

Ахмадеева Лейла Ринатовна

Профессор кафедры неврологии

Руководитель Центра неврологии, реабилитационной медицины и ботулинотерапии в Клинике БГМУ

www.ufaneuro.org

БОЛЕЗНЬ МЕЛКИХ ЦЕРЕБРАЛЬНЫХ СОСУДОВ

(SMALL VESSELS DISEASE)





**Данное выступление не спонсировано
никакими производителями фармацевтических
препаратов либо оборудования**

Автор не получает за него гонорар

Конфликт интересов отсутствует



**«Сосудистое поражение головного мозга для
клинической неврологии является проблемой
номер один»**

Е.В.Шмидт, 1968



Цереброваскулярные заболевания



Острые



Хронические



Хронические цереброваскулярные заболевания

могут быть вызваны заболеваниями

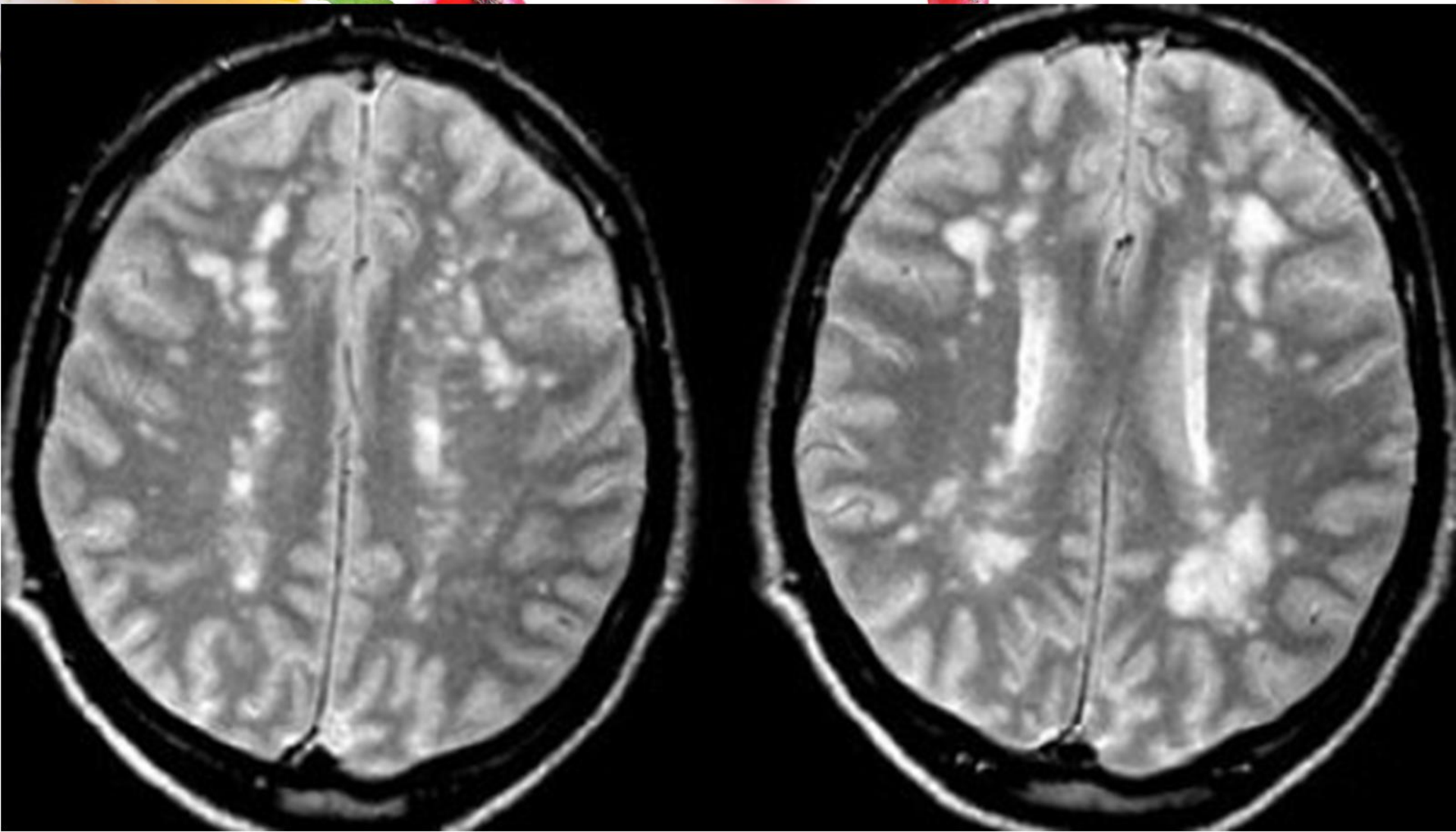
- Сердца
- Крови
- Крупных и **мелких** сосудов



ВАЖНО!!!



**УСТАНОВИТЬ
ЭТУ ПРИЧИНУ**



<https://www.arthriticchick.com/diagnoses/non-autoimmune-diseases/brain-disorders/chronic-small-vessel-disease-of-the-brain>



«Наибольший в настоящее время вызывает группа заболеваний с определенными нейровизуализационными изменениями на МРТ томограммах головного мозга — болезнь мелких сосудов (БМС).»

**Дисциркуляторная энцефалопатия и
болезнь мелких сосудов**

Р.Г. Есин¹, 2*, О.Р. Есин², И.Х. Хайруллин²

Журнал неврологии и психиатрии, 8, 2016

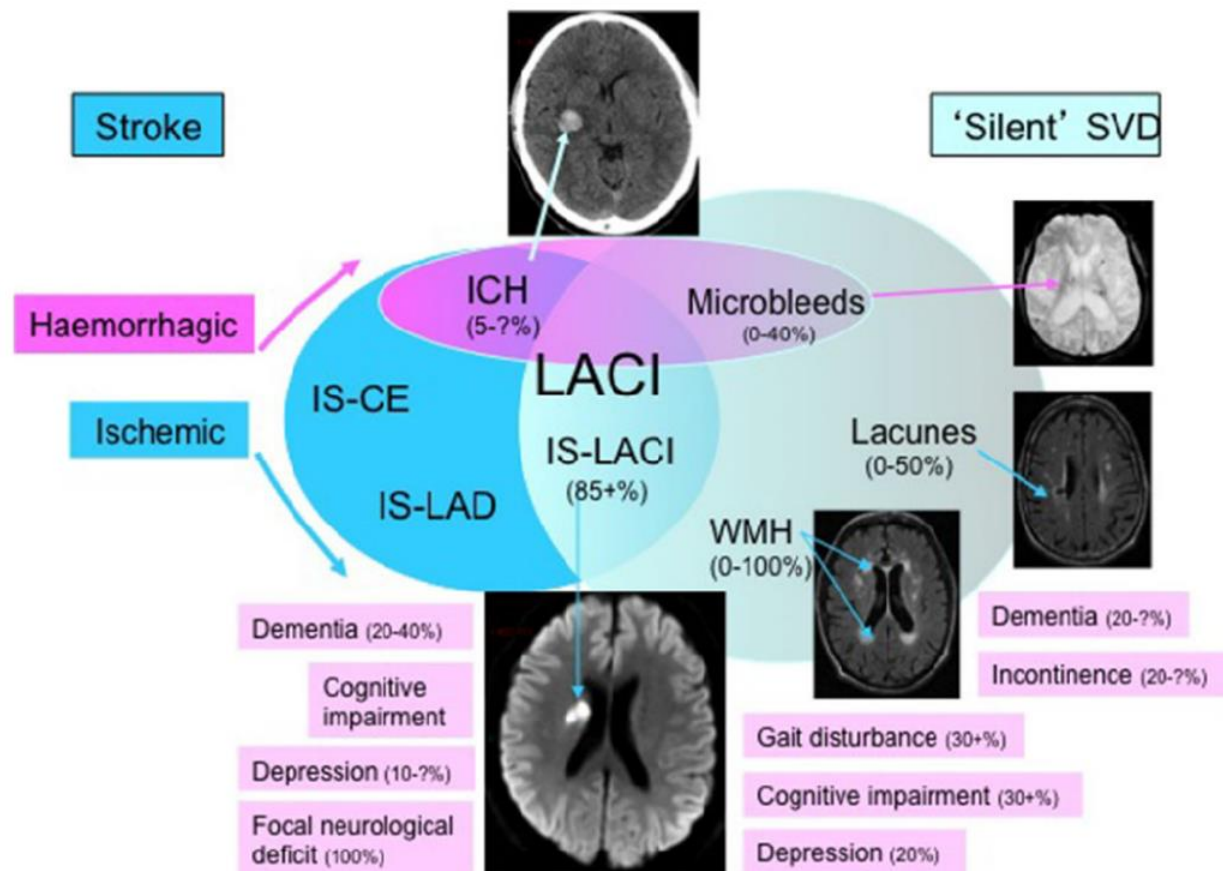


Fig. 1 Venn diagram showing relationship between small vessel disease and other forms of stroke. The embedded neuroimages show, clockwise from the top: intracerebral haemorrhage (ICH), microbleeds, lacunes ('lakes' of cerebrospinal fluid), white matter hyperintensities (WMH), and an acute lacunar infarct (LACI). Percentages relate to SVD etiologies and complications and are approximate: '?' indicates a lack of data.

Pharmacological treatment and prevention of cerebral small vessel disease: a review of potential interventions

Philip M. Bath^{1*} and Joanna M. Wardlaw²



Conclusions

There are no established therapeutic strategies for either preventing or treating SVD although more information about *strategies to avoid* and that *show promise* are emerging (4). A number of routine vascular prophylaxis strategies, especially lowering BP, might reduce SVD but their current guideline use means any future trials would have to test intensity of treatment. Other potential interventions are not in routine use post stroke and have multiple activities with the potential for targeting mechanisms of SVD formation. It is clear that further trials dedicated to preventing the development or worsening of SVD are now required.



Основные тезисы

- **Не** существует установленных стратегий для профилактики или лечения БМС
- НО появляются стратегии, указывающие ЧЕГО СЛЕДУЕТ ИЗБЕГАТЬ и что может звучать *многообещающим*
- Ряд рутинных подходов, особенно **снижение артериального давления** может уменьшать проявления БМС, но предстоит еще уточнить необходимую интенсивность терапии
- Ясно то, что требуется больше новых исследований, посвященных предотвращению развития или ухудшения течения БМС



Согласно современным представлениям, в понятие БМС включают:

- лакунарный инфаркт (ЛИ),
- гиперинтенсивность белого вещества (ГИБВ),
- лакуны,
- микрокровоизлияния,
- расширение периваскулярных пространств и
- церебральную атрофию

Pantoni L. Cerebral small vessel disease: from pathogenesis and clinical characteristics to therapeutic challenges. *The Lancet Neurology*. 2010;9(7):689-701.



Предполагается, что

БМС является частой причиной сосудистой деменции, вносит значительный вклад в развитие сочетанной деменции, а также является причиной почти 50% **инсультов**

Norrving B. Lacunar infarcts: no black holes in the brain are benign. *Practical Neurology*. 2008;8(4):222-228.

Warlow C, Sudlow C, Dennis M, Wardlaw J, Sandercock P. Stroke. *The Lancet*. 2003;362(9391):1211-1224.

Debette S, Markus H. The clinical importance of white matter hyperintensities on brain magnetic resonance imaging: systematic review and metaanalysis. *BMJ*. 2010;341(1):3666-3666.

Morris Z, Whiteley WN, Longstreth WT, Weber F, Lee Y, Tsushima Y, Alphas H, Ladd SC, Warlow C, Wardlaw JM, Al-Shahi Salman R. Incidental findings on brain magnetic resonance imaging: systematic review and metaanalysis. *BMJ*. 2009;339(1):3016-3016.



Significance of Cerebral Small-Vessel Disease in Acute Intracerebral Hemorrhage

Shoichiro Sato, MD*; Candice Delcourt, MD*; Emma Heeley, PhD; Hisatomi Arima, MD; Shihong Zhang, MD; Rustam Al-Shahi Salman, MD; Christian Stapf, MD; Daniel Woo, MD; Matthew L. Flaherty, MD; Achala Vagal, MD; Christopher Levi, MD; Leo Davies, MD; Jiguang Wang, MD; Thompson Robinson, MD; Pablo M. Lavados, MD; Richard I. Lindley, MD; John Chalmers, MD; Craig S. Anderson, MD; on behalf of the INTERACT2 Investigators†

Background and Purpose—The significance of structural changes associated with cerebral small-vessel disease (SVD), including white matter lesions (WML), lacunes, and brain atrophy, to outcome from acute intracerebral hemorrhage is uncertain. We determined associations of computed tomographic radiological manifestations of cerebral SVD and outcomes, and in terms of any differential effect of early intensive blood pressure–lowering treatment, in the large-scale Intensive Blood Pressure Reduction in Acute Cerebral Hemorrhage Trial (INTERACT2).

Methods—We graded WML (van Swieten scale), the presence of lacunes, and brain atrophy (2 linear measurements and visual rating) for 2069 of 2839 patients with available baseline brain computed tomography (<6 hours of intracerebral hemorrhage onset) by 3 independent neurologists blind to clinical data.

Results—WML grade and 2 linear measurements of brain atrophy were associated with death or major disability at 90 days: multivariable-adjusted odds ratios for WML (grade 3 and 4 versus 0), frontal ratio, and third ventricle Sylvian fissure distance (most versus least severe atrophy quartile) were 1.42 (95% confidence interval, 1.02–1.98), 1.47 (1.08–1.99), and 1.64 (1.21–2.22), respectively (all *P* for trend <0.05). There was no association between lacunes and outcomes. There were no significant differences in the effects of intensive blood pressure–lowering across subgroups of cerebral SVD.

Conclusions—Preexisting cerebral SVD manifestations of WML and brain atrophy predict poor outcome in acute intracerebral hemorrhage. There is no apparent hazard of early intensive lowering of blood pressure according to the INTERACT2 protocol, in patients with underlying cerebral SVD.

Clinical Trial Registration—URL: <http://www.clinicaltrials.gov>. Unique identifier: NCT00716079.

(*Stroke*. 2016;47:00-00. DOI: 10.1161/STROKEAHA.115.012147.)



Основные тезисы

- Значимость структурных изменений, связанных с болезнями мелких церебральных сосудов, включая поражение белого вещества головного мозга, лакуны и атрофию мозга, остается неопределенной
- Томограммы 2069 из 2839 пациентов ДО геморрагического инсульта
- 3 невролога (слепое исследование)
- Наличие БМС являлось *предиктором плохого исхода при острых внутримозговых кровоизлияниях*

Поражение мелких церебральных сосудов и риск развития летального исхода, ишемического инсульта и сердечно-сосудистых заболеваний у пациентов с атеросклеротическим поражением сосудов.

Исследование The Second Manifestations of ARterial disease-Magnetic Resonance (SMART-MR)

Источник. M.M.A. Conijn, R.P. Kloppenborg, A. Algra, W.P.Th.M. Mali, L.J. Kappelle, K.L. Vincken, M.I. Geerlings for the SMART Study Group. Cerebral small vessel disease and risk of death, ischemic stroke, and cardiac complications in patients with atherosclerotic disease. The Second Manifestations of ARterial disease-Magnetic Resonance (SMART-MR) Study. Stroke 2011;42:6:3105–3109.

Department of Radiology, University Medical Center Utrecht, Utrecht, the Netherlands; the Julius Center for Health Sciences and Primary Care, UMCU, Utrecht, the Netherlands; the Department of Neurology, Academic Medical Center, Amsterdam, the Netherlands; the Department of Neurology and Rudolf Magnus Institute of Neuroscience, UMCU, Utrecht, the Netherlands; and the Image Sciences Institute, UMCU, Utrecht, the Netherlands.

Предпосылки и цель исследования. Поражение мелких церебральных сосудов может быть связано с сосудистой и несосудистой патологией. Проанализировали влияние наличия лакунарных инфарктов и очагов поражения в белом веществе головного мозга, по результатам магнитно-резонансной томографии (МРТ), на повышение риска развития летального исхода от сосудистых и несосудистых причин и развитие цереброваскулярных заболеваний в будущем у пациентов с церебральным атеросклерозом. **Методы.** МРТ головного мозга выполнили 1309 пациентам с церебральным атеросклерозом, отобранным из базы данных исследования Second Manifestations of ARterial disease-Magnetic Resonance (SMART-MR). Очаги ишемии оценивали визуально, а очаги поражения белого вещества головного мозга оценивали волюметрическим методом. В среднем в течение 4,5 лет (интервал от 0,2 до 7,1 года) проводили наблюдение за пациентами для регистрации летального исхода, ишемического инсульта и ишемических сердечно-сосудистых осложнений. **Результаты.** Регрессионные модели Кокса показали, что наличие лакунарных инфарктов ($n=229$) повышает риск развития смерти от сосудистых (отношение шансов [ОШ]=2,6, 95% доверительный интервал [ДИ] от 1,4 до 4,9) и несосудистых (ОШ=2,7, 95% ДИ от 1,3 до 5,3) причин после внесения поправок на возраст, пол, сосудистые факторы риска, наличие нелакунарных инфарктов и поражение белого вещества головного мозга. Эти риски не различались у пациентов с бессимптомными лакунарными инфарктами. Объем поражения белого вещества головного мозга (по отношению к общему внутричерепному объему) повышал риск смерти от сосудистых причин (ОШ для увеличения на 1 миллилитр 1,03, 95% ДИ от 1,01 до 1,05), а поражение белого вещества в верхней квинтиль по сравнению с более низкой квинтилью повышало риск развития ишемического инсульта (ОШ=2,6, 95% ДИ от 1,3 до 4,9). **Выводы.** Поражение мелких церебральных сосудов, вне зависимости от наличия цереброваскулярных заболеваний, ассоциировано с повышением риска развития летального исхода и ишемического инсульта у пациентов с церебральным атеросклерозом.

July 2017

Association of Microvascular Dysfunction With Late-Life Depression

A Systematic Review and Meta-analysis

Marnix J. M. van Agtmaal, MD^{1,2}; Alfons J. H. M. Houben, PhD^{1,2}; Frans Pouwer, PhD³; [et al](#)

» [Author Affiliations](#)

JAMA Psychiatry. 2017;74(7):729-739. doi:10.1001/jamapsychiatry.2017.0984

Key Points

Question Are both the peripheral and cerebral forms of microvascular dysfunction associated with late-life depression, as suggested by the vascular depression hypothesis?

Findings This systematic review and meta-analysis of 48 studies comprising 43 600 participants, including 9203 individuals with depression, shows that the cerebral and peripheral forms of microvascular dysfunction were associated with increased odds for (incident) late-life depression, independent of cardiovascular risk factors.

Meaning These findings support the hypothesis that microvascular dysfunction is causally linked to late-life depression. This finding may have clinical implications because microvascular dysfunction might provide a target for the prevention and treatment of depression.



Основные тезисы

- **ВОПРОС:** Связана ли микроваскулярная дисфункция с депрессией у пожилых?
- **Мета-анализ:** 48 исследований, 43600 участников, из них 9203 с депрессией, в среднем 3.7 лет наблюдений



Results A total of 712 studies were identified; 48 were included in the meta-analysis, of which 8 described longitudinal data. Data from 43 600 participants, 9203 individuals with depression, and 72 441 person-years (mean follow-up, 3.7 years) were available. Higher levels of plasma endothelial biomarkers (soluble intercellular adhesion molecule-1: OR, 1.58; 95% CI, 1.28-1.96), white matter hyperintensities (OR, 1.29; 95% CI, 1.19-1.39), cerebral microbleeds (OR, 1.18; 95% CI, 1.03-1.34), and cerebral (micro)infarctions (OR, 1.30; 95% CI, 1.21-1.39) were associated with depression. Among the studies available, no significant associations of albuminuria and retinal vessel diameters with depression were reported. Longitudinal data showed a significant association of white matter hyperintensities with incident depression (OR, 1.19; 95% CI, 1.09-1.30).

Conclusions and Relevance This meta-analysis shows that both the peripheral and cerebral forms of microvascular dysfunction are associated with higher odds of (incident) late-life depression. This finding may have clinical implications because microvascular dysfunction might provide a potential target for the prevention and treatment of depression.



РЕЗУЛЬТАТЫ И ВЫВОДЫ

- Как периферические, так и центральные формы микроваскулярной дисфункции связаны с учащением проявления депрессии в позднем возрасте
- *Гиперинтенсивность белого вещества – OR=1.29*
- *Церебральные микрокровоизлияния – OR=1.18*
- *Церебральные (микро)инфаркты – OR=1.30*



AHA/ASA Scientific Statement

Vascular Contributions to Cognitive Impairment and Dementia

A Statement for Healthcare Professionals From the American Heart Association/American Stroke Association

The American Academy of Neurology affirms the value of this guideline as an educational tool for neurologists.

The Alzheimer's Association participated in the development of this statement to advance knowledge and understanding of the causes of dementia and the factors that contribute to its progression.

Philip B. Gorelick, MD, MPH, FAHA, Co-Chair; Angelo Scuteri, MD, PhD, Co-Chair;
Sandra E. Black, MD, FRCPC, FAHA*; Charles DeCarli, MD*;

Stroke. 2011;42:2672–2713

Table 1. Applying Classification of Recommendations and Level of Evidence

		SIZE OF TREATMENT EFFECT												
ESTIMATE OF CERTAINTY (PRECISION) OF TREATMENT EFFECT		CLASS I <i>Benefit >>> Risk</i> Procedure/Treatment SHOULD be performed/ administered	CLASS IIa <i>Benefit >> Risk</i> Additional studies with focused objectives needed IT IS REASONABLE to per- form procedure/administer treatment	CLASS IIb <i>Benefit ≥ Risk</i> Additional studies with broad objectives needed; additional registry data would be helpful Procedure/Treatment MAY BE CONSIDERED	CLASS III <i>No Benefit</i> or CLASS III <i>Harm</i>									
					<table><tr><th></th><th>Procedure/ Test</th><th>Treatment</th></tr><tr><td>COR III: No benefit</td><td>Not Helpful</td><td>No Proven Benefit</td></tr><tr><td>COR III: Harm</td><td>Excess Cost w/o Benefit or Harmful</td><td>Harmful to Patients</td></tr></table>		Procedure/ Test	Treatment	COR III: No benefit	Not Helpful	No Proven Benefit	COR III: Harm	Excess Cost w/o Benefit or Harmful	Harmful to Patients
		Procedure/ Test	Treatment											
	COR III: No benefit	Not Helpful	No Proven Benefit											
	COR III: Harm	Excess Cost w/o Benefit or Harmful	Harmful to Patients											
LEVEL A Multiple populations evaluated*	■ Recommendation that procedure or treatment is useful/effective ■ Sufficient evidence from multiple randomized trials or meta-analyses	■ Recommendation in favor of treatment or procedure being useful/effective ■ Some conflicting evidence from multiple randomized trials or meta-analyses	■ Recommendation's usefulness/efficacy less well established ■ Greater conflicting evidence from multiple randcmized trials or meta-analyses	■ Recommendation that procedure or treatment is not useful/effective and may be harmful ■ Sufficient evidence from multiple randomized trials or meta-analyses										
LEVEL B Limited populations evaluated*	■ Recommendation that procedure or treatment is useful/effective ■ Evidence from single randomized trial or nonrandomized studies	■ Recommendation in favor of treatment or procedure being useful/effective ■ Some conflicting evidence from single randomized trial or nonrandomized studies	■ Recommendation's usefulness/efficacy less well established ■ Greater conflicting evidence from single randcmized trial or nonrandomized studies	■ Recommendation that procedure or treatment is not useful/effective and may be harmful ■ Evidence from single randomized trial or nonrandomized studies										
LEVEL C Very limited populations evaluated*	■ Recommendation that procedure or treatment is useful/effective ■ Only expert opinion, case studies, or standard of care	■ Recommendation in favor of treatment or procedure being useful/effective ■ Only diverging expert opinion, case studies, or standard of care	■ Recommendation's usefulness/efficacy less well established ■ Only diverging expert opinion, case studies, or standard of care	■ Recommendation that procedure or treatment is not useful/effective and may be harmful ■ Only expert opinion, case studies, or standard of care										
Suggested phrases for writing recommendations		should is recommended is indicated is useful/effective/beneficial	is reasonable can be useful/effective/beneficial is probably recommended or indicated	may/might be considered may/might be reasonable usefulness/effectiveness is unknown/unclear/uncertain or not well established	COR III: No Benefit is not recommended is not indicated should not be performed/ administered/ other is not useful/ beneficial/ effective	COR III: Harm potentially harmful causes harm associated with excess morbidity/mortality should not be performed/ administered/ other								
Comparative effectiveness phrases [†]		treatment/strategy A is recommended/indicated in preference to treatment B treatment A should be chosen over treatment B	treatment/strategy A is probably recommended/indicated in preference to treatment B it is reasonable to choose treatment A over treatment B											

A recommendation with Level of Evidence B or C does not imply that the recommendation is weak. Many important clinical questions addressed in the guidelines do not lend themselves to clinical trials. Although randomized trials are unavailable, there may be a very clear clinical consensus that a particular test or therapy is useful or effective.



8.2.9. Recommendations

- 1. In people at risk for VCI, smoking cessation is reasonable (*Class IIa; Level of Evidence A*).**
- 2. In people at risk for VCI, the following lifestyle interventions may be reasonable: moderation of alcohol intake (*Class IIb; Level of Evidence B*); weight control (*Class IIb; Level of Evidence B*); and physical activity (*Class IIb; Level of Evidence B*).**
- 3. In people at risk for VCI, the use of antioxidants and B vitamins is not beneficial, based on current evidence (*Class III; Level of Evidence A*).**



Основные рекомендации АНА/АSА при риске сосудистых деменций

- Отказ от курения (уровень А)
- Коррекция употребления алкоголя, массы тела, физической активности (уровень В)
- Использование антиоксидантов и витаминов группы В при риске сосудистых деменций – **НЕ ПРИНОСИТ ПОЛЬЗЫ** (уровень А)



8.4.6. Recommendations

- 1. In people at risk for VCI, treatment of hypertension is recommended (*Class I; Level of Evidence A*).**
- 2. In people at risk for VCI, treatment of hyperglycemia may be reasonable (*Class IIb; Level of Evidence C*).**
- 3. In people at risk for VCI, treatment of hypercholesterolemia may be reasonable (*Class IIb; Level of Evidence B*).**
- 4. In people at risk for VCI, it is uncertain whether treatment of inflammation will reduce such risk (*Class IIb; Level of Evidence C*).**



Основные рекомендации АНА/АSА при риске сосудистых деменций

- **Рекомендуется лечение артериальной гипертензии (уровень А)**
- **Может быть разумным лечение гиперхолестеремии (уровень В) и гипергликемии (уровень С)**



Recommendations

1. A Mediterranean-type dietary pattern has been associated with less cognitive decline in several studies and may be reasonable (*Class IIb; Level of Evidence B*).
2. Vitamin supplementation is not proven to improve cognitive function, even if homocysteine levels have been positively influenced, and its usefulness is not well established (*Class IIb; Level of Evidence B*).
3. Physical activity might be considered for the prevention of cognitive impairment (*Class IIb; Level of Evidence B*), but the usefulness of other lifestyle or vitamin interventions is uncertain (*Class IIb; Level of Evidence B*).
4. The effectiveness of antiaggregant therapy for VCI is not well established (*Class IIb; Level of Evidence B*).



Основные рекомендации АНА/ASA при риске сосудистых деменций

- Средиземноморская диета может быть обоснована (уровень В)
- Использование витаминов не доказано для коррекции когнитивных функций (уровень В)
- Физическая активность может быть рассмотрена как средство профилактики когнитивных нарушений (уровень В)



Update on cerebral small vessel disease: a dynamic whole-brain disease

Yulu Shi,^{1,2} Joanna M Wardlaw¹

To cite: Shi Y, Wardlaw JM. Update on cerebral small vessel disease: a dynamic whole-brain disease. *Stroke and Vascular Neurology* 2016;1:e000035. doi:10.1136/svn-2016-000035

ABSTRACT

Cerebral small vessel disease (CSVD) is a very common neurological disease in older people. It causes stroke and dementia, mood disturbance and gait problems. Since it is difficult to visualise CSVD pathologies in vivo, the diagnosis of CSVD has relied on imaging findings including white matter hyperintensities, lacunar ischaemic stroke, lacunes, microbleeds, visible perivascular spaces and many

Generally, including in this review, CSVD is used to describe a series of imaging changes in the white matter and subcortical grey matter, including recent small subcortical infarct, lacunes, white matter hyperintensities (WMHs), prominent perivascular spaces (PVS), cerebral microbleeds (CMBs) and atrophy.⁴ Usually, recent small subcortical



Основные тезисы

- Болезни мелких церебральных сосудов часто встречаются у пожилых
- Они вызывают
 - инсульты,
 - деменции,
 - изменения настроения
 - Изменения ходьбы/походки
- Основы Ds – изменения, найденные на МРТ
 - Гиперинтерсивность белого вещества
 - Лакунарные ишемические инсульты
 - Лакуны
 - Микрокровоизлияния
 - Др

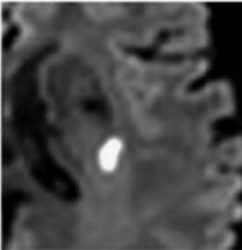


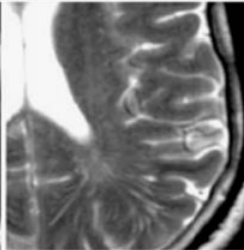
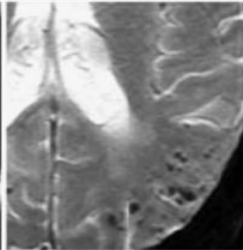
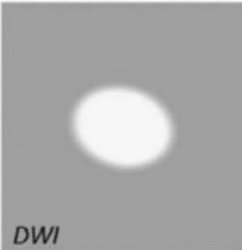

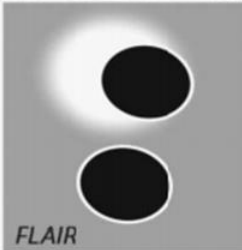

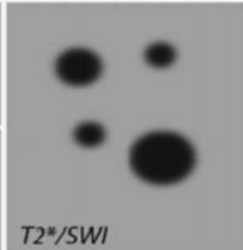
	Recent small subcortical infarct	White matter hyperintensity	Lacune	Perivascular space	Cerebral microbleeds
Example image					
Schematic	 DWI	 FLAIR	 FLAIR	 T2 T1/FLAIR	 T2*/SWI
Usual diameter ¹	≤ 20 mm	variable	3-15 mm	≤ 2 mm	≤ 10 mm
Comment	best identified on DWI	located in white matter	usually have hyperintense rim	usually linear without hyperintense rim	detected on GRE seq., round or ovoid, blooming
DWI	↑	↔	↔/(↓)	↔	↔
FLAIR	↑	↑	↓	↓	↔
T2	↑	↑	↑	↑	↔
T1	↓	↔/(↓)	↓	↓	↔
T2* / GRE	↔	↑	↔ (↓ if haemorrhage)	↔	↓↓

Figure 1 STRIVE, STandards for Reporting and Imaging of Small Vessel Disease example findings (upper), schematic representation (middle) and a summary of imaging characteristics (lower) of MRI features for changes related to small vessel disease.⁴ DWI, diffusion-weighted imaging; FLAIR, fluid-attenuated inversion recovery; SWI, susceptibility-weighted imaging; GRE, gradient-recalled echo.



Prevention and Management of Cerebral Small Vessel Disease

Vincent Mok,^a Jong S. Kim^b

Lacunar infarcts/lacunes, white matter hyperintensities (WMH), and cerebral microbleeds (CMBs) are considered various manifestations of cerebral small vessel disease (SVD). Since the exact mechanisms of these manifestations differ, their associated risk factors differ. High blood pressure is the most consistent risk factor for all of these manifestations. However, a "J curve" phenomenon in terms of blood pressure probably exists for WMH. The association between cholesterol levels and lacunar infarcts/lacunes or WMH was less consistent and sometimes conflicting; a low cholesterol level probably increases the risk of CMBs. Homocysteinemia appears to be associated with WMH. It is noteworthy that the risk factors profile may also differ between different lacunar patterns and CMBs located at different parts of the brain. Thrombolysis, antihypertensives, and statins are used to treat patients with symptomatic lacunar infarction, just as in those with other stroke subtypes. However, it should be remembered that bleeding risks increase in patients with extensive WMH and CMBs after thrombolysis therapy. According to the Secondary Prevention of Small Subcortical Strokes trial results, a blood pressure reduction to < 130 mmHg is recommended in patients with symptomatic lacunar infarction. However, an excessive blood pressure decrease may induce cognitive decline in older patients with extensive WMH. Dual antiplatelet therapy (aspirin plus clopidogrel) should be avoided because of the excessive risk of intracerebral hemorrhage. Although no particular antiplatelet is recommended, drugs such as cilostazol or triflusal may have advantages for patients with SVD since they are associated with less frequent bleeding complications than aspirin.



Основные тезисы

- Лакунарные инфаркты/лакуны, гиперинтерсивность белого вещества и церебральные микрокровоизлияния считаются различными проявлениями болезни мелких церебральных сосудов
- Самым значимым фактором этих проявления является атериальная гипертензия
- Не ясна связь между этими МРТ-явлениями и уровнем холестерина
- Низкий уровень холестерина, вероятно, увеличивает риск церебральных микрокровоизлияний



Основные тезисы

- Тромболизис, антигипертензивные препараты и статины используются для лечения БМС
- **НО** при БМС увеличивается риск кровоизлияний после тромболизиса
- Пациентам с симптоматикой лакунарных инфарктов мозга рекомендуется АД < 130 мм рт ст
- **НО** чрезмерное снижение АД увеличивает когнитивный дефицит у пожилых с БМС



Although the harm of dual antiplatelet therapy (aspirin plus clopidogrel) was correctly identified in patients with SVD, the appropriate antiplatelets in SVD require investigation. In particular, the efficacy of drugs such as cilostazol or triflusal, which are known to be associated with less frequent bleeding complication than aspirin, should be further investigated. Finally, as discussed above, subcortical infarction is heterogeneous. Proximal subcortical infarction, especially those associated with parental artery stenosis, has characteristics of atherosclerosis, whereas distal subcortical infarction has SVD characteristics. In this regard, treatment may have to be tailored; for example, statins and dual antiplatelets may work better in the former group of patients but may be harmful in the latter group. Previous studies disregarded the heterogeneity of small subcortical infarction, so further studies that consider this aspect should be performed.



Основные тезисы

- Доказан **ВРЕД** двойной антиагрегантной терапии при БМС (аспирин + клопидогрель)
- Цилостазол и трифлузал являются препаратами, с которыми связан **меньший** риск кровоизлияний, и по которым продолжаются исследования



Основные тезисы

- Субкортикальные инфаркты гетерогенны!
 - Проксимальные связаны со стенозом и атеросклерозом
 - **Дистальные** – с БМС
- **ПОЭТОМУ** лечение их будет разным!
 - При проксимальных субкортикальных инфарктах могут быть полезны статины и двойная антиагрегантная терапия,
 - А при *дистальных* эта терапия может быть **вредна!**



Ограниченные знания

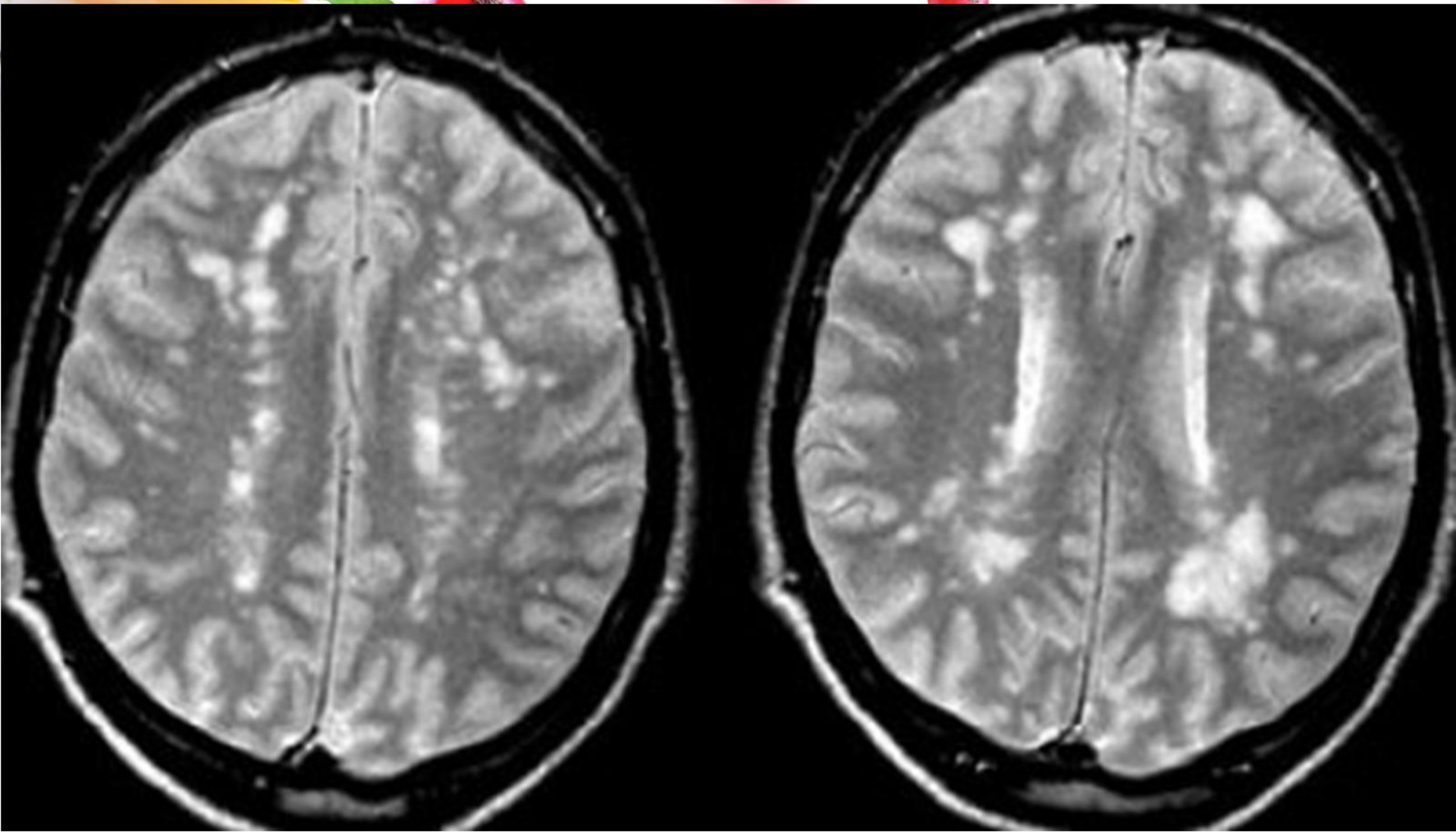
Hugh Markus summarized the treatment of SVD patients. He emphasized that **we have limited knowledge** from clinical trials and therefore a great demand for new trials. In addition, there is a need for better stroke subtyping in large stroke trials as many classification systems cannot discriminate between SVD-related infarcts and small infarcts of other etiology. **Some specific recommendations:** Double platelet inhibition should be avoided, also in the acute phase. Recent data from an Austrian registry suggest that thrombolysis is equally beneficial in SVD-related stroke compared with other stroke subtypes. *Antihypertensive treatment is very important, also in SVD patients without stroke.* But blood pressure lowering might be harmful in late disease stages, as it can augment hypoperfusion. **Ultimately, a better understanding of SVD pathomechanisms is needed in order to develop new therapies.**

<https://eso-stroke.org/eso/cerebral-small-vessel-disease-esoc-2017-session-highlights/>



Рекомендации ESO (2017) при БМС – результаты сессии:

- Избегать двойной антиагрегантной терапии
- Тромболизис эффективен при разных подтипах инсультов, в том числе и вызванных БМС
- Антигипертензивная терапия важна при БМС, в том числе и у пациентов без инсультов
- НО снижение АД на поздних стадиях БМС может принести вред из-за гипоперфузии
- Требуется лучшее понимание патогенетических механизмов для разработки методов лечения



<https://www.arthriticchick.com/diagnoses/non-autoimmune-diseases/brain-disorders/chronic-small-vessel-disease-of-the-brain>



www.ufaneuro.org