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Biological and imaging predictors of cognitive impairment after stroke: systematic review.

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BACKGROUND

Cognitive impairment after stroke accounts for an important part of its disability. Dementia may be due to stroke lesions, underlying Alzheimer pathology or coexistence of both (1-3). Stroke and Alzheimer's disease share similar risk factors and a biological interaction between the two types of pathologies occurs at the level of the neurovascular unit (Fig 1). The association is way beyond the mere coexistence of the two pathologies. Therefore, stroke patients at highest risks for developing dementia should be identified at the acute stage, because they may need a more aggressive secondary prevention strategy, and a longer follow-up with a focus on cognition. Several stroke-related factors have already been associated with an increased risk of dementia being most of them related to the stroke itself (4, 5). Predictors independent from index stroke showed contradictory results. The aim of our study was to systematically review biological and imaging predictors of post stroke cognitive impairment.

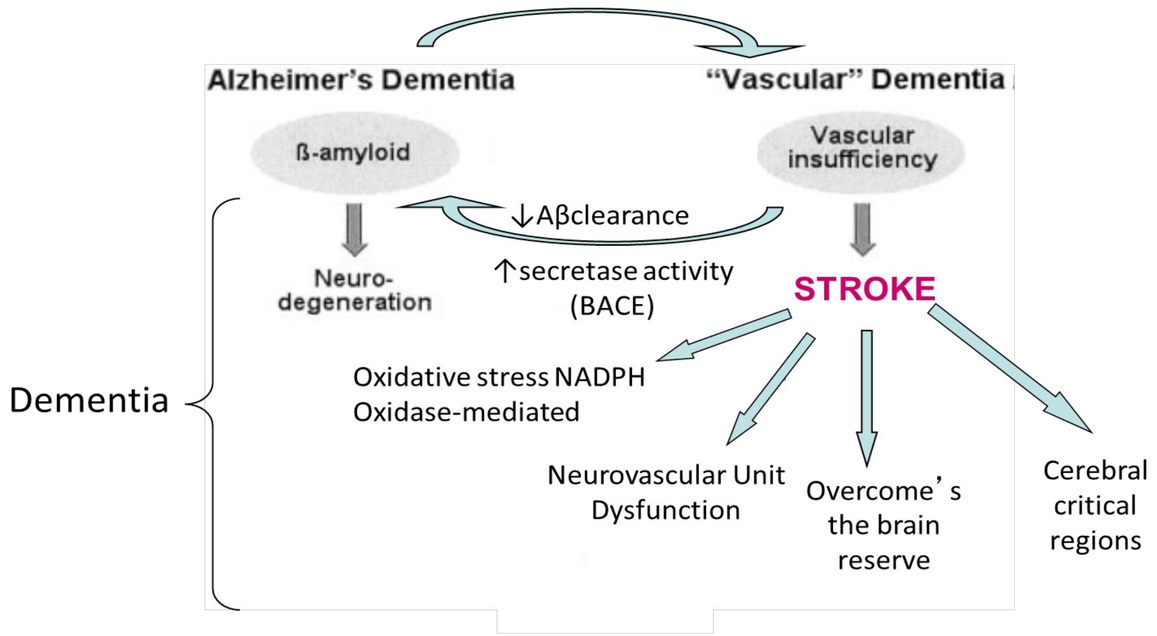

Fig. 1 Modified from Iadecola C, *Stroke*, 2003

METHODS

On January 26th, 2015 we searched Ovid Medline from 1966, Embase from 1980, and Cochrane library using an electronic search strategy. We used the following key words: "stroke or cerebral infarct or brain infarct or cerebral haemorrhage or cerebral ischaemia or cerebral haematoma or brain haemorrhage" and "dementia or cognitive decline or cognitive impairment or cognition" (title/abstract/Mesh terms). We considered as inclusion criteria articles on at least 30 patients with clinical manifestations of ischaemic stroke or transient ischaemic attack (TIA) and with a cognitive follow up 3 months or more after stroke. We considered only factors pre-existing to stroke (not related with the index stroke) that were studied in at least two articles.

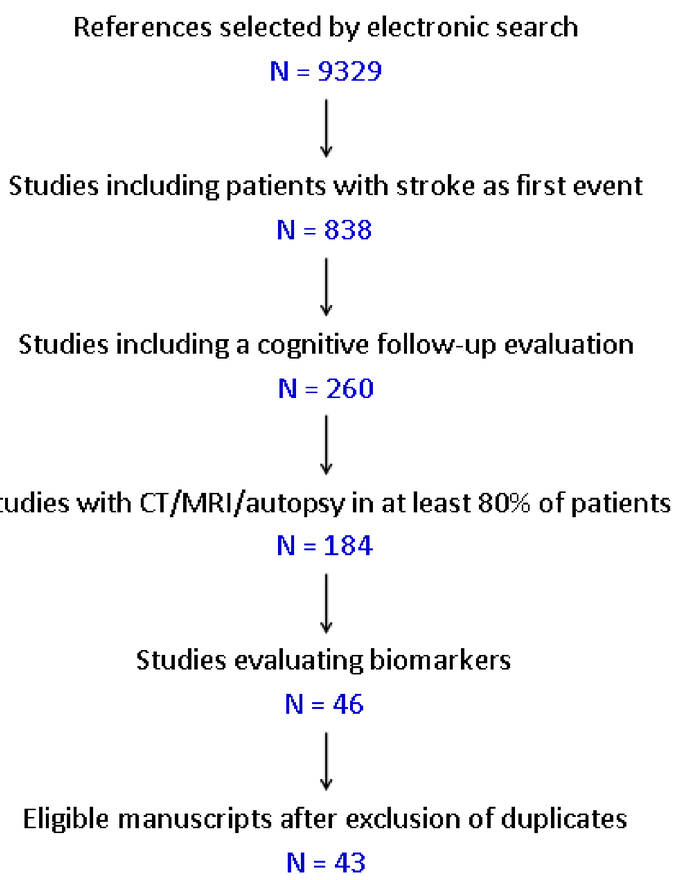

Fig 2 Selection process of the 43 eligible articles.

RESULTS

We identified 9329 abstracts (Fig 2). Forty-three articles met selection criteria for the study. Global cerebral or medial temporal lobe atrophy, silent infarcts, lacunes or white matter changes were found associated with an increased risk of cognitive impairment or dementia, in most of the studies. APO E4 genotype and angiotensin converting enzyme (ACE) genes polymorphisms were related to dementia with inconsistencies among the studies.

DISCUSSION

The predictors of post-stroke cognitive impairment (i.e. apo E4 genotype, global cerebral atrophy, medial temporal lobe atrophy, silent infarcts and white matter changes) have all shown contradictory results in at least one article among those selected. It could be surprising the evidence that many known strong markers of dementia in the population have not been found associated with post stroke cognitive impairment in all studies. A reason explaining the discrepancy could be the presence of methodological differences between reports. Moreover, the prominent effect leading to cognitive impairment after stroke is in relation with the characteristics of the acute stroke

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5. Leys D, Henon H, Mackowiak-Cordoliani MA, Pasquier F. Poststroke dementia. Lancet Neurol. 2005;4(11):752–759.