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Role of magnetic susceptibility-weighted imaging in characterization of cerebral microbleeds in acute ischemic stroke Egyptian obese patients

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Abstract

Objective: The study was done to investigate the role of susceptibility-weighted imaging (SWI) in characterization of cerebral microbleeds (CMBs) and incidence of CMBs in acute ischemic stroke obese patients.

Materials and methods: We recruited 120 acute ischemic stroke obese patients. All participants or their relatives gave written informed consent. MRI of the brain with SWI sequences was done for all patients.

Results: SWI showed 30% of the patients have CMBs. The total numbers of CMBs were statistically significant higher in small vessel diseases (SVDs) than large vessel diseases (LVDs) ($P = 0.008$). According to the severity scale, CMBs were more prominent in patients with grade 0 (70%) followed by grade III (25.8%) then grade II (4.2%). BMI was independent risk factors for developing CMBs as shown in simple regression analysis ($x^2 = 6.008$, $P = 0.002$). SVDs and hypertension were statistically significant independent predictors and had higher odds to exhibit CMBs. Patients with SVDs had 5.3 times higher odds to exhibit CMBs ($P = 0.002$) while patients with hypertension had 3.9 times higher odds to exhibit CMBs ($P = 0.032$).

Conclusion: SWI is a gold standard MR imaging technique in diagnosis and characterization of CMBs with more prevalence especially in obese patients suffering from acute ischemia stroke due to SVDs. Hypertension was an independent risk factor for development of CMBs but IHD, use of antiplatelet medications, increased BMI, and dyslipidemia must be taken into consideration.

Keywords: Cerebral microbleeds, SWI, Ischemic stroke, Obesity, Vessel disease

Introduction

Obesity has a detrimental effect on health; it increases the risk of developing hypertension, cardiovascular disease, and cerebral stroke [1, 2]. Obesity is considered a modifiable risk factor for both hemorrhagic and ischemic stroke in both sexes [3].

Several studies reported the linear relationship between body mass index (BMI) and ischemic stroke but not hemorrhagic stroke [4].

Cerebral microbleeds (CMBs), white matter hyperintensities (WMH), and lacunes; all are common radiological imaging biomarkers of cerebral small vessel diseases (SVDs) [5].

CMBs have been observed in patients with ischemic and hemorrhagic stroke with frequency between 4 and 5% in healthy people, and can increase to 50–70% in patients with cerebrovascular disease [6].

Susceptibility-weighted imaging (SWI) is a newly developed MRI technique. SWI can differentiate the magnetic susceptibility effect of different components like blood products, deoxygenated blood, calcium, and iron. SWI employs a fully flow-compensated, high-resolution, 3D gradient-echo pulse using either separate or

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combined phase and magnitude images, in order to characterize brain tissue. Postprocessing of SW imaging includes combination of magnitude images with phase images [7].

SWI allows better detection of CMBs compared with 2D using a gradient-echo (GRE) pulse sequence. The high sensitivity detection rate of CMBs by SWI is due to the longer scan time as well as the high-spatial resolution 3D imaging [8].

In SWI, CMBs appear as small hypointense foci with size ranged from 2 to 10 mm. CMBs occur as a result of focal accumulations of hemosiderin-containing macrophages that have a paramagnetic effect causing signal loss due to susceptibility effects [9].

In MRI, phase images can differentiate between calcification from CMBs as iron is a paramagnetic material while calcium is a diamagnetic material with opposing phase shifts so CMBs appear either low signal or high signal on phase image depending on the type of MR machine, whether the imager uses a left- or right-handed coordinate system [8].

CMBs are commonly asymptomatic but are considered a predictive factor of future recurrent ischemic and hemorrhagic stroke and considered to be clinically correlated with chronic hypertension, aging, and WMH [10, 11].

CMBs were classified into three groups according to their locations in SWI: lobar (cortical-subcortical white matter), deep (deep gray matter, basal ganglia, thalamus, the white matter of corpus callosum, internal, external and extreme capsule), and infra-tentorial (brain stem and cerebellum) [12, 13].

The distribution pattern of CMBs indicates different mechanisms, CMBs located in the deep white matter associated with hypertensive arteriopathy, whereas those in the lobe are more likely associated with cerebral amyloid angiopathy (CAA), with recent research shows that lobar CMBs are associated with changes in cognitive function [14, 15].

Many studies have shown that CMBs have not an important impact on hemorrhagic transformation only but they also consider as an independent risk factor for new ischemic stroke, transient ischemic attack, and stroke recurrence [16]. Therefore, the role of CMBs is important for the prevention and treatment of various strokes [17].

The aim of this study was to investigate the role of SWI in characterization of CMBs and incidence of CMBs in acute ischemic stroke obese patients.

Patients and methods

This is a cross sectional study that was approved by Research Ethics Committee (REC) for Human Subject and Animal Research at the Faculty of Medicine, Helwan University, Cairo, Egypt (serial13-2020). We recruited 120 patients admitted to the neurology department of Helwan University Hospitals from February to April 2020, these patients were obese and presented with acute ischemic stroke diagnosed by computed tomography (CT) brain, and all participants or their relatives gave written informed consent. The procedures we performed were in accordance with our protocol; brain MRI with SWI sequences was done for all patients.

We recruited obese patients of both sexes, ≥ 60 years of age; patients that were before this age, showed mimic ischemic stroke diseases, refused consent, or who were contraindicated to brain MRI were excluded. All patients underwent full history taking and complete neurological examination stressing on medication drugs especially anticoagulants and antiplatelet therapy with high focus on vascular risk factors. Blood samples for laboratory investigation were obtained after overnight fasting and serum total cholesterol (TC), high-density lipoprotein (HDL-C), low-density lipoprotein cholesterol (LDL-C), triglyceride (TG), and fasting plasma glucose levels.

Acute ischemic stroke subtype SVDs and large vessel disease (LVDs) were diagnosed on the basis of cranial CT and MRI findings together with medical history, neurological signs and symptoms, and physical examination obtained at admission.

Vascular risk factors

Cerebral small vessel disease refers to a diverse range of clinical and neuroimaging findings resulting from pathological changes of various etiologies affecting the cerebral small vessels, particularly small veins, venules, capillaries, arterioles, and small arteries [18]. Cerebral large vessel disease is defined as disease affecting the proximal intracranial anterior and posterior circulation [19].

Hypertension was defined as self-reported hypertension, treatment with antihypertensive medication, a systolic blood pressure (SBP) ≥ 140 mmHg, and/or diastolic blood pressure (DBP) ≥ 90 mm Hg [20]. Diabetes mellitus was defined as a fasting blood glucose level 126 mg/dL, a random blood glucose level 200 mg/dL, or treatment with oral antidiabetic drugs or insulin [21].

Dyslipidemia was defined as a serum TC level 200 mg/dl, serum TG ≥ 150 mg/dL, HDL-C ≤ 40 mg/dL in males and ≤ 50 mg/dL in female, LDP-C ≥ 100 mg/dL, or treatment with lipid-lowering drugs [22]. A current smoker was defined as a person who smoked at least 1 cigarette per day habitually or some days in the last 30 days preceding an interview [23]. Body mass index (BMI) calculated as weight in kilograms divided by height in meter squared and classified patients into overweight ≥ 25(pre-obese 25–29.99) and obese ≥ 30 (grade I (30–34.99), grade II (35–34.99), and grade III ≥ 40) [24].

MRI and SWI sequence protocol

All 120 patients performed conventional MRI with axial SWI images by 1.5-T MR machine (Philips, Achieva 1.5

T, Philips health care, Netherlands Holland); the following sequences were done: axial TSE T1-axial-weighted image (TR 400 ms TE 14 ms flip angle 90°), axial and coronal TSE T2-weighted image (WI, TR 2500 ms TE 88 ms flip angle, 90°), and axial and sagittal FLAIR FS (TR 9000 ms TE 127 ms). Diffusion-sensitizing gradients were done, axial SWI (TR 78 ms TE 47 ms flip angle 15°slice thickness 1.5 mm) both the magnitude and phase images were done, and then post processing was done by combining the phase and magnitude images to create SWI and also created minimum intensity projection image (MIP)

Image analysis

CMBs were described as small round foci with hypointense signals that ranged from 2 to 10 mm in diameter on the SWI images.

Other areas of hypointense signal loss as vascular flow voids, areas of symmetric hypointensity, and areas in the basal ganglia, that represented calcification or non-hemorrhagic iron deposits, were excluded. Small foci of calcifications displayed hypointense signal loss in SWI images were excluded by phase images.

CMBs were classified into 3 types according to their location: lobar type that includes cortical or subcortical location (Fig. 1); deep type that included deep gray matter, thalamus, basal ganglia as well as white matter of the external, internal, extreme capsule, corpus callosum, cerebellum and brain stem (Fig. 2); and mixed type (Fig. 3) [11].

CMBs foci number was reported for each patient. According to the number of foci, the CMB severity was classified into 4 grades: in which grade 0 = absent CMBs foci, grade 1 = 1 CMBs foci lesion, grade 2 = 2 to 5 CMBs foci lesions, grade 3 = more than 6 CMBs foci lesions [25].

Statistical analysis

Data were entered and analyzed using IBM-SPSS software (IBM Corp. released 2017; IBM SPSS Statistics for Windows, Version 25.0. Armonk, NY: IBM Corp.). Qualitative data were expressed as frequency and percentage. Chi-square test or Fisher's exact test according to sample size of cells (expected counts) was used for comparing qualitative data. Chi-square test was used if sample size was ≥ 5 units per cell while Fisher's exact test was used if sample size was < 5 units in ≥ one cell. Quantitative data were initially tested for normality using Shapiro-Wilk's test with data being normally distributed if $P > 0.050$. Presence of significant outliers (extreme values) was tested for by inspecting boxplots. Quantitative data were expressed as mean ± standard deviation (SD) if normally distributed or median and interquartile range (IQR) if not. To compare quantitative data for two groups, Mann-Whitney U test was used. Binary logistic regression analysis was used to predict the likelihood of occurrence of microbleeds. Crude odds ratio was initially calculated for each predictor and then a model was run to get the odds ratios of independent predictors. For any of the used tests, results were considered as statistically significant if P value ≤ 0.050.

Results

Qualitative and quantitative characteristics of the study cohort: Our study included 120 patients. Fifty-four patients were (45%) males and 66 patients were (55%) females; the participants aged from 60 to 88 years and their median age was 68 years [IQR was 64–75]. As regards BMI, 43 (35.8%) patients were overweight, 77 (64.2%) patients were obese [grade I = 53, grade II = 18, grade III = 6 patients]. The median BMI was 31.1(maximum was 45 and minimum was 25.8) and IQR 29.1–34.7.
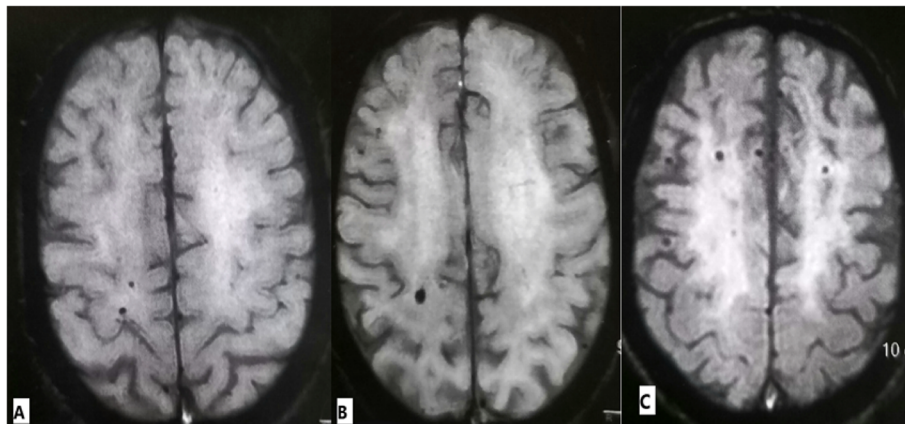

Fig. 1 Lobar type cerebral microbleeds. Axial brain SWI images (**a**–**c**) show multiple signal loss areas of cerebral microbleeds in the cortical gyri and subcortical white matter
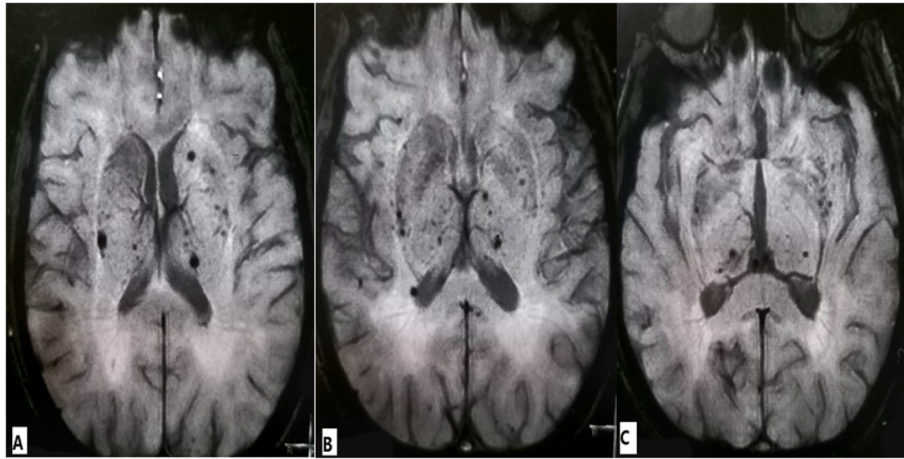
Fig. 2 Deep type cerebral microbleeds. Axial brain SWI images (**a**–**c**) show multiple signal loss areas of cerebral microbleeds in the periventricular deep white matter, basal ganglia, and both thalami

In our study, there were 62 (51.7%) hypertensive patients, 48(40%) diabetes mellitus (DM) patients, 53(44.2%) IHD patients, 17 patients (14.2%) were suffering from atrial fibrillation (AF), 25 patients (20.8%) were smokers, 51patients (42.5%) were on antiplatelet medications, 17 patients (14.2%) were on anticoagulant medication, and 54 patients (45%) suffered from dyslipidemia.

Brain MRI was done to all patients; 32 patients (26.7%) suffered from SVDs and 88 patients (73.3%) suffered from LVDs. SWI showed 36 patients (30%) of cases suffered from CMBs (31 cortical, 35 deep, 36 mixed, and 18 infra-tentorial) (Table 1).

Frequency of CMBs
By SWI lobar type, CMBs ranged from 0 to 57; its median was 5 and IQR was 3 to 9 in SVDs, while in LVDs, CMBs ranged from 0 to 33, its median was 2 and IQR was 1–8. Lobar CMBs were statistically significantly higher in SVDs than LVDs ($P = 0.066$).

Deep type CMBs ranged from 0 to 80, its median was 10, and IQR was 7 to 23 in SVDs while it ranged from 1 to 48, its median was 4, and IQR was (2 to 8) in LVDs. Deep CMBs were statistically significantly higher in SVDs than LVDs ($P = 0.008$).

Mixed type CMBs ranged from 6 to 125, its median was 15, and IQR was 9 to 32 in SVDs, while LVD ranged from 2 to 81, its median was 7, and IQR was 3 to 14. Mixed type CMBs were statistically significantly higher in SVDs than LVDs ($P = 0.009$).

Infra-tentorial type CMBs ranged from 0 to 18, its median was 3, and IQR was 0 to 5 in SVDs, while it ranged from (0 to 3), its median was 0, and IQR was (0 to 2) in LVDs. Infra-tentorial CMBs were statistically significantly higher in SVDs than LVDs ($P = 0.025$).
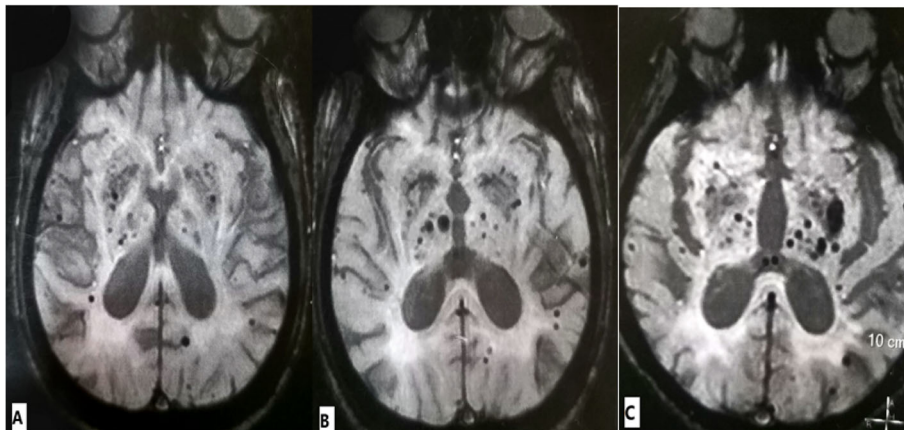

Fig. 3 Mixed type cerebral microbleeds. Axial brain SWI images (**a**–**c**) show bilateral multiple signal loss areas of cerebral microbleeds in the, basal ganglia, both thalami, deep periventricular and subcortical white matter, and cortical gyri

Table 1 Characteristics of the study cohort ($n = 120$)

Qualitative data	Frequency (%)
Sex	
Male	54 (45)
Female	66 (55)
BMI category	
Overweight	43 (35.8)
Obese	77 (64.2)
Grade I	53
Grade II	18
Grade III	06
Presence of hypertension	62 (51.7)
Presence of diabetes	48 (40)
Presence of dyslipidemia	54 (45)
Presence of IHD	53 (44.2)
Presence of AF	17 (14.2)
A current smoker	25 (20.8)
On antiplatelet medications	51 (42.5)
On anticoagulant medications	17 (14.2)
Brain MRI	
SVDs	32 (26.7)
LVDs	88 (73.3)
SWI	
CMBs	36 (30)
lobar	31
Deep	35
Mixed	36
Infra-tentorial	18
Quantitative data	Median (IQR), minimum, maximum
Age (years)	68 (64–75), 60, 88
BMI (kg/m^2)	31.1 (29.1–34.7), 25.8, 45
Serum TC (mg/dL)	186 (152–249), 103, 275
Serum TG (mg/dL)	139.5 (109.3–162), 49, 280
Serum LDL-C (mg/dL)	44 (39.3–56), 23, 106
Serum HDL-C (mg/dL)	97 (69–151.5), 32.8, 184

IHD ischemic heart disease, *AF* atrial fibrillation, *MRI* magnetic resonance imaging, *SWI* susceptibility-weighted imaging, *BMI* body mass index, *SVDs* small vessel disease, *LVDs* large vessel disease, *CMBs* cerebral microbleeds, *TC* total cholesterol, *TG* triglycerides, *LDL* low-density lipoprotein, *HDL* high-density lipoprotein

In comparing total numbers of CMBs in SVD and LVD, there were statistically significant higher number of CMBs in SVDs than LVDs ($P = 0.008$) (Table 2).

Severity of CMBs

According to severity of CMBs by SWI, there were 84 patients (70%) with grade 0, 5 patients (4.2%) grade II, and 30 patients (25.8%) grade III and there was no patient in grade I.

Univariate analysis of predictors of CMBs

Simple logistic regression test was run to ascertain the effects of sex, BMI (kg/m^2), hypertension, DM, dyslipidemia, AF, IHD, smoking history, antiplatelet drugs, anticoagulant therapy, MRI, serum TC (mg/dl), serum TG (mg/dl), LDL-C (mg/dl), and serum HDL-C (mg/dl) on the likelihood that participants CMBs. Of all univariate predictors of CMBs, the simple logistic regression model was statistically significant by comparing obese versus overweight ($x^2 = 6.008$, $P = 0.002$) and has 3.107 higher odds to exhibit CMBs. An increase in BMI category by one class has 2.223 higher odds to exhibit CMBs. The simple logistic regression model was statistically significant as univariate predictors of CMBs for dyslipidemia versus non-dyslipidemia ($x^2 = 7.414$, $P = 0.008$) has 3.025 odds to exhibit CMBs; also the simple logistic regression model was statistically significant as univariate predictors of CMBs for IHD, antiplatelet drug therapy ($P = 0.016$ and $P = 0.023$), respectively. The simple logistic regression model for brain MRI showed SVDs versus LVDs was the strongest univariate predictors of CMBs ($x^2 = 31.202$, $P < 0.001$) and had 11.629 higher odds to exhibit CMBs followed by hypertension versus normotensive patients ($x^2 = 20.652$, $P < 0.001$) and had 8.125 higher odds to exhibit CMBs as in Table 3

Predictors of the likelihood of occurrence of CMBs

A binomial logistic regression was run to ascertain the effects of presence of SVDs on MRI, presence of hypertension, BMI (kg/m2), serum TC (mg/dl), LDL-C (mg/dl), presence of IHD, and use of antiplatelet medications on the likelihood that participants have microbleeds. There were 5 studentized residuals with values of 2.167, 2.167, 2.477, 2.002, and 2.272 SD which were kept in the analysis. The logistic regression model was statistically significant, $\chi^2 (7) = 39.376$, $P < 0.001$. The model explained 39.7% (Nagelkerke R^2) of the variance in microbleeds and correctly classified 82.5% of cases. Sensitivity was 61.1%, specificity was 91.7%, positive predictive value was 75.9%, and negative predictive value was 84.6%. Of the eight predictor variables, only presence of small vessel disease on MRI and presence of hypertension were statistically significant independent predictors. Patients with small vessel disease on MRI had 5.3 times higher odds to exhibit microbleeds ($P = 0.002$) and patients with hypertension had 3.9 times higher odds to exhibit microbleeds ($P = 0.032$) as in Table 4.

Discussion

In this study, SWI showed 36 patients suffered from CMBs (31 cortical, 35 deep, 36 mixed and 18 infra-tentorial); according to that, the prevalence of CMBs in this study was 30% (68.8% in SVDs &15.9% in LVDs) with high statistically significantly difference in SVDs (χ^2

Table 2 Frequency and location of CMBs

Location	SVD ($n = 32$)		LVDs ($n = 88$)		Z value	P value
	Median (IQR)	Range	Median (IQR)	Range		
Lobar	5 (3–9)	0–57	2 (1–8)	0–33	− 1.845	0.066
Deep	10 (7–23)	0–80	4 (2–8)	1–48	− 2.621	0.008
Mixed	15 (9–32)	6–125	7 (3–14)	2–81	− 2.584	0.009
Infra-tentorial	3 (0–5)	0–18	0 (0–2)	0–3	− 2.399	0.025
Total	16 (12–34)	7–143	8 (3–16)	2–81	− 2.618	0.008

P value: Mann-Whitney U test. SVDs small vessel diseases, LVDs large vessel diseases

= 31.201, P < 0.001); while Potigumjon and his colleagues reported that the prevalence of CMBs in ischemic stroke patients was 20%, Kwa and his colleagues found that the incidence of CMBs in ischemic stroke patient was 6% [26, 27].

In our study, the number of CMBs was statistically significantly higher in SVDs versus LVDs in comparing location: deep, mixed (lobar and deep), infra-tentorial, and total number in both groups (P = 0.008, P = 0.009, P = 0.025, P = 0.008), respectively, but not in lobar location (P = 0.066).

In this study, we observed the majority of the cases presented with CMBs were mixed, deep, lobar then infra-tentorial location; this was inconsistent with Pantoni who reported that CMBs were mainly observed in lobar brain regions [28].

As regards MBI, CMBs were higher significantly difference in the obese group when compared with the overweight group which had 3.107 higher odds to exhibit CMB (x^2 = 6.008, P = 0.017), furthermore, an increase in BMI category by one grade has 2.223 higher odds to exhibit CMB, (grade II = 4.446, grade III = 6.669) (P = 0.002). This is consistent with Mitaki and his colleagues; they found that increased BMI was associated with increased incidence of deep and infra-tentorial CMBs; however, the positive association between incidence of CMBs and BMI is still controversial [11].

In our study hypertension was strong independent risk factor in development of CMBs (x^2 = 20.652, P < 0.001); this was consistent with Vernooij et al. [10] as they reported CMBs were independently associated with higher systolic blood pressure and inconsistent with Mitaki and his colleagues who found that the hypertension were not associated with increased incidence of CMBs [11].

In this study, we found that CMBs were statistically significantly higher in dyslipidemic patients (x^2 = 7.414, P = 0.008). Serum TC and serum LDL-C were significantly associated with increased incidence of CMBs (P = 0.002 and P = 0.004), respectively, with no association with serum TG and serum HDL-C. This is inconsistent with Mitaki et al. [11] that dyslipidemia was not associated with increased incidence of CMBs.

In this study, we founded that patients who suffered from IHD and received antiplatelet drugs were more prone to develop CMBs with higher statistically significant difference when compared with nonusers (x^2 = 5.988, P = 0.016); this is consistent with Huo and his colleagues and also with Vernooij and his colleagues that the prevalence of CMBs were higher in patients treated with antiplatelet drugs used in the primary and secondary prevention of thrombotic cardiovascular disease, including acute coronary syndrome compared with nonusers [29, 30].

We founded in our research that the prevalence of CMB was higher in patients who received antiplatelet with a statistically significant difference when compared with nonuser (58.3%), P = 0.023. while Lihong and his colleagues reported that CMBs occurred in 40.0% of patients taking aspirin for at least 1 year and 30.7% of patients taking clopidogrel for at least 1 year [31].

In our study, we founded that patients with SVDs on brain MRI were statistically significantly higher when compared with LVDs to develop CMBs (x^2 = 31.201, P < 0.001).

In this study, we did not find any statistically significant difference as regards sex (female and male), diabetic and non-diabetic, and AF and non AF patients; patients received anticoagulant therapy, serum TG, and serum HDL-C in prevalence of CMBs. Mitaki et al. [11] found that elevated fasting glucose was not associated with increased incidence of CMBs, inconsistent with Saito and his colleagues who reported that the incidence of CMBs in patients with AF is significantly higher than that in patients without AF [32].

In our research, we found that a binomial logistic regression to ascertain the effects of presence of SVDs on MRI, presence of hypertension, BMI (kg/m2), serum TC (mg/dl), LDL-C (mg/dl), presence of IHD, and use of antiplatelet medications on the likelihood that participants had microbleeds. Of the seven predictor variables, only the presence of small vessel disease on MRI and presence of hypertension were statistically significant independent predictors to develop CMBs. Patients with SVDs on SWI had 5.3 times higher odds to exhibit microbleeds (P =

Table 3 Univariate analysis of predictors of CMBs

Predictor	No microbleeds (n = 84)	Microbleeds (n = 36)	Test of significance		Simple logistic regression	
			Statistic	P1	Crude odds ratio	P2
Sex						
Female	45 (53.6%)	21 (58.3%)	$\chi^2 = 0.231$	0.631	R	0.631
Male	39 (46.4%)	15 (41.7%)			0.824	
BMI						
Overweight	36 (42.9%)	7 (19.4%)	$\chi^2 = 6.008$	0.014	R	0.017
Obese	48 (57.1%)	29 (80.6%)			3.107	
Grade I	37 (44.0%)	16 (44.4%)	FET	0.010	2.223	0.002
Grade II	09 (10.7%)	09 (25.0%)				
Grade III	02 (2.4%)	04 (11.1%)				
Hypertension						
No	52 (61.9%)	6 (16.7%)	$\chi^2 = 20.652$	< 0.001	R	< 0.001
Yes	32 (38.1%)	30 (83.3%)			8.125	
DM						
No	49 (58.3%)	23 (63.9%)	$\chi^2 = 0.324$	0.569	R	0.570
Yes	35 (41.7%)	13 (36.1%)			0.791	
Dyslipidemia						
No	53 (63.1%)	13 (36.1%)	$\chi^2 = 7.414$	0.006	R	0.008
Yes	31 (36.9%)	23 (63.9%)			3.025	
AF						
No	69 (82.1%)	34 (94.4%)	$\chi^2 = 3.136$	0.077	R	0.094
Yes	15 (17.9%)	2 (5.6%)			0.271	
IHD						
No	53 (63.1%)	14 (38.9%)	$\chi^2 = 5.988$	0.014	R	0.016
Yes	31 (36.9%)	22 (61.1%)			2.687	
On antiplatelet drugs						
No	54 (64.3%)	15 (41.7%)	$\chi^2 = 5.276$	0.022	R	0.023
Yes	30 (35.7%)	21 (58.3%)			2.520	
On anticoagulant drugs						
No	69 (82.1%)	34 (94.4%)	$\chi^2 = 3.136$	0.077	R	0.094
Yes	15 (17.9%)	2 (5.6%)			0.271	
Brain MRI						
LVDs	74 (88.1%)	14 (38.9%)	$\chi^2 = 31.201$	< 0.001	R	< 0.001
SVDs	10 (11.9%)	22 (61.1%)			11.629	
Age (years)	68.5 (63–75)	68 (65.3–73.8)	$Z = -0.077$	0.938	0.988	0.689
BMI (kg/m^2)	30.6 (28.7–33.97)	33.5 (30.9–37.2)	$Z = -3.196$	0.001	1.195	0.001
Serum TC (mg/dl)	179.5 (144.5–227)	233 (175–251)	$Z = -3.064$	0.002	1.013	0.002
Serum TG (mg/dl)	129 (109–161)	146.5 (110.5–169.5)	$Z = -0.465$	0.642	1.000	0.918
Serum HDL-C (mg/dl)	43 (35.3–51.8)	44.5 (41.3–59)	$Z = -1.750$	0.080	1.029	0.037
Serum LDL-C (mg/dl)	96 (68–137.8)	141 (69–175.8)	$Z = -3.112$	0.002	1.014	0.004

R reference category, *COR* crude odds ratio, *P1, P2* simple logistic regression, *DM* diabetes mellitus, *AF* atrial fibrillation, *IHD* ischemic heart disease, *LVDs* large vessel diseases, *SVDs* small vessel diseases, *BMI* body mass index, *TC* total cholesterol, *TG* triglyceride, *HDL-C* high-density lipoprotein-C, *LDI-C* low-density lipoprotein-C

Table 4 Predictors of the likelihood of occurrence of CMBs

Predictor	B	SE	Wald	P value	OR (95% CI)
Brain MRI	1.669	0.551	9.176	0.002	5.307 (1.802–15.627)
LVDs					
SVDs					
Hypertension	1.363	0.637	4.586	0.032	3.909 (1.123–13.612)
No					
Yes					
BMI (kg/m^2)	− 0.015	0.080	0.037	0.848	0.985 (0.842–1.152)
Serum TC (mg/dl)	0.004	0.012	0.082	0.775	1.004 (0.980–1.028)
Serum LDL-C (mg/dl)	0.001	0.014	0.003	0.958	1.001 (0.874–1.028)
IHD	0.386	1.586	0.059	0.808	1.471 (0.266–32.910)
No					
Yes					
On antiplatelet medications	0.426	1.597	0.071	0.790	1.531 (0.067–35.021)
No					
Yes					

B binary logistic regression coefficient, *SE* standard error, *OR* odds ratio, *CI* confidence interval, *LVDs* large vessel diseases, *SVDs* small vessel diseases, *BMI* body mass index, *TC* total cholesterol, *LDI-C* low-density lipoprotein, *IHD* ischemic heart disease

0.002), and patients with hypertension had 3.9 times higher odds to exhibit microbleeds (*P* = 0.032)

Conclusion

SWI is a gold standard MR imaging technique in diagnosis and characterization of CMBs with more prevalence especially in obese patients suffering from acute ischemia stroke due to small vessel disease. Hypertension is an independent risk factor for the development of CMBs but IHD, use of antiplatelet medications, increased BMI, and dyslipidemia must be taken into consideration.

Abbreviation
AF: Atrial fibrillation; BMI: Body mass index; BP: Blood pressure; CAA: Cerebral amyloid angiopathy; CMBs: Cerebral microbleeds; CT: Computed tomography; DBP: Diastolic blood pressure; DM: Diabetes mellitus; GRE: Gradient recall echo; HDL: High-density lipoprotein; IHD: Ischemic heart disease; IQR: Interquartile range; LDL: Low-density lipoprotein; LVDs: Large vessel diseases; MRI: Magnetic resonance imaging; SBP: Systolic blood pressure; SD: Standard deviation; SVDs: Small vessel diseases; SWI: Susceptibility-weighted imaging; TC: Total cholesterol; TG: Triglyceride; WI: Weighted imaging; WMD: White matter disease

Authors' contributions
AAM and CAZ carried out the work. AAM performed the procedure, did the statistical analysis, and was responsible for collecting the scientific data and writing the initial draft of the manuscript. CAZ designed the protocol shared in collecting the scientific data. CAZ interpreted the radiology of all patients. Both authors read and approved the final version to be published.

Availability of data and materials
The data supporting the results of this article are included within the article.

Ethics approval and consent to participate
The authors obtained permission to conduct this study that was approved by Research Ethics Committee (REC) for Human Subject and animal Research at the Faculty of Medicine, Helwan University, Cairo, Egypt (serial 13-2020). All participants gave written informed consent. The procedures followed were in accordance with our protocol. We recruited 120 patients admitted to the neurology department of Helwan University Hospitals from February to April 2020.

Consent for publication
Not applicable.

Competing interests
The authors declare that they have no competing interests.

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