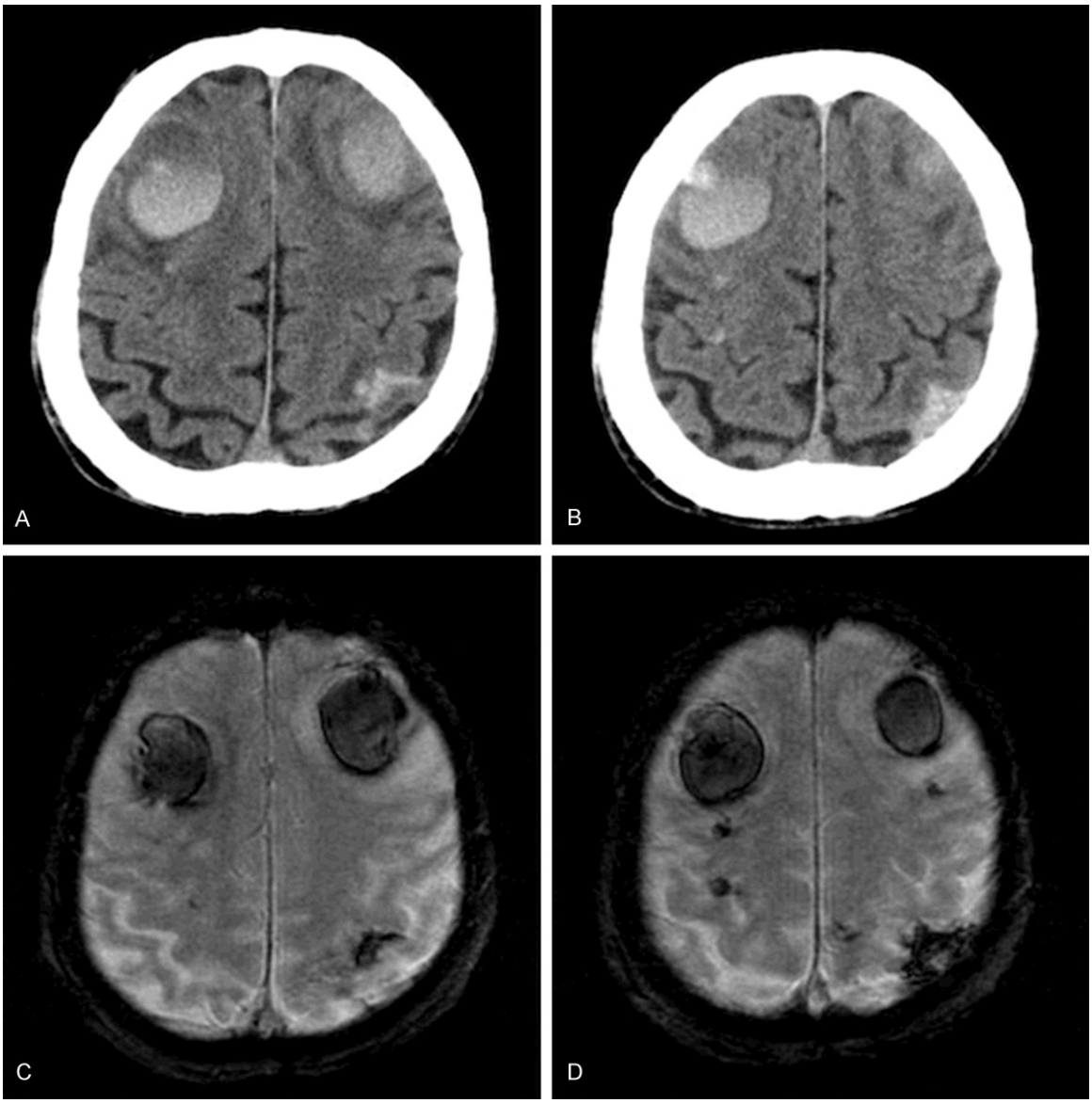

Figure 5. CT and MRI of a 65 year old male patient who had malignant mesothelioma and developed unconsciousness and syncope. A and B. Simultaneous hematomas of different sizes in both cerebral hemispheres are observed in CT images. C and D. Axial T2*-weighted gradient echo sequence MR images show hypointense hemorrhagic products within metastatic masses.

MSICHs were studied and it was found that 94 (63.8%) were of deep origin, 27.7% of lobar and 8.5% of infratentorial origin [5]. Unlike the literature, of the localities of the 24 hematomas in seven primary MSICH cases in the present study, 63.7% were of lobar origin, 20.8% of deep and 12.5% of infratentorial origin. Higher percentage of deeply located primary MSICHs in literature could be due to higher percentage of hypertensive MSICHs in great majority of the cases in those studies [1, 3, 5, 8]. Five of the seven hematomas with deep location in the present study belonged to hypertensive MSICHs, while all hematomas of infratentorial location except for four were observed in hypertensive MSICH cases. This finding might indicate that great majority of MSICHs due to etiologies other than hypertension tends to have supratentorial or lobar locations. Secondary MSICHs were found to be the most common type of simultaneous intracerebral hemorrhages, accounting for up to 71.4% of MSICHs. [3, 5]. Sixty five per cent of the cases with MSICHs were secondary MSICHs. Higher incidence of

Table 1. Demographic features, etiological factors, localizations, deep or lobar localities, and volumes of hematomas in MSICH patients

Patient	Sex	Age	Etiology	Location	Deep or lobar	Volume (ml)
1	M	60	Warfarin	R parietal	Lobar	1.69
1				R occipital	Lobar	0.17
1				L temporal	Lobar	2.25
1				R frontal	Lobar	0.09
2	F	83	Hypertension	R occipital	Lobar	0.09
2				L occipital	Lobar	0.44
3	M	40	Hypertension	R cerebellar	Cerebellar	5.76
3				Pons	Brain stem	15.75
4	M	75	Bronchogenic carcinoma	L frontal	Lobar	30.65
4				R temporal	Lobar	1.68
4				R frontal	Lobar	0.50
5	M	65	Bronchogenic carcinoma	R occipital	Lobar	1.68
5				L occipital	Lobar	0.67
6	M	47	malignant mesothelioma	R frontoparietal	Lobar	4.20
6				L parietal	Lobar	12.86
7	M	62	Sinus thrombosis	L parietooccipital	Lobar	25.72
7				L temporal	Lobar	2.28
7				R frontal	Lobar	0.10
7				R parietal	Lobar	0.22
8	F	1	Factor X deficiency	L frontotemporal	Lobar	36.72
8				R frontal	Lobar	0.09
8				L occipital	Lobar	0.07
8				L occipitoparietal	Lobar	0.20
9	F	57	Acute myeloblastic leukemia	L temporoparietal	Lobar	27.54
9				L inferior colliculus	Brain stem	0.18
10	F	78	Hypertension	L frontal	Lobar	3.93
10				L parietal	Lobar	0.60
11	M	46	Bronchogenic carcinoma	R frontal	Lobar	28.56
11				R temporal	Lobar	0.12
11				R occipital	Lobar	3.84
11				R occipitoparietal	Lobar	29.92
11				R parietal	Lobar	0.12
11				L frontal	Lobar	0.44
11				L frontal	Lobar	27.90
12	F	49	Hypertension	L cerebellar	Cerebellar	24.75
12				L thalamus	Deep	1.53
13	M	75	Amyloid angiopathy	R temporal	Lobar	0.06
13				L external capsul-insular	Lobar	0.07
13				R frontal	Lobar	0.30
13				R frontal	Lobar	0.33
13				R frontal	Lobar	0.15
14	M	79	Hypertension	L basal ganglia-thalamus	Deep	111.10
14				L parietal	Lobar	8.10
14				L frontal	Lobar	0.60
14				L frontal	Lobar	0.90
14				L frontal	Lobar	0.30
14				L frontal	Lobar	5.89
14				L frontal	Deep	0.08
14				R frontal	Deep	0.05

14				R basal ganglia	Deep	1.54
15	M	64	Bronchogenic carcinoma	L cerebellar	Cerebellar	0.01
15				R temporal	Lobar	0.04
15				R parietal	Lobar	0.12
15				Corpus callosum	Deep	0.28
15				L frontal	Lobar	0.09
15				L frontal	Lobar	0.19
15				R frontal	Lobar	0.04
16	M	69	Hypertension	L frontal	Lobar	0.52
16				L frontal	Lobar	11.55
17	M	66	Colon neuroendocrin carcinoma	L frontal	Lobar	0.04
17				L frontal	Lobar	0.38
17				R frontal	Lobar	0.06
17				R frontal	Lobar	0.04
18	F	57	Signet ring cell carcinoma of omentum	Brainstem	Brainstem	31.39
18				R temporal	Lobar	6.48
18				R temporal-lentiform nucleus-thalamus	Deep	0.30
18				R parietal	Lobar	5.23
18				L occipital	Lobar	0.28
18				R parietal	Lobar	0.07
19	M	41	HELLP syndrome	L occipital	Lobar	1.08
19				R parietal	Lobar	0.09
20	M	72	malignant mesothelioma	R occipital	Lobar	4.49
20				L occipital	Lobar	0.44
20				L frontal	Lobar	28.90
20				L parietal	Lobar	3.90
20				L parietal	Lobar	0.07
20				L occipital	Lobar	0.07
20				R parietal	Lobar	0.06
20				R frontal	Lobar	0.09
20				R frontal	Lobar	0.08
20				R frontal	Lobar	18.28
20				L cerebellar	Cerebellar	0.03
20				R frontal	Lobar	0.54
20				L frontal	Lobar	0.86

primary MSICHs was reported in males with a male to female ratio of 1.56:1 in the literature [3]. In the present study, primary MSICH incidence rates in males and females were similar with the literature.

Except for the secondary MSICHs patients with etiologies such as subarachnoid hemorrhage, traumatic hemorrhage, hemorrhagic transformation of arterial or venous infarcts, and underlying tumors, pathophysiologies of primary or idiopathic MSICHs are not known [5]. Cerebrovascular degeneration induced by hypertension in different irrigation areas of the brain could explain recurrent ICH in hypertensive patients. However, this mechanism cannot fully explain bilateral MSICHs. Coincidental rupture of bilateral micro-aneurysms or perforating arteries undergoing hyaline degeneration may be possible cause [1, 9]. It has been reported that structural and hemodynamic changes mediated by primary hemorrhage focus of MSICHs might induce other hemorrhage foci [1, 9]. Although arterial hypertension alone is the biggest risk factor for solitary spontaneous hemorrhage, MSICH incidence in hypertensive patients have been reported to vary from 0.3 to 1.0% [1, 6, 8, 10]. MSICH incidence in hypertensive patients in the present study (22.6%) was much higher than the literature reports. It has been reported that hypertensive MSICHs are predominantly of deep gray matter origin

(thalamus, basal ganglia) and that the frequencies of deep and lobar localities in hypertensive solitary hemorrhages and MSICHs are similar [1, 5]. In their studies with 21 hematomas in 10 hypertensive MSICH patients, Yen et al. [1] found that 81% of hematomas were of deep, 14.3 of lobar and 4.8% of brain stem location. Six patients in the present study (30%) had hypertensive MSICHs, and unlike the literature, 5 hematomas (26.3%) were of deep location, 2 (10.5%) of cerebellar, 1 (5.3%) of brain stem and 11 (57.9%) of lobar location. In a study with 11 hypertensive MSICH patients by Shiomi et al. [10], both infra- and supratentorial hemorrhages were observed in 80% of the patients. In the present study, on the other hand, only 16.7% of hypertensive MSICH patients had both infra- and supratentorial hemorrhage.

MSICH, a relatively rare form of brain hemorrhage, might pose a diagnostic dilemma. However, most MSICH cases are accompanied by evident risk factors or underlying morbidities. In MSICH cases for which etiology is not known, on the other hand, imaging and determining the risk factors constitute the base for identification of underlying etiology [2]. Awareness of the medical conditions, especially of hypertension, trauma, drug use, patient's age, intracranial hemorrhage and deep vein thrombosis, risk factors for cancer such as smoking and presence of known malignancies might help classify the patients [2]. In patients with no underlying vascular pathology or coagulopathy, malignity based hemorrhagic metastases are one of the most possible reasons for MSICHs detected in brain CT [2]. Patients with malignity constituted one of the largest groups of MSICH patients (33.3%) in the present study. Among the malignancies commonly resulting in MSICH are bronchogenic carcinoma, renal cell carcinoma and carcinoma or malign melanoma metastases [2] and among the rarer ones are hemorrhagic metastases of different types of malignancies [7]. Bronchogenic carcinoma constituted 44.4% of the all malignancies causing MSICHs in the present study. When a hemorrhagic metastasis is observed in patients with malignancies, it is possible that the primary tumor is not known [2]. In two of our cases, malignity diagnoses were made after hemorrhagic metastases were detected in brain CT examinations. Primary tumor diagnosis of the case with malignant mesothelioma was made after evacuating of one of the hematomas detected in CT, while the malignity diagnosis in another case with bronchogenic carcinoma was made by other examinations after brain CT (contrasted brain MRI, thorax CT).

Tumors presenting as a high density mass could be misdiagnosed as a hematoma in initial CT scan. One of the reasons of the high attenuation in CT is due to the elevated protein content in tumor [11]. Diagnosis could be made evaluating cases with brain MRI including T2*-weighted gradient echo sequence. In the present study, one of the patients was excluded from the study after diagnosed as lung metastasis mimicking parenchymal hematoma.

Intense vasogenic edema that can be detected by CT or MRI can be observed around the cerebral metastatic masses. In the present study, density decreases due to intense edema in CT which were unrelated to hematoma sizes were observed around MSICH of malignancy origin (**Figure 2A**, **2B**). This finding can be considered as a factor strengthening the malignancy etiology of MSICHs which have intense vasogenic edemas around them despite their small sizes.

In patients over sixty years of age who have no risk factors, amyloid angiopathy could be an etiological factor as important as malignancies [2]. Izumihara et al. [12] studied 37 patients with intracerebral hemorrhages that developed secondary to amyloid angiopathy and found an MSICH incidence of 14%. Hemorrhages secondary to amyloid angiopathy could be in different sizes and they might be located in different lobes of cerebral hemispheres, predominantly in cortical-subcortical white ore areas [12]. Basal ganglia, brain stem and cerebellum are generally protected in amyloid angiopathy [13]. Although cortical-subcortical location of lobar hemorrhages in MSICHs might indicate amyloid angiopathy, other causes of hemorrhage cannot be eliminated using CT imaging characteristics. However, compared to CT, MRI could provide better information to identify the etiological factor especially in amyloid angiopathy. For example, gradient echo sequence on MRI can be beneficial in detecting prior silent hemorrhage suggestive of underlying amyloid angiopathy as well as direct clot visualization as an area of hypointensity in cerebral venous thrombosis [2]. In a 75 year-old patient with amyloid angiopathy in the present study, lobar hemato-

mas volumes of which varied from 0.06 to 0.16 ml were observed in five different foci (**Figure 3A-E**). The facts that the patient was normotensive, that he had chronic renal failure history and that there were hypointensities belonging to hemosiderin other than the hemorrhage foci detected in gradient T2A series in CT observed in MRI performed after CT were the factors pointing to amyloid angiopathy diagnosis.

Though rare, MSICHs can be seen in patients using oral anticoagulants [11, 14]. Studying 21 patients on oral anticoagulants, Gökçe et al. [15] found an MSICH incidence of 4.7%. In another study by Tejero et al. [14], one of the seven MSICH patients had oral anticoagulant use history. Stemmer et al. [5] reported hypertension and anticoagulant use in four of the 29 MSICH patients, while a total of seven patients had anticoagulant use (warfarin or heparin). In the present study, on the other hand, only one patient (5.0%) was found to have MSICH due to anticoagulant use in four different foci in cerebral parenchyma (**Figure 4A-C**). This patient, whose tension was kept under control by medical treatment, also had co-morbidities such as diabetes and coronary artery disease.

Though rare, there are reports of acute myeloblastic leukemia (AML) dependent MSICHs in the literature. Yabumoto et al. [16] detected MSICH in three different areas in the first day brain CT of a patient with AML, and observed MSICHs in nine additional foci in the second day brain CT. In the present study, two hematomas were observed in two different foci, one infratentorial and the other supratentorial, in a case with AML dependent MSICH.

Treatment methods and prognosis of MSICHs vary depending on neurological grading at the onset of the condition, and localizations and maximum diameters of the hematomas also affect surgical approaches [10, 17]. For example, if the hematomas of supratentorial localities have smaller sizes than in cerebellar hematomas, surgical evacuation of cerebellar hematomas can be performed [10, 17]. Accordingly, in a case with hypertensive MSICH having thalamic and cerebellar hematomas (case 12), cerebellar hematoma was evacuated since thalamic hematoma was small and cerebellar hematoma was large enough to cause ascending transtentorial herniation.

Conclusion

As observed in the present study, with the increasing use of thinner slice cranial CT examinations, a higher incidence of MSICH detection is observed. Number and size of MSICHs could vary independent of etiological factor. In cases with MSICHs, careful questioning and examination as well as and without treatment programs based on radiological imaging findings should be implemented.

Disclosure of conflict of interest

None.

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