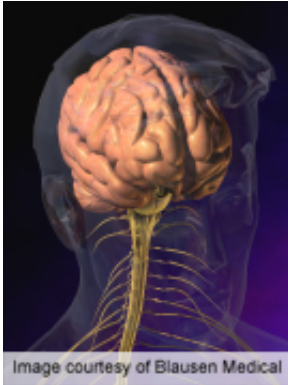


Less Alzheimer's pathology with angiotensin receptor blocker use

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In autopsy findings, patients treated with angiotensin receptor blockers show less Alzheimer's disease-related pathology, according to a study published online Sept. 10 in the *Archives of Neurology*.

(HealthDay)—In autopsy findings, patients treated with angiotensin receptor blockers (ARBs) show less Alzheimer's disease (AD)-related pathology, according to a study published online Sept. 10 in the *Archives of Neurology*.

Ihab Hajjar, M.D., from the University of Southern California in Los Angeles, and colleagues evaluated the impact of ARB treatment on the neuropathology of AD using data from autopsy samples from 890 patients with hypertension (mean age at death, 81 years). [Vascular injury](#) markers, neuritic plaque, and neurofibrillary tangle measures were compared for those taking ARBs and those treated with other antihypertensive medications and untreated individuals.

The researchers found that participants treated with ARBs, with or without AD, showed significantly fewer amyloid deposition markers than those treated with other antihypertensive medications (lower Consortium to Establish a Registry of Alzheimer Disease score: odds ratio, 0.47; Alzheimer Disease and Related Disorders

Association score: odds ratio, 0.43; Braak and Braak stage: odds ratio, 0.52; neuritic plaques: odds ratio, 0.59). Compared with untreated hypertensive patients, patients treated with ARBs also had less AD-related pathology. Participants who received ARBs had more frequent pathologic evidence of large vessel [infarct](#) and [hemorrhage](#), as they were more likely to have had a stroke.

"This autopsy study suggests that treatment with ARBs is associated with less amyloid accumulation and AD-related pathology independent of other AD risk factors," the authors write. "To our knowledge, it is the first human evidence to suggest that treatment with ARBs may have a selective beneficial effect on amyloid metabolism."

More information: [Abstract](#)
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