J Korean Radiol Soc 1997;37:195-200

Signal Void Dots on T2-weighted Brain MR Images in Patients with Hypertensive Intracerebral Hemorrhage : Its Nature and Clinical Significance[1]

Sang Joon Kim, M.D., Dong Soo Yoo, M.D., Seung Chul Kim, M.D.
Tae Hoon Kim, M.D., Jae Seung Kim, M.D., Jae-Il Kim, M.D.[2]

Purpose : To describe the signal void dots found on T2-weighted magnetic resonance (MR) images of the brain in hypertensive patients.

Materials and Methods : Conventional T2-weighted MR images of 11 patients with hypertensive intracerebral hemorrhage (ICH), 14 with lacunar infarction and 11 comprising a normal control group aged over 60 were analyzed with regard to the presence, location, number and size of signal void dots. We also evaluated their relationship to hypertension. We performed time-of-flight or phase contrast MR angiography, gradient echo pulse sequences, or conventional cerebral angiography in some hypertensive ICH patients and compared them with corresponding T2-weighted images.

Results : Signal void dots were found in all patients with hypertensive ICH. Six of 14 patients with lacunar infarction showed these dots ; all six suffered from hypertension. The dots were located in the thalami, pons and basal ganglia, and were measured as 1 to 4mm in diameter, mostly 2mm ; they looked larger on gradient echo images. In the normal control group there were no signal void dots, and on MR or conventional angiography, no vascular ectasia was noted at the site corresponding to the signal void dots.

Conclusions : Signal void dots were not considered to be part of the normal aging process, but appeared to be closely related to hypertension and ICH. The dots were thought to be due to the susceptibility effect of blood degradation product rather than to flow artifact or enlarged vessels. The thrombosed microaneurysm with or without surrounding microleakage of blood may explain the nature of signal void dots on T2-weighted images of hypertensive brain.

Index Words : Brain, hemorrhage
Brain, MR

In spite of aggressive diagnosis and treatment, hypertension is still the leading cause of spontaneous intracerebral hemorrhage (ICH) (1), which is usually diagnosed on the basis of clinical signs and symptoms. A history of hypertension and bleeding into a site at which hypertensive ICH typically occurs, such as the putamen, thalamus, cerebellum, or pons is sufficient to demonstrate the existence of this condition.

On conventional T2-weighted magnetic resonance (MR) images of the brain in patients with hypertensive ICH, punctate signal void (or dark signal) dots have been frequently noticed in areas vulnerable to hypertensive ICH. We also found similar dots in patients with lacunar infarction without hemorrhage. There have been very few reports in the literature concerning this kind of hypointense lesion in association with chronic hypertension, and the nature and underlying mechanisms of the hypointense dots are not yet clearly understood (2, 3).

Our purpose was to describe these signal void dots and to elucidate the nature of these findings and their relationship to hypertensive ICH or lacunar infarction.

[1]Department of Radiology, Dankook University College of Medicine
[2]Department of Neurology, Dankook University College of Medicine
Supported by a grant from Dankook University
Received January 14, 1997 ; Accepted May 2, 1997
Address reprint requests to : Sang Joon Kim, M.D., Department of Radiology, Dankook University College of Medicine, # 29 Anseo-Dong, Cheonan, ChungNam, 330-714, Korea Tel : 82-417-550-6921 Fax : 82-417-552-9674

Materials and Methods

We retrospectively reviewed conventional spin echo T2-weighted brain MR images of 11 consecutive patients with hypertensive ICH, and compared them with those of 14 patients with lacunar infarction or deep white matter ischemia and 11 patients with 'normal' brain MR findings.

The inclusion criterion for hypertensive ICH patients was a history of systemic hypertension concomitantly with a recent or old ICH at a typical site such as the putamen, thalamus, cerebellum or pons. The criteria for systemic hypertension were systolic blood pressure over 140 mmHg and diastolic pressure over 90 mmHg. Fourteen other patients with lacunar infarcts or ischemic changes in the basal ganglia or periventricular white matter, with or without systemic hypertension, were randomly selected; those with lobar infarcts were excluded. To determine whether the presence of signal void dots is a sign of physiological aging, the MR images of 11 age-matched normal control subjects were also reviewed. They were over 60 years old and had 'normal' brain MR findings and no history of hypertension. The age and sex of each group were summarized in Table 1.

Table 1. Patients Age and Sex Distribution

Group	Sex (M : F)	Age(years)
ICH	6 : 5	44—74 (mean 61.0)
Infarct	10 : 4	55—82 (mean 67.3)
Normal	4 : 7	60—73 (mean 64.4)

Notes : ICH = intracerebral hemorrhage, M = male
F = female

MR imaging was carried out using a 1.5 T MR system (Signa; GE Medical Systems, Milwaukee, U.S.A.). Conventional T2-weighted images (repetition time (TR) 2,000—2,500 msec; echo time (TE) 80—100 msec; number of excitations, 1—2) were used to analyze the location, number and size of signal void dots in the basal ganglia, thalami, and pons. Because we could not consistently separate them from vascular signal void, signal void lesions in the cortical/subcortical areas were not included. The relationship between signal void dots and hypertension was also analyzed.

In the ICH group we also obtained and analyzed gradient echo images in one patient, time-of-flight (TOF) MR angiography (MRA) and their source images in nine, phase contrast (PC) MRA in three, gadopentetate dimeglumine enhanced TOF MRA in one and conventional angiography in one, and compared them with T2-weighted images.

Table 2. Number of Patients Who had Signal Void Dots in Each Group

Group	No. of SV(+) Pts.
ICH	11/11
Infarct	6/14
with hypertention	6/9
without hypertension	0/5
Normal control	0/11

Notes : ICH = intracerebral hemorrhage
SV(+) = signal void present
pts. = patients, No. = number

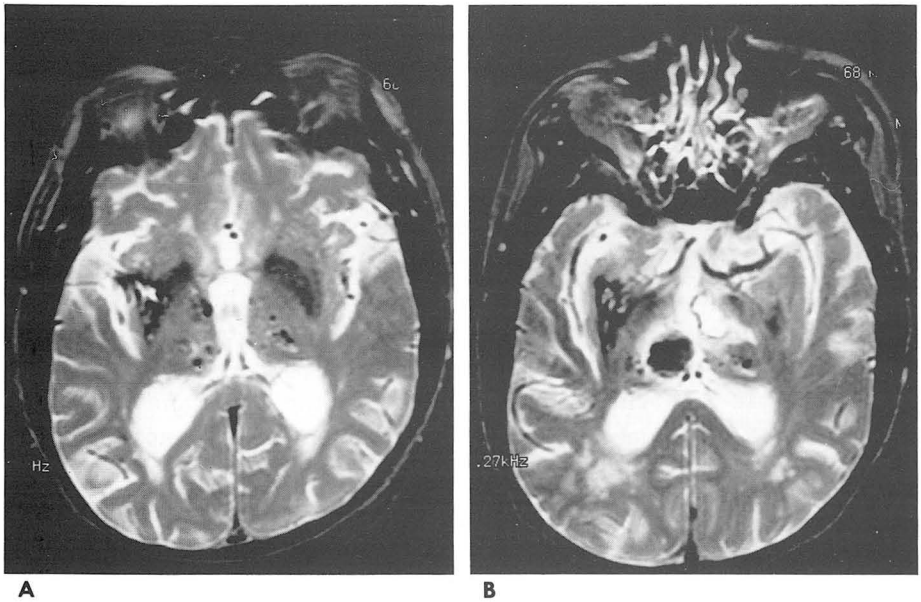


Fig. 1. T2-weighted images of a 68-year-old man with repeated hypertensive hemorrhages.
A. Multiple signal void dots are noted in both side thalami along with old hemorrhage in right putamen. This patient also had an acute stage hemorrhage in the dentate nucleus(not shown here).
B. Two months follow-up MR image shows newly developed acute and subacute stage hemorrhages in both thalami in the areas of previous signal void dots.

Results

Signal void dots were found in all patients in the hypertensive ICH group (Fig. 1). Among 14 patients with lacunar infarction or deep white matter ischemia, six with hypertension showed signal void dots. These were not found in any normal control subjects (Table 2). Among 20 hypertensive patients, including both the ICH and infarct group, 17 (85%) showed signal void dots, whereas 16 normotensive patients, including those in the normal control group, showed no dots. There was a significant difference in the prevalence of dots between the hypertensive and normotensive group (Fisher's exact test, $p < 0.01$), and they were found more frequently in the ICH group (100%) than in the lacunar infarct group (42.9%) (Fisher's exact test, $p < 0.01$).

The signal void dots were round to ovoid in shape and were between 1 and 4 mm, mostly 2 mm, in diameter; most were located in the thalamus. The pons and basal ganglia were the next most common locations, and in both, the number of dots was similar (Table 3).

Table 3. Location and Number of Signal Void Dots

Locations	No. of Patients		Total No. of SV dots
	ICH group (11)	Infarct group(6)	
Thalamus	10	5	60
Pons	5	4	21
Basal ganglia	5	3	21
Int. Capsule	1	—	1

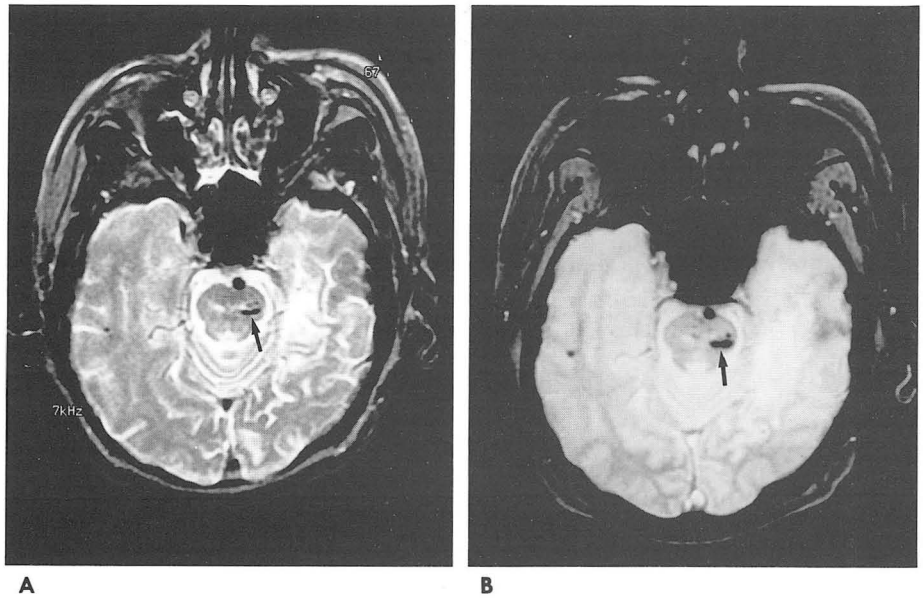
Notes : Int. = internal, ICH = intracerebral hemorrhage
No. = number, SV = signal void

In one patient who underwent gradient echo imaging, the dots were seen to be larger than on T2-weighted spin echo images (Fig. 2). On conventional angiography, no ectatic vessel or aneurysmal sac was found in a patient who on T2-weighted images showed signal void dots in the basal ganglia. TOF MRA, PC MRA, and gadolinium-enhanced TOF MRA did not reveal flow-related enhancement at the site corresponding to the dots; instead, dots were noted on source images of MRA (Fig. 3).

Discussion

The concomitant presence of signal void dots on T2-weighted images of the brain in patients with hypertensive hemorrhage or lacunar infarction prompted us to investigate the nature of these dots and to evaluate the relationship between them and systemic hypertension.

The dots do not seem to be merely part of the aging process, because they are not found in normal brain MR images of the control group; possible explanations for their presence are flow-related signal voids, focal depositions of blood degradation products, or calcification. Because no dilated vessels were seen on the conventional cerebral angiography or TOF- or PC-MRA in the matching areas of the signal void dots, the possibility of flow-related signal voids in the dilated vessel could be ruled out. Instead, the dots on T2-weighted images were also of low signal intensity on the corresponding source images of MRA, and on gradient echo images, were slightly larger. This suggests that they are



A B

Fig. 2. A 67-year-old man with hypertension. MR images showed acute stage infarction in left basal ganglia and ischemia in the deep hemispheric white matter(not shown here)
A. T2-wighted image shows punctate high signal intensities in pons and two contiguous signal void dots in left side pons(arrow)
B. On the gradient echo image (TR/TE/Flip Angle, 500/13/30°) at the same level as A, the contiguous signal void dots look larger than on T2-weighted image, probably due to stronger susceptibility effect, suggesting blood degradation product (arrow).

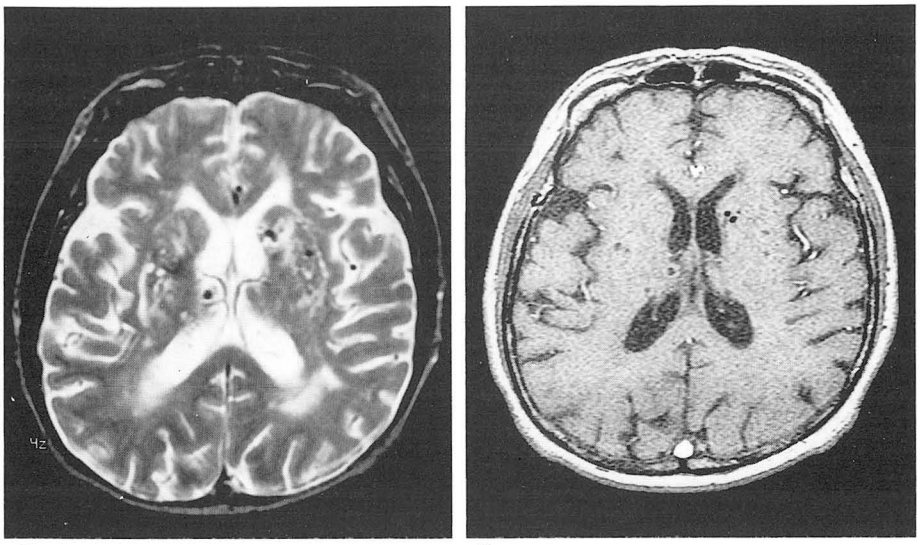


Fig. 3. A 63-year-old man with deep white matter and basal ganglia ischemia
A. Multiple signal void dots are noted in left basal ganglia and right thalamus along with punctate or patchy high signal in basal ganglia and deep white matter on T2-weighted image
B. The source image of 3D TOF MRA(45/5.2/20°) at the same level as A reveals similar dark signal suggesting that they are not caused by flow.

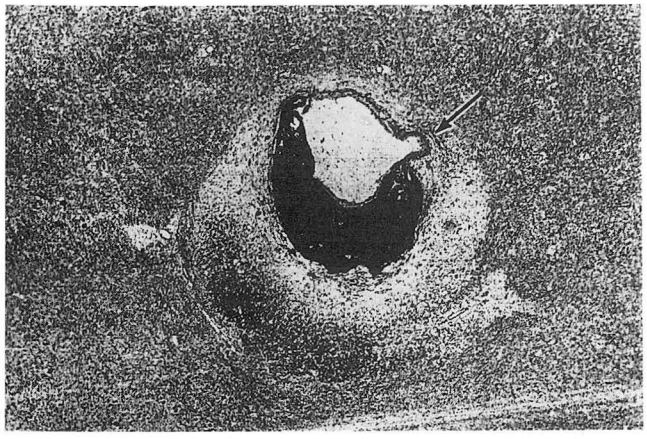


Fig. 4. Miliary saccular aneurysm in thalamus. The parent artery is the small protrusion on the right(arrow). The black material is a fibrin thrombus. The surrounding material is a hemorrhagic extravasation(PTAH, ×30).(From reference 7, with permission)

caused by the susceptibility effect of hemosiderin or blood degradation products (4). On CT images obtained in some patients, no evidence of calcification was found in areas corresponding to the dots.

Although it seems evident that dark signal spots represent blood degradation products, the underlying mechanisms of the spots are not clear. Scharf et al. (2) believed they were hemorrhagic lacunes, frequently mentioned in literature describing the pathology of the hypertensive brain. They diagnosed hemorrhagic lacunes if lacunar lesions with central or peripheral areas suggesting the presence of hemosiderin-laden macrophages were seen on MR images, though they failed to show anatomic correlation. Whether the hypointense dots may be regarded simply as hemorrhagic lacunes is open to question. We believe the dark signal dots may be more than mere hemorrhages because (a) they were rather uniform in size (1 to 4 mm in diameter), (b) they were uniformly round-to-oval shaped and had a smooth margin and (c) in our series there were no definite acute or subacute lesions of hemorrhages similar in size to the dots. We suggest that these spots may be thrombosed microaneurysms with surrounding microhemorrhages.

Pathologically, the brains of hypertensive patients show lipohyalinosis and microaneurysms of penetrating arteries 50 to 200 μm in diameter (1). Lipohyalinosis is a subintimal accumulation of a lipid-rich hyaline material that finally weakens and destroys artery (5). The cerebral miliary aneurysm was originally described by Charcot and Bouchard (6), and although the debate concerning the existence of cerebral microaneurysms and their role in hypertensive hemorrhage has continued even to the present time, many authors have reported the presence of microaneurysms in brains which have suffered this type of hemorrhage (1, 5, 7—11). The microaneurysms were 300—1100 μm in diameter and lay on vessels between 40—160 μm in diameter (Fig. 4)(7). Fibrin and mural thrombus usually lined the inner surface of the wall; red blood cells had penetrated to the outside to form a coating of blood 20—300 μm thick, which showed that microrupture had occurred. Hemosiderin-laden macrophages and free iron were often scattered and surrounding brain parenchyme showed heavy astroglial proliferation (7, 9). Russel (9) reported these microaneurysms in 14 of 15 hypertensive brains, but also in some normotensives. They were found mainly in putamen, thalamus, pons and sometimes in cerebral subcortical white matter (1, 7) echoing the characteristic anatomic distribution of hypertensive ICH. The exact mechanism of

microaneurysm formation in the arteries of a hypertensive patient is not clear, but one explanation is weakness of a wall due to lipohyalinosis, resulting in local arterial dilatation(7). These aneurysmal dilatations were obliterated due to derangement of the wall, fibrin deposits or fibrosis.

We consider that signal void dots on T2-weighted images may represent these thrombosed microaneurysms together with surrounding red blood cells and macrophages which are due to microrupture. The basis of this conclusion is as follows : (a) the predominant location of signal void dots exactly corresponds to that of the pathologically reported microaneurysms, (b) the signal void dots were about 2 mm in diameter, and this matches the size of microaneurysms and surrounding microhemorrhages, (c) the signal intensity could be explained as the susceptibility effect of blood degradation products.

In our series, signal void dots were found exclusively in hypertensive patients; they were not in 16 normotensive patients, including the normal control group. Although the number of patients is small, the dots appear to be closely related to hypertension, and to be more closely related to ICH than to lacunar infarct. The frequency of dots in the ICH group or hypertensive patients was much higher in our series than in a previous report by Scharf et al(2). This may be because they used mid-field strength MR(1.0T) and/or their criteria for dark signal dots may be different from ours ; in addition, the study population in our series was small, some bias may have existed.

In conclusion, the signal void dots found on conventional T2-weighted images of hypertensive brain may be due to susceptibility effect of blood degradation product, and on the basis of neuropathologic findings reported in the literature, can be explained as microaneurysms with or without surrounding microhemorrhages. They are, in addition, closely related to systemic hypertension and ICH. We have not shown pathologic correlation, and to confirm the nature and underlying mechanisms of the signal void dots, and to demonstrate the pathologic basis of our observations, further investigation is needed.

References

1. Feldmann E. Intracerebral hemorrhage. *Stroke* 1991 ; 22 : 684-691
2 Scharf J, Brauherr E, Forsting M, Sartor K. Significance of hemorrhagic lacunes on MRI in patients with hypertensive cerebrovascular disease and intracerebral hemorrhage. *Neuroradiology* 1994 ; 36 : 504-508
3. Multifocal hypointense cerebral lesions on gradient-echo MR are associated with chronic hypertension. Chan S, Kartha K, Yoon SS, Hilal SK. *AJNR* 1996 ; 17 : 1821-1827
4. Bradley, Jr WG. MR appearance of hemorrhage in the brain. *Radiology* 1993 ; 189 : 15-26
5. Fisher CM. Pathological observations in hypertensive cerebral hemorrhge. *J Neuropathol Exp Neurol* 1971 ; 30 : 536-550
6. Charcot JM, Bouchard C. Nouvelle recherches sur la pathog ne de l'hemorrhagie cérébrale. *Arch Physiol Normale Pathol* 1868 ; 1 : 110-127,643-665
7. Fisher CM. Cerebral miliary aneurysms in hypertension. *Am J Pathol* 1972 ; 66 : 313-330
8. Phillips SJ, Whisnant JP. Hypertension and the brain. *Arch Intern Med* 1992 ; 152 : 938-945
9. Russell RWR. Observations on intracerebral aneurysms. *Brain* 1963 ; 86 : 425-442
10. Cole FM, Yates PO. The occurrence and significance of intracerebral micro-aneurysms. *J Pathol Bacteriol* 1967 ; 93 : 393-411
11. Spangler KM, Challa VR, Moody DM, Bell MA. Arteriolar tortuosity of the white matter in aging and hypertension. A microradiographic study. *J Neuropathol Exp Neurol* 1994 ; 53 : 22-26

대한방사선의학회지 1997;37:195-200

고혈압성 뇌실질출혈환자의 T2강조 자기공명영상에서 나타나는 신호소실반점:정체와 임상적 의의[1]

[1]단국대학교 의과대학 진단방사선과학교실
[2]단국대학교 의과대학 신경과학교실

김상준 · 유동수 · 김승철 · 김태훈 · 김재승 · 김재일[2]

목 적 : 고혈압 환자의 뇌의 T2 강조 자기공명영상에서 발견되는 신호소실 반점을 기술하고 임상적 중요성을 알고자 하였다.

대상 및 방법 : 고혈압성 뇌실질 출혈 환자 11명, 열공(lacunar) 뇌경색 환자 14명, 60세 이상의 정상 대조군 11명의 T2 강조영상에서 신호소실 반점의 존재여부, 숫자, 크기에 관해 분석하였고 신호소실반점과 고혈압과의 상관관계를 알아보았다. 고혈압성 뇌실질 출혈 환자 일부에서 TOF 또는 PC 자기공명혈관조영술, gradient echo 영상, 고식적 뇌혈관조영술을 시행하고 이를 T2 강조영상과 비교 분석 하였다.

결 과 : 신호소실 반점은 고혈압성 뇌실질 출혈 환자 11명 모두에서 발견되었고 14명의 열공 경색환자 중 6명에서 발견되었으며 이 6명은 모두 고혈압 환자였다. 정상 대조군에서는 신호소실 반점이 관찰되지 않았다. 신호소실 반점은 대부분 시상, 뇌교 및 기저핵에서 발견되었고 gradient echo 영상에서 약간 커보였다. 크기는 1-4 mm 로 대부분 2 mm 내외였다. 자기공명혈관조영술이나 고식적 뇌혈관조영술상 신호소실 반점이 있는 위치에서 혈관의 확장 소견은 발견되지 않았다.

결 론 : 신호소실 반점은 정상적인 노화과정에서 나타나는 것은 아니며 고혈압 및 고혈압성 뇌실질출혈과 밀접한 관계가 있었다. 신호소실 반점은 혈류 인공물이나 확장된 혈관이 아니고 혈액 성분에 의한 자화율효과(susceptibility effect)에 의한 것으로 생각된다.