Al-Azhar Med. J. *Vol. 48(4), October, 2019, 351-362*
DOI : 10.12816/amj.2019.64943
h ps://amj.journals.ekb.eg/ar cle_64943.html

ASSOCIATION BETWEEN CEREBRAL SMALL VESSEL DISEASE AND MILD MOTOR PARKINSONIAN SIGNS IN PATIENTS WITH LACUNAR STROKES AND TRANSIENT ISCHEMIC ATTACKS

By

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ABSTRACT

Background: Both cerebral small vessel disease (SVD) and Mild Parkinsonian signs (MPS) are serious conditions affecting elderly patient, but, the association between them remain unclear.

Objective: Examine the association between SVD, Total SVD burden and MPS in patients with Transient Ischemic Attacks (TIA) and Lacunar Strokes.

Patients and Methods: We performed a cross-sectional study among 36 patients admitted with either TIA or Lacunar Stroke, but not known to have Parkinson's disease or Parkinsonism. Patients were recruited from Al-Azhar University Hospitals during the period from June, 2016 to May 2018. Mean age was 73.38 ± 8.3 years, 58.3% males. MPS was evaluated via Unified Parkinson's Disease Rating Scale Part III. Brain MRI was used to determine SVD (lacunar infarctions [LIs], white matter hyperintensities [WMH], cerebral microbleeds [CMBs], and Enlarged Perivascular spaces [EPVSs]). Total SVD score was calculated based upon presence/absence of these four SVD markers.

Results: In a multivariate analysis, we found that the presence of LIs, WMHs, CMBs, and total SVD score were significantly associated with MPS, whereas EPVSs were not. We found a strong correlation between SVD score & MPS score. We also found a significant association between total SVD score and Rigidity & Bradykinesia. GFR was found to significantly correlate with SVD score & MPS score.

Conclusion: Our results provided additional evidence that SVD and especially total SVD burden, might be a surrogate marker for MPS and support the support the hypothesis of a vascular contribution to MPS in older adults.

Keywords: Small vessel disease – total SVD score – mild parkinsonian signs.

INTRODUCTION

Cerebral Small vessel disease (SVD) is a disorder affecting the small perforating vessels in the brain *(Pantoni, 2010)*, and is considered one of the most prevalent pathologic processes encountered by neurologists in clinical practice *(Nam et al., 2017)*. It causes up to 45% of dementia, 25% of ischaemic strokes *(Shi*

et al., 2016). In addition to, gait and balance dysfunction and dysfunction of the bladder *(Wardlaw et al., 2013)*.

SVD is diagnosed on the basis of brain imaging. On brain MRI, four closely correlated features are now considered markers of SVD: Lacunar infarcts (LIs), white matter lesions (WMLs), cerebral microbleeds (CMBs), and enlarged perivascular spaces (EPVS). *(Huijts et al., 2013, Staals et al., 2014* and *Cannistraro et al., 2019)*.

A total SVD burden score was proposed, which incorporates these four established markers of SVD *(Lau et al., 2017)*. There is increasing evidence that this score, might better reflect the global overall effect of SVD on the brain than any individual marker separately *(Pinter et al., 2017)*.

Another known cause of functional impairment in the elderly is the development of mild motor Parkinsonian signs (MPS), which refer to subtle motor features of four domains; bradykinesia, rigidity, tremor as well as gait and postural abnormalities *(Wada-Isoe et al., 2016)*. These signs are related to adverse health outcomes including development of functional disability, cognitive impairment, dementia, increased risk of Parkinson's disease (PD), and increased mortality *(Berg et al., 2013)*.

The identification of SVD-related MPS at an early stage might be an important strategy for preventing and delaying functional disability, progression to Parkinsonism or Parkinson's disease *(Li et al., 2011)*.

The present work aimed to assess the association between cerebral small vessel disease, total SVD burden and mild parkinsonian signs in patients with lacunar strokes and transient ischemic attacks.

PATIENTS AND METHODS

Patients were recruited from Al-Azhar university hospitals (Al-Hussain and Sayed Galal hospitals), during the period from June, 2016 to May 2018. All subjects enrolled in our study were older than 60 years, admitted to hospital either with transient ischemic attack (TIA), or with first ever lacunar stroke with mild neurological deficit; national institutes of health stroke scale (NIHSS) ≤ 4. Patients with history of previous strokes, large non-lacunar strokes, significant neurological deficit (NIHSS > 4), debilitating illness, or with a known diagnosis of Parkinson's disease or Parkinsonism were excluded.

Assessment of all subjects included full medical past history, detailed clinical examination with specific stress on assessment of NIHSS score, and careful search for the presence of MPS.

Assessment of Mild Parkinsonian Signs: MPS were assessed for all study subjects, using an abbreviated (9-item) version of motor section (section III), of the unified parkinson disease rating scale (UPDRS), addressing mainly evaluation of speech, facial expression, tremor at rest, rigidity, finger taps, leg agility, posture, gait and body bradykinesia, with later further categorization of these clinical features into the main categories of MPS, i.e. rigidity, bradykinesia, gait and postural disturbances, and tremors.

As in previous large-scale studies, MPS were defined as present when any 1

of the following conditions was met (De Laat et al., 2012):

- 1 UPDRS rating of 2 or higher.
- 2 or more UPDRS ratings of 1 or higher.

MRI scanning and Evaluation: All MRI scans of all study subjects were acquired on a single 1.5 T MRI scanner. The protocol included axial diffusion-weighted imaging (DWI), with apparent diffusion coefficient (ADC) map, standard axial T2–weighted, axial fluid–attenuated inversion recovery (FLAIR), standard T1–weighted, and susceptibility-weighted images (SWI).

All images were carefully evaluated and rated for the presence of the four previously specified markers of SVDs, i.e. lacunar infarcts, white matter Hyperintensities, cerebral microbleeds, and enlarged perivascular spaces (figure 1).

LI was defined as hyperintense lesions on T2-weighted images that have corresponding hypointense lesions with a hyperintense rim on FLAIR images located in the basal ganglia, thalamus, internal or external capsule, or brain stem with a diameter from 3 to 15 mm (Hatate et al., 2016). One point was awarded if ≥1 asymptomatic lesion was present *(Klarenbeek et al., 2013)*.

WMHs were identified into periventricular WMHs and deep WMHs according to site, and both types were graded on a scale of 0-3 according to Fazekas Scale. One point was awarded if (early) confluent deep WMHs (Fazekas score 2 &3) or irregular periventricular WMHs extending into deep white matter (Fazekas score 3) were present *(Fazekas et al., 1987)*.

CMBs were classified into strictly Lobar or Deep CMBs (Miwa et al., 2011). One point was awarded if ≥1 deep CMBs were present. Only deep CMBs were considered because available evidence suggests they are more specifically related to arteriosclerotic SVD *(Staals et al., 2009)*.

EPVSs were counted at the level of BG, in one hemisphere, on the slide with the biggest number of them, and then graded them according to number through a 3-category ordinal scale (Mild:0–10; Moderate:10–25; Extensive: >25). One point was awarded if moderate to extensive (10–25 or >25) EPVSs were present *(Klarenbeek et al., 2013)*.

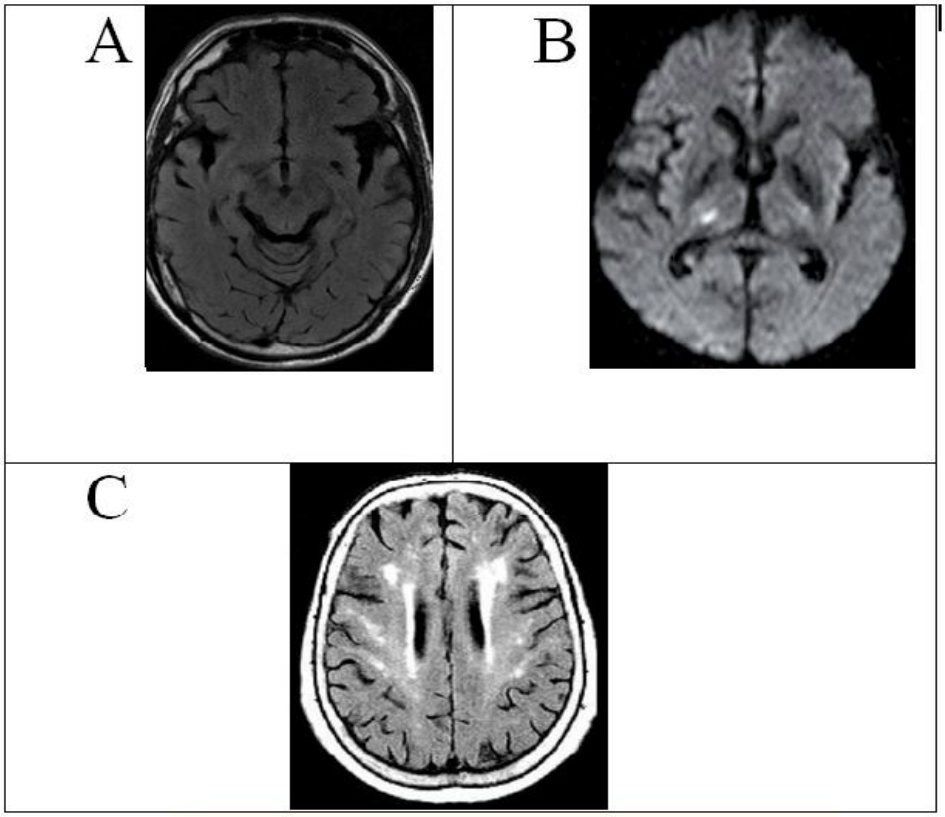

Figure (1): Neuroimaging markers of cerebral small vessel disease

(A)Asymmetrically dilater Virchow-Robin Space at the left basal ganglia Level on FLAIR image, (B) Recent acute tight thalamic lacunar infarction on DWI image, (c) Fazekas type II white matter hyperintensity on FLAIR image

Calculation of Total SVD score: The presence of each of the four SVD markers (as previously defined) was awarded with 1 point, then the summation of points was calculated, producing a minimum score of 0 and a maximum of 4, representing the total Burden of SVD *(Hatate et al., 2016)*.

Routine laboratory investigations including complete blood count, BUN, creatinine, sodium, potassium, INR, lipid profile and glycosylated hemoglobin, in addition to doppler study of both carotids and vertebro-basilar systems, ECG and echocardiography were done to all patients.

Ethical approval: The current investigation was executed based on the recommendations of the ethical committee, Faculty of Medicine, Al-Azhar University, Cairo, Egypt. All clinical interventions were illustrated obviously to all participants prior to study processing. Possible adverse events were exemplified, whereas, a written informed consent after a clear explanation of all study steps was obtained.

Statistical analysis:

Data were collected, revised coded and entered to the statistical package for social sciences **(SPSS)**, version 20. Qualitative data were presented as number and

percentages, while quantitative data were presented as mean, standard deviations and ranges. The comparison between two groups with qualitative data were done using **Chi-square test**. The comparison between two groups regarding quantitative data with parametric distribution was done using Independent t-test. **P-value** was considered significant when P was < 0.05.

RESULTS

The study included 36 patients admitted to hospital during period from January 2017 to March 2019, with either TIAs or first lacunar stroke. Mean age of our subjects was 73.389 ± 8.350 SD. Twenty one patients (58.33%) were males and 15 patients (41.67%) were females. Nine patients (25%) were admitted with lacunar strokes, while 27 patients (75%) with TIAs (Table 1).

Table (1): Characteristics of Study Subjects

Characteristics	All (N=36)
Age (mean ± SD)	73.389 ± 8.350
Male (%)	21 (58.3%)
DM (%)	26 (72.2%)
HTN (%)	31 (86.1%)
HLP (%)	31 (86.1%)
SVD (%)	24 (66.7%)
LI (%)	15 (41.7%)
WMH (%)	14 (38.9%)
CMB (%)	11 (30.6%)
EPVS (%)	14 (38.9%)
SVD score (mean ± SD)	1.528 ± 1.320
MPS (%)	17 (47.2%)
Rigidity (%)	9 (25.0%)
Bradykinesia (%)	7(19.4%)
Gait & postural disturbances (%)	8 (22.2%)
Tremors (%)	3 (8.3%)
MPS score (mean ± SD)	1.167 ±1.404

Regarding Prevalence of SVD among our study patients, 24 patients (66.6%) were found to have at least one marker of SVD, whereas, 12 patients (33.3%), had no marker of SVD at all.

Prevalence of each SVD marker was as following; LIs were found in 15 patients (41%), WMHs in 14 patients (38%), CMBs in 11 patients (30%), and EPVSs in 14 patients (38%). SVD was significantly related to MPS (P value = 0.009 - Table 2). A highly significant correlation was also found between SVD and MPS score (P value = 0.0010).

Table (1): Relation between Presence of SVD and Presence of MPS

SVD / MPS	Absent		Present		Total		Chi-Square
	N	%	N	%	N	%	P value
Absent	10	83.3	9	37.5	19	52.8	0.009*
Present	2	16.7	15	62.5	17	47.2	
Total	12	100.0	24	100.0	36	100.0	

Relation between SVD and age was found to be significant, while its relation with gender was non-significant. No significant difference was found between patients admitted with TIA and patients admitted with stroke regarding presence of SVD. Of the four categories of MPS, the only one found to be significantly related to SVD was Rigidity (P value = 0.022).

Mean SVD score of study subjects was found to be 1.528 ± 1.320, (Table 1). Different SVD scores were as following; 6 patients had only one marker of SVD (SVD score 1), 9 patients had 2 markers (SVD score 2), 6 patients had 3 markers (SVD score 3), while only 3 patients had all four markers of SVD (SVD score 4).

A highly significant correlation was found between SVD score and MPS score (r=0.768 & P-value=<0.001). A similar significant relation was found between SVD score, Rigidity and bradykinesia (P-value = <0.001) (Table 3).

Table (2): Relation of SVD score to different Parkinsonian Parameters

SVD Score / parameters		**N**	**Mean Rank**	**Sum of Ranks**	**Mann-Whitney U test**
					Asymp. Significance
Sex	**Male**	21	1.46	30.3	0.82
	Female	15	1.7	20.4	
Rigidity	**No**	27	1.4	54.1	**<0.001***
	Yes	9	3.0	12.2	
Bradykinesia	**No**	29	1.07	33.1	**<0.001***
	Yes	7	2.5	10.3	
Gait & Posture	**No**	28	1.31	30.2	0.05
	Yes	8	2.1	19.2	
Tremors	**No**	33	1.9	34.2	0.52
	Yes	3	2.6	23.4	

As regard, presence of MPS among subjects, MPS were found in 17 patients (47.2%). Prevalence of the four different MPS subcategories were as following; 9 patients (25%) had rigidity, 7 patients (19.4%) had Bradykinesia, 8 patients (22%) had Gait & Postural manifestations, and 3 patients (8.3%) had Tremors. Three of SVD markers (LIs, WMHs & CMBs) were significantly associated with presence of MPS (table 4), while EPVS had no significant association. A highly significant correlation was found between MPS and SVD score, as also with Age (P-value <0.001). No significant difference was found between patient with TIAs and patients with strokes as regard presence of MPS (Table 4). Mean MPS score was 1.16 ±1.40 SD (Table 1). A highly significant relation was found between MPS score, Age, SVD, and total SVD scores.

Table (3): Study characteristics with respect to presence of MPS

MPS / Characteristics	MPS present N(%) N=17	MPS Absent N(%) N=19	**Mann-Whitney U test Asymp. Significance**
Age (Years)	78.353±7.818	68.947±6.096	<0.001
Male (%)	9 (52.9%)	12 (63.2%)	>0.05
LI (%)	11 (64.4%)	5 (26.4%)	0.021*
WMH (%)	11 (64.1%)	3 (15.2%)	0.003*
CMB (%)	10 (63.8%)	0 (0.0%)	<0.001*
EPVS (%)	8 (46.9%)	6 (31.1%)	<0.03*
SVD score	2.412±1.228	0.737±0.806	<0.001*

DISCUSSION

In our study, prevalence of SVD among our study subjects was 67%. 17% of study subjects had only one SVD marker (SVD score 1), 25% of them had 2 SVD markers (SVD score 2), 17% had 3 SVD markers, and 8% had all four SVD markers, while, 33% of our study subjects had no SVD marker.

Nearly similar prevalence rates were found by 2 cohort studies. *Staal et al. (2014)* found SVD prevalence of 61%, and *Loos et al. (2017)* found prevalence of SVD to be 68%, with 26% of patients having only one SVD marker, 24 % having 2 markers, 13% with 3 markers, 5% having all the four markers and 32% with no marker of SVD.

Another study by *Klarenbeek et al. (2013)* found rather higher prevalence rate, where SVD prevalence was 85%, which can be attributed to selection criteria of patient in that study, as all patient had Strokes. On the other hand, *Uiterwijk et al. (2016)* found lower prevalence rate (52%), which may be explained by the younger age of the patients of this study.

Regarding prevalence of different SVD markers among our study subjects, LIs was the most common found in 41.6% of patients, followed by WMHs in 39%. EPVSs in 38.8%, and CMBs in 30% of them.

Comparable rates were found by *Li et al. (2015)* where LIs was found in 44%, WMHs in 43%, CMBs in 13% and EPVSs in 32%. Prevalence of SVD markers showed some variability in other large studies *(Staals et al., 2014* and *Hatate et al., 2016)*. These variabilities might be explained by different age groups, different selection criteria of patients, in addition to genetic and ethnic differences.

In our study, we found a significant relation between SVD and age. These results were consistent with those elicited by *Klarenbeek et al. (2013)* and *Hilal et al. (2017)* who also found that prevalence

of SVD increases with age, but with no significant sex differences.

No significant difference between patients admitted with TIAs and patients admitted with strokes regarding the prevalence of SVD.

There was a highly significant relation between presence of SVD and presence of MPS, and higher SVD score to be significantly correlated to higher incidence of MPS. Similar result was described by *Hatate et al. (2016)* who found that SVD significantly contributes to the presence of MPS in patients with vascular risk factors. They also found that total SVD score, deep CMBs, mixed LIs, as well as PVH, were independently associated with MPS at any age.

SVD was significantly related to rigidity, but not to other MPS subcategories (bradykinesia, gait and postural disturbances or tremors). Total SVD score was significantly associated with presence of rigidity, bradykinesia, gait and postural disturbances. This indicated that SVD might be a better predictor of different MPS features than each individual SVD marker.

The relation between SVD, gait and postural disturbances showed only a trend towards significance. This could be explained by the smaller number of patients in our study patients. This was consistent with *Loos et al. (2018)* who did not find a significant association between total CSVD burden, objectively measured gait & postural disturbances. On the other hand, *de Laat et al. (2011)* found that both WMLs and LIs are independently associated with gait disturbances.

The mean SVD score in our study was comparable to mean SVD scores that was found by *Li et al. (2015)*.

GFR was found to be significantly correlated with SVD score and MPS score. These results were consistent with *Wada et al. (2008)* that CKD is an independent risk for cerebral SVD lesions, and also by *Makin et al. (2015)* who found that worse renal functions was associated with having more SVD features on imaging, which remained significant after controlling for hypertension.

MPS was found in 47.2% of our study subjects. Prevalence of different MPS categories was as following; Rigidity was found in 25%, Bradykinesia in 19.4%, Gait & postural disturbances in 22% while Tremors was found in 8.3%.

Similar prevalence of MPS was found by cross sectional study done by *Hatate et al. (2016)*, with some variations of the prevalence of different Categories. This was also consistent with a large cohort study done by *Mahoney et al. (2014)* which demonstrated that MPS is present in 47%. Another large prospective study by *de Laat et al. (2012)* demonstrated lower prevalence rates. MPS were present in 21.4%. Lower prevalence rates in that study could be explained by the lower mean age of its subjects.

A highly significant relation was found between MPS and SVD, SVD score and age. MPS and MPS scores were significantly related to presence of each of LIs, WMHs and CMBs, but not to EPVSs.

These results were consistent with the univariate analysis done by *Hatate et al. (2016)* who found that patients with MPS were significantly older, with more severe

SVD, and had a baseline history of stroke than patients without MPS.

Mean MPS score in our study was found to be significantly related to SVD, as well as SVD score.

CONCLUSION

These findings supported the hypothesis of a vascular contribution to MPS in older adults, and provided additional evidence that SVD, and especially total SVD burden, might be a surrogate marker for MPS. Control of risk factors for SVD, might halt progression of these lesions and hence prevent the development of (vascular) Parkinsonism.

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العلاقة بين مرض الأوعية الدموية المخية الصغيرة والعلامات الباركنسونية الحركية الخفيفة في مرضى السكتة الدماغية الجوبية والنوبات الإقفارية العابرة

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خلفية البحث: يعد كلا من مرض الأوعية الدموية الدماغية الصغيرة والعلامات الباركنسونية الخفيفة من الحالات الخطيرة التي تؤثر كبار السن من المرضى، ولكن العلاقة بينهما لا تزال غير واضحة. ترتبط هذه الحالات بمجموعة واسعة من النتائج الصحية الخطيرة بما في ذلك زيادة خطر الإصابة بمرض الشلل الرعاش، وضعف الإدراك، والخرف، وكذلك تطور الإعاقة الوظيفية وزيادة الوفيات.

الهدف من البحث: دراسة العلاقة بين مرض الأوعية الدموية الدماغية الصغيرة، والعلامات الباركنسونية الحركية الخفيفة في المرضى الذين يعانون من النوبات الإقفارية العابرة، والسكتات الدماغية الجوبية.

المرضى وطرق البحث: أجرينا دراسة مقطعية بين 36 مريضًا تم حجزهم بمستشفيات جامعة الأزهر في الفترة بين يونيو 2016 ومايو 2018، إما بنوبة إقفارية عابرة أو بسكتة دماغية جوبية في الفترة من يناير 2017 إلى مارس 2019، ممن لا يُعرف قبلا أنهم مصابون بمرض الشلل الرعاش أو الشلل الرعاش. قد تم تقييم العلامات الباركنسونية الخفيفة من خلال مقياس تصنيف مرض الشلل الرعاش الموحد (الجزء الثالث). كما تم إستخدام التصوير بالرنين المغناطيسي للدماغ لتحديد العلامات الدالة على وجود مرض الأوعية الدموية الدماغية الصغيرة (الجلطات الجوبية، فرط كثافة المادة البيضاء، النزف الدماغي

المجهري، وتمدد المساحات المحيطة بالأوعية الدموية)، ثم تم حساب مجموع العبء الكلي لمرض الأوعية الدموية الدماغية الصغيرة بناءً على وجود أو عدم وجود هذه العلامات الأربعة.

نتائج البحث: كل من الجلطات الجوبية، فرط كثافة المادة البيضاء، النزف الدماغي المجهري، بالإضافة إلى مجموع العبء الكلي لمرض الأوعية الدموية الدماغية الصغيرة، مرتبط بشكل كبير مع وجود العلامات البارکنسونية الخفيفة. كما يوجد إرتباط قوي بين مجموع العبء الكلي لمرض الأوعية الدموية الدماغية الصغيرة وبين كل من التصلب وبطء الحركة في المرضى كبار السن.

الاستنتاج: أظهرت الدراسة أن مرض الأوعية الدموية الدماغية الصغيرة وبخاصة مجموع العبء الكلي له يرتبط ارتباطا وثيقا بوجود العلامات البارکنسونية الخفيفة، مما يدعم فرضية المساهمة الوعائية كمسبب لحدوث العلامات البارکنسونية الخفيفة في المرضى كبار السن.